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Beyond population regulation and limitation

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Abstract.   The study of population dynamics addresses three questions that are not always separated in discussions with empirical data. Two questions address population regulation. *What stabilises population density* is the first question, and, in spite of much theory, little progress has been made in answering this question empirically. The assumption of an equilibrium density is impossible to test and direct experimental tests to answer this question are rare. *What prevents population growth* is a second question, and is the classic question of population regulation. To answer this question requires an increasing population, and, with adequate experimental manipulations, the density-dependent factors preventing increase can be identified. Surprisingly, answering this question has provided little assistance in solving practical problems in population dynamics, possibly because most populations are rarely in the state of growth and show a limited range of densities. *What limits population density in good and poor habitats* is a third question, which addresses population limitation rather than regulation, and has been the most useful question for empirical ecologists to ask. Population limitation admits of little theory and no elegant models, and highlights the gap between theory and practice in much of ecology. Defining the question clearly and adopting an experimental approach with clear alternative hypotheses will be essential to avoiding the controversies of the past while building useful generalisations for the practical problems of population management.

Introduction

The problems of population dynamics contain, without question, the most controversial and long-standing issues in population ecology. The battles between the density-dependent school and the density-independent school began in the 1920s, peaked in the 1950s and early 1960s, and arose again in the 1990s. Long-standing controversies in science are typically caused by scientists who operate under different paradigms (Kuhn 1970), and thus normal types of empirical tests cannot resolve the controversies. Since many of these controversies have been over words that have been used in different ways, I begin by defining terms following Sinclair (1989).

Population regulation: the process by which a population returns to its equilibrium density.

Population limitation: the processes that set the equilibrium density.

Population dynamics: the analysis of the causes for change in population density, including both regulation and limitation.

Given that we already have extensive reviews of population regulation and limitation (Sinclair 1989; Krebs 1995, Turchin 1999; Murray 1999; White 2001), the first question that I must answer is what new could possibly be said on this issue. Why should anyone keep reading this paper? The answer is that in this review I will adopt an *empirical* view of the problems of population dynamics and ask three questions:

(1) What are the questions we wish to answer about population limitation and regulation?

(2) What data are relevant to answering these questions?

(3) How successful have ecologists been in achieving these goals of understanding population changes?

I adopt this empirical approach because the theory of population regulation and limitation has been elegantly presented in many papers beginning with Nicholson (1933) and formalised more clearly by Enright (1976), May (1981, 1989), Murdoch (1994), Sinclair (1989) and Sinclair and Pech (1996), among others. The problem does not rest in the theory but in the application to real populations, and that will be my point of departure. What is striking about most theoretical discussions of population limitation and regulation is that they present no data, only idealised graphs (e.g. Berryman *et al.* 1987; Sinclair and Pech 1996). This is the first clue that there is a gaping chasm between the theory and the real world. We clearly need to close this gap by bringing theory and data together, as was achieved by Caughley *et al.* (1987) in their study of kangaroo populations. I will discuss here the issues of population dynamics within a single-species population. Some of these same issues arise in community ecology in comparisons between species.

What are the questions we wish to answer?

There are three questions about a population that are often grouped under the aegis of population dynamics.

(1) *What factors stabilise population density?*   If populations are regulated toward an equilibrium density, this can be an interesting practical question. For example, in the management of fish populations a stable population can provide a more stable economic harvest.

(2) *What factors prevent population growth?*   This is the classical statement of the question of population regulation addressed first by Howard and Fiske (1911) and later by Nicholson (1933). It has been the central issue of population ecology for more than half a century.

(3) *What factors cause high population density in some habitats and low population density in other habitats?*   This is the problem of population limitation, which is sometimes confused with the first two questions.

   If the world were simple, all these three questions would have the same answer. For example, one might argue that predators keep deer populations relatively stable, prevent them from growing exponentially, and, when differing in abundance, cause differences in average deer density between habitats. But there is no reason to think that answers will always be this simple, and we should begin by considering these three questions as independent issues. I have been unable to find any empirical study of population dynamics that is not focused on one or more of these questions. Each of these questions may be broken down into a variety of more detailed questions for particular populations.

