Two Paradigms of Population Regulation

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Abstract

No population increases without limit and ecologists have utilised two paradigms to find out why. The *density-dependent paradigm* assumes that birth, death and movement rates will be related to population density. In many cases they are not, and the search for density dependence has become a holy grail. A better approach is through the *mechanistic paradigm* which searches for relationships between birth, death and movement rates, and the mechanisms controlling populations, such as disease, predation, food shortage and territoriality. Seven suggestions are made for analysing the role of disease in population regulation in mammals. Useful progress will flow more quickly from the mechanistic paradigm without the need to search for density dependence.

Introduction

Population regulation has been a central problem in animal ecology for the last 50 years. Controversy about regulation erupted in the 1950s and 1960s but then quietened down. The ecological establishment for the most part accepted with minor modifications the Nicholson-Lack view of density-dependent regulation. Hassell (1986) and Sinclair (1989) are two typical recent apostles of this dominant view. Most ecologists now seem to assume that the proper approach to population regulation is through density dependence, and the two terms regulation and density dependence are virtual synonyms in much of the current literature.

The purpose of this review is to point out that there are two paradigms of population regulation, and that the consequences of adopting the conventional paradigm of density dependence has not helped us to achieve an understanding of ecological processes. Population regulation plays a central but partly hidden role in ecology, underlying many of the problems of community and ecosystem ecology and the practical problems of conservation biology and global change. Decisions about how to study population regulation have consequences for practical matters.

A paradigm defines a research agenda and, as Kuhn (1962) pointed out, scientific revolutions involve the overthrow of paradigms that have ceased to be useful in explaining problems in a scientific field. The choice of paradigms is not a scientific choice because paradigms cannot be tested by experiments and accepted or rejected. There are no rigid criteria for making these judgments, and they are the net result of many scientists' judgments over many years. A paradigm is a way of looking at the world, and in science it includes implicit instructions about the kinds of observations one should make and the vocabulary one should use. Proponents of competing paradigms are usually at loggerheads because they do not use the same words in the same ways and do not approach problems with the same questions. To a neutral observer trying to choose among paradigms only one judgment is important: which paradigm is more useful in making testable predictions and solving the key problems of the day? Since not everyone will agree what the key problems are in ecology, there is clearly much room for argument about paradigms.

Another method of evaluating a paradigm is to ask how successful it has been in the past at explaining puzzles or understanding a system. Paradigms unlike hypotheses are not true or false.

I will attempt to compare in this paper two paradigms of population regulation and to enumerate their strengths and weaknesses. I will apply these ideas to the specific problem of the role of disease in population regulation in mammals.

The Problem

The central problem of population regulation is that no population goes on increasing without limit. How can we find out what prevents unlimited increase in a population? The broad features of the two paradigms that ecologists have suggested for answering this question are summarised in Table 1. The *density-dependent paradigm* is the classical approach pioneered by L. O. Howard and W. F. Fiske and brought to completion by A. J. Nicholson and H. S. Smith in the 1930s (Krebs 1994, Chapter 16). The *mechanistic paradigm* arose from the work of H. G. Andrewartha and L. C. Birch, D. Chitty, A. Watson and R. Moss and had its early roots in the writings of F. S. Bodenheimer and B. P. Uvarov in the 1920s. How do these two paradigms differ and what agenda do they dictate for studies of the role of diseases in population regulation in mammals?

**Table 1.   Comparative features of the two paradigms of regulation**

	Density-dependent paradigm	Mechanistic paradigm
Stability	equilibrium oriented	non-equilibrial
Mechanisms	not necessary	essential
Approach	observational	experimental
Orientation	backward in time	forward in time
Duration of study	long	short
Utility for modelling	high	low
Management utility	low	high
Generality of theory	high	low
Causes of death	simple, additive	complex, compensatory

Stability

The density-dependent paradigm begins with a central assumption that there is a point equilibrium toward which the population moves if displaced. This oversimplified model can be replaced with more complex models that produce limit cycles or chaotic dynamics, but the assumption remains that there is a set of deterministic relationships (e.g. Fig. 1) between population density and rates of birth, death and movements. What is the evidence that this is a good assumption to make for mammals? In this discussion I will assume that we can measure all these population parameters precisely and accurately, so that deviations from this ideal model cannot be attributed to measurement errors.

