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9

Population Cycles

Inferences from Experimental, Modeling, and Time Series Approaches

Xavier Lambin, Charles J. Krebs, Robert Moss, and Nigel G. Yoccoz

9.1 Introduction

Some of the most interesting debates in population ecology have taken place within the context of population cycles. Their study has been a fertile ground for the development of ideas on how population models should be formulated and confronted with data. It is the setting in which the use of field experiments became established in ecology (e.g., Krebs and DeLong 1965), and also the context of many methodological and conceptual developments in the fields of population demography (Leslie and Ranson 1940), pest management (Berryman 1982), and community dynamics (Sinclair et al. 2000). Yet, as with many other issues in population dynamics, identifying without ambiguity the causes of population cycles in general, and for any organism in particular, continues to prove an extraordinarily difficult task.

The major purpose of this book is to review recent research developments on the role of food web architecture, and more specifically on the effects of food, predators, and pathogens in population cycles. Its stated aim is to present evidence that population cycles could be caused by food web architecture in some natural systems. Whereas in chapter 1 Alan Berryman promotes a research program centered on the analysis of time series data for formulating, selecting, and even testing hypotheses on population cycles, the case studies encompass a much broader diversity of research approaches. The authors and coworkers of the seven case studies have combined time series analysis, model building, natural history observation, and experiments in different proportions to reach the conclusion that trophic interactions play an important role in generating cyclic dynamics. This diversity of approaches

reflects, in part, a taxonomic divide between vertebrates and invertebrates, experiments being more common with the former, but also profound differences in research traditions. Indeed, the investment required to estimate population size and quantify the causes of mortality of moths and beetles is substantially less than that required for estimating the abundance of voles, hares, and grouse and their predators. From these practical constraints, divergent research traditions have evolved. Despite reaching a broad consensus that trophic interactions are "important" to population cycles of diverse taxonomic groups, the varying strength of the conclusions reached betrays profound disagreements on what inferences ecologists should be willing to make from different strands of data. The statement that trophic interactions are "important" has the benefit of being uncontroversial, but cannot be defined operationally. A key contentious issue, which forms the thread of this chapter, is whether experiments are necessary for separating correlation from causation or whether time series analysis provides an alternative way to eliminate false hypotheses and, given appropriate data, eventually identify the cause of population cycles.

In this chapter we critically review the findings of the empirical chapters, while focusing on the contribution of the research approaches being used. We accept the conclusion that every chapter of this book presents convincing evidence that trophic interactions *could* cause population cycles in the species and populations under study. However, we hold the view that, even in the absence of a well-formulated competing hypothesis, scientific rigor demands that we subject our hypotheses to critical testing, whether through experiment, deductive logic, or other means. Failure to do this has led to the multiplication of poorly scrutinized hypotheses or to the acceptance of untested explanations. Throughout our discussion, we highlight the various stages at which hypotheses erected as explanations for population cycles should be subjected to attempted falsification.

Our discussion is divided into three sections. First, we distinguish between different types of questions in need of answering. Second, we consider the strength and limitations of approaches based on time series, and the contribution of modeling to the formalization and testing of hypotheses on the cause of population cycles. Finally, we review the contribution that experiments have made and should make to subjecting hypotheses dealing with the causes of population cycles to rigorous testing.

9.2 Cyclical Population Dynamics and the Dynamics of Cyclical Populations

The primary question that contributors to this book aim to answer is: What is (are) the specific causal factor(s) responsible for the oscillatory nature of cyclic population fluctuations? Many species have populations with both cyclical and noncyclical populations, thus species-specific features are unlikely to hold the answer. In contrast, features of ecosystems could differentiate

populations with cyclical oscillations from those whose numbers fluctuate, sometimes violently, but without indication of a regular period. Thus trophic interactions form a plausible answer. A second, related question seeks to determine: What processes explain spatial and temporal variation in the dynamics of cyclical populations? Populations of several species display cycles over large geographical areas, but amplitudes and periods often vary markedly over such areas. Whether the process accounting for the oscillatory nature of the dynamics also accounts for the spatial variation in dynamics, or whether two or more processes interact in shaping the dynamics observed in a given region, is an important question.

Successive fluctuations of the same population in the same location may differ in amplitude and in the abundance of natural enemies (figure 9.1). Populations of the autumnal moth and small rodents in Fennoscandia offer striking examples. Oscillations of autumnal moths in Lapland sometimes reach outbreak densities with associated widespread defoliation and tree death at a given site. Subsequent cycles at the same location may be of such low amplitude as to be nearly undetectable (chapter 8). The fluctuations of small rodent populations also vary in amplitude along geographical gradients and over time. Up to five small rodent species in Finnish Lapland experienced high amplitude, largely synchronous cycles with low trough densities from 1950 to 1985. More recently, and at the same sites, *Microtus* voles that were previously abundant have become scarce and *Clethrionomys* voles now experience much lower amplitude, yet still distinctly cyclical oscillations (figure 9.1a).