   My first recommendation is that if you are studying population dynamics, you should state clearly which of these three questions you are attempting to answer.

What data are relevant to answering these questions?

Before we can decide what data are needed to answer one of these questions, we will have to agree on some empirical rules of procedure. First, we need to adopt an experimental approach before we can proceed. This experimental approach can be either a comparative or a manipulative approach, following standard scientific procedures (Hurlbert 1984; Damon and Harvey 1987; Underwood 1997). The chief argument at this stage involves whether a comparative approach involving time-series analysis can be used to answer these questions without the need for experimental studies of demographic variables. I will assume, along with Berryman and Turchin (2001), that, while time-series analysis can suggest hypotheses about regulation, experimental studies are essential to test them. It is important to separate the origin of hypotheses from their testing, and there are many routes to finding successful hypotheses in science. I am not primarily concerned here with the origin of hypotheses, but rather with their subsequent testing in field populations.

What factors stabilise population density?

The theoretical answer to this question is quite simple: density-dependent processes (or negative feedback) tend to stabilise population size, and if there are time delays of varying magnitudes, populations are destabilised (Berryman *et al.* 1987). Stability is defined here as the absence of population fluctuations. Note that this question does not assume that there is some stable or equilibrium population density that is equivalent to the set point on machines that have negative feedback designs, only that some processes tend to stabilise population size. This question dissolves into the problem of determining how processes affect population growth rates as density changes. If there is no clear equilibrium density for a population, or if the population changes in size continually, this question can lead you into a quagmire of circular arguments (Wolda 1989). To avoid circularity you must adopt an experimental approach, as follows.

   Given a series of replicated controls (unmanipulated) and manipulated populations, you can determine from sequential measurements if the manipulated populations are less stable than the controls. Stability is measured by the coefficient of variation of population density over time. Manipulations may involve processes like births, deaths, or dispersal, or changes in factors like food supplies, predation, diseases, parasites, and physical factors like temperature, or changes in social factors like relatedness, and genetic factors like gene frequencies, or landscape factors like habitat patchiness.

   There would seem to be no difficulty to carrying out these kinds of experiments, but, in fact, few such studies have been done on field populations to answer this question directly. Instead, many ecologists have relied on modelling studies with assumed density-dependent processes to determine whether or not the suggested processes are strong enough to affect population stability (Anderson and May 1978; Abrams and Roth 1994; Holyoak and Sachdev 1998). The theoretical analyses have proceeded far ahead of empirical studies, and with too little connection between the two.

   The question of stability can be turned upside down to ask what processes destabilise population density. Again the theoretical answer is clear: delays in density dependence are destabilising (Berryman 1981). The search for delayed density-dependence has itself been a focus of concentration in the theoretical literature (Turchin 1990; Holyoak 1994; Berryman and Turchin 2001). One good example of the application of these ideas is the explanation for a gradient in vole cycles from southern to northern Fennoscandia. According to Hanski *et al.* (1991), specialist predators in northern Fennoscandia destabilise vole populations and cause cycles, while generalist predators in southern areas stabilise vole populations. Time-series analyses of population trends can provide a useful description of the relative strength of density-dependent effects and delayed density-dependent effects (Stenseth 1999).

The key question that has arisen out of these analyses of population stability is how these explanations of stability and instability can be tested empirically. Time-series analysis and population models involving delays in density dependence can supply patterns that mimic changes in natural populations but how can we proceed from patterns to processes? This answer is far from clear. Detailed models like those of Hanski and Korpimäki (1995) and King and Schaffer (2001) specify parameters that can be measured empirically. Time-series models do not specify empirical parameters (Stenseth *et al.* 1998), and must be tested more directly by experiments.

A related question regarding the stability of population density is how rapidly a population that is perturbed from its current density will return to its former level. This measure of stability is sometimes referred to as resilience (Lewontin 1969; Ives 1995). Ives (1995) has pointed out the difficulty of determining resilience for stochastic systems that have no readily determined equilibrium density. The experimental perturbation of population density and the measurement of its return trajectory has been used as a critical test for density dependence (Karels and Boonstra 2000).