There is usually no conceptual problem for any particular organism in measuring birth, death or movement rates, but there is a conceptual problem with measuring population density, recognised long ago by Sang (1950). For mammals density might include breeding adults, non-breeding individuals, young juveniles or neonates in the nest or pouch. Density might be measured at any season of the year, and if you wish to plot birth rate against population density it is not at all clear which of these densities you ought to use.

There are many examples from mammalian studies in which density is clearly related to rates of birth or death, and many examples in which it is not (Fig. 2). When there is a close relationship, the data fit the density-dependent paradigm. But when there is no relationship, how
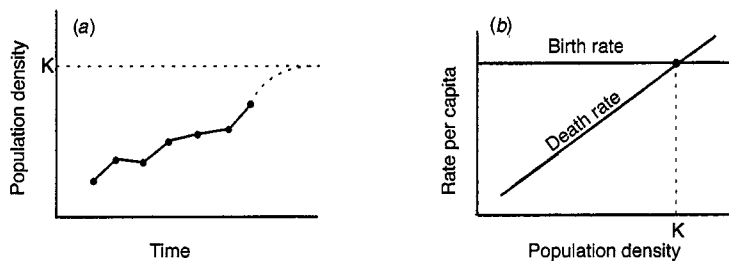
Fig. 1. The population processes postulated in the density-dependent paradigm. *a*, A population increasing toward carrying capacity (K). *b*, If the birth rate per capita is constant and independent of density, the death rate must rise until an equilibrium is reached (birth rate = death rate), at the density defined as the carrying capacity.

should we proceed? Ecologists take two approaches to this problem. First, we invoke the 'measurement error' explanation, and assert that sampling error causes deviation from a perfect relationship (cf. Peters 1991, p. 192). If we do not permit the 'measurement error' explanation, the second approach is to invoke a delayed-density-dependent model (Turchin 1990). In some cases this approach does work but in other cases it does not. Since a density-independent relationship can be postulated for any scatter of points, there is always a way to explain these results within the density-dependent paradigm; however, perhaps one should look at the problem from a different angle.

The equilibrium view (Fig. 1) has always been recognised as the Achilles heel of the density-dependent paradigm (Andrewartha and Birch 1954; Wolda 1989). In much of current ecological theory the equilibrium outlook has been replaced by a non-equilibrial view (DeAngelis and Waterhouse 1987). The important question for a student of population regulation is how can one analyse a population system if non-equilibrial dynamics is the rule? Trying to separate a density-dependent regulatory 'signal' from obscuring environmental 'noise' is one approach (May 1989), but for these systems the mechanistic paradigm offers a simpler viewpoint.

The mechanistic paradigm does not assume that there are stable relationships between population density and birth and death rates (Fig. 1). It avoids these problems by postulating that *density is not the relevant variable to be used in analysing a population system*. This postulate undercuts the central assumption of the density-dependent paradigm and asks the question: what is the relevant variable? To answer this question we need to consider the mechanisms of population regulation.

Mechanisms

One of the most important modern thrusts of ecology is toward an understanding of mechanisms (Tilman 1987). Population density is not a mechanism, and one of the key problems in trying to apply the density-dependent paradigm to field populations is that it is not initially concerned with mechanisms of regulation. Population density is used in effect as a surrogate measure for food supply, territoriality, predation, disease, or other factors in the environment. If the birth rate is a function of food supply and if food supply is only loosely correlated with population density because of time-lags, it will not be surprising if birth rate shows only a density-vague relationship to population density.