Oscillations that vary in amplitude or period, but nevertheless remain distinctly cyclic, suggest that factors responsible for variation in amplitude or period are not necessary to explain the cyclical nature of dynamics, even though they influence the dynamics. For instance, changes in the prevalence of parasitism in a cyclic host population regulated by a predator–prey interaction might result in different dynamics than would occur if only the predator–prey interactions shaped its dynamics. This could take the form of steeper, shorter population declines if epidemics occurred following high-density years, even if the parasite–host interaction itself did not cause the cycles. A related view is that specialist mammalian predators might modify the shape of cyclic population trajectories by deepening or prolonging the low phase without causing their cycles (Pearson 1966, MacLean et al. 1974, Fitzgerald 1977), a view consistent with recent experimental data with cyclic small rodents (Korpimäki and Norrdahl 1998). This could occur, for example, if predators were subject to density-dependent mortality that precluded them from showing a numerical response. A further example of such a potentially amplifying process might be found in red grouse populations in Britain. Low amplitude, long (6–11 years) and symmetrical (equal time for up and down) cycles of red grouse have been documented in northeast Scotland where average rainfall is low (e.g., Moss and Watson 2001) and, presumably, parasite transmission is low and variable because free-living stages of the parasite require high humidity to survive (Hudson 1986b, Moss et al. 1993,

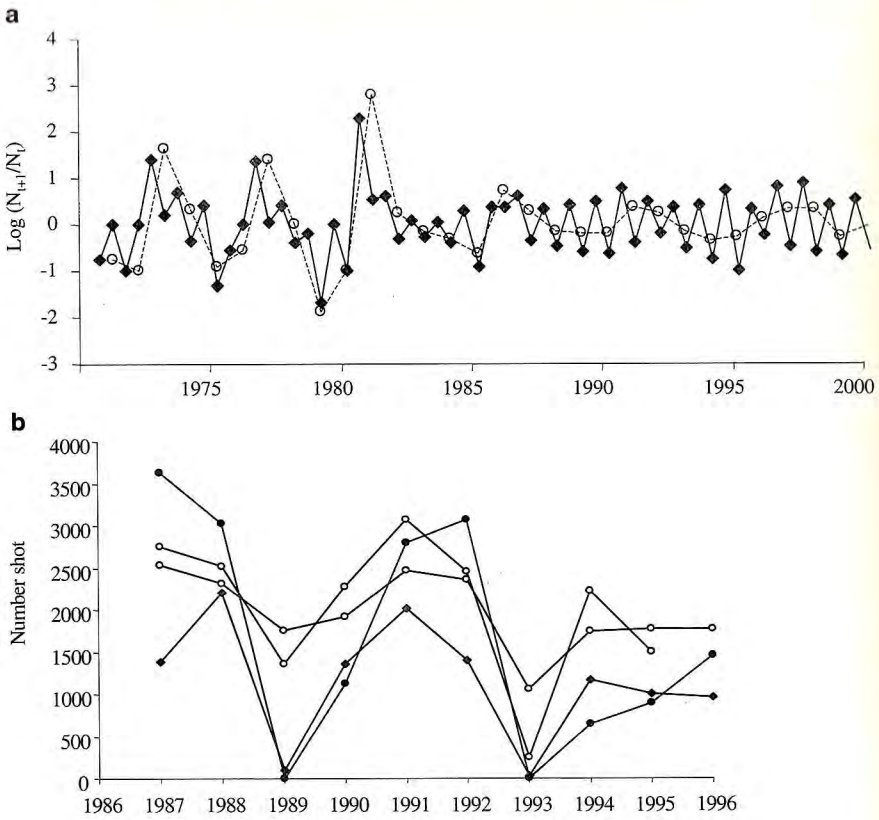

Figure 9.1 Two examples of cyclic populations with changing amplitude, suggesting that separate processes simultaneously determine the amplitude and the cyclical nature of fluctuations. (a) Changes in per-capita growth rates of bank voles (*C. glareolus*) at Pallasjärvi, Finnish Lapland remain distinctly oscillatory despite a marked change in the seasonal dynamics and amplitude of oscillations circa 1985 when, for unknown reasons, *Microtus* voles, the primary prey of least weasels, became scarce (Henttonnen 2000). White circles are spring surveys, black diamonds are late summer surveys. (b) Number of red grouse (*L. lagopus scoticus*) shot on two British moors where parasite burden was reduced experimentally in 1989 and 1993 (white circles) and on two control moors (black circles) (Hudson et al. 1998). Although no attempt was made to shoot birds on the control areas in 1989 and 1993, cycle amplitude was apparently lower on the treated area relative to controls, but trajectories remained cyclical in all areas.

Saunders et al. 1999). Thus, it seems unlikely that parasites could be responsible for the long, low-amplitude cycles in the north (Moss et al. 1993, 1996). Further south, where rainfall is substantially higher, and parasite transmission presumably higher, shorter (3–5 years), high-amplitude cycles are asymmetrical (slow up, fast down) because precipitous declines are associated with

a high prevalence of cecal threadworms (Hudson et al. 1992). There are three possible explanations for this observed pattern. (1) Cycles are caused by a parasite–host interaction but the period and amplitude are affected by variations in rainfall (or some other related variable). (2) Cycles are caused by some other delayed density-dependent process (e.g., kin selection) but the amplitude and period are affected by parasitism in wetter regions and/or times; for example, in areas of low average rainfall, parasite outbreaks would tend to occur when wet summers coincide with high grouse density, resulting in precipitous declines in the grouse population, an increase in the amplitude and, possibly, a shortening of the period of the cycle. (3) Cycles in the north are caused by a different mechanism than in the south, which may be the result of host–parasitoid interactions; for example, different feedback mechanisms dominate the dynamics in the north (chapter 1, Berryman 1993). An experiment capable of differentiating between these hypotheses has yet to be performed.

We see two main reasons for stressing the distinction between factors influencing the dynamics of cyclical populations and those responsible for the cyclical nature of their dynamics. For brevity we focus on amplitude, while noting that some of the same considerations apply to period. First, focusing on the capacity of causal processes to account for observed variations in population growth rates, instead of on those responsible for the cyclical nature of dynamics, rules out a priori a large number of processes that may nevertheless be causally involved. Variations in life history and demographic traits not caused by resources or natural enemies are almost always subtle and hence, by definition, unlikely to be the sole cause of wide amplitude fluctuations such as those observed pre-1982 in bank voles in Lapland (figure 9.1a). Yet, variations in those subtle factors, rather than more potent agents of mortality, may distinguish cyclical populations from those which experience erratic fluctuations (e.g., Oli and Dobson 2001). Second, given geographical variability in cycle amplitude, even between populations of the same species, restricting the scope of specific causal hypotheses to cycles with similar amplitude precludes much scope for refutation. The assumption that cycles with different amplitude result from different causal processes would automatically lead to an accumulation of explanations that could not be refuted by data.