A good example of the empirical study of population stability for agricultural systems can be cited. Calderini and Slafer (1998) showed that wheat yields from 21 countries around the globe have increased dramatically during the last century, and this increase in yield has not been associated with any reduction in yield stability. In general, there has been a tendency for wheat yields to be slightly more stable in recent years, even at high yields, a desirable attribute for human agroecosystems.

Population stability has become increasingly subsumed under the more general problem of community stability, and the general question of whether community complexity leads to community stability (May 1973; Haydon 1994). Population stability is affected by food web structure and the resulting community interactions. Whether stability can be understood more readily within a community context or a population context is not yet clear (Hone 1999). Of particular importance is the stability of ecological communities when some component species reach very low densities (Murdoch *et al.* 1995).

For many populations for which stability cannot be experimentally analysed, we can only infer the possible agents contributing to stability from modelling or time-series data. We can also compare different populations of the same species and ask why one population is more variable than another (Connell and Sousa 1983). Such studies, while valuable, suffer from all the problems of inferring causes from correlations. In general, there has been little practical progress in understanding real populations by asking what stabilises population density, and this question by itself is the least interesting of the three questions typically subsumed under the aegis of population dynamics. The analysis of population stability *per se* has been, for the most part, a black hole of ecological effort with little payoff for the solution of practical problems.

What factors prevent population growth?

The factors preventing a population from growing would appear to be the most significant fact to know about any population, and, as such, ought to be the most important of the three problems of population dynamics. For pest populations we wish to know what factors we can manipulate to stop population growth, and for endangered species we would like to know what factors might increase population growth. How can we obtain this information most readily?

The theoretical answer to this question is relatively simple: density-dependent factors or processes that involve negative feedback are both necessary and sufficient to stop populations from increasing (Berryman 1981). Nevertheless, this simple conclusion has been the root of many of the vituperative controversies over population dynamics, and we need to understand why this has happened. There are at least six sources of problems with this simple theory that prevent it from being very useful in managing real-world populations:

- Problem 1: An observed population is stable or declining. We cannot infer the factors that prevent population growth in populations that are not observed to be growing. We require for a scientific assessment a set of control populations that are stable or declining and a set of populations that are increasing. Alternatively, we could manipulate a set of replicate populations to cause them to increase (for example, by adding food supplies or reducing predation).
- Problem 2: No process or factor that produces negative feedback can be found despite extensive study. The search for density-dependent factors or processes is sometimes successful (Dennis and Taper 1994) and sometimes not (Turchin 1990). The critical question is, what do we infer from the failures to verify theory?
- Problem 3: Feedback processes are only vaguely related to population density. Vague density dependence (Strong 1986) is common in field studies, and we need to determine whether the scatter is due to measurement error or process error. There is an implicit assumption in many cases that the entire scatter is due to measurement error.
- Problem 4: We have no way of knowing how general any observed negative feedback relationships are. If we observe a negative relationship between fecundity and population density, will this same relationship apply in another geographic area at another time?
- Problem 5: Multiple factors interact to produce negative feedback. Joint action of factors is rarely considered in testing for regulation (Lidicker 2000) and there is often an implicit assumption that all the other factors

impacting on a particular population will remain constant over all densities.

- Problem 6: Populations are rarely closed, and open populations provide the possibility that changes in densities are driven by immigration and emigration as well as by births and deaths. Metapopulations are one example of this complication (Hanski 1998).

The result has been that extensive attempts have been made to search for density-dependent relationships in field populations with only modest success and only limited understanding of the resulting population dynamics (Sale and Tolimieri 2000).

Fig. 1 illustrates a purely empirical approach to answering the question of what stops population growth. The first step should be to determine which of the primary demographic components change as the population increases. The second step is to isolate the potential regulating (density-dependent) factors, and to study them experimentally either by comparative methods or by manipulative experiments. The emphasis in this approach is on measuring and understanding how density is affected by these regulating factors, as well as by those factors that are not density dependent.