The mechanistic paradigm ignores density dependence because it focuses directly on mechanisms of regulation. Plot birth and death rates directly against the factors affecting them, it proposes, and then you will have some understanding of how the system works. In particular, if a disease regulates a mammal population, plot disease mortality against population growth rate and see whether there is a negative relationship. There is little room for argument with this
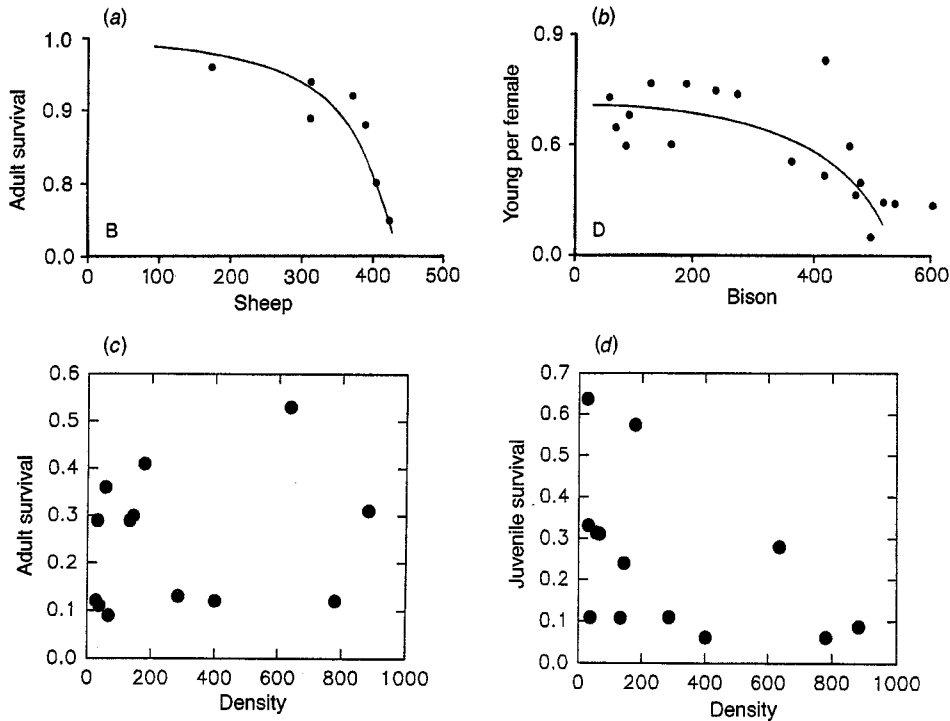
Fig. 2. Density-dependent relationships in some mammal populations. *a*, Survival of adult Soay sheep (*Ovis aries*) (from Fowler 1981). *b*, Birth rate of American bison (*Bison bison*) (from Fowler 1981). *c*, Adult survival in snowshoe hares (*Lepus americanus*). *d*, Juvenile survival in snowshoe hares (from Keith and Windberg 1978).

approach, in contrast to the never-ending arguments over density-dependent relationships in, for example, insect populations (Hassell *et al.* 1989; Wolda and Dennis 1993) and mammal populations (see Boutin 1992 for a critique). Caughley and Gunn (1993) make this same point about the lack of utility of density-dependent approaches.

Approach and Orientation

The density-dependent paradigm approaches the problem of regulation through observation. As May (1989) quotes approvingly from Dempster and Pollard (1986):

'the best hope of unraveling the roles of different factors in the population dynamics of animals, still rests in analyses of long-term, life-table data'.

If this is correct, the density-dependent paradigm is always backward looking, analysing previous year's data and trying to understand changes in populations in the past. There is no logical reason why this needs to be and, as Sinclair (1989) has emphasised, some who support this paradigm also advocate experimental studies.

The mechanistic paradigm by contrast recommends experiments, particularly manipulative experiments, as the *modus operandi*. For this reason it is always forward-looking because hypotheses about regulation are actively tested by experiments. Multiple working hypotheses and strong inference are key concepts in this approach. An example from disease studies would be that if a disease regulates the abundance of a mammal population, removing the disease will produce exponential increase in the host. This experimental outlook is one strong contrast between the practitioners of the two paradigms.

This difference in approach between the two paradigms is reflected further in the recommended duration of study under each paradigm. If regression analyses of the type shown in Fig. 2 and in Turchin (1990) are the rule for the density-dependent paradigm, the message must be to study an unmanipulated system for at least 10 generations and preferably at least 20 generations before you attempt any analysis. By contrast, the mechanistic paradigm permits shorter, specific experimental studies to test specific hypotheses about regulation. Long-term experiments are not precluded in this approach (Krebs 1991) since questions about long-term dynamics require long-term experiments. But we must not glorify long-term studies *per se*. In particular, after 28 years of data on the spruce budworm (*Choristoneura fumiferana*), Turchin (1990) reports from his analysis no evidence of any type of density dependence or delayed density dependence for this system. This type of approach completely failed to reach a conclusion after 28 years, whereas an experiment would have given a clear positive or negative result.

