Rapid evolution, seasonality, and the termination of parasite epidemics

MEGHAN A. DUFFY,1,5 SPENCER R. HALL,2 CARLA E. CACERES,3 AND ANTHONY R. IVES4

1School of Biology, Georgia Institute of Technology, 310 Ferst Drive, Atlanta, Georgia 30332-0230 USA
2Department of Biology, Indiana University, 1001 E. 3rd Street, Bloomington, Indiana 47405 USA
3School of Integrative Biology, University of Illinois, Urbana, Illinois 61801 USA
4Department of Zoology, University of Wisconsin, Madison, Wisconsin 53706 USA

Abstract. Why do epidemics end? This simple question has puzzled ecologists and epidemiologists for decades. Early explanations focused on drops in host density arising from highly virulent parasites and, later, on the effects of acquired immunity. More recently, however, two additional epidemic-ending mechanisms have surfaced: environmental change (including seasonality) and rapid evolution of increased resistance of hosts to infection. Both mechanisms, via either decreasing seasonal temperatures or evolution of resistance, act by altering transmission rates. To explore these possibilities, we tracked five epidemics of a virulent yeast parasite in lake populations of Daphnia dentifera from late summer through autumn. We then fit and compared performance of time-series models that included temperature-dependent and/or evolutionary changes in transmission rates. The analyses show evolution to be the better explanation of epidemic dynamics. Thus, by integrating data and models, this study highlights the potential role of evolution in driving the termination of epidemics in natural populations.

Key words: contemporary evolution; eco-evolutionary dynamics; epidemic fade-out; infectious disease; Metschnikowia bicuspidata; parasitism; state–space model; susceptibility.

INTRODUCTION

An array of field and laboratory studies show that evolution can operate on sufficiently short time scales to strongly influence ecological interactions (Thompson 1998, Hairston et al. 2005, Carroll et al. 2007). Nonetheless, surprisingly few examples of such eco-evolutionary dynamics exist (Fussmann et al. 2007). Such dynamics should arise most prominently when rapidly evolving traits strongly influence ecological interactions (Hairston et al. 2005, Carroll et al. 2007). Resistance to infection by virulent parasites is a likely example of such a trait. Numerous studies have documented rapid increases in host resistance to virulent parasites (e.g., Dwyer et al. 1990, Lohse et al. 2006, Duncan and Little 2007), and clearly this trait should influence host–parasite interactions (Anderson and May 1991, Godfray 2000).

We have recently proposed that rapid evolution can shape host–parasite interactions in a plankton system on ecological time scales and can even terminate parasite epidemics (Duffy and Sivars-Becker 2007, Duffy et al. 2008). This conclusion stems from a combination of data and theory. Empirically, we found evidence for high resistance arising in Daphnia populations that had recently suffered epidemics of a virulent yeast parasite; meanwhile, populations without epidemics retained low resistance. An evolutionary epidemiological model provided additional support, suggesting directional selection for increased resistance should qualitatively alter parasite dynamics. Without evolution, the parasite should persist with the host (that is, become endemic), yet with evolution, outbreaks should terminate within a season (Duffy and Sivars-Becker 2007, Duffy and Hall 2008).

Other factors, however, might also drive dynamics of infections in natural populations and could also contribute to the termination of epidemics. In particular, seasonality can potentially affect both the onset and end of epidemics (Pascual and Dobson 2005, Altizer et al. 2006). Seasonality shapes epidemics via host immunity (Nelson et al. 2002, Hosseini et al. 2004), effects of seasonal weather patterns on host susceptibility (Sultan et al. 2005), or, for vector-borne diseases, changes in abundance of vectors (Hoschen and Morse 2004). In Daphnia systems, seasonal declines in water temperature can especially diminish transmission rates of parasites (Mitchell et al. 2004, Hall et al. 2006).