Two examples will illustrate some of these problems. Fig. 2 shows the expected relationship between rate of population increase (r) and population density from the standard model of population regulation (e.g. Krebs 2001, p. 281), and data on the house sparrows living on four islands off northern Norway. It is clear in the four years of study that the observed data cannot be fitted to the equilibrium, density-dependent paradigm. The reason for this can be seen most clearly by looking at Fig. 3, which illustrates the same plot for song sparrows on Mandarte Island, British Columbia. Song sparrows are a classic example of a regulated population that shows density-dependent declines in reproductive output and juvenile survival (Arcese *et al.*

1992). Because the song sparrow ranges over a greater span of densities and shows several episodes of population growth and decline, it is possible to see some evidence of density-dependent decline of r values at high densities.

A second set of examples of the failure of the general model for population regulation is shown in the analysis by Sinclair *et al.* (1998) of the impacts of predation on endangered species in Australia. In spite of the elegant theory of density-dependent predation rates, not a single example of empirical data in that paper shows any signs of density dependence, i.e. of rates of population increase falling as density rises. Populations of endangered species like the rock-wallaby *Petrogale lateralis* may decline at low density or at high density, and population density alone tells us nothing about their predicted dynamics. Predation is inferred to be severe enough at all levels of density to cause population declines (Kinnear *et al.* 1998).

The question of what stops population growth has been addressed extensively in populations that cycle in abundance because the increase phase of the cycle is a repeated sequence of unlimited growth that can be contrasted with the other phases of density change (Chitty 1960). Populations that do not cycle in abundance and do not go through large density changes are more difficult to analyse. Since a majority of populations are non-cyclic, trying to answer this question for them has proven frustrating. Analysing density dependence in populations that show a limited range of densities (Fig. 2) is largely futile, and asking this second question for these populations is not profitable.

There is an unstated assumption that if we understand the regulating factors that stop population growth we will also know how to manage the population if it is not growing but is declining in density. This is an equilibrium fallacy that is particularly dangerous in the assessment of conservation problems, as Sale and Tolimieri (2000) have pointed out.
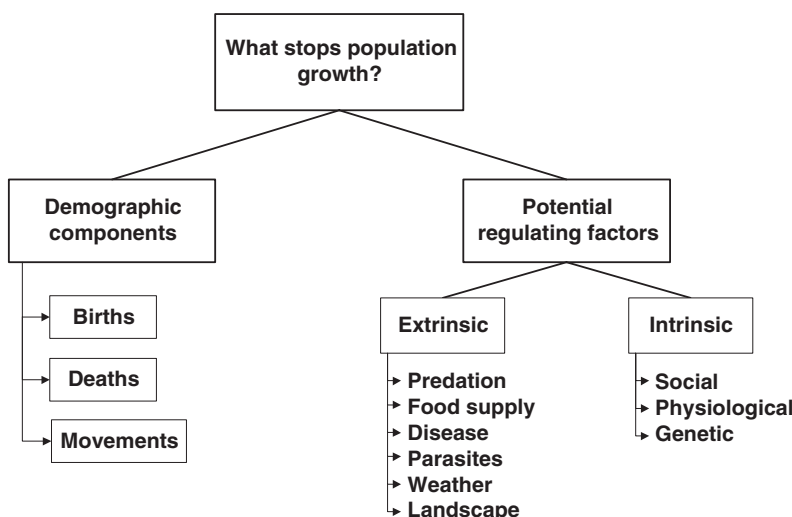