Modelling Population Processes

The utility of density-dependent processes for facilitating population modelling is one of the great strengths of this paradigm. If birth, death and movement rates depend clearly on population density, a mathematical model of the population system can be written simply (Berryman 1991; Grenfell *et al.* 1992). If, however, birth and death rates depend on other factors such as food supplies, predation rates or disease, the population model will rarely be simple and cannot be written without detailed studies of these mechanisms. The net result is that most modellers utilise the density-dependent paradigm in analysing population dynamics.

The key question here is the utility of these models for future understanding. If the models do not capture the mechanisms driving population dynamics, they may fail to be useful for devising management strategies or conservation strategies for rare species (Peters 1991).

Causes of Death

The density-dependent paradigm views mortality as a simple, additive process in which (in principle) a factor can be assigned responsibility for each death in the population. The death rate is an aggregate of these additive effects in this view, and with sufficient fieldwork one should be able to list these causes of death. Compensatory mortality (Krebs 1994, p. 334) causes complications with this view, and factor interaction in mammals makes this approach difficult to apply to many populations (Chitty 1960).

The mechanistic paradigm recognises the complexity of the factors that interact to set birth and death rates in natural populations. It does not seek to 'explain' the birth or death rate but rather asks how these rates change as we manipulate a factor of interest. For example, if helminth parasites are reduced in rabbits, does the birth rate increase?

The orientation of the mechanistic paradigm is toward explanations of particular population systems, and because of this it never achieves the proported generality of regulation theory espoused by the density-dependent paradigm. Limitation is the main conceptual ideal of the mechanistic paradigm and experimentation its main tool.

Criticisms of this Viewpoint

Two major criticisms of my point of view in distinguishing these two paradigms have been raised by ecologists who have refereed this paper. First, I reject the density-dependent paradigm and thus by definition I must be supporting the density-independent paradigm of regulation. This objection illustrates how difficult it is to move from one conceptual paradigm to a new one. The dichotomy between density dependence and density independence is relevant only if you accept the density-dependent paradigm of regulation. I am attempting here to move outside this paradigm and to replace it with a broader, more utilitarian approach to population dynamics. This criticism thus begs the question by assuming the density-dependent paradigm.

A second objection is that the density-dependent paradigm is an attempt to explain the *regulation* of numbers, while the mechanistic paradigm is an attempt to explain the *limitation* of numbers, or what causes year-to-year fluctuations in numbers. But the density-dependent paradigm has always included both regulation and limitation (Sinclair 1989), and I propose that the mechanistic paradigm will also address these two problems but in a quite different way. The mechanistic paradigm asserts that the way to achieve an understanding of what prevents unlimited increase and what causes fluctuations in numbers is to study the mechanisms behind population changes. It thus shortcuts the conventional approach to population dynamics by searching for the mechanisms that prevent population growth on the assumption that only these relationships are valid predictors of how populations will change and why they stop growing. The most successful applications of the density-dependent paradigm are in large mammals in stable environments (Fig. 2a, b; Messier 1991) in which population density is a close surrogate for food supplies. In other large mammals, like kangaroos, that live in less stable environments, the density-dependent paradigm is not useful (Caughley *et al.* 1987, pp. 179–84).

Application to Disease Studies

If you wish to apply these ideas to host-population regulation by parasites or diseases, the key question you must answer depends on which paradigm you support: (*a*) is the mortality or reduced reproduction caused by the parasite or disease density-dependent? or (*b*) does the change in mortality (or reduced reproduction) caused by the parasite or disease change the numbers of the host species?

The first question, which arises from the density dependence paradigm, argues for a large-scale data collection to measure these variables, or alternatively for an experimental reduction or increase in host density. In neither of these approaches is there is any insight provided into mechanisms. Furthermore there is potential confusion in these approaches between transient and permanent effects, because the paradigm is equilibrium-based. The second question by contrast arises from the mechanistic paradigm and suggests a less theory-burdened approach to disease and parasite studies.

There are few data available to evaluate the density-dependent paradigm for diseases and parasites of mammals. Scott and Dobson (1989) list five prerequisites for demonstrating regulation under the density-dependent paradigm.

1.   'As parasite density increases, the ability of the host to survive or reproduce is reduced.' This may occur and is an important question, but is not a necessary part of the evidence that parasites can regulate host abundance.

2.   'As host density increases, the impact of the parasite on host survival and/or fecundity increases.' This is the key question for regulation under the density-dependent paradigm. The increased impact must be measured as a per capita rate.

3.   'The host population must be able to grow' (by immigration or births). I am not clear what this requirement has to do with regulation. It seems to be a condition for existence of any population.

