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DYNAMICAL EFFECTS OF PLANT QUALITY AND PARASITISM ON POPULATION CYCLES OF LARCH BUDMOTH

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Abstract. Population cycles have been remarkably resistant to explanation, in part because crucial experiments are rarely possible on appropriate spatial and temporal scales. Here we show how new approaches to nonlinear time-series analysis can distinguish between competing hypotheses for population cycles of larch budmoth in the Swiss Alps: delayed effects of budmoth density on food quality, and budmoth–parasitoid interactions. We re-examined data on budmoth density, plant quality, and parasitism rates. Our results suggest that the effect of plant quality on budmoth density is weak. By contrast, a simple model of budmoth–parasitoid interaction accounts for 90% of the variance in budmoth population growth rates. Thus, contrary to previous studies, we find that parasitoid–budmoth interaction appears to be the dominant factor driving the budmoth cycle.

Key words: *forest insects; larch budmoth; nonlinear time-series analysis; parasitism; plant quality; population cycles; Switzerland; trajectory matching;* Zeiraphera diniana.

INTRODUCTION

Population dynamics of forest insects have been a focus of numerous controversies in ecology. One area of contention is whether outbreak cycles are driven by density dependent processes or by exogenous forces (Royama 1981, Turchin 1990, Hunter and Price 1998). Within the density-dependence camp, arguments rage between proponents of intrinsic factors, e.g., maternal effects (Ginzburg and Taneyhill 1994) vs. trophic hypotheses (Berryman 1995).

Because dynamics occur on large spatial and temporal scales, in many cases it is not feasible, or at least it is very difficult and expensive, to test hypotheses about dynamic mechanisms experimentally. This is probably why there are so few experimental studies that successfully tested mechanistic hypotheses of population cycles, and only one, as far as we know, in a forest insect (snowshoe hares, Krebs et al. 1995; Red Grouse, Hudson et al. 1998; voles, Korpimäki and Norrdahl 1998; southern pine beetles, Turchin et al. 1999). If the only methodologically valid approach for elucidating the causes of population cycles is manipulative experiments in the field, then we should expect very slow progress in this area of ecology. On the other hand, Kendall et al. (1999) showed, for a laboratory population, how a combination of time-series analysis and mechanistic modeling could serve as an equally valid approach for distinguishing between alternative hypotheses to explain population cycles. In this paper we show that this approach can be applied successfully to populations in the field, to explain the cause of cycles in a forest insect pest, the larch budmoth (LBM).

Larch budmoth (*Zeiraphera diniana*) populations in the Swiss Alps are among the most remarkable recorded cycles, oscillating with a period of 8–9 yr and ~100 000-fold change between peak and trough density (Fig. 1). Ecological theory offers several potential explanations for such regular population cycles in forest insects: parasitoid–host interaction (Hassell 1978), delayed effects of plant quality (Fischlin and Baltensweiler 1979, Edelstein-Keshet and Rausher 1989), pathogen–host interaction (Anderson and May 1980), and maternal effects (Ginzburg and Taneyhill 1994). A large empirical research effort has been devoted to studying the LBM cycle and trying to identify the causal mechanisms (see review in Baltensweiler and Fischlin 1988). As a result of this effort, the last two hypotheses can be rejected in this system. There is no empirical support for maternal effects, and although the granulosis virus infection was observed to cause substantial mortality during the first two intensively studied outbreaks (50% in the peak year of 1955 and

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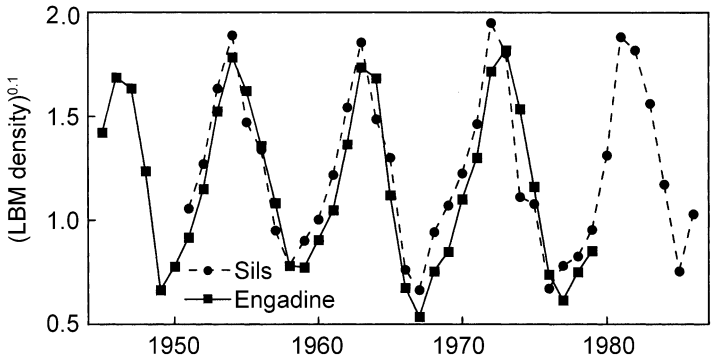

FIG. 1. Population fluctuations of larch budmoth density in the Upper Engadine Valley (for explanation of ''Engadine'' vs. ''Sils'' data, see *Methods: Sources of data*).