Fig. 1. Decomposing the elements for the analysis of what stops population growth. Note that the potential regulating factors will include some factors that will later be determined to be density dependent, and others that will be determined to be density independent.
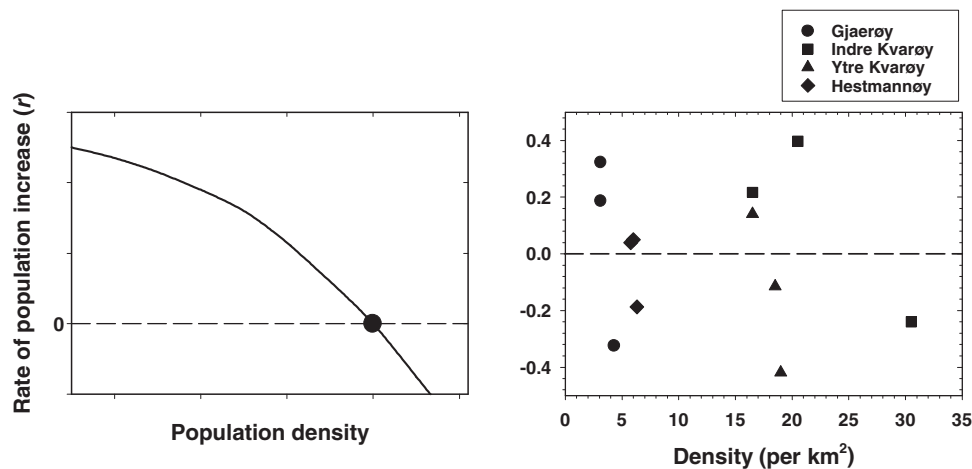
Fig. 2. Theoretical plot of density-dependent rate of population growth with an equilibrium point when $r = 0$, contrasted with data from four populations of the house sparrow (*Passer domesticus*) on islands off north Norway during 1993–96 (Saether *et al.* 1999). There is no trace of the expected curve in the empirical data, and the question is, how do we interpret these kinds of inconsistencies?

There has been a great deal of energy invested in trying to answer this second question about population regulation without much light being shed on important empirical problems of population dynamics. Although the question of what stops population growth is interesting and challenging, there are few populations whose management has been improved by studies that concentrate on trying to identify the density-dependent and delayed density-dependent factors that stop growth.

What factors limit population density?

What limits population density has always been considered as separate from the problem of population regulation but is sometimes confused with it (Sinclair 1989). This question deserves more attention because it is the central question of

Fig. 3. Rate of population increase in the song sparrows of Mandarte Island, British Columbia, in relation to population density, 1975–2000. (Data courtesy of P. Arcese and J. N. M. Smith.)

population dynamics and has a greater empirical importance and a greater management importance than either of the previous two questions dealing with population regulation. Few populations show periods of unlimited increase, and thus are not suitable for finding out what prevents population growth. But they are all suitable for determining the factors that limit density in time and space.

The problem of population limitation has been neglected in ecology because it is theoretically very dull. Enright (1976) showed that any factor, density dependent or density independent, could affect average density. This neglect is well illustrated by Sinclair (1989), who suggested that everything that caused mortality limited population density, a theoretically uninteresting proposition, but one that is very important in applied ecology.

The question of population limitation is most readily illustrated by comparisons in space. Good habitats, which support high population densities, can be contrasted with poor habitats, which support low densities. Any of the factors listed in Fig. 1 may be the cause of these habitat differences, and from a management perspective the key factor can be utilised for management manipulations. It is more difficult to address the question of population limitation in a time perspective. If a population is lower in one year than it was the year before, we can ask what processes caused this demographic collapse. There is no interest in considering whether the relevant process is density-dependent or not – the key is to understand the mechanism of change in birth, death, or movement rates.

The question of equilibrium density, or 'carrying capacity', is central to this problem. As Wolda (1989) pointed out, it is impossible to separate a fluctuating population size from a fluctuating carrying capacity by
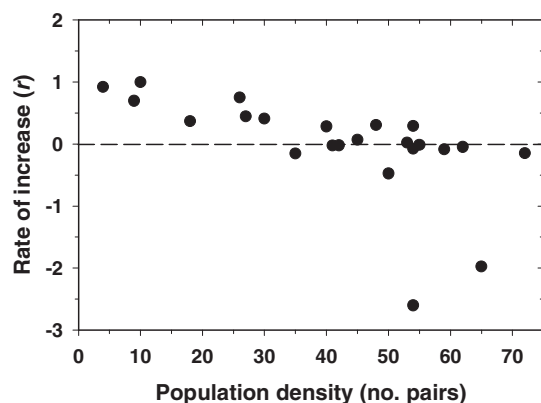
simple observations of population numbers. To avoid this issue, we answer this question by comparing populations both in time and in space. If we can identify a high incidence of disease or parasitism with low population density, we can test the hypothesis that this disease or parasite is the cause of low density by a removal experiment or by comparing disease incidence in low- and high-density populations. The key here is on experimental manipulations.