9.3 Testability and Paradigms in the Study of Population Cycles

Two highly contrasting paradigms have been invoked when tackling these problems, and they call for different research agendas. One paradigm is summarized by Berryman (chapter 1). Briefly, Berryman first distinguishes between cycles that result from unidirectional causal processes (*exogenous factors*) and those driven by delayed negative feedback loops (*endogenous factors*). Determining the causes of endogenous cycles involves the principle

of feedback dominance (limiting factors), which implies that only one of many possible delayed feedback loops will dominate the dynamics of a species at one time and place (Berryman 1993). It therefore follows that the research challenge is to distinguish *dominant* feedback loops from those that are *subsidiary*. One way to achieve this using time series is to determine the proportion of the variation in the realized per-capita growth rate of a population that can be statistically explained by a time series of another species. If one feedback loop is broken, another may take effect. Accordingly, blocking the causal feedback may change the nature of the cycle and the causal process but not necessarily stop the cycles. This paradigm calls for time series of abundance of as many species as possible, for these are required to establish the strength of the feedback, as determined by correlation analysis, between the various components of the web. The strongest negative feedback loop is considered to dominate the dynamics and, thereby, is most likely to be responsible for the observed population cycles. Feedback loops that account for a lower proportion of the variance in the realized per-capita growth rate are deemed *subsidiary*. The capacity of models of trophic interaction parameterized from time series to reproduce the observed population trajectory is then compared with the output of a model based on the explanatory variable that showed the strongest correlation with the growth rate of the focal population. In this context, the role of experiments is largely restricted to establishing the existence of feedback loops and testing the predictions of feedback models. According to the feedback dominance paradigm, experiments aimed at stopping the cycle may be doomed to failure because removing a dominant feedback process may lead to a previously subsidiary loop dominating the system.

An alternative paradigm, embraced by many experimentalists, has been to search for conditions that are *necessary* or *sufficient* for a given population or group of populations to show regular cycles. A necessary condition must always be present for an effect to occur, but may not be sufficient to cause the effect. A cause may be sufficient to result in an effect, but if the same effect occurs in its absence, the cause is not necessary (Chitty 1960, Moss and Watson 2001). Accordingly, a process that accounts for a large proportion of the variation in a population's growth rate need not be responsible for the cyclical nature of its dynamics if, in its absence, the population nevertheless experiences cyclical oscillations, albeit with a lower amplitude. Thus, the approach is based on refutation of hypotheses and centers on explaining the cyclical nature of dynamics instead of the overall variance in population growth rates. An assumption underlying the search for causes necessary and sufficient for cyclicity is that several processes may act, additively or not, in shaping observed trajectories. Some processes not responsible for the cyclical nature of a population trajectory may amplify, dampen, or otherwise modify oscillations caused by the necessary and sufficient processes. The challenge is to experimentally identify the subset of processes contributing to the dynamics of a population that are actually responsible for its cyclical nature.

If one subscribes to Berryman's (chapter 1) argument that delayed density dependence is the only necessary condition for generating endogenous cycles, the search is then for the specific process necessary to generate delayed density-dependent feedback in given cyclical populations. Beyond the fact that it is more satisfying to identify an ecological process rather than to detect the signature of delayed density dependence, searching for necessary and sufficient conditions provides an obvious way to generate predictions that can be tested by experiments, observational data, and time series. Assume, for example, a predator–prey cycle characterized by a delayed negative feedback loop. If experimentally preventing the delayed numerical response of a specialized predator succeeded in preventing regular cycles from taking place, then the specific endogenous feedback loop caused by predation would have been shown to be a necessary part of the explanation for the observed cycles. The test would be even more convincing if reinserting the predator's numerical response into the system was followed by a resumption of cycles. Conversely, if removing or preventing the numerical response of a predator did not substantially alter the dynamics of the prey, or if the resulting prey dynamics remained cyclical, although with a reduced amplitude, then predation would be viewed as not necessary for the cyclical dynamics of that population at that time. In the case of the thought experiment above, this outcome would not imply that predation does not contribute to the observed dynamics, but instead would indicate that some other process is responsible for the cyclical nature of the dynamics.

Identifying which process(es) are necessary for cyclical dynamics and associated density dependence need not be restricted to data obtained by manipulative experiments. Indeed, not all variables invoked as part of hypothetical explanations of cycles are amenable to experimentation. This does not preclude the application of a Popperian (refutation) approach. Under some circumstances, observational data may allow for strong inference on the cause of population cycles. If hypotheses are to remain testable in the absence of manipulative experiments, it is essential that their predictions should be spelled out quantitatively, and sufficiently diverse predictions should be generated, such that they can be confronted with empirical evidence. Constructing and parameterizing mathematical models is one way to meet these requirements. Berryman (chapter 1) discusses some of the unresolved controversies on the most appropriate formulations. Note that if specific model predictions are tested, the chosen formulation used to capture the process invoked, as well as the parameter values chosen or estimated, then become part of a specific hypothesis refutable by any inconsistent data. Another requirement for maintaining the testability of hypotheses in the absence of experiments is that their spatial and taxonomic scope should be spelled out explicitly. Many species with cyclic population dynamics have broad geographical ranges and researchers collect data and perform experiments in different locations. There is, of course, no need to presume that the same process is responsible for delayed density dependence in demographic parameters throughout the range of cyclical dynamics, although some prefer this as a starting assumption (Krebs 1996, Moss and Watson 2001). However, unless the spatial scope of a hypothesis is spelled out, it is impossible to test it.

