Detecting the causes of population cycles by analysis of R-functions: the spruce needle-miner, *Epinotia tedella*, and its parasitoids in Danish spruce plantations

Mikael Münster-Swendsen and Alan Berryman

Explaining the causes of regular multiannual oscillations (cycles) in animal populations has been a major problem for ecology, partly due to a lack of methodological rigor. In this paper we show how the analysis of R-functions, the functional relationship between the per capita rate of change of a species and components of its environment, can be used to detect the causes of population cycles. Analysis of the R-functions enables one to separate cycles due to negative feedback between species (endogenous causes) from those forced by one-way effects (exogenous causes). We illustrate the approach by reference to the spruce needle-miner inhabiting Danish spruce plantations, and conclude that population cycles in this insect are probably caused by interactions with two species of parasitic hymenoptera.

M. Münster-Swendsen and I were working on this paper at the time of his untimely death in 2003. The purpose of the paper was to show how my approach to population analysis (Berryman 1990, 1999, 2001) could be used to detect the causes of population cycles in his data on the spruce needle-miner, *Epinotia tedella* (CL.) (Münster-Swendsen 1979, 1982, 1985). I should point out here that, at the time we first met, Mikael was not too impressed with my approach, being a proponent of the life table–key factor approach (Varley et al. 1975). In time, however, he was to change his mind, and the reason for that is one of the lessons of this paper. I, on the other hand, was very impressed with his data. For 19 years he measured, with considerable precision (Münster-Swendsen 1985), the density of spruce needle-miners and their primary insect parasitoids emerging annually from the litter of a Danish spruce plantation. He then constructed detailed life tables, including all suspected mortality factors, for 9 of those years. Finally, he repeated the sampling for various shorter periods of time at 10 additional locations. Others may have longer time series, more detailed life tables, or more spatial replication, but none to my knowledge have more of all three. Mikael recently reviewed his analysis in chapter 2 of a book that I edited (Münster-Swendsen 2002), so there is no need for me to repeat it here. Suffice to say that the needle-miner population was found to exhibit spatially synchronous 6–7 year cycles of abundance, and that the key factor affecting the population change was reduced fecundity. He also briefly mentions how my approach led to a different conclusion, that insect parasitoids were mainly responsible for the cyclic dynamics, and that the discovery of pseudoparasitism reconciled this conflict. However, he did not cover details of our analysis, referring instead to the present paper, which was
interrupted in such an untimely and tragic manner. Thus, I am lead to complete this paper, partly to fulfill my obligation to Mikael, and partly as a tribute to my friend and colleague and a great Danish entomologist.

The problem

Many animal populations exhibit spectacular fluctuations in abundance that reoccur with almost clockwork regularity (Elton 1942, Keith 1963), but explaining the cause of these periodic oscillations has not been an easy problem for ecology. Many hypotheses have been proposed and argued about – e.g. climatic and sunspot cycles, predator–prey interactions, plant–herbivore interactions, maternal effects, genetic feedback (reviewed by Krebs and Myers 1974, Finerty 1980, Myers 1988, Berryman 1996) – but no clear picture has emerged. Part of this problem may be a lack of methodological rigor and consistency. Here we propose a general methodology for attacking the problem of population cycles.

In a general sense, there are only two ways by which regular cycles can be induced in a dynamic system. The first and most obvious is that they are driven by, or follow after, some independent external variable that cycles with the same frequency as the variable of interest (e.g. sunspot cycles). In this case the cycles are said to result from exogenous forcing. The second way involves the idea of circular causality (Hutchinson 1948). It is well known that cycles can be caused by time-delays in the operation of negative feedback loops, and that the length of the time delay is proportional to the number of variables involved in the loop (Milsum 1966). In this case the dynamics are the result of the internal structure of the system and are, therefore, due to an endogenous process. Thus, the first question that needs to be answered when confronting cyclical population data is whether the cycles are the result of exogenous or endogenous processes. Given an answer to this general question, then the next problem is to identify the specific mechanism(s) involved. For example, specific exogenous mechanisms may involve cyclical weather effects on per capita birth and/or death rates, while specific endogenous mechanisms may involve genetic or phenotypic feedback or interactions between trophic levels.