25% in the 1964 peak), subsequent outbreaks collapsed without being accompanied by any viral mortality (Baltensweiler and Fischlin 1988).

Plant quality (e.g., raw fiber and protein content) and parasitism have the necessary delayed effects to induce cycles. It takes two or more years for foliage quality to recover after heavy defoliation, which accompanies a budmoth population peak. Furthermore, field and laboratory bioassays show that poor food quality has a strong effect on budmoth survival and reproduction (Benz 1974, Omlin 1977, Fischlin 1982). Thus, the current explanation of budmoth cycles is based on their interaction with foliage quality (Baltensweiler and Fischlin 1988, Berryman 1999).

Measured parasitism rates vary between lows of 1–5% and highs of 80–90% (Delucchi 1982), and maximum parasitism rates are achieved ~2 yr after budmoth peaks. Previous studies concluded that mortality by parasitoid wasps does not cause the cycle, but merely tracks the budmoth population (Delucchi 1982, Baltensweiler and Fischlin 1988). However, as our analysis will show, this rejection of the parasitoid hypothesis was inappropriate.

The goal of our paper is to empirically distinguish between the two rival hypotheses for which there is empirical evidence in the larch budmoth (LBM) system: plant quality vs. parasitism. To do this, we formulated dynamic models of the LBM system that embody the rival hypotheses. We then use these mechanism-based models as statistical models for the purpose of testing hypotheses about which mechanism is best supported by the available data. Conceptually, we are simply using the standard approach to statistical hypothesis testing, but the highly nonlinear nature of the models complicates the implementation of the approach, as described below in *Methods: Models*.

METHODS

Sources of data

Systematic population census of larch budmoth in the Upper Engadine Valley (Switzerland) started in 1949 and with minor modifications continued until 1977 (Auer 1977, Baltensweiler and Fischlin 1988) (lesser quality data are also available for 1945–1948). Data were collected at multiple sites throughout the valley separately (Auer 1977; these data are tabulated in Fischlin 1982), but because all sites oscillated in close synchrony, we can average them into one time series that we call ''Engadine.'' The density of budmoth larvae (third instar) is expressed in number per unit of 1 kg of branches with foliage. For the period of 1952–1976 (with one year, 1968, missing) we have data on the percentage of larvae parasitized, also averaged over multiple sites (Delucchi 1982) omitting several data points that were interpolated by the author. Although numerous parasitoids are associated with larch budmoth, parasitism is dominated by the ichneumonid *Phytodietus griseanae* and three eulophid species, whose fluctuations are correlated ($R^2 = 0.94$ between log *Phytodietus* abundance and log eulophid abundance, $P < 0.001$). After 1977, sampling in the Upper Engadine valley continued on a reduced scale. At one site, Sils, data were collected in an uninterrupted sequence from 1951 to 1992: we refer to these as the ''Sils'' data (Baltensweiler 1993; A. Fischlin, *unpublished data*). During the period of 1961–1992, needle lengths of trees at Sils were also measured. Needle length is a good index of plant quality because it is well correlated with raw fiber and protein content of larch needles (Omlin 1977, Fischlin 1982). Furthermore, bioassay data of Benz (1974) indicated that needle length has a strong effect on larval survival and pupal mass (and pupal mass is closely connected to adult fecundity). Turchin (2003) calculated that the needle length of foliage with which LBM larvae were fed in the Benz (1974) bioassays explained 86% variance in a measure of LBM fitness (the product of larval survival and adult fecundity).

Models

To decrease the chance that an inappropriate modeling choice would bias our results against the plant quality hypothesis, we modeled the effect of plant quality on budmoth dynamics with two alternative functional forms. The first (the ''nonlinear'' version) is a modified Ricker model in which the discrete rate of budmoth population increase is a hyperbolic (saturating) function of plant quality (for equations, see Table

TABLE 1. Dynamic models and model equations for larch budmoth populations in Switzerland.