Where biogeographical gradients are present, such as in red grouse, voles, and autumnal moths (Hanski et al. 1991, Hudson 1992, Bjørnstad et al. 1998a), parsimony dictates that the same process should be invoked to account for the cyclical nature of the dynamics in adjacent populations. A second process, or a change in the parameters of the same process, must therefore be invoked to account for the geographical variation in dynamics. For example, red grouse populations experience population cycles of varying amplitude and period ranging from 4 to 11 years in a 300 km region from northern England to northern Scotland (Williams 1985). The important question is whether the observed increase in period is caused by a change in the mechanism responsible for the cycle, or by a change in the modifying factors. Moss et al. (1993) found that worm burdens in the north were too low to generate cyclic dynamics in a parameterized parasite–host model, implying that parasites are a modifying factor of a cycle caused by some other process, or that different processes are responsible for the cycle in the north and south.

When mathematical models are parameterized using data from manipulative experiments, or gleaned from appropriate studies performed in a given geographical area, such as those of chapters 3, 5, and 8, a minimum requirement is that model predictions, and hence the hypothesis as captured by the model, should be falsifiable by any contrary data from the geographical area used for parameterizing the model. For instance, models of the specialist predation hypothesis for small mammal cycles include weasel growth parameters derived from studies conducted in Britain and Fennoscandia (chapter 3, Turchin and Hanski 1997). They must therefore be consistent with all empirical patterns in those two geographical areas. On the other hand, observations such as that of lemming cycles on Wrangel Island in Siberia, in the complete absence of any mustelid predators (Litvin and Ovsyanikov 1990), does not refute the specialist predation hypothesis as spelled out for Fennoscandia. Instead, it demonstrates that predation by mustelids is not a necessary condition for the occurrence of 3–4-year cycles in small rodents, and suggests that there may be many sufficient but no geographically widespread necessary conditions for cycles in small rodents.

Finally, care is needed when assessing the degree of support from nonexperimental data for hypotheses invoking more than one process such as, for instance, specialist and generalist predators in causing and dampening population cycles in Fennoscandia, respectively. In chapter 3, Hanski and Henttonen describe six features of rodent dynamics that may be explained by predation. These include the presence of a geographical gradient in cyclicity, interspecific and spatial synchrony of fluctuations, and the recent reduction in the amplitude of fluctuations. Although the ability to explain so many aspects of the dynamics is impressive, the fact that predation could explain these six aspects of rodent dynamics does not provide support for or against the hypothesis that predation by specialists is the necessary process for causing population cycles per se in Fennoscandia. Indeed, if a gradient in generalist predation can dampen cycles caused by specialist predators, then the same gradient in generalists should be able to dampen cycles caused by other processes. The converse is of course true. The observation that cycles occur in vole populations subjected to generalist predation pressure much higher than that predicted by the model of Turchin and Hanski (1997), in which a gradient in generalist predation added to the interaction between least weasels (specialist) and voles reproduces the Fennoscandian gradient in rodent cyclicity, is a challenge to the generalist component of the hypothesis and not its specialist component (Lambin et al. 2000). Note, however, that the most productive function of a hypothesis formalized in a model is to derive new predictions and not simply account for known observations.

9.4 Time Series Analysis as a Tool for Formulating, Selecting, and Testing Hypotheses

Three case studies in this book (chapters 2, 5, and 7) illustrate well the potential of multispecies time series for developing hypotheses when appropriate data are available (see also Royama 1992, Kendall et al. 1999). When multispecies time series are available, attempts can be made to identify the structure in the food web that could create the observed dynamics, and Berryman (chapter 1) advocates the use of the *R*-function to this effect. For instance, the application of time series analysis, in the manner advocated by Berryman, to Münster-Swendsen's needleminer data was clearly more effective than *k*-factor analysis, and pointed toward the existence of a hitherto unidentified factor associated with parasitism and influencing miner fecundity. The hypothesis generated from this particular time series analysis (TSA) led to new experiments being performed and to the eventual demonstration of the existence of pseudoparasitism. Stenseth et al. (1998) applied empirical, statistical, and mathematical modeling to trapping records from the Hudson's Bay Company for hares and lynxes, as well as to extensive detailed field data, to suggest that phase dependencies in hunting success by lynx through the cycle resulted in nonlinear processes in the predator–prey interaction.

A difficult issue, however, is what inference can be drawn when, as is often the case, sparse and possibly non-independent time series are used for characterizing feedback, estimating parameters, and evaluating the performance of parameterized models. Does obtaining a high correlation between model output and observed changes in abundance (Kendall et al. 1999) indicate that the process most likely to drive the dynamics of the species has been identified? Is it possible to rank the plausibility of competing hypotheses on the basis of their relative ability to reproduce trajectories? Does the failure to achieve a high correspondence between predicted and observed population trajectories amount to a refutation of a hypothesis or the model? Everyone agrees that, when a precise quantitative match between observational data and model prediction is observed, the hypothesis is supported until a confounding variable that would explain this match is identified. However, such a quantitative approach is only rarely possible due to uncertainty in parameter estimation, model structure, and sampling error. Here, we take the view that,

like any descriptive data, time series analysis is intrinsically unable to separate correlation from causation although, when appropriate data are available, it is a powerful tool for describing patterns, generating and refining hypotheses, and making predictions useful in a management context. However, a close correspondence between predictions and observations might be achieved for the wrong reason. Thus, where a quantitatively good fit can be obtained with time series data, the case for performing experiments is reinforced, not weakened. In the absence of experimental tests, inferring the causes of any natural phenomenon from time series analysis alone is an act of faith. Our skepticism about the kind of inference that can be drawn from TSA should not be confused with a negative attitude to the approach. Indeed, several of us have embraced the approach with the above objectives in mind (Steen et al. 1990, Stenseth et al. 1997, 1998, 1999, Lambin et al. 1998, Watson et al. 1998, 2000, Yoccoz et al. 1998).