4.   'The experimental design should include either separate uninfected control populations, or should follow individual populations before and after the introduction of the parasite, or before and after elimination of the parasite from the host population.' This is the experimental criterion for population *limitation* by parasites, rather than regulation, and is exactly what the mechanistic paradigm would suggest.

5.   'The parasite and the host population should be able to interact with one another without intervention.' This would seem to be a condition appropriate to any study conducted under either paradigm and does not specifically refer to regulation.

Of these five prerequisites for demonstrating regulation, only one is necessary according to the density-dependent paradigm.

Studies by Scott (1987, 1990) have clearly shown that the addition of a nematode parasite to a laboratory population of house mice reduced mouse density by more than 90%. However, her

data show no evidence of regulation in the sense defined by Sinclair (1989) and Scott and Dobson (1989). No data show that host survival or host reproduction declines with increasing host density as a function of the parasites present. This laboratory mouse system seems to exist in two states: at high mouse density with no parasites and at low mouse density with the nematode parasite. The nematode certainly causes mouse mortality but the regulation paradigm demands evidence that the parasite-induced mortality is dependent on mouse density if parasitism is to be called a regulatory factor. There is in Scott's studies excellent experimental evidence of population limitation, and her results can be accommodated more readily under the mechanistic paradigm. I suspect that the most exciting work being done now on the role of disease in population regulation is being done intuitively under the mechanistic paradigm.

The impact of helminth parasites on their mammalian hosts clearly increases with parasite density, although these effects on survival and reproduction may be non-linear (Singleton and Spratt 1986; Scott and Lewis 1987; Gregory 1991). These parasite-density-related effects should not be confused with the density-dependent effects required for population regulation under the density-dependent paradigm. There are two issues. (1) Is the probability of survival (or reproduction) of the host related to parasite load? This is an important question but it has no direct relevance to density-dependent regulation. (2) Is the probability of survival (or reproduction) of the host, as affected by the parasite, related to density of the host population? This question is relevant to the density-dependent paradigm, and is, I suggest, a much less interesting question than the first one. There is enormous confusion stemming from the term density dependent; many physiological and behavioural processes are related to density but these should not be labelled density dependent because of all the theoretical baggage associated with this paradigm.

Botflies in Townsend's Vole

Many species of rodents in North America are parasitised by larvae of cuterebrid botflies. We have described the effect of botflies (*Cuterebra grisea*) on the vole *Microtus townsendii* near Vancouver, Canada, from 1971 to 1978 (Boonstra *et al.* 1980). Botflies reduced the survival rate of all sex and age classes of voles from August to October, and they reduced the reproductive rate of both males and females over this time period. Growth rates in body size were also depressed by botfly infestations.

We can use this example to illustrate the differences in approach of the two paradigms of regulation. For the density-dependent paradigm the major question is whether the impact of botflies on survival and reproduction increases with vole density. For survival the impact of botflies was density dependent in male voles ($r = 0.62$, $n = 21$) but not in female voles ($r = 0.19$) (Boonstra *et al.* 1980) (Fig. 3). For both sexes there was so much variation with population density that we decided to abandon this conventional approach. We could detect no density dependence in the reproductive effects of botflies.

There is little insight provided into the dynamics of this host–parasite system by analysing for density dependence. By contrast if we adopt the mechanistic paradigm, we first proceed to show the impacts of botflies on survival, reproduction and movements (as in Boonstra *et al.* 1980). We then proceed to a key experiment: what is the impact on the population of eliminating botfly parasitism in these voles? This experiment has now been done on *M. townsendii* by Lambin (unpublished data) but the results are not yet analysed. We have speculated that one possible explanation for the fact that Townsend's vole does not always have 3–4-year cycles is that botflies cause mortality that disrupts social organisation, and the social processes that control population cycles cannot operate (Krebs 1985). It is also possible that botflies selectively eliminate the socially dominant voles. These ideas about the *mechanisms* of botfly impact are all speculative, but illustrate how the experimental paradigm assists in dissecting a problem in population regulation. The mechanisms involved in population regulation in *M. townsendii*, and how botfly parasitism may operate to influence the mechanisms by which regulation is achieved, have been discussed by Taitt and Krebs (1985) and Lambin and Krebs (1991).

