Density Dependence and Independence

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Advanced article

Article Contents

Density dependence occurs when the population growth rate, or constituent gain rates (e.g. birth and immigration) or loss rates (death and emigration), vary causally with population size or density (*N*). When these parameters do not vary with *N*, they are density-independent. Direct density dependence, where the population growth rate or gain rates vary as a negative function of *N*, or the loss rates vary as a positive function of *N*, is necessary but not always sufficient for population regulation. The opposite patterns, inverse density dependence or the Allee effect, may push endangered populations towards extinction. Direct density dependence is caused by competition, and at times, predation. It is detected observationally by analysis of abundance time-series; however, experimental field manipulations provide the most rigorous analytical methods for both detecting and understanding underlying mechanisms. Future directions include expanded and more detailed mechanistic field data integrated with more sophisticated population models.

Introduction: Concepts and Importance in Ecology

In simplest terms, density dependence occurs when an ecological or behavioural parameter varies as a causative function of density (number or cover of organisms per unit area) or abundance (number or cover within a particular habitat), signified as *N* (Royama, 1977). Most often, the parameter of interest involves population dynamics, including the population growth rate and the four primary demographic (or vital) rates – birth, death, immigration and emigration – although related parameters, such as growth and fecundity, are also investigated. **See also**: Population Dynamics: Introduction

Use of the words 'density dependence' alone normally means 'direct density dependence' (or compensation): the per capita (proportional) gain rate (population or individual growth, fecundity, birth or immigration) decreases as *N* increases (**Figure 1a**) or the loss rate (death and/or emigration) increases as *N* increases (**Figure 1b**). The opposite patterns are called inverse density dependence (or depensation). When the parameter of interest does not vary as a function of *N*, then that parameter is said to be density-independent. Note that a demographic rate may have both a density-dependent component, the strength of which is defined by its slope, and a density-independent component, defined by its *y*-intercept (**Figure 1c**).

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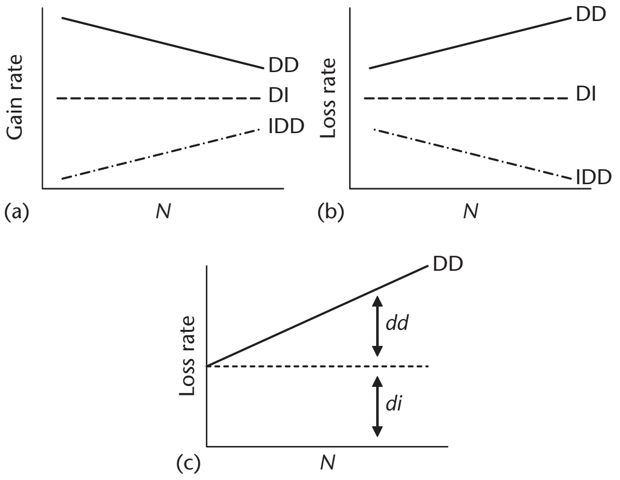

Figure 1 Possible effects of population density on per capita demographic rates. Panels A and B illustrate density-dependent (DD), density-independent (DI) and inversely density-dependent (IDD) gain rates (e.g. population or individual growth, fecundity, birth or immigration) and loss rates (e.g. death or emigration), respectively. Panel C illustrates both density-dependent (*dd*) and density-independent (*di*) components of a density-dependent loss rate. The *dd* component is estimated by the slope of the curve, whereas the *di* component is estimated by the *y*-intercept.

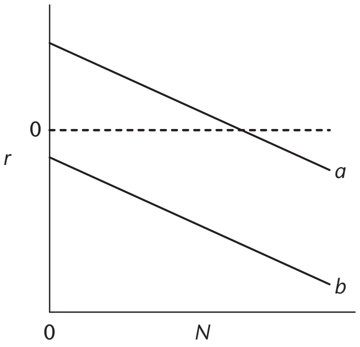

Figure 2 Density dependence does not necessarily result in population regulation. Curves *a* and *b* both illustrate direct density dependence, because the instantaneous population growth rate (*r*) decreases with population density (*N*). However, only the population represented by curve *a* is regulated, because growth is positive at low densities and negative at high densities (i.e. the density-dependent curve crosses the dashed zero-growth line). Despite density-dependent growth of population *b*, this population never exhibits a positive growth rate and will eventually go extinct.

Demographic density dependence is a concept of fundamental importance in ecology (Kingsland, 1995), fisheries biology (Rose *et al*., 2001), wildlife management (Fowler, 1987), pest control (Walde and Murdoch, 1988) and conservation biology (Ginzburg *et al*., 1990) because it is necessary for population regulation: the persistence of a population via bounded fluctuations and return tendency that preclude extinction (Murdoch, 1994; Turchin, 1999). Thus, density dependence that results in the population growth rate being positive at low values of *N* and negative at high values of *N* causes a population to increase before declining to extinction and decrease before overshooting any carrying capacity (**Figure 2**, curve a). The concept of density dependence in the context of population regulation was first modelled as the logistic equation of Verhulst (1838), with many subsequent elaborations. In fisheries and other harvesting models, density dependence is incorporated into various stock-recruitment functions that are the basis of predicting sustainable yields (Beverton and Holt, 1957), albeit with questionable accuracy (Holt, 2009). **See also**: Sustainable use of Populations and Overexploitation

It is important to note that density dependence is necessary but not always sufficient for population regulation because density dependence can be under- or overcompensating (Turchin, 1995). Therefore, the detection of density dependence is not necessarily tantamount to demonstrating population regulation (**Figure 2**).

Inverse density dependence can manifest as the Allee effect, in which a population declines past a lower threshold where extinction becomes inevitable (Courchamp *et al*., 1999). This effect often occurs when declining population density causes the birth rate to decline due to decreasing frequency of contact between potential mates. Understanding the circumstances under which inverse density dependence occurs is therefore a primary focus of conservation biology for preventing endangered species from going extinct.

Mechanisms of Density