Here, we explore the joint roles of rapid evolution of host resistance and seasonal temperature declines in driving epidemic dynamics. Specifically, we ask which of these factors can better explain dynamics of infections observed in five intensively sampled epidemics in lake Daphnia populations. We pursue this question by analyzing the time course of the epidemics, fitting
models that incorporate the effects of rapid evolution of increased host resistance and/or seasonal changes in temperature on transmission rates. After estimating parameters from the data, we then statistically compared models. Via this comparison, we found that evolution of increased host resistance, rather than temperature, better described the decline of epidemics.

**Methods**

**Study system**

*Daphnia dentifera* (formerly *Daphnia galeata mendoetae* and *Daphnia rosea*) is a common, large, important freshwater zooplankter in temperate lakes in North America (Hebert 1995) and often dominates the plankton in stratified lakes surrounding the Kellogg Biological Station in southwest Michigan, USA (Tessier and Woodruff 2002) where this study was performed. *D. dentifera* migrates vertically, residing in cold deep waters during the day and in warm surface waters at night (Hall et al. 2005). It is cyclically parthenogenetic, reproducing asexually throughout summer and early autumn, and switching to sexual reproduction in late autumn (Cáceres and Tessier 2004, Duffy et al. 2008). Sexually produced eggs are dormant, hatching in the spring of the following or later years.

*D. dentifera* populations are host to numerous internal parasites (Hall et al. 2005), one of the most common being the yeast *Metschnikowia bicuspidata* (see Plate 1). *Metschnikowia* is transmitted horizontally between individuals when spores released from dead *Daphnia* are consumed by uninfected *Daphnia* (Ebert 2005, Hall et al. 2007). Water temperature has a strong effect on transmission rate, most likely due to temperature dependence of physiological rates of both host and parasite (Hall et al. 2006). The strong effect of temperature on transmission rate stems in part from effects of temperature on consumption rates of *Daphnia*. *Daphnia* eat less with decreasing temperature, leading to the ingestion of fewer spores and lower transmission rates at lower temperatures (Hall et al. 2006). In the temperature range observed during our current study, we expect transmission to increase exponentially with increasing temperature (Hall et al. 2006).

*Metschnikowia* reduces host fecundity and life span (Ebert et al. 2000, Hall et al. 2006, Duffy and Hall 2008), increases susceptibility to fish predation (Duffy and Hall 2008), and affects the evolution of *D. dentifera* populations (Duffy and Sivars-Becker 2007, Duffy et al. 2008). *D. dentifera* do not recover from *Metschnikowia* infections (Ebert 2005). Therefore, *Metschnikowia* is an “obligate killer” (Ebert and Weisser 1997).

**Field sampling**

We sampled five *Metschnikowia* epidemics in four lakes: Baker (see Plate 1), Bassett, Bristol, and Warner (Barry County, Michigan, USA). Two of these epidemics occurred in Baker and Bassett Lakes in 2003; in 2004, epidemics occurred in Bassett, Bristol, and Warner Lakes. These lakes are part of a set of 18 lakes that we monitor for infections (Cáceres et al. 2006); we selected these for intensive monitoring because they had a high probability of having *Metschnikowia* epidemics based in part on lake basin shape (Cáceres et al. 2006). We measured parasite prevalence and host density every three to four days during summer and autumn. In 2003, Baker and Bassett Lakes were sampled on average every four days, beginning in early July and ending in late October (Baker Lake) or early November (Bassett Lake). Unfortunately, the complete termination of this Bassett Lake epidemic could not be tracked due to the beginning of deer hunting season. In 2004, Bassett, Bristol, and Warner Lakes were sampled every three days during most of the epidemic but less frequently at the beginning of the epidemic. On each lake-date, we collected four samples using a 153-µm mesh Wisconsin net following methods in Duffy and Hall (2008). Each of these samples combined four whole-water-column, vertical net tows taken at four different sites within the deep basin of each lake. Three of these samples were preserved in alcohol and later counted to determine *D. dentifera* density. The fourth sample was examined live (within 12 hours of collection) under a stereomicroscope to determine infection prevalence following the methods in Cáceres et al. (2006).