Model and parameters	Equation
Plant quality I (the ''nonlinear'' version)	
N_t: budmoth density	$N_{t+1} = \lambda_0 N_t \frac{Q_t}{\delta + Q_t} \exp[-gN_t]$
Q_t: plant quality index	$Q_{t+1} = (1 - \alpha)\left(1 - \frac{N_t}{\gamma + N_t}\right) + \alpha Q_t$
Plant quality II (alternative N_t equation, the ''linear'' version)	
N_t: budmoth density L_t: needle length	$N_{t+1} = N_t \exp[u + vL_t - gN_t]$
Parasitoid–host	
N_t: budmoth density	$N_{t+1} = \lambda_0 N_t \exp\left[-gN_t - \frac{aP_t}{1 + ahN_t + awP_t}\right]$
P_t: parasitoid density	$P_{t+1} = \phi N_t \left\{1 - \exp\left[-\frac{aP_t}{1 + ahN_t + awP_t}\right]\right\}$
Tritrophic I (full model)	
N_t: budmoth density	$N_{t+1} = \lambda_0 N_t \frac{Q_t}{\delta + Q_t} \exp\left[-gN_t - \frac{aP_t}{1 + ahN_t + awP_t}\right]$
P_t: parasitoid density	$P_{t+1} = \phi N_t \left\{1 - \exp\left[-\frac{aP_t}{1 + ahN_t + awP_t}\right]\right\}$
Q_t: plant quality index	$Q_{t+1} = (1 - \alpha)\left(1 - \frac{N_t}{\gamma + N_t}\right) + \alpha Q_t$
Tritrophic II (simplified ''linear'' version) N_t: budmoth density P_t: parasitoid density L_t: needle length	$N_{t+1} = N_t \exp\left[u + vL_t - \frac{aP_t}{1 + awP_t}\right]$

Notes: All state variables and parameters are bounded below by 0. In addition, Q_t, α, ϕ, γ, h, and w are bounded above by 1. The proportion of larvae parasitized was computed as $S_t = [1 - \exp\{-aP_t/(1 + ahP_t + awN_t)\}]$. We assumed that the field data underestimate (some instances of parasitism are missed because field-collected larvae must be reared in the lab) so the expected observed parasitism rate was assumed to be γS_t where $\gamma \leq 1$ is a parameter to be fitted. The measure of plant quality in our data is needle length, and we assumed a linear relationship between needle length and actual plant quality, $Q_t = b + cL_t$, where b and c are parameters to be fitted.

1). The parameter g within the exponent quantifies direct density dependence. The second (the ''linear'' version) assumes that the realized per capita rate of budmoth population change ($r_t = \log(N_t/N_{t-1})$ is a linear function of the plant quality index and LBM density. In the plant quality equation, quality next year is decreased by current-year herbivory in a hyperbolic fashion. Once herbivore damage becomes negligible (as a result of collapse of the budmoth population) quality increases gradually to the maximum, and the speed of recovery is regulated by the parameter α.

The host–parasitoid model is based on the standard Nicholson-Bailey framework, but with self-regulation in the host and a general form of functional response (Beddington 1975) that combines effects of prey saturation and parasitoid mutual interference.

The tritrophic model simply combines the nonlinear version of the plant quality model and the parasitoid–host model. In addition, we investigated a simplified version of the tritrophic model, which combined the ''linear'' version of the plant-quality model with the simplified form of parasitoid functional response (all equations in Table 1).

General philosophy of analysis

Our approach is based on the standard logic of statistical hypothesis testing. However, because our hypotheses are rich in mechanism, the mathematical models embodying these hypotheses are substantially more nonlinear and detailed than is usual in statistical hypothesis testing. As noted in the *Introduction,* we are focusing on two ''monocausal'' hypotheses (food quality and parasitism). In addition, it is possible that a composite explanation is correct (both food and parasitism jointly explain the LBM cycle). Once we have translated all hypotheses into models, we ask whether the observed data could plausibly have been generated by either of the simpler models or whether we require the more complicated model to explain them. The process involves fitting each model to the observed data

(estimating its parameters), so that model comparisons can be made with each model performing as well as its basic structure will allow (of course, both fitting and comparison must be performed using the same measure of fit). This is precisely the approach that underlies almost all statistical hypothesis testing.