The data

We illustrate a solution to this problem by reference to data on the spruce needle-miner (Epinotia tedella (Cl.); Lepidoptera: Tortricidae) inhabiting Danish spruce plantations (Münster-Swendsen 1985, 2002). The needle-miner is a univoltine insect whose adults emerge in June to lay eggs on the old needles of Norway spruce trees (Picea abies Karst.). Larvae mine the needles from July through October and descend from the canopy to hibernate in cocoons in the forest floor in November. Pupation takes place in early May. Young needle-miner larvae are attacked by a complex of univoltine, well synchronised, endoparasitic wasps (Hymenoptera). In November, parasitised larvae descend from the canopy with their healthy cohorts. The parasitoids eventually take over and kill their hosts in late April of the following year. Adult parasitoids emerge from the forest floor a week or so after the needle-miner moths.

The density of mature needle-miner larvae was estimated for 19 years in a particular Norway spruce plantation by placing funnels to intercept larvae as they descended from the canopy in November (Table 1). All larvae were dissected to determine if they contained parasitoids. The number of primary parasitoids, Apanteles tedellae Nix. and Pimplopterus dubius (Hgn.), were identified for all years, and the secondary parasitoids Campoplex cursitans and Mesochorus silvarum were recorded for nine years (Table 1). The time series suggest that spruce needle-miners and their two primary parasitoids exhibit more or less synchronous cycles of abundance (Fig. 1). The question is: is one population merely tracking the abundance of the other (exogenous cause) or are cycles the result of feedback between the parasitoids and their host (endogenous cause)?

The analysis

A common approach to search for causes to population cycles is to look for correlation between variables. For example, we could calculate the correlation between the spruce needle-miner and parasitoid time series shown in Fig. 1. When we do this we obtain large coefficients, suggesting that all populations are closely related; i.e. $r = 0.92$ between $E. tedella$ and $A. tedellae$, 0.81 between $E. tedella$ and $P. dubius$, 0.70 between $A. tedellae$ and $P. dubius$, and 0.95 between $E. tedella$ and the total number of primary parasitoids. However, the correlation coefficients alone provide little information about the causal structure since they cannot tell us whether the populations are tracking an exogenous cycle or are driven by feedback between parasitoid and host populations. Answering this question requires a different approach.

Detecting feedback between variables

Changes in populations of living organisms are caused by factors that affect the birth and death rates of individual organisms. In other words, it is individuals that are born and die, and it is these individual processes that cause changes in population density. From this perspective, it makes sense to look for associations between the per capita birth and death rates and the
suspected causal variable(s) (Royama 1977, 1992, Berryman 1999, 2001). Even though these rates may not be directly measured, we can estimate their net effect by calculating the realized per capita rate of change from a time series (Berryman 1999)

\[ R^N_t = \ln (1 + B - D) = \ln N_t - \ln N_{t-1} \]  

where \( N_t \) is the density of population \( N \) (say spruce needle-miners) at time \( t \), \( B \) and \( D \) are per capita birth and death rates, respectively, and \( R^N_t \) is the realized logarithmic per capita rate of change of the population over the time interval. Natural logarithms are used for biological and statistical reasons (Royama 1992, Berryman and Turchin 2001, Berryman 2003). The correlation between \( R^N_t \) and the value of another variable (say a population of parasitoids) provides an estimate of the effect of that variable on the per capita rate of change of individual needle-miners. For example, the correlation between needle-miner R-values and the density of the parasitoid \( A. tedellae \) was found to be \( r = -0.46 \). An almost identical effect was found with the other primary parasitoid, \( P. dubius \). Using the natural logarithm of parasitoid density improved the correlation to \( r = -0.61 \) and \( -0.47 \), respectively. Using the sum of parasitoids as the independent variable, or entering them separately in multiple regression, produced little improvement. Parasitism, it seems, resolves no more than 40% of the variation in needle-miner rates of change. However, part of the reason for this poor determination may be that the wrong model is being used for the parasitoid–prey interaction (Berryman 1992).