Botflies in Townsend voles
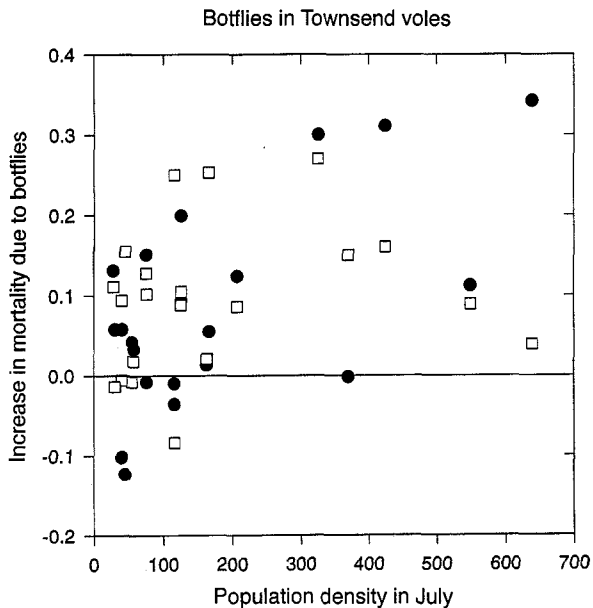
Fig. 3. Density-dependent effects of botfly parasitism on survival rates of *M. townsendii* near Vancouver, Canada. ●, males; □, females. The impact of botflies on survival is measured as the difference in survival rates per 2 weeks for infested voles and non-infested voles. Spacing behaviour plays a central role in population regulation for this species, and the impact of botfly parasitism on social organisation may be an important link in causing most populations of this vole to fluctuate annually instead of in 3–4-year cycles. (Data from Boonstra *et al.* 1980)

The observation that parasite prevalence shows no positive correlation with host density is fatal to the density-dependent paradigm (e.g. Singleton *et al.* 1993) but is not relevant to the mechanistic paradigm. It is quite possible that population regulation by parasites is achieved without density dependence, and the critical experiment is to reduce or increase the parasite experimentally in field populations. Until the detailed mechanisms of regulation are studied experimentally, the question of the role of parasites in regulation must remain unanswered.

Conclusions

I advocate abandoning the density-dependent paradigm of population regulation not because it is wrong but because it is not useful in achieving an understanding of population dynamics in the field. One might argue persuasively for this paradigm from a modelling perspective because it simplifies population analysis, and one might find it a useful paradigm in the laboratory. However, in the field it has proven to be a monumental obstacle to progress as ecologists have looked at populations for decades trying to find density-dependent relationships. Students of population dynamics of small rodents abandoned this paradigm over 30 years ago (Chitty 1960) and recently even some fishery ecologists have expressed doubt about this approach (Shepherd and Cushing, 1990). There is the beginning of a revolt among some insect ecologists against the density-dependent paradigm (Murdoch *et al.* 1985), and in theoretical ecology some glimmerings of rejection of the contemporary wisdom (Schaffer and Kot 1986).

If the impact of parasites and diseases on population dynamics is to become an important focus for students of population dynamics, more progress will be made more quickly by the use of the mechanistic paradigm in which population regulation is achieved as a by-product of the mechanisms causing birth and death rates to change. Density dependence can rarely be studied directly in mammal populations and, even when it can, more effective understanding could be achieved with the mechanistic approach.

For some practical advice on analysing the role of disease in population regulation, I offer the following seven principles.

1.   Ask a precise question and frame a precise answer (hypothesis) and at least one alternative.

2.   Seek mechanisms by which population effects are achieved. Density is not a mechanism.

3.   If parasites or diseases are to affect population dynamics, they must affect birth, death or movement rates. Do not assume the converse. The fact that a parasite reduces the reproductive rate of the host does *not* mean that it must reduce population size.

4.   Do experiments, either comparative or manipulative.

5.   Avoid mathematical models. They are more seductive than useful at this stage of the subject (Krebs 1988).

6.   If you are addicted to models, at least do not believe them until all the assumptions can be tested (Bradley 1982) and their predictions verified. There is no such thing in population dynamics as a 'reasonable assumption' without data.

7.   Be optimistic yet humble. These problems are enormously difficult and will not be solved in a few years. Lay a good foundation for your intellectual grandchildren.

By addressing questions about mechanisms in these ways we can begin to understand the complex processes that produce the simple result described by Malthus and Darwin that no population grows without limit.

Acknowledgments

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