The fact that standard statistical methods follow the framework that we use here can be seen by considering the theory underlying a familiar one-way ANOVA. In this case two models are compared, a null model that the data are all observations of random variables having the same mean, and an alternative model that they are observations of random variables whose mean depends on the level of some factor. Both models are fit to the data by least squares, and their fit compared by the difference in their residual sum of squares. The *P* value of the ANOVA is obtained by considering the distribution of difference in sum of squares that would be expected if the null model were true: we have to abandon the simpler model only if the improvement in model fit of the alternative is larger than could plausibly have occurred if the null model is true.

The models that we want to compare are highly nonlinear and this causes some difficulty in formulation and fitting. In particular, we must deal with two types of stochasticity: process error and measurement error. Process error is variability due to factors that are not explicitly modeled (''noise''). It affects the true values of population density. Measurement error is variability due to imprecisely measuring population density. Simultaneous treatment of both types of variability with highly nonlinear models is a relatively new development, requiring computationally intensive methods that have not yet received much testing (Ellner et al. 2002). We therefore chose to work with two complementary measures of fit, each addressing only one source of error. The first measure of model fit (one-step-ahead fitting) considers the average ability of the model to predict the population change over a year, given measurements of the system state at the beginning of the year: this amounts to neglecting measurement error. The second measure of model fit (global fitting, also known as ''trajectory matching'') measures the ability of the model to predict the entire series of data when iterated forward deterministically from starting conditions, which are also estimated by fitting: this amounts to neglecting process error, but yields accurate estimates if the period of oscillations is nonetheless constant or nearly so, as in LBM (Kendall et al. 1999, Wood 2001*b*). If conclusions based on both measures concur then we can have some confidence that neither of their key approximations is critical (Hilborn and Mangel 1997).

A key practical issue (especially for the trajectory-matching approaches) is that model fitting and inference are not straightforward for highly nonlinear, parameter-rich models when using relatively small data sets. In particular, the model fitting objectives (which measure model fit) can show a good deal of structure, including local minima, at a variety of scales. Much of this variability is of no statistical significance, in the sense that it is of a scale below that of the sampling variability of the fitting objective itself. Nonetheless, such fine-scale structure can make model fitting difficult and tends to spoil the performance of the large-sample approximations such as Likelihood Ratio tests that would usually allow for statistical inferences about the fitted models. We deal with this issue by using bootstrapping methods, both as a means of escaping from small-scale local minima during fitting (see Wood 2001*a*) and for statistical comparison of models.

Formally, then, we treat each model comparison as testing a null hypothesis that a simple model is correct against an alternative that a more complicated model is needed. The sequence of steps is as follows:

1) Both models are fitted to data, and their difference in fit, Δ, is noted.

2) We create replicate simulated data sets under the null hypothesis that the simpler model is true by bootstrap resampling the residuals from the simpler model and adding this resample to the fitted values from the simpler model.

3) Both models are fitted to each bootstrap-simulated data set and the difference in their fit Δ^* is noted for each replicate.

4) The Δ value is compared to the distribution of Δ^* values generated by simulation—if it lies well within the distribution then the null hypothesis that the simple model is correct can be accepted, otherwise it will be rejected in favor of the alternative model. A *P* value can be obtained by assessing the proportion of Δ^* values which are more favorable to the alternative model than the observed Δ.

One-step-ahead fitting

The response variable was defined as the realized per capita rate of population change, defined as $r_t = \ln(N_t) - \ln(N_{t-1})$ (Turchin 1990, Berryman 1991, Royama 1992). Each model used (see Table 1) could be recast to predict this quantity given measurements of some or all of N_{t-1}, L_{t-1}, and P_{t-1}(LBM density, needle length [an index of food quality], and parasitism rate, respectively), depending on the model in question. Model parameters were estimated by minimizing the mean square difference between model predictions of r_t and observed r_t using the nonlinear regression routines in STATISTICA (Statsoft 1999).