The model implied by linear regression of \( R^N_t \) on the density of \( A. tedellae \) is

\[ R^N_t = a^N + b^N A_{t-1} \]  

with \( a \) and \( b \) the parameters estimated by regression (\( r = -0.46 \)), and \( A_{t-1} \) the density of \( A. tedellae \) at the beginning of the period over which the rate of change was measured (in our case a year). Since the needle-miner may be influenced by the density of its own population, the model

\[ R^N_t = a^N + b^N N_{t-1} + c^N A_{t-1} \]  

may be more appropriate. Once again the constants \( a, b, \) and \( c \) are estimated by multiple linear regression, but the coefficient of multiple correlation (\( r_m = 0.50 \)) indicates little improvement over (2). We could also add the other primary parasitoid, \( P. dubius \)

\[ R^N_t = a^N + b^N N_{t-1} + c^N A_{t-1} + d^N B_{t-1} \]  

improving things slightly (\( r_m = 0.62 \)). Finally, we can use the sum of the parasitoids as the independent variable

\[ R^N_t = a^N + b^N N_{t-1} + c^N (A_{t-1} + B_{t-1}) \]  

and obtain the same effect (\( r_m = 0.62 \)).

<table>
<thead>
<tr>
<th>Year</th>
<th>Epinotia tedella</th>
<th>Apanteles tedellae</th>
<th>Pimplopterus dubius</th>
<th>Campoplex carstians</th>
<th>Mesochorus silvarum</th>
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<tr>
<td>1970</td>
<td>5708</td>
<td>233</td>
<td>2369</td>
<td>178</td>
<td>1051 (134, 917)</td>
</tr>
<tr>
<td>1971</td>
<td>21197</td>
<td>1130</td>
<td>7709</td>
<td>327</td>
<td>1393 (308, 1085)</td>
</tr>
<tr>
<td>1972</td>
<td>33654</td>
<td>3924</td>
<td>13788</td>
<td>193</td>
<td>560 (248, 312)</td>
</tr>
<tr>
<td>1973</td>
<td>10064</td>
<td>2290</td>
<td>2884</td>
<td>14</td>
<td>42 (12, 30)</td>
</tr>
<tr>
<td>1974</td>
<td>2557</td>
<td>312</td>
<td>743</td>
<td>59</td>
<td>93 (50, 43)</td>
</tr>
<tr>
<td>1975</td>
<td>6957</td>
<td>1100</td>
<td>952</td>
<td>223</td>
<td>116 (71, 45)</td>
</tr>
<tr>
<td>1976</td>
<td>10554</td>
<td>2795</td>
<td>1754</td>
<td>466</td>
<td>274 (201, 73)</td>
</tr>
<tr>
<td>1977</td>
<td>24200</td>
<td>8785</td>
<td>3165</td>
<td>802</td>
<td>1000 (690, 310)</td>
</tr>
<tr>
<td>1978</td>
<td>21525</td>
<td>11578</td>
<td>5196</td>
<td>21</td>
<td>26 (15, 11)</td>
</tr>
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<td>1979</td>
<td>773</td>
<td>346</td>
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<td></td>
</tr>
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<td>1980</td>
<td>134</td>
<td>30</td>
<td>45</td>
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</tr>
<tr>
<td>1981</td>
<td>105</td>
<td>2</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>1982</td>
<td>1324</td>
<td>241</td>
<td>15</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1983</td>
<td>3550</td>
<td>196</td>
<td>313</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1984</td>
<td>19352</td>
<td>2227</td>
<td>1986</td>
<td></td>
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</tr>
<tr>
<td>1985</td>
<td>69864</td>
<td>22901</td>
<td>7706</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1986</td>
<td>55947</td>
<td>22877</td>
<td>17170</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1987</td>
<td>7697</td>
<td>2596</td>
<td>1524</td>
<td></td>
<td></td>
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<tr>
<td>1988</td>
<td>1278</td>
<td>322</td>
<td>509</td>
<td></td>
<td></td>
</tr>
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</table>