To characterize variation in temperature during the season, we measured vertical temperature profiles biweekly throughout summer and autumn. Since *D. dentifera* migrate vertically, we used these temperature profiles and information on vertical migration in these populations (Hall et al. 2005; M. A. Duffy and S. R. Hall, *unpublished data*) to calculate time-weighted temperatures experienced by the *D. dentifera* (as in Duffy et al. 2005).

**Modeling**

We fit time-series data from the five epidemics to a modified version of an evolutionary epidemiological model that we have previously used to describe *D. dentifera–Metschnikowia* dynamics (Duffy and Sivars-Becker 2007, Duffy and Hall 2008). In the model used here, the transmission rate declines exponentially with decreasing temperature and also diminishes via evolution of resistance. *D. dentifera* populations show substantial genetic variation in susceptibility and evidence for evolution of resistance (Duffy and Sivars-Becker 2007, Duffy et al. 2008). Surprisingly, we have not found significant genetic variation in the virulence or infectivity of *Metschnikowia* collected in different lakes and different years (Duffy and Sivars-Becker 2007). As a result, we do not consider parasite evolution in our current study.

The dynamics of infected *D. dentifera*, *I*, and transmission rate, *β*, follow the equations
The spread of disease (Eq. 1a) is described as in a modified “SI” model where the density of infected hosts (individuals/m$^2$) at sample $t$, $I_t$, depends on the densities of infected and susceptible hosts at sample $t - L$, respectively $I_{t-L}$ and $S_{t-L}$. We included a time lag $L$ in the model to match the biology of infection, because the incubation period of *Metschnikowia* is roughly 9–12 days (i.e., about three sampling visits; Hall et al. 2007). In the model all infected *D. dentifera* die between $t - L$ and $t$ (i.e., within 9–12 days; Eq. 1a); this assumption is reasonable given the high virulence of *Metschnikowia* and the high selectivity of fish predation on infected hosts (Duffy and Hall 2008). Thus, $I_t$ is the density of infected hosts that have fully developed infections at sample $t$, having been infected $L$ samples previously. Note that we do not explicitly model the dynamics of the susceptible hosts. We have data on the densities of susceptible hosts, and because we are not interested in explaining their dynamics, we use these data as input variables for the dynamics of the epidemic.

Disease transmission has several components. First, exponents $b_S$ and $b_I$ allow infection rates to scale non-linearly with densities of susceptible and infected hosts (Eq. 1a). If, for example, $0 < b_S < 1$ (as we find in this study; see the Appendix), then the density of infected hosts $I_t$ increases with the density of infecteds in the sample at $t - L$, $I_{t-L}$, but at a less-than-linear rate. The mass action model of disease transmission is recaptured when exponents $b_S$ and $b_I$ equal one. Second, the per capita transmission rate $\beta_t$ can depend on temperature, $T$, as governed by the exponential function in Eq. 1b, where the higher the value of $\beta_t$, the greater the decline in transmission with decreasing temperature from its nominal (i.e., temperature-independent) value $\beta_t^0$. Finally, the nominal per capita transmission rate $\beta_t^0$ can evolve, with hosts becoming more resistant as $\beta_t^0$ drops (Eq. 1c). Eq. 1c is derived under a model that treats the transmission rate $\beta_t$ as a quantitative genetic trait that depends on a measure of the clonal variance in susceptibility, $v$, and the ratio of infecteds to susceptibles at time $t$ when the sample is taken, $I_t/S_t$. The ratio $I_t/S_t$ is a measure of mortality of infected hosts, since infected hosts are invariably killed, and hence $I_t/S_t$ is proportional to the strength of selection for resistance. Note that in Eq. 1c, the lag is $t - 1$, not $t - L$ as in Eq. 1a; see the Appendix for a full derivation of Eq. 1c.