As explained in *Methods: General philosophy of analysis,* bootstrap resampling of the model residuals was performed by resampling with replacement from the model residuals. We selected the subset of years (1962–1978) for which we had both needle length data and parasitism data. We used Sils budmoth data as the response variable in the regression analysis (thus, this procedure biases our results in favor of the plant quality hypothesis). Furthermore, we used the linear model r_t

$= u + vL_{t-1}$, because our analysis showed that it fitted the data better than the alternative model in Table 1 (given the one-step-ahead fitting objective). We also fitted the data with the parasitism model $r_t = r_0 - aP_{t-1}/(1 + awP_{t-1})$, and the tritrophic model $r_t = u + vL_{t-1} - aP_{t-1}/(1 + awP_{t-1})$ (this is Tritrophic Model II in Table 1). Note that the test we constructed is conservative with respect to finding for the parasitism hypothesis.

Global fitting

Global fitting was performed using the methods described in Wood (2001*b*). Each model was fitted to all available data predicted by that model, using a least squares objective. In this case the model is not making predictions of the next year's population change given observations of state variables at the start of the year and the parameters, but is predicting each of the entire series of data given the model parameters and some starting conditions (which like the parameters, must be estimated). To stabilize variances, the LBM density was raised to the power 0.1 for fitting, and a linear transformation was applied to needle length in order that needle length residuals were comparable to transformed LBM and parasitism rate residuals. Parasitism rates were fitted on the usual arcsine square-root transformed scale. Bootstrapping of the residuals was performed by resampling with replacement on a series by series basis. Local minima were avoided during fitting by bootstrap restarting (Wood 2001*a*).

Again, the computer intensive methods described in the section *Methods: General philosophy of analysis* were used to test reduced models against the tritrophic model, using difference in residual sum of squares between the alternative models as Δ. First, all four time series (Engadine, Sils, needle length, and parasitism rate) were fitted with the trajectory-matching method (1) by the tritrophic model and (2) by a reduced model in which food quality has no influence on the host but hosts have an impact on food quality (i.e., $\delta = 0$ in the tritrophic model of Table 1). The effect of plant quality was modeled using the first alternative in Table 1, because we found that it fitted data better than the linear model. Again, this procedure biases results in favor of the plant quality model. Secondly, the three series other than the parasitism rate were fitted (1) by the full model, and (2) by the plant quality model of Table 1. (The plant quality model cannot be used to predict parasitism rate: when we remove from the model the effects of parasitism on LBM, we also remove the effects of LBM on the parasites because both are functions of the same parasitism rate).

RESULTS

Exploratory regression analysis

The regression analysis indicated that the plant quality index has a surprisingly weak effect on budmoth dynamics. The linear model fits the data better than the nonlinear alternative, but still explains only 31% of the variance in the realized per capita rate of change of budmoth, $r_t = \ln(N_t) - \ln(N_{t-1})$ (Table 2a; the nonlinear model explains 15% of the variance). Interestingly, the last documented budmoth peak (in 1989, not shown in our graphs) was curtailed due to several years of unfavorable weather in winter and spring (Baltensweiler 1993). As a result, budmoth did not achieve the usual peak density and there was no widespread defoliation. The plant quality index did not collapse, but budmoth density did, suggesting that poor plant quality (as measured by needle length) may not be necessary for driving the budmoth population to a low level.

TABLE 2. Results of analyses of time-series data.

a) Nonlinear regression

Model	Dependent variable	R^2
Plant quality	larch budmoth (r_t)	0.312
Plant quality	plant quality (Q_t)	0.615
Parasitoid–host	larch budmoth (r_t)	0.880
Parasitoid–host	parasitoid (P_t)	0.712

b) Results from trajectory matching

Model	R^2	P value
Full tritrophic	0.839	...
No feedback from plant quality to host	0.828	0.025
Plant quality only	0.443	<0.003

Notes: R^2 is the coefficient of determination, and $r_t = \log N_{t+1}/N_t$. Other variables and equations are the same as in Table 1.

By contrast, parasitism rates explain almost 90% of variance in the per capita rate of change of budmoth numbers (Table 2a). In fact, a simple model $r_t = r - aP_{t-1}/(1 + awP_{t-1})$ with only three fitted parameters explains the bulk of the effect of parasitism on budmoth rate of population change ($R^2 = 86.5\%$). Using a different metric, $\log N_t$ instead of r_t, and the same model, we find that 94.1% of the variance in $\log N_t$ is explained by parasitism rates (see Fig. 2). We are not aware of any other field data sets in population ecology in which such a simple model can explain a comparable amount of variation. Interestingly, the effects of budmoth density on plant quality and on parasitism rates are almost equally strong, with $>70\%$ of variance explained in each case (Table 2a).