Table 1. Data for the spruce needle-miner and its insect parasitoids converted to numbers per 100 m$^2$ of forest soil (Münster-Swendsen 1985 for details on sampling procedures). Data in parentheses indicate Mesochorus found in Apanteles (including Campoplex) and Pimplopterus, respectively.
The relationship between the per capita rate of change of a particular species and the variables that affect it has been called the R-function (Huffaker et al. 1999, Berryman 1999). R-functions defined by Eq. 3 and 4 are of the Lotka–Volterra type because the parasitoid effect is independent and additive. When the independent variables are transformed to natural logarithm, they become Gompertz modifications of the Lotka–Volterra model.

An alternative approach to modeling trophic interactions is to use a logistic R-function, in which case the rate of change is related to the ratio of consumers to their resources (Berryman 1990, 1999, 2004, Berryman et al. 1995a, Berryman and Gutierrez 1999). The logistic R-function for a host attacked by 2 parasitoids in the absence of alternative prey is

\[ R^N_t = a^N + b^NN_{t-1} + c^N\frac{A_{t-1} + B_{t-1}}{N_{t-1}} \]  

(6)

(Note that this is an approximation since *P. dubius* attacks other hosts.) When this model is fit to the data, the coefficient of multiple correlation increases to \( r_m = 0.86 \), which is a considerable improvement. Omitting the intra-specific effect (the second term) from the regression gives a simple correlation \( r = -0.85 \) between *Epinotia* R-values and the parasitoid/host ratio (or the proportion of larvae parasitized), suggesting that this variable explains almost all the variation in the multiple regression. Entering the parasitoid species separately

\[ R^N_t = a^N + b^NN_{t-1} + c^N\frac{A_{t-1}}{N_{t-1}} + d^N\frac{B_{t-1}}{N_{t-1}} \]  

(7)

marginally improves the coefficient of multiple correlation to \( r_m = 0.90 \). It is worth noting that the logistic R-function arises from Thompson's (1924) model for the relationship N/K with N the density of consumers (in this case parasitoids) and K proportional to the supply of food (in this case needle-miner larvae; Berryman 2004 for an empirical example). Entering the parasitoid species separately increases the coefficient to \( r_m = 0.84 \). Repeating the analysis with *P. dubius* yields coefficients of \( r_m = 0.51 \) and 0.67 for the Lotka–Volterra model, \( r_m = 0.68 \) and 0.84 for the Gompertz variant, and \( r_m = 0.86 \) and 0.87 for the logistic R-function.

Effects of each species (or combination of species) on the rate of change of the other is summarized in Fig. 2 for the Lotka–Volterra and logistic R-functions. It is clear that the logistic form provides the best fit to the data. Also apparent is the strong and more or less symmetrical reciprocal interaction (feedback) between the needle-miner and its primary parasitoids, supporting the hypothesis that the observed population cycles are due to an endogenous mechanism resulting from the feedback between parasitoid and host populations. Before accepting this result, however, we need to make sure that the detected interactions make biological sense.

The first requirement for biological consistency in a parasitoid/host model is that parasitoids have a negative effect on the per capita rate of change of their prey (i.e. parasitoids should harm their hosts) (Royama 1992, Berryman et al. 1995b). Thus, the interspecific interaction coefficient in the prey equation should be negative in both Lotka–Volterra and logistic models; i.e. \( c^A < 0 \) and \( d^N < 0 \) in Eq. 3–7. The second requirement is that parasitoid rates of change be positively related to the density of their prey (i.e. hosts should benefit parasitoids). This means that the interspecific interaction coefficients in the parasitoid equation should be positive in the Lotka–Volterra model and negative in the logistic; e.g. \( c^A > 0 \) in Eq. 8 and \( c^A < 0 \) in Eq. 9. In addition, we would normally expect the intraspecific coefficient to be negative or zero in all models; e.g. \( b^N \leq 0 \) and \( b^A \leq 0 \).