We fit the dynamics described by Eq. 1 by placing the model in state–space form (Harvey 1989). The state space approach allowed us to estimate the change in transmission rate, a variable that we otherwise could not observe through time, and to incorporate both environmental variability and measurement error. The overall structure of the model is

$$U_t = F[U_{t-1}] + \varepsilon_t$$  \hspace{1cm} (2a)

$$\log I^*_t = \log I_t + \eta_t$$  \hspace{1cm} (2b)

where in Eq. 2a, $U_t = (\log I_0, \log I_{t-1}, \log I_{t-2}, \beta_t)^T$ is the vector of lagged log densities of infected hosts and the transmission rate, $\varepsilon_t = (\varepsilon_{1,t}, \varepsilon_{2,t-1}, \varepsilon_{3,t-2}, 0)^T$ is the vector of environmental random variables (process errors, with variances of $\varepsilon_t$ all equal to $\sigma^2$) causing variability in log densities of infected hosts. We assume that process error variability in the equation describing the evolution of transmission rates (Eq. 1c) is vanishingly small, so the last element of $\varepsilon_t$ is zero. We make this assumption because the anticipated variance through time in the mean population-level transmission rate is small given the size of the *D. dentifera* population in a lake. The function $F$ describes dynamics of the system given in Eq. 1. In Eq. 2b, $I^*_t$ is the observed density of infected hosts in sample $t$, and $\eta_t$ is a normal random variable describing measurement error in the observations (i.e., the difference between observed and actual densities on a given sampling date). From repeated samples during our monitoring program, we calculated the error standard deviation in densities, which was 0.09445 individuals/m$^2$.

Because Eqs. 1a–c are nonlinear, we fit the state–space model (Eq. 2) using an extended Kalman filter (Harvey 1989) that gives the approximate log likelihood function. We fit the data from all five epidemics simultaneously, letting the initial transmission rates for each epidemic, $\beta_j^0$ ($j = 1, \ldots, 5$ epidemics) differ by treating them as parameters estimated in the fitting process. Thus, there were 10 parameters estimated in the full model: $b_S, b_I, \beta_{1,T}, v, \beta_j^0$ ($j = 1, \ldots, 5$), and $\sigma^2$.

We tested two hypotheses: (1) that the transmission rate $\beta_t$ depends on temperature ($H_0: b_T = 0$) and (2) that $\beta_t$ declines over the course of infection in a manner consistent with evolution ($H_0: v = 0$). We tested these hypotheses using likelihood ratio (LR) tests, comparing the full model containing all 10 parameters with the two reduced models, the first without an effect of temperature ($b_T = 0$) and the second without temporal changes in the nominal transmission rate ($v = 0$). Statistical inference was obtained by computing the maximum log likelihood for the full model, LL, and one of the reduced models, LL$_0$. Under mild statistical assumptions, the distribution of 2(LL – LL$_0$) is given asymptotically by a chi-square distribution with degrees of freedom equal to the difference in number of parameters between models, in our case 1 (Judge et al. 1985). Thus, for example, if 2(LL – LL$_0$) > 3.84 then we could reject the null hypothesis that a parameter is zero at the 0.05 significance level.
information-theoretic approach to model selection (Burnham and Anderson 2002). To compare models, we calculated AICc differences, where $\Delta_i = AICc_i - AICc_{\text{min}}$; the $\Delta_i$ of the best performing model equals zero and models with $\Delta_i < 2$ have substantial empirical support (Burnham and Anderson 2002). We also present the Akaike weights that give the relative probabilities that each of the three models is the correct model. We recognize that hypothesis testing and information-theoretic model selection are conceptually different statistical approaches (Burnham and Anderson 2002) and that some feel they should not be combined (Stephens et al. 2005, 2007, Lukacs et al. 2007). Nonetheless, they give complementary conceptual ways of interpreting the data that, in our case, are consistent with each other.

We also computed two measures of goodness of fit: the prediction $R^2$ and the process error prediction $R^2$. The prediction $R^2$ values resemble the values usually yielded by one-step ahead (here $L$-step ahead) procedures that ignore measurement error. In contrast, the process error prediction factors out unavoidable measurement error from the prediction $R^2$ (see Appendix for more details).