Australian studies on the European rabbit (*Oryctolagus cuniculus*) provide an excellent illustration of the ideas of population limitation (Newsome *et al.* 1989; Pech *et al.* 1992; Banks 2000). Removing foxes from an area allowed rabbits to increase dramatically in several experiments, showing that fox predation limited rabbit numbers. Changes in rabbit numbers are driven by changes in food supplies, primarily caused by rainfall (Pech and Hood 1998).

The question of population limitation has been exceedingly useful in the management of both desirable and undesirable species, and is the major tool of wildlife and fisheries management. Biological control is the application of the principles of population limitation to the control of invertebrate and vertebrate pests. The declining population paradigm of conservation biology (Caughley and Gunn 1996) is the embodiment of these ideas for the conservation of endangered species. The secret of success in investigating population limitation is that you do not need to determine whether a particular factor is density-dependent or not. Some of the most spectacular successes of biological control, for example, apparently involve no density-dependent processes, a fact that has greatly puzzled theorists in this area (Murdoch *et al.* 1995). Density-dependence is a red herring in questions of population limitation.

The analysis of population limitation can be criticised as a simple exercise in correlation. Is the density of rabbits correlated with the amount of food present in their habitat? Clearly, this question can be answered with a manipulative experiment, as well as addressed with descriptive studies and correlations. As with all questions in population dynamics, if we are to use them as predictions for management, we need to find out how general these kinds of results are.

The major difficulty with this approach to problems of population dynamics is that it permits of no general theory at the present time. This is indeed a defect because ecologists, as all scientists, wish to achieve general theory. The problem is what to do in the absence of general theory. In a historical sense, ecologists have developed elegant theory divorced from the real world, and while this is not a problem if there is continual feedback between theory and reality, in ecology the feedback loop from much ecological theory to the real world does not exist. Theory has become a dead end for which there is little empirical interest. Ecological problems are being solved largely by trial and error empiricism.

A second difficulty with this approach is that it is not conducive to long-term modelling of population dynamics.

Modellers are overly concerned with density dependence because of the need to prevent runaway population growth. But it would be more useful to develop models for population limitation that are short-term process models and to forget the long-term models that require some form of negative feedback to remain within bounds. This raises the larger issue of what types of models are most useful in understanding population dynamics, a critical question that is too little debated in ecology.

Discussion

The controversies over population dynamics are partly a controversy over words but also a controversy over how one should go about analysing population processes. There are approximately nine distinct potential regulating factors (Fig. 1) and if one includes all their potential interactions as well as multiple-factor hypotheses, there are well over 1000 possible scenarios of regulation. If one is a manager interested in manipulating the density of a species, there is too much potential choice about what to do. To counter this problem, ecologists have often argued that there is typically only one major factor regulating or limiting population density, and our job as ecologists is to finger the correct factor. Thus, one might postulate that caribou populations are regulated by wolf predation, and to solve caribou population problems one need only reduce or eliminate the wolves (Seip 1992; Bergerud and Elliot 1998).

The key to a useful theory of population limitation and regulation is to have a detailed understanding of the mechanisms affecting a population. We have made considerable progress in this direction for specific groups of organisms, and we can speed up progress by asking the right question about general issues. I will illustrate three questions here that can be addressed directly:

Do populations routinely reach food-limited carrying capacity?

Define for each particular population we wish to consider the carrying capacity of the habitat with respect to food resources. This is easier said than done, partly because the carrying capacity varies seasonally and food supplies vary in quality (White 1993). There are two general approaches, one through nutritional ecology and bioenergetics and a second through agricultural-type experiments used in pasture management. Given that you can do these studies, you can now determine whether or not your population ever reaches the carrying capacity of the habitat. Mammalian ecologists, at least, seem completely divided on this issue. If food supplies are unutilised, it is possible to argue that the crunch period comes at a different season of the year, or that you have not adequately defined *food*. Skogland (1983) argued that wild reindeer herds in Norway did, in fact, operate at the food-limits of their winter ranges, and that this extrinsic limitation operated through calf mortality. Mduma *et al.*

(1999) argue persuasively that food shortage both limited and regulated the Serengeti wildebeest population during the last 20 years.