Figure 2 indicates that the conditions for biological plausibility are met in both Lotka–Volterra and logistic spruce needle-miner R-functions (i.e. negative slopes), but that the former is not consistent in the parasitoid R-functions (i.e. negative slopes when they should be positive). This inconsistency holds for both simple and multiple regression and for the Gompertz form. Hence, the logistic R-function appears to be the superior model for describing parasitoid–host dynamics, not really a surprising conclusion since it was derived from an analysis of parasitoid searching (Thompson 1924, Berryman and Gutierrez 1999).
Feedback within the parasitoid complex
In the above analysis we used the 19 year time series (Table 1) consisting of data for the needle-miner and its two primary parasitoids, *A. tedellae* and *P. dubius*. However, for nine years of this study (1971–1979), Münster-Swendsen (1985) recorded all species in the parasitoid complex (Table 1), enabling us to examine the separate and combined effects of the different species on each other’s R-functions. In addition to the two primary parasitoids (above), the complex contained the clepto-parasitoid, *Campoplex cursitans* (Hgn), that only attacks *E. tedella* larvae that have previously been parasitized by *A. tedellae*, and the hyper-parasitoid, *Mesochorus silvarum* Curt, that attacks all the other parasitoid species. Although these time series are rather short, they still offer a chance to extend our analysis. For reasons discussed above, the logistic model was used to determine the relative contribution of each species to the four species-specific R-functions. The results are summarized in Table 2. The first thing to notice is that 81% of the variation in needle-miner per capita rates of change can be explained by total primary parasitism (row 8 under $R_N$ in Table 2), a value somewhat higher than that found in the longer (19 year) time series. When the two primary parasitoids are separated, however, *A. tedellae* explains a much higher percentage of the variability than *P. dubius* (Table 2; row 6 and 7 under $R_N$). Notice that the best multiple regression model explains no more of the variability in the needle-miner R-function than total primary parasitism alone, again implying that total parasitism is the most important causal variable in needle-miner dynamics (c.f. row 8 and 13).

Turning our attention to the R-function for *A. tedellae* (Table 2 under $R_A$), we see that the largest effect again comes from the ratio of total parasitoids to host numbers (row 8). There is also a fairly strong association between *A. tedellae* and its own rate of change (row 2), suggesting that interference competition may be important in this species (something that we will encounter again). Note that the proportion of hosts parasitized by *P. dubius* contributes little by itself to *Apanteles* rates of change but improves the combined effect (row 7 and 8), suggesting that competition between parasitoid species may be important. It is rather interesting that the clepto- and hyper-parasitoids had little...
Table 2. Simple coefficients of determination (100 $r^2$) for needle-miner and parasitoid R-values regressed against selected independent variables and the best, biologically consistent, multiple regression (* marks variables used in the multiple regression and blanks cells indicate biologically inconsistent coefficients). $R^2 = R$-function for the ith species: $N = \text{Epinotia tedella, } A = \text{Apanteles tedellae, } B = \text{Pimplopterus dubius, } C = \text{Campoplex curstians, } M = \text{Mesochorus silvarum.}$ Analyses of the 9 year time series (1971–1979) for all species and (in parentheses) the 19 year time series for N, A and B.