The analysis of joint effects of quality and parasitism in the reduced data set showed that the plant quality model explained 48.0% of the variance in budmoth rate of population change (the higher proportion of variance explained than in the full data set is primarily due to the reduced data set not including the last oscillation). The parasitism and tritrophic models explained 65.6% and 66.1% of variance, respectively.

The trajectory matching (global fitting) analysis yielded very similar results. The full tritrophic model provided an excellent fit to the data (Fig. 3), marginally

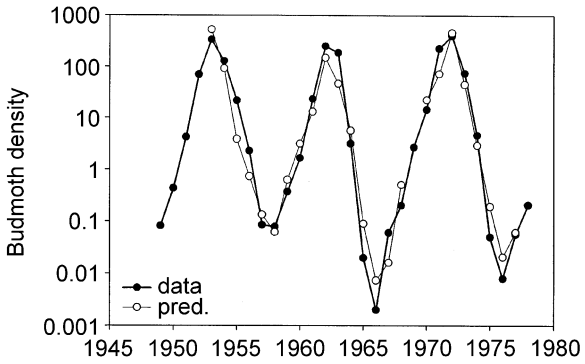

FIG. 2. Comparing predictions of the fitted parasitoid model to data. Predictions are obtained according to the following formula: $\hat{N}_t = N_{t-1} \exp[r - aS_{t-1}N_{t-1}/(1 + awS_{t-1}N_{t-1})]$ where N_t and S_t are observed budmoth density and proportion parasitized, and r, a, and w are fitted parameters.

better then the model omitting the effect of plant quality on budmoth (Table 2b). By contrast, the food quality model fitted poorly (Table 2b).

To summarize, exploratory one-step-ahead regression analysis indicated that parasitoids have a strong effect on budmoth dynamics, but it did not detect any effect of plant quality. Global fits suggested similar results albeit hinting at the possibility of a slight effect of plant quality. These results are consistent with the hypothesis that budmoth population cycles are driven primarily (or solely) by the moth's interactions with parasitoids, with plant quality fluctuating in response to changes of budmoth numbers.

Formal hypothesis testing

Using one-step-ahead fitting the parametric bootstrap test, described in *Methods*, indicated that the tritrophic model fitted data significantly better than plant quality model ($P = 0.029$). By contrast, the parasitism model fitted the data as well as the tritrophic model ($P = 0.64$). In other words, with this measure of fit, the analysis yields no evidence for an effect of plant quality on the rate of larch budmoth population growth. This result is particularly striking because our statistical test was designed to be biased in favor of plant quality hypothesis (see *Methods: Models*).

Using global fitting and testing the full tritrophic model against the null hypothesis of a reduced ''parasitoid only'' model (in which the feedback from plant quality to budmoth is eliminated) yielded an estimated P value of 0.025, indicating that the small effect of plant quality on host dynamics seen in the one-step-ahead regression analysis is nonetheless real and plays a role in shaping budmoth cycles. Testing the full model against the plant-quality-only model, yielded an estimates of $P < 0.003$ with this measure of fit. Thus, there is strong evidence that plant quality alone cannot account for the observed host cycles, which lends firm support to both the exploratory results and the formal testing with the one-step-ahead approach.

DISCUSSION

Summary of LBM results

Our re-examination of data on budmoth density, plant quality, and parasitism rates suggests that the effect of plant quality on budmoth density is weak. By contrast, a simple model of budmoth–parasitoid interaction accounts for 90% of the variance in budmoth population growth rates. Thus, contrary to previous studies, we find that parasitoid–budmoth interaction appears to be the dominant factor driving the budmoth cycle. The fact that parasitism does not peak until budmoth is already in decline was previously taken as evidence against the parasitism hypothesis (Delucchi 1982). However, our model shows that this phase lag is consistent with parasitism driving the cycles (Fig. 3).

Our results, however, should not be interpreted as suggesting that plant quality plays no role in larch budmoth cycles. Although the regression approach could not detect a statistically significant effect of plant quality on larch budmoth population rate of change, the trajectory matching results indicate that the tritrophic model fits data slightly (and statistically significantly, $P = 0.025$) better than the parasitism-only model.