Below, we elaborate in turn on three main limitations of time series analysis that restrict the strength of inferences that can be made on the basis of TSA, and on our unwillingness to stop short of performing experiments even when TSA-based investigation provides strong support for a hypothesis.

First, long time series are the product of a commitment to long-term monitoring of biological populations as well as the variables that affect them. Long time series are scarce and may lack measurements of things that we now think are part of the relevant ecological architecture. Thus, what we deduce about the structure of time series is based upon what we chose to start to measure many years ago. Hence, time series analysis is backward looking and likely to be confirmatory in nature instead of challenging theory. Most importantly, time series of sufficient length are highly biased in favor of large species and only rarely include macroparasites, even less microparasites. Thus, time series of the prevalence of bacteria and viruses are nearly nonexistent (but see Begon et al. 1999). Robust tests of, for example, the sign of partial derivatives requires long time series from populations with low exogenous variation or sampling noise. The larch budmoth example has by far the lowest exogenous variation of 14 insect time series, and the series is a very long one (Turchin and Taylor 1992). Whereas fitting mathematical models to Nicholson's laboratory blowfly populations time series uncovered the correct answer and illustrated the real potential of the approach (Kendall et al. 1999), the ratio of signal to noise in field populations is likely to be substantially lower. Thus, whereas the well-known classic data sets of population ecology such as Nicholson's blowflies, with both long time series and low exogenous noise, are of some interest, single and even multispecies time series data from most animal populations are lacking.

Second, the process(es) responsible (necessary and sufficient) for the cyclical nature of population dynamics may not be those accounting for most of the variance in population growth. Hence, relying on the capacity of a model parameterized from a time series to reproduce that time series cannot unambiguously identify the cause of cyclicity. Chapter 8 illustrates

this issue well. One interpretation of the time series on autumnal moth is that all populations experience decadal peaks, but at most localities these are so low as to remain unnoticed. Thus, unless data are pooled over wide geographical areas, time series for this species will necessarily be dominated by the occasional large outbreaks. Ruohomäki et al. (2000) considered that climatic factors have the potential to turn low-amplitude oscillations into occasional highly visible outbreaks. Because one model of parasitism with parameters estimated from time series, and further fine-tuned, accounted for 55% of the variance in the same time series and generated cycles of appropriate periods, they favored parasitism as a likely explanation. But if two processes are involved in generating the cyclical dynamics and the occasional outbreaks of autumnal moth, using the capacity of single-factor models to reproduce the observed dynamics seems unproductive and likely to lead to incorrect conclusions.

Third, all time series consist of *estimates* of true abundance. They are often not error-free, not based on statistically robust estimation methods such as capture–recapture, nor a sample of any previously defined target population. Yoccoz et al. (2001) reviewed the consequences of these limitations for the nature of the inferences that can be drawn. Given these sources of bias, relying on quantitative agreement between model simulations and time series to rank and eliminate hypotheses is a risky exercise. The following four issues are of greatest concern.

(1) The longest time series data from vertebrates are derived from harvesting schemes and hence contaminated to various extents by *sampling error* and *bias*. Such biases at low density are common in wildlife harvest data (Royama 1992). For instance, the amplitude of snowshoe hare densities in intensive studies in the Yukon is approximately 14-fold (chapter 4), whereas fur return statistics suggest amplitudes in excess of 1000-fold. Counts of the number of red grouse hens in spring in northern England show amplitudes of 6-fold (Hudson et al. 1992), whereas shooting bag statistics suggest amplitudes in excess of 1000-fold (Hudson et al. 1998). Time series of the abundance of forest insects and their parasitoids are more straightforward to collect, although they also tend to be based on indices of abundance that have rarely been shown to be unbiased estimates of the true population size—hence, these suffer from similar, if less acute, problems as vertebrate harvest indices. For all species, the greatest source of discrepancy between real and estimated fluctuations is the difficulty of sampling species at low density (Hudson et al. 1999).

(2) Time series are typically *not spatially replicated*. Thus measures of uncertainty around any estimates of parameters derived from time series not only include sampling and process error, but also do not consider spatial variability, itself a further source of biases. For instance, Turchin's (chapter 7) "final verdict" that larch budmoth cycles are caused by feedback interactions with both parasitoids and plant quality is based on the analysis of a single, unreplicated time series of parasitoid abundance, and a single, unreplicated set of measurements of larch needle length taken in a different plot some

unspecified distance away from the former sampling area. Without any estimation of spatial variation in the process, reaching such a conclusion is as unwarranted as drawing statistical inference from a nonreplicated experiment. Instead, the analysis is extremely useful in formulating a strong hypothesis, and the testable prediction that neither removing parasitoids nor preventing changes in food abundance should stop the cycle.

(3) The precision with which relevant variables can be measured differs, precluding quantitative comparison of how well models of different processes reproduce time series. For instance, the relationship between an index of a relevant variable for which data are available (e.g., larch needle length) and the dynamically important variable (e.g., larch budmoth larval nutrition) may be less than perfect. Thus, even if the right model was specified and perfectly parameterized, time series models or models of hypotheses would necessarily account for a lower proportion of the variation in the rate of change of the proxy variable than would an equally appropriate model that includes a variable more directly related to the process modeled. In these circumstances, finding a weak correlation in a time series cannot be used to reject a causal hypothesis, nor can finding a weaker relationship for one variable than for another be used to conclude that the process in which it is involved is subsidiary (chapter 1).