<table>
<thead>
<tr>
<th>Row</th>
<th>Variable</th>
<th>$R^N$</th>
<th>$R^A$</th>
<th>$R^B$</th>
<th>$R^C$</th>
<th>$R^M$</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>N</td>
<td>22* (12*)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>A</td>
<td>54 (21)</td>
<td>47* (11*)</td>
<td>35 (11)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>B</td>
<td>14 (21)</td>
<td>5 (8)</td>
<td>18* (20)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>C</td>
<td></td>
<td>57</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td></td>
<td></td>
<td>22</td>
<td></td>
<td>39</td>
<td>50*</td>
</tr>
<tr>
<td>6</td>
<td>A/N</td>
<td>45 (68*)</td>
<td>49 (62*)</td>
<td>30 (34*)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>B/N</td>
<td>4 (6*)</td>
<td>2 (4*)</td>
<td>13 (32*)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>(A+B)/N</td>
<td>81* (74)</td>
<td>77* (61)</td>
<td>78* (73)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>C/A</td>
<td></td>
<td>1*</td>
<td></td>
<td></td>
<td>0*</td>
</tr>
<tr>
<td>10</td>
<td>M/A</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0*</td>
</tr>
<tr>
<td>11</td>
<td>M/B</td>
<td></td>
<td>52*</td>
<td></td>
<td></td>
<td>80*</td>
</tr>
<tr>
<td>12</td>
<td>M/(A+B)</td>
<td></td>
<td>6*</td>
<td>9</td>
<td>12*</td>
<td>22</td>
</tr>
<tr>
<td>13</td>
<td>Multiple</td>
<td>81 (81)</td>
<td>93 (71)</td>
<td>85 (75)</td>
<td>86</td>
<td>92</td>
</tr>
</tbody>
</table>

direct effect on the dynamics of their hosts (row 9 and 10) but contributed to the multiple regression (omitting these variables reduced the coefficient of multiple determination by 15%).

The R-function for $P. \text{dubius}$ (Table 2 under $R^B$) was again most strongly affected by the total proportion of host larvae parasitized (row 8), suggesting that interspecific competition with $A. \text{tedellae}$ may be quite important. Notice that the hyper-parasitoid, $M. \text{silvarium}$, has an important direct effect on $P. \text{dubius}$ (row 11) and also contributes significantly to multiple determination (row 13).

The R-function for the clepto-parasitoid $C. \text{curstians}$ (Table 2 under $R^C$) was most strongly affected by its own density (row 4), suggesting that interference between searching parasitoids may be important. It is surprising that, as a specialist on $A. \text{tedellla}$, the clepto-parasitoid was so little affected by the abundance of its host, or the parasitoid/host ratio (row 2 and 9). In fact the former relationship was negative and, therefore, deemed implausible for biological reasons. On the other hand, the hyper-parasitoid, $M. \text{silvarium}$, seems to have a detectable effect on the clepto-parasitoid R-function, particularly in the multiple regression, where the inclusion of the proportion of total primary parasitoids attacked by the hyper-parasitoid improved the coefficient of determination by 20%.

Finally, the R-function for the hyper-parasitoid $M. \text{silvarium}$ (Table 2 under $R^M$) was very strongly affected by the proportion of $P. \text{dubius}$ it had parasitized (row 11), suggesting that interference between searching hyper-parasitoids may be important.

Because of these weak bottom-up effects we would not expect strong negative feedback between these secondary species and their hosts, and it seems unlikely that the cyclic dynamics in the system could be generated by their interactions. It seems more plausible that the secondary parasitoids merely follow the cyclic dynamics generated by the interaction between the needle-miner and its primary parasitoids (i.e. they are exogenous cycles).

Discussion

Questions about the causes of regular cycles in animal populations have plagued and confused ecologists for decades. Particularly prevalent has been the search for a universal mechanism, and the endless arguments that this has precipitated. The approach advocated in this paper is relatively objective, in the sense that no a priori assumptions are made concerning the causal process, and plausible explanations are only hypothesized after data analysis. It is very important to realize that the data needed for this kind of analysis require long-term commitments to monitoring biological populations, and that short term approaches, no matter how detailed, cannot expose the dynamic relationships between causal variables.