RESULTS

Metschnikowia showed strong epidemic dynamics in all five lake-years (Fig. 1). In all cases, infection prevalence was extremely low in July, then rose to a peak in late summer or autumn. Peak infection prevalence during these five epidemics was 8–11% in 2003 and 17–46% in 2004 (Fig. 1A). In four of the five epidemics, infection prevalence decreased substantially by the end of October. The exception was Bassett Lake in 2003. Although it appears that infection prevalence was beginning to wane, we were unable to sample through the complete termination of the epidemic; however, no infected D. dentifera were found in this lake when we resumed sampling in early summer in the 2004 season (S. R. Hall and M. A. Duffy, unpublished data).

Fits of the full model to the data (Fig. 2) show good agreement with the data. For the full model including both evolutionary changes in transmission and temperature-dependent transmission, the prediction $R^2$ value was 0.59 and the process error prediction $R^2$ value was 0.76 (Table 1). The process error prediction $R^2$ is necessarily higher than the prediction $R^2$ because it factors out the loss of predictive power due to measurement error.

The analyses of the three models show that evolution (which reduces transmission rates through time) provides a better explanation of the epidemic dynamics than temperature-dependent transmission rates alone. The null hypothesis that $v = 0$ (i.e., that there is no evolutionary change in transmission rate) was rejected (Table 1; LR test, $\chi^2 = 10.6, P = 0.0011$), whereas the null hypothesis that $b_T = 0$ (i.e., that there is no temperature-dependent change in transmission) could not be rejected (Table 1; LR test, $\chi^2 = 3.16, P = 0.07$). Taking an information-theoretic approach, differences in AICc values show that the full model and the model excluding temperature-dependent transmission have essentially the same support, whereas both of these models provided much better fit to the data than the model excluding evolution. The Akaike weights indicate that the full model and the model excluding tempera-
ture-dependent transmission have roughly the same probabilities of being correct (0.59 vs. 0.40, respectively), yet the probability of the model excluding evolution is much lower (0.01). Finally, both measures of goodness of fit, the prediction $R^2$ and the process error prediction $R^2$, show the full model to have the best fit followed closely by the model excluding temperature-dependent transmission (Table 1). However, the model excluding evolution was a distinct third.

The lack of a significant effect of temperature on infection dynamics can be explained by differences in the timing of epidemics (Fig. 1B). For three of the epidemics (Baker, Bassett 2004, Bristol), infection prevalence began to decrease when the lakes began to cool. For the other two epidemics (Warner and especially Bassett 2003), however, infection prevalence continued to increase even after the lakes had cooled substantially. Given this observation, it is useful to know whether the timing of the five epidemics used in this study was typical of that for Metschnikowia epidemics in general. To address this, we compared the timing of these epidemics with those of all 25 Metschnikowia epidemics observed during a larger lake survey conducted between 2002 and 2006 (Cáceres et al. 2006; C. E. Cáceres, M. A. Duffy, S. R. Hall, and A. J. Tessier, unpublished data). We described the timing of epidemics by using the date on which the maximal infection prevalence was observed (see Cáceres et al. 2006 for details on sampling methods). According to this comparison, the five epidemics sampled in this study were typical of all of the observed epidemics in their timing (Fig. 3). This lends support to our analysis of the five lake-years for which we have detailed time-series data; the pattern causing the better performance of the evolutionary model in the five lakes (i.e., the lack of correspondence between seasonality and declines in epidemics) is also seen in a broader sample of less-intensively sampled lakes.