(4) The type of dynamic pattern exhibited by a population may change over time or be nonstationary. When long runs of abundance estimates allow for their detection, long-term trends in dynamics are commonplace. They have been observed in snowshoe hares (Sinclair et al. 1993, Ranta et al. 1997), small mammals (Hanski and Henttonen 1996, chapter 3), and red grouse (chapter 6, Moss et al. 1996, Watson et al. 1998). Explanations range from near chaotic dynamics (Hanski et al. 1993), to variations in the strength of entraining exogenous cycles (potentially giving rise to *phase-forgetting* exogenous cycles such as with tree rings and snowshoe hares, Sinclair et al. 1993), changes in exogenous (precipitation) variables influencing dynamic variables (larval fecundity) (chapters 2 and 8), the inherent complexity of multispecies prey–predator assemblages (Hanski and Henttonen 1996), or following invasion by an alien predator (Oksanen and Henttonen 1996). Thus, true parameter values may drift over time, or the process responsible for the dynamics may change. If clear-cut changes in dynamics occur, and if time series data are available under each set of conditions, as is now the case with microtine rodents in Finnish Lapland (Henttonen 2000, chapter 3, and figure 9.1a), TSA may serve to test hypotheses by comparing the structure of time series of *Clethrionomys* voles in the absence of *Microtus* voles and of weasels, whose interaction is said to entrain the cycles of *Clethrionomys* voles.

Whereas statistical developments may help address some of the above issues by explicitly including the sampling process in the estimation of parameters of theoretical models fitted to data (e.g., Bjørnstad et al. 1998b), this is most often overlooked. While there is no doubt that substantial advances have been made in producing models that give good predictions of population trajectories, the limitations of time series analysis indicate the use of a pluralistic approach, which includes descriptive, statistical, theoretical, and experimental procedures.

9.5 Experimental Tests of Underlying Assumptions

The only known way to separate causation from spurious correlation is to resort to experiments. In the present context of population cycles, the most clear-cut experiment that would demonstrate the role of a trophic interaction in causing population cycles in given study populations would be to arrest these oscillations by removing or suitably interfering with some aspects of the interactions. Such an experiment would demonstrate that the presence of predators was *necessary* for population cycles to occur in the experimental setup. However, experimental tests of population-level predictions of hypotheses on population cycles, performed at a meaningful spatial scale (May 1999) and with acceptable levels of replication (Krebs et al. 1993, Hudson et al. 1998), require enormous effort and cost. Even so, if designed to test predictions that exclude other hypotheses, and if accompanied by accurate measurements of demographic parameters (chapter 4), population-level experiments remain the most promising avenue for separating correlation from causation.

Given their cost, it is more efficient if population-level experiments are preceded by strenuous attempts to test the plausibility of hypotheses by testing their logic through analysis of available time series, and modeling and testing their constituent assumptions through smaller scale experiments. Whereas hypotheses may have arisen from time series modeling, several of the hypotheses examined in this book had been formulated long ago, based on natural history observations (chapters 4 and 6). This approach is followed in chapters 3–6, although not all research programs traveled equally far along the road of attempted refutation.

Experiments attempting to refute the basic assumptions of a hypothesis are invariably easier to perform than attempts to modify population trajectories by manipulative experiments. It is therefore likely to be more efficient to do them first. That the empirical refutation of the assumptions of a hypothesis precludes the need to test its predictions has been little appreciated. The consequence of prematurely focusing on testing the population-level predictions of a hypothesis in the absence of support for its assumption is well illustrated by Krebs' (1978) genetic-based version of Chitty's polymorphism hypothesis. Over 20 years, much effort was expanded in testing the prediction of genetic changes in vole populations (Krebs 1970, 1979, Gaines and Krebs 1971) before any support for the assumptions of the heritability of life history and behavioral traits was available. Only 30 years after the hypothesis was formulated was the key experiment performed, and the assumption of a strong heritability of life history traits rejected (Boonstra and Boag 1987). A recent transplant experiment with field voles provided a further refutation of this and other hypotheses assuming that life history traits reflect the past environment experienced by voles, through the influence of maternal devel-

opmental effects or genetic inheritance, and not their present environment (Ergon et al. 2001).

A powerful way to strengthen the plausibility of a given mechanistic model is to provide experimental and statistical support for its constituent assumed relationships. In fact, McCallum (2000) considers that it is difficult to generalize from ad hoc models, which he defines as those not relying on experiments for characterizing functional responses and on statistical analysis of extensive data for parameter estimation. Whereas McCallum (2000) used the model of vole–weasel–generalist predator by Turchin and Hanski (1997) as his prime example of such an ad hoc model, Sundell et al. (2000) recently provided experimentally derived parameter estimates for the functional response of weasels that deviated only slightly from those assumed initially. A further five parameters in this seven-parameter model still require formal estimation. Given the spatial variation in the number and diversity of prey species available to predators, testing the assumption that functional responses are constant would also be valuable. We are not aware of any instance where the measurement of functional responses has been replicated outside laboratory conditions.

Testing whether parameter values assumed when optimizing the fit between simulated data and time series are compatible with empirical data is another way to test mechanistic hypotheses. For instance, Münster-Swendsen (chapter 2) reports that a complex model based on *k*-factors, and assuming that 75% of attacks by parasitoids of spruce needleminer result in pseudoparasitism, accounts for 78% of the variation in the per-capita rates of change of needleminers. Note that two-species logistic modeling captured the effect of parasitoids on the per-capita rate of change of the needleminer without any knowledge of how its birth rate was affected. On that basis, he concluded that "it is difficult to escape from the conclusion that the specific cause of population cycles is its interaction with parasitoids, including the effect of pseudoparasitism on the birth rate." In fact, he formulated a quantitative hypothesis based on a parameter value and an assumed relation of direct proportionality between the prevalence of pseudoparasitism and parasitoid abundance. This quantitative hypothesis should now be tested using ovary dissection in field populations, preferably not in the same population as that used to formulate the hypothesis, so as to provide evidence on the generality of the assumed relationship.