If food limitation is the ultimate form of population regulation, populations must regularly bump against carrying capacity. Many populations of mammals are limited by food supplies but are not regulated by food shortage (Boutin 1990). Predation is one of several processes that can keep a population from ever reaching food-defined carrying capacity. Diseases, parasites, territorial behaviour or weather may also regulate or limit a population in this manner. There is at present no agreement on how often these factors operate to hold populations below food limits.

The experimental approach to population regulation immediately suggests a series of manipulations to test whether a particular population is at, or substantially below, carrying capacity determined by food supplies.

Is regulation better studied in widely fluctuating populations by looking for negative feedbacks rather than density dependence?

There are two models of density-dependent regulation. The *strong model* asserts that there is a set of deterministic relationships between population density and birth, death or movement rates. These relationships are postulated to be repeatable in space and time, so that they can form the basis of a predictive theory of population dynamics. The strong model asserts that all variation found in these deterministic relationships is due to measurement error. These relationships may, in turn, be broken down into a further set of density-dependent relationships for mortality caused by predation, food shortage and other factors.

By contrast, the *weak model* of density dependence recognises that there will be some negative-feedback mechanism operating in populations so that, as numbers build up, growth rate will eventually stop. But these negative-feedback relationships are only loosely tied to population density, so that when you make the conventional density-dependent graphs you obtain highly scattered data (e.g. Fig. 2). The scatter or variation in these graphs is interpreted in this model as a message that population density is not the determining variable. Instead, one should plot birth, death and movement rates against some particular ecological variable such as food biomass, predator numbers, or incidence of disease. Density-vague relationships (Strong 1986) will be the rule under the weak model of density-dependence.

For many populations one might argue that we should use the strong model because we do not have the data available to apply the weak model. Thus *density* becomes a surrogate measure of food supplies, social space or predation pressure. If this is the case we should be careful to recognise that we do not expect our density-dependent relationships to be predictive since density is not a close predictor of food supplies or predation pressure or any ecological parameter for many species.

We also need to remember that population density is not a simple variable to measure. In migratory animals, like monarch butterflies or caribou, summer ranges and winter ranges differ and it is not clear what area to use to determine density, even if one knows exactly the number of animals in the population (e.g. Skogland 1983). If predators concentrate on certain age groups in the prey population, the relevant density may be restricted to the very young and very old individuals. In territorial mammals or birds, breeding territorial numbers may determine population density, and non-breeding surplus individuals may be irrelevant to the ecological interactions that determine population density.

Can we assume simple linear cause–effect chains for the analysis of population limitation and regulation?

One of the central assumptions of many animal population ecologists has been that we can determine causes of death for individuals and use these data to understand population limitation and regulation. This has partly been fostered by the technological revolution of methods such as radio-telemetry for vertebrates, which allows one to find animals very quickly after death. If we ignore for the moment the problem of scavenging and assume that we can be there very shortly after an individual dies, we can with due care assess an immediate cause of death. More often than not, predators are the main cause of mortality (e.g. Boutin *et al.* 1986; Steen *et al.* 1997). The key question we have to answer to use these data to discuss limitation or regulation is whether or not there are predisposing factors. Errington (1946) pointed out the classic case of muskrats being eaten by mink in which the individuals were social subordinates that could not obtain a territory. Intrinsic processes operating through territorial behavior could thus result in predation being judged to be the regulating or limiting factor for this population.

Parasites and diseases are often considered to be debilitating conditions that may expose individuals to a higher probability of mortality from starvation, predation, or exposure to bad weather (Murray *et al.* 1997). These interactions between the factors listed in Fig. 1 can be critical, and it is important to try to measure these effects in field populations and to manipulate them experimentally.

A distinction between additive and compensatory mortality has been one attempt to get around the problem of assigning causes of death (Bartmann *et al.* 1992). Although it is possible in principle to determine whether mortality is additive or compensatory (Nichols *et al.* 1984), in practice this distinction has been mainly applied to hunting mortality in comparison to natural sources of loss.