**DISCUSSION**

Empirical examples of eco-evolutionary dynamics remain surprisingly scarce (Fussmann et al. 2007). Here, we found a dynamical signature consistent with rapid evolution during five intensively sampled epidemics. By fitting three models to time-series data, we found that

![Fig. 2. Fitted transmission rates and infected densities for the five epidemics. Epidemic time series are arranged side by side, as indicated at the top of the figure. (A) Log density of Metschnikowia-infected D. dentifera (measured as individuals/m²); x’s represent observed data from lake populations; the solid line shows the outcome of the full model incorporating evolution of host resistance and effects of temperature on transmission rate (i.e., the “full model” of Table 1). (B) Estimates of transmission rate obtained by fitting the full state-space model to the data.](image)

**Table 1.** Statistical comparison between temperature and evolutionary changes in transmission rates as explanations for epidemic dynamics.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Full model</th>
<th>Excluding evolution ($v = 0$)</th>
<th>Excluding temperature ($b_T = 0$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>$k$</td>
<td>10</td>
<td>9</td>
<td>9</td>
</tr>
<tr>
<td>log likelihood</td>
<td>-157.6</td>
<td>-163.0</td>
<td>-158.7</td>
</tr>
<tr>
<td>LRT (vs. full model)</td>
<td>$\chi^2 = 10.6, P = 0.0011$</td>
<td>$\chi^2 = 3.16, P = 0.07$</td>
<td></td>
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<tr>
<td>AIC</td>
<td>8.21</td>
<td>0.81</td>
<td></td>
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<tr>
<td>Akaike weights</td>
<td>0.59</td>
<td>0.01</td>
<td>0.40</td>
</tr>
<tr>
<td>Prediction $R^2$</td>
<td>0.588</td>
<td>0.563</td>
<td>0.586</td>
</tr>
<tr>
<td>Process error prediction $R^2$</td>
<td>0.756</td>
<td>0.726</td>
<td>0.752</td>
</tr>
</tbody>
</table>

**Notes:** For each model, we provide the number of parameters per model ($k$), log likelihoods, results from a likelihood ratio (LRT) test, Akaike’s information criterion differences (A), Akaike weights, and two types of coefficients of determination ($R^2$; see Appendix for details); $b_T$ is an exponent in Eq. 1 scaling transmission with temperature, and $v$ is the clonal variance in susceptibility. Parameter values are given in Appendix Table A1.
models including rapid evolution of increased resistance (i.e., lower transmission rates) fit better than models based on the alternative hypothesis that transmission rate decreases with seasonal drops in temperature. Our previous theoretical work shows that such declines in temperature-dependent transmission could extinguish epidemics (Hall et al. 2006). Nonetheless, the statistical evidence here more strongly points to rapid evolution as a factor driving the end of epidemics. This result complements previous empirical evidence for high resistance of several of these host populations after the 2003 epidemics and theory demonstrating that parasite-mediated directional selection could terminate disease outbreaks (Duffy and Sivars-Becker 2007).

In our models, evolution occurs as a decrease in the transmission rate over the course of the epidemic. Therefore, we infer that this change stems from evolution, but it is possible that other, non-genetic factors (e.g., phenotypic plasticity) also play a role. Nonetheless, our previous, experiment-based results have demonstrated rapid evolution of D. dentifera for resistance to Metschnikowia during the course of epidemics (Duffy and Sivars-Becker 2007, Duffy et al. 2008). Thus, when combined with previous experimental evidence, the statistical modeling developed here creates a compelling case for rapid evolution as an important component of the ecological dynamics in this planktonic disease system.

The role of evolution in host–parasite ecology has only been demonstrated in a few other cases. In gypsy-moth–virus interactions, evolution of host resistance also appears to be important to ecological host–parasite dynamics. However, in this case evolution appears to drive fluctuations in host density rather than termination of epidemics (Elderd et al. 2008). Pathogen evolution, rather than host evolution, can also affect ecological dynamics. For example, antigenic evolution of influenza is associated with periods of anomalously high mortality in human populations (Koelle et al. 2006). In our system, however, laboratory experiments have shown much higher potential for evolution in the host, D. dentifera, than in the parasite, Metschnikowia. While D. dentifera show ample genetic variation in susceptibility and evidence for evolution of transmission rate (Duffy and Sivars-Becker 2007, Duffy et al. 2008), we have not found significant genetic variation in the virulence or infectivity of Metschnikowia collected in different lakes and different years (Duffy and Sivars-Becker 2007). For that reason, we excluded parasite evolution from our analyses here. However, for many other host–parasite systems, the prominence of rapid host–parasite coevolution would require an epidemiological model that explicitly includes coevolution.