Models that predict population size invariably generate other, intermediate, predictions such as the size of constituent parameters. Testing such predictions with experiments or other data is yet another way to subject a hypothesis to proper empirical scrutiny. One such prediction of standard two-species predator-prey models is that predators should have a delayed density-dependent response to their prey because of the difference in growth rates or developmental delays. This intermediate prediction may not hold in real data if the predator is itself the prey of a higher level predator, or if the predator population is subjected to strong density dependence. For instance, current models of weasel–vole interactions assume that weasel survival is

strictly density-independent (Turchin and Hanski 1997). Weasel territoriality resulting in density-dependent dispersal or predation on weasels by larger, generalist predators are two plausible mechanisms that would explain the absence of any delay in the numerical response of weasels (Graham and Lambin unpublished results). Because a delayed numerical response is required for predator–prey interactions to cause cycles, the failure to observe such a response in a cyclical population amounts to a refutation of the predator–prey hypothesis as a necessary process for cyclicity for a given system, without having to resort to a population-level experiment. The experiment reported in chapter 5 provides support for such a key "intermediate prediction" of the hypothesis that cycles in southern pine beetle (*Dendroctonus frontalis*) are caused by predator–prey interactions. Turchin et al. (1999) estimated the proportion of bark beetle larvae, protected from predators or exposed to natural predation, surviving to become emerging adults over the course of a whole 6-year cycle. Predator-imposed mortality was negligible during the increase phase, grew during the year of peak population, and reached a maximum during the period of population decline. Whereas the delayed pattern was as expected if predation by natural enemies played a driving role in driving *D. frontalis* oscillations, Reeve and Turchin (chapter 5) are well aware that further experiments would be required to rule out other mechanisms that can generate delayed density dependence. Nevertheless, the experiment was a useful step.

Strong exogenous influences may also weaken the underlying negative feedback that would otherwise arise in a trophic interaction. For example, Moss et al. (1993) found that most of the year-to-year variation in red grouse parasite burdens was explained by rainfall with only a weak influence of red grouse density, implying that processes other than parasitism were responsible for the grouse cycle in that study area. Thus, dissecting hypotheses on the cause of cyclicity into components and subjecting each in turn to empirical scrutiny has the potential to exclude false hypotheses without resorting to the more difficult and expensive large-scale experiments. Studies of lynx and coyote numerical and functional responses are exemplary in providing field data on components of predation on cyclic snowshoe hares (O'Donoghue et al. 1997, 1998). An investigation into the causes of cyclicity in a given system is, however, not complete until a hypothesis that survives preliminary tests is subjected to the ultimate test of its population-level predictions.

9.6 Experimental Test of Population-level Predictions

Despite calls for more experiments on cycles having been made repeatedly from many corners (Hanski and Korpimaki 1995, Korpimaki and Krebs 1996, May 1999, chapter 1), remarkably few have been attempted and two of the most significant are reported in this book (chapters 4 and 6, but see also Moss et al. 1996). Both experiments had as the main objective to transform periodic oscillations into nonperiodic erratic fluctuations, and tested the

hypotheses that parasitism and predation were necessary and sufficient processes responsible for population cycles in red grouse and snowshoe hare, respectively. If these experiments were to be judged by their ability to put an end to controversies, they could be viewed as failures. The two experiments differ greatly in the level of replication achieved and in the nature and intensity of the response variable measured, yet each has been informative. Together, they highlight important pitfalls and suggest what experiments are likely to be most fruitful in the future.

The experiment by Hudson et al. (1998) is exceptional in being well replicated, conducted on a large spatial scale, based on a relationship demonstrated experimentally (Hudson et al. 1992), and testing predictions generated, although a posteriori, by a parameterized mechanistic model. The last feature is exceptional for field experiments with cyclic populations, although more readily achieved with laboratory populations (e.g., McCauley et al. 1999). Hudson et al. used anthelmintics to reduce worm burdens in cyclic red grouse managed for shooting. They drugged grouse in the springs of 1989 and 1993, each the first year of two separate declines. On two areas birds were drugged in 1989 and 1993, on two areas birds were drugged in 1989 only, and two areas served as controls (no drug). The response variable, grouse population size, was estimated indirectly as the number of grouse shot. No demographic data such as grouse survival or emigration, or worm burden before or after the experiment, were recorded. The number of birds shot declined 2- to 4-fold on the treated areas relative to peak time (figure 9.1b). No attempt was made to shoot grouse in low-density troughs in five out of the six untreated controls because counts of brood size in mid-July indicated to estate managers that too few grouse were present for shooting to be economical. The problem is that, despite having performed a large experiment, comparatively little energy was expended in accurately measuring the response variables. Had data on changes in worm burdens on control or experimental areas, or data on grouse survival, fecundity, or emigration been recorded, the experiment would have been more decisive (Lambin et al. 1999). As a general rule, and given that levels of replication for large-scale experiments will always be limited, collecting demographic and other ancillary data will always increase the value of experiments. Some measure of the magnitude of any experimentally induced difference between treatments and controls in the prevalence and burden of pathogens, or the abundance of predators, is also important if models are to be used to help interpret ambiguous outcomes of experiments. Although Hudson et al. point out that a decrease in cycle amplitude is the outcome predicted by a model of the parasitism hypotheses, this prediction is not unique to the parasitism hypothesis (see section 9.2). This ambiguity of outcome is likely to arise with any experiment designed to reduce the amplitude of the cycle rather than completely suppressing it. Thus, it is important to determine (with models) the magnitude of the treatment to be imposed so as to generate clear-cut predictions. The use of a parameterized model to interpret the outcome of the experiment raises another general issue. The model used to generate predictions (Hudson et al. 1998) did not include any stochasticity. It predicted that dampening cycles, similar to those observed, will occur following an approximately 20% reduction in worm prevalence. However, it is well established that, in model simulations, dampening cycles may be perpetuated by stochastic noise (e.g., chapters 1 and 2). Given that stochastic noise in parameter values is always present in natural populations, it is unclear whether real cycles should dampen or persist when experimentation with deterministic models predicts a dampening cycle.