The interaction of factors affecting populations has provided the greatest challenge to the conventional density-dependent view of population regulation (Rodenhouse *et al.* 1997). The result of many interactions is the production of

time-lags in responses, and the resulting failure of current population density to provide much information on future trends in numbers. All of this makes for much more interesting biology, if less simple mathematical models.

Conclusions

How successful have ecologists been in understanding population changes? All the controversy over population regulation and limitation may lead one to believe that no progress has been achieved and, while this view may have been correct in the 1950s, it is completely wrong today. Ecologists have made important progress in the last 50 years in working out the broad principles of population dynamics and uncovering empirical generalisations about the factors that drive population changes. For almost every group of animals and plants we have achieved a good understanding of the mechanisms that may limit or regulate density, and where there is controversy it is usually over whether factor *a* or factor *b* is dominant. Moreover, the experiments needed to decide these controversies are now clearly laid out, and are waiting to be done. A set of paradigms is firmly in place, but there is still much to do.

Paradoxically, population ecology has suffered from too much theory and too little empiricism. In particular, papers on population limitation and regulation abound with theoretical diagrams with no data, and theoretical predictions that could never be tested (Sinclair and Pech 1996; Murray 1999). Theoretical diagrams can have great heuristic value but they can also be confused with the real world. Confusion in all our discussions about population dynamics could be reduced if we followed four simple precepts.

(1) *Distinguish between the three broad questions of population dynamics*. Much confusion could be eliminated from the start by addressing specific questions and recognising that the issue of population dynamics involves at least three broad questions that may have quite different answers for the same population system. Within these three broad questions some approaches have proven to be more useful than others (Table 1).

(2) *Focus on manipulative experiments to answer the key questions*. Our major focus in studies of population dynamics should be this: what factor or process could we manipulate to produce large changes in population dynamics? If predation is necessary for limitation or regulation, you should consider manipulating predator numbers. Manipulative experiments are no panacea but they cut through much of the muddle in discussions of population dynamics.

(3) *Do not draw conclusions about factors you have not studied*. A student of population dynamics is always on the horns of a dilemma because he or she cannot study everything at once. We pick what seems most promising and analyse it. There is no problem with this unless these choices are forgotten when the study is reported in the literature. There are errors in both directions. Diseases are often rejected as causal agents in limitation or regulation when they have not been studied. Spacing behavior is often rejected as a potential regulatory agent when it has not been studied. There are too many examples of this in the literature, and we should be less sweeping in our conclusions and more modest in our generalisations about population limitation and regulation.

(4) *Finish your analysis with a clear statement of the alternative hypotheses and the experiments that could test them*. We should always view science as an on-going process, so that at the end of every paper we should consider what needs to be done next and what the alternatives are. Many authors feel that this is obvious, given the paper. I have found by giving the same paper to several graduate students to analyse that what to do next is seldom obvious. I think our goal here is to build up a growing hypothesis-tree that defines operationally what we mean by terms like predator-regulation, and to devise clever predictions that follow from our hypotheses. Perhaps in molecular biology one could not do this for

Table 1. Some examples of questions on population regulation that are useful to ask and others that have been found over the past 50 years not to be very useful for solving empirical problems of population dynamics

Profitable questions	Unprofitable questions
Why is population A more stable in density than population B?	Is population A stabilised by density-dependent factors?
How variable is population density in population A?	Is population A above or below its equilibrium density?
Is factor X always absent when populations are growing rapidly?	Is the impact of factor X density dependent or density independent?
Are immigration and emigration related to population growth rate?	Is population A an open population?
Are birth and death rates related to density in a repeatable manner?	Are birth and death rates density dependent?
How much does density change when factor Y is reduced or eliminated?	Is factor Y affected by population density?
Is there a time trend in the density of population B?	Is population B in a state of balance?
Do population fluctuations show a common pattern of demography?	Are population fluctuations chaotic?
What factors affect the demographic rates of population A?	Is population A regulated?
Do introduced biological control agents reduce average densities of a pest?	Do biological control agents cause density-dependent mortality?

fear of our intellectual property being stolen, but in ecology we should be glad if someone takes up our ideas, since it requires years of work to obtain a single data point and we need to test our ideas in a variety of systems in different geographical regions.

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