The fact that the regression (one-step-ahead fitting) and trajectory matching (global fitting) approaches are not in perfect agreement about the effect of plant quality reflects the different model fitting criteria, which have different strengths and weaknesses. The regression analysis deals well with process noise by concentrating on short time scales, but consequently has low power to detect weaker interactions whose effects on the dynamics only become apparent over longer time

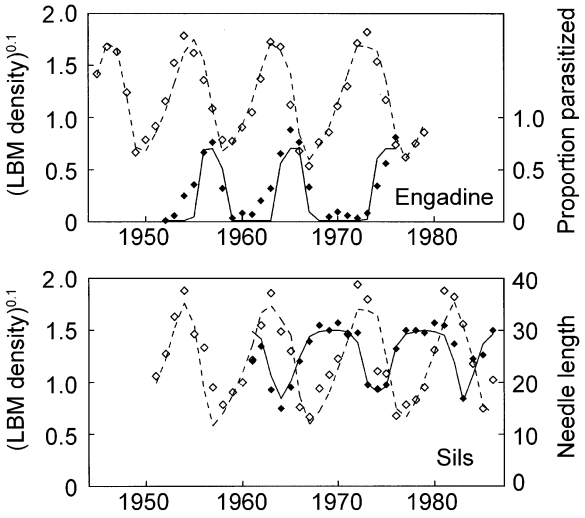

FIG. 3. Results of trajectory matching. Upper panel: Engadine data set (hollow symbols indicate budmoth density; filled symbols indicate proportion parasitized). Lower panel: Sils data set (hollow symbols indicate budmoth density; filled symbols indicate needle length).

scales. It is also sensitive to measurement errors, which are usually nontrivial in any ecological field study. In contrast, trajectory matching deals well with measurement error noise, and is sensitive to long time-scale patterns in the data, but makes an unrealistic assumption that process noise is absent. The observation that the two approaches agree on the role of parasitism in larch budmoth dynamics greatly strengthens this result.

Another feature of our approach that strengthens the main conclusion is that we did everything we could to avoid biasing the analysis against the food quality hypothesis. In fact, we consistently made choices that could be regarded as biasing the analysis in favor of the food-quality hypothesis, and nonetheless it failed to perform better than the parasitism hypothesis.

General implications

Models based on very different ecological mechanisms can produce dynamics that are qualitatively similar (Hunter and Dwyer 1998). For example, pathogen, plant quality, and parasitism models can all generate second-order oscillations (that is, cycles characterized by delayed density dependence) with periods of 7–10 yr typical of forest insect populations. Traditional approaches using qualitative comparisons between model predictions and data are unable to distinguish between dynamically plausible alternative hypotheses. Furthermore, since experimental manipulations at appropriate spatiotemporal scales are often impossible or prohibitively expensive, it would seem that we are unable to make further progress in elucidating the causes of forest insect cycles.

In a previous paper (Kendall et al. 1999), we argued that a way out of this apparent impasse is to develop approaches that combine mechanistic modeling with rigorous time-series statistics. Kendall et al. (1999) showed that in a laboratory setting such a synthetic approach can distinguish among rival mechanisms that predict qualitatively similar dynamics. In this paper, we show that the same approach can be successfully used in a field situation. The clear message from our analysis is that the dynamic data best support the hypothesis that LBM cycles are driven by parasitism, but modified slightly by an interaction with food quality. The reason we can obtain such a strong result is because we utilize the data more fully (performing detailed quantitative comparison of models with data) and because we employ not only data on LBM dynamics, but also on the dynamics of potential interactors (food quality and parasitoids).

Although technically quite involved, our approach has the same logical structure as standard hypothesis tests such as ANOVA: two models (e.g., effect present vs. effect absent) are compared based on their ability to fit the data, and the simpler is rejected in favor of the more complex if the improvement in goodness of fit is large enough. Few ecologists would dispute that an ANOVA table with $P < 0.003$ (this is the P value associated with rejecting the food quality hypothesis, see Table 2) should be regarded as a decisive result. The same force of evidence emerging from a dynamical model should be equally decisive. In fact, we would argue that it should count for more; our result is based on biologically plausible and empirically grounded dynamical models, while the assumptions of ANOVA are rarely subject to scrutiny.

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