A crucial part of this approach is the choice of a theoretical model to describe the interaction between dynamic variables. Because of the nature of biological systems we, like others (Royama 1977, Turchin and Taylor 1992), think that the model should be based on the R-function. However, the explicit form of this R-function is open to debate (Berryman 1992). Models can be chosen on the basis of their goodness-of-fit (coefficient of multiple determination), their ability to describe the correct feedback structure, or their appropriateness. For example, the logistic food web model (Berryman et al. 1995a) may be more appropriate for analyzing parasitoid–host interactions because it is explicitly derived from this type of interaction (Thompson 1924, Berryman and Gutierrez 1999). Once a model has been chosen and fit to the data, the relative importance of the independent variables can be assessed by comparing their coefficients of determination, alone and in combination. The detection of strongly symmetrical interac-
tions (feedback) between species supports an endogenous explanation for the cyclical population dynamics, while strongly asymmetrical interactions (little feedback) support an exogenous explanation. As a result of this process, it may be possible to build a plausible interaction structure for the system under investigation, and to make an informed interpretation of the possible causes of the cyclic population dynamics (Berryman 2001). In the case of the spruce needle-miner system, it is difficult to avoid the conclusion that population cycles are the result of interactions between the needle-miner population and its guild of primary parasitoids. As clepto- and hyper-parasitoids seem to have little or no effect on these dynamics, we have to conclude that the cycles observed in these top predators are driven from below (exogenously) by the interaction between the needle-miner and its primary parasitoids. However, although the clepto- and hyper-parasitoids seem to have negligible effect on the dynamics, they may add to the stability of the system, as suggested by Münster-Swendsen (1985).

Our analysis indicates that *A. tedellae* is the dominant, host-specific, parasitoid in this system. This conclusion is in perfect agreement with Cape (1969), who stated that *A. tedellae* was the only absolutely host specific parasitoid, and with Münster-Swendsen (1979, 1982), who demonstrated that *A. tedellae* was the obligate winner in interference competition, and that its searching behavior was better adapted to this host.

Recent studies indicate that the effects of parasitoids on spruce needle-miner dynamics are manifested, not only through direct mortality caused by successful larval parasitism, but also through sterilization of needle-miners following unsuccessful parasitism, or what is called pseudoparasitism (Münster-Swendsen 1994, 2002). Sterilization occurs when parasitoids are disturbed (say by interference competition) before depositing an egg but after sterilizing agents have been injected into the host (Brown and Reed 1997). It is the total combined impact of parasitism on mortality and fecundity that apparently provides the strong negative feedback needed to drive population cycles in all species of this community. This example illustrates one of the advantages of the approach advocated in this paper, for it captures the total impact of a factor or factors on the per capita rate of change (R-function) of the subject population, including unknown effects. In fact, our analysis captured the total effect of parasitoids on the needle-miner before their effect on fecundity (pseudo-parasitism) was discovered. Previous life table analysis had found reduction in fecundity to be the key factor in spruce needle-miner population fluctuations (Münster-Swendsen 1985), while our R-function analysis suggested that parasitoids were responsible. These conflicting results stimulated the search for an explanation, and the eventual discovery of pseudoparasitism in this species (Münster-Swendsen 1994). I should note in closing that it was the success of R-function analysis and the failure of traditional life table analysis that finally convinced Mikael of the utility of my approach.

In general, life tables are not suited to detecting the total effect of environmental factors on individual performance, since they focus attention on individual rather than combined effects, and on mortality rather than fecundity. According to Price (2003), they would better be called “death tables”.

**Note: Mikael Münster-Swendsen and his contributions to Nordic science**

With the untimely death of Associate Professor Mikael Münster-Swendsen on 28 August 2003, at the age of 62 years, Nordic ecology lost one of its most prominent profiles. From 1974 until a malign cancer forced him to retire in 2001, he was employed at the Zoological Institute, University of Copenhagen, where he was much appreciated by his colleagues and the numerous students he supervised. Most of Mikael’s research was dedicated to forest entomology, in particular to studies of the biology of the spruce needle-miner (*Epinotia tedella*) and its complex of natural enemies. His research resulted in more than 50 publications. Mikael held several honorary positions, including service on the board of the Danish Ecological Society (OIKOS) and Danish representative to the board of The Nordic Ecological Society from 1983–1991. He is sorely missed by his friends and colleagues around the world. A summary of Mikael’s life and work, including details of the data used in this paper, can be found at http://www.bi.ku.dk/mmswendsen

**References**