The experiment reported by Boutin et al. (chapter 4) on the snowshoe hare cycle in the southwestern Yukon employed a set of manipulations of predator abundance, food supplies, nutrient additions to vegetation, and a combined predator manipulation with food addition. As such, it was a large-scale experiment done with no explicit mathematical model, but based on a factorial approach to experimentation. Predictions from previous studies by Keith and associates (Keith 1983) were stated before the experiments were carried out (Krebs et al. 1993), but a formal model of the system is only now available, after the experiments were completed (King and Schaffer 2001). A strength of the Kluane experiments was the detailed demographic data gathered on all the major vertebrate species in the ecosystem and, as such, it was in some sense the converse of the red grouse experiment. The implicit aim of the manipulations was to stop the 10-year cycle of snowshoe hares. This was not achieved, as reviewed by Boutin et al. in chapter 4, and the key arguments for the conclusion that predation is both necessary and sufficient to cause the hare cycle were based on changes in demographic parameters under the different experimental treatments. Density changes in fact were misleading in many of the treatments, because of local immigration and emigration, a problem even with relatively large-scale manipulations. The Kluane experiments did not conform to the view that a mathematical model is a necessary step before experimental manipulations are designed, and it is not clear what other manipulations would have been suggested by modeling exercises. Indeed, the alternative and more traditional view that motivated the Kluane experience is that we need first to do experiments on vague, verbal hypotheses and then proceed to model the system to search for missing assumptions to test at a later date.

9.7 Conclusions

Whereas it may seem obvious to many that the whole toolkit available to ecologists should be used to tackle the issue of population cycles, field experiments with cyclic populations are difficult because of the spatial and temporal scale over which they take place. Instead of believing that experiments on population cycles will necessarily produce indeterminate results, we favor striving to overcome the problems of implementation, together with the collection and use of time series to generate hypotheses, and model building to explore their plausibility. In fact, we are in complete agreement with Turchin

(1999) when he writes that "progress in ecology results from collecting and analyzing data using innovative techniques, developing insightful mathematical models, performing well-designed experiments, and, particularly, from interaction between the different approaches." We strenuously disagree with the view that experiments are inherently ambiguous and that support for a hypothesis may be so strong that experiments are unnecessary.

Whereas the reasoning underlying the experimental approach may be simplistic, it has the benefit of providing a basis for generating clear, testable predictions for experiments. If otherwise strongly supported hypotheses were consistently not supported by the failure to stop cycles in well-executed experiments, this would possibly lead to the rejection of the experimental paradigm and its replacement by a less simplistic alternative, such as a search for the feedback structure that dominates the observed dynamics advocated by Berryman (chapter 1). In our view, however, to date, too few experimental tests of population-level predictions of simple hypotheses on population cycles have been performed satisfactorily to justify abandoning the experimental paradigm.

Many chapters in this book end with the conclusion that trophic processes are *important* in generating the observed dynamics. Whereas such conclusions have the benefit of being uncontroversial—who would argue to the contrary?—ecology suffers from having no operational way of deciding if a factor is "important." The trophic hypotheses considered in this book have all been shown to be plausible, in many cases the most likely hypotheses for the cyclical nature of the observed dynamics. So far, in all cases, they remain just that: plausible hypotheses. We argued that using an approach based on the search for processes that are necessary for the cyclical nature of dynamics is more rigorous than searching for processes able to account for a high proportion of the variance in change in population size. By demonstrating causal links between processes, experiments offer a firmer basis than correlational evidence on which to build theories. This is because correlations usually have more than one possible explanation. Finally, a major virtue of experiments is that they can provide unexpected results, which can lead to the development of new hypotheses. The real world is much more surprising than models have envisioned, and forces us to consider ideas not previously imagined, indeed, as Mark Twain (1897) wrote, "Truth is stranger than fiction, but it is because Fiction is obliged to stick to possibilities. Truth isn't."

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10

Do Trophic Interactions Cause Population Cycles?

Alan A. Berryman

10.1 Introduction

My motivation in editing this book has been to present as compelling and credible a story as possible. Although I am personally convinced of the soundness of our argument, that food web architecture plays a key role in the cyclic dynamics of many animal populations, I am not sure that others will be so convinced. In this final chapter, therefore, I exercise my prerogative as editor to have the last word, a final attempt to convince the skeptics and to answer the critics.

10.2 The Spruce Needleminer Cycle

Perhaps the most compelling case comes from the Mikael Münster-Swendsen monumental study of a needleminer infesting Danish spruce forests (chapter 2). Mikael is the only person I know of who has, almost single-handedly, and with considerable precision, measured all the variables suspected of affecting the dynamics of a particular population over an extended period of time (19 years) and in several different localities (seven isolated spruce stands). Others have longer time series from more places, but none has been so complete in terms of the number of variables measured. This exhaustive study enabled him to build a model of the complete needleminer life system, and use this model to home in on the factors responsible for the cyclical dynamics. However, the story would not have been complete without multivariate time series analysis, which led to the discovery of parasitoids as the cause