![Plate 1](image)

**PLATE 1.** (Left) Baker Lake in autumn. The Daphnia dentifera population in Baker Lake had an epidemic in the autumn of 2003. (Right) Uninfected and infected Daphnia dentifera collected from Baker Lake. The Daphnia on the left is uninfected, while the Daphnia on the right is infected with the yeast Metschnikowia. The bright white areas of the infected Daphnia are the regions where the yeast asci have collected. Photo credits: (left) M. A. Duffy, (right) Alan Tessier.
Our analyses centered on changes in transmission rates during epidemics within a season, rather than changes between epidemics (i.e., from year to year). However, year-to-year variation in epidemics creates an interesting puzzle for this system. If increased host resistance terminates epidemics, how do epidemics occur in subsequent, and even consecutive, years (this study; Cáceres et al. 2006)? Consider the case of Bassett Lake; the 2003 epidemic (during which transmission rate declined; Fig. 2) was followed by a large epidemic in 2004 (with accompanying high transmission rate detected by our modeling; Fig. 2). Apparently, the rapidly evolved resistance (low transmission) becomes lost between epidemics (years); this observation suggests that a trade-off might maintain highly susceptible genotypes. We have found a significant positive correlation between transmission rate and reproduction (i.e., a significant trade-off between resistance and reproduction; S. R. Hall, C. C. Cáceres, C. R. Becker, and M. A. Duffy, unpublished data). Theory suggests that, in some cases, such a trade-off between resistance and fecundity could catalyze disruptive selection (Boots and Haraguchi 1999). In a more detailed examination of the 2004 Bristol epidemic (Duffy et al. 2008), we characterized the signature of parasite-mediated disruptive selection (rather than directional selection as modeled here). Our present model did not allow for bimodal trait distributions, so our current approach could not detect disruptive selection. Exploration of this tension between disruptive and directional selection will motivate our future research, in part because it might strongly influence year-to-year variation in epidemics.

The results of our statistical comparison among models emphasized the importance of rapid evolution rather than temperature dependence of transmission rate. This finding seems surprising, given that we had previously detected strong temperature dependence of transmission of this yeast parasite in laboratory and field experiments (Hall et al. 2006). It is possible that temperature does exert an effect, but that it is indirect. There are at least three such indirect effects of temperature that may be important in the dynamics of this system. First, we have previously argued that temperature can be an important driver of disease dynamics, mediated largely by temperature-driven changes in selective predation from fish (Hall et al. 2006). In addition, temperature indirectly affects both food quality and the density of competitors in these lake populations, and such changes also have the potential to cause the end of epidemics (Hall et al. 2009a, b). Unfortunately, estimating such indirect effects of temperature via species interactions would require considerably more data than we could collect. Therefore, we did not include these factors when creating the evo-epidemiological model used here.

As examples of rapid evolution continue to emerge, it becomes increasingly clear that the traditional view of a partition between ecological and evolutionary timescales can be inaccurate (Hastiorst et al. 2005, Carroll et al. 2007). Instead, we are beginning to realize that evolution occurs on ecological timescales and can affect the dynamics of populations and communities (Hastiorst et al. 2005, Carroll et al. 2007, Fussmann et al. 2007). Our research supports this emerging view and suggests that we must simultaneously consider both ecological and evolutionary processes to understand host–parasite dynamics.

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Literature Cited


APPENDIX

Derivation of Eq. 1c, an explanation of the two types of $R^e$ using the Kalman filter, details on the transmission of Metchnikowia, and a table showing parameter estimates for the three models of epidemic dynamics (Ecological Archives E090-095-A1).

SUPPLEMENT

Matlab computer code and data for analyses of the full model (Ecological Archives E090-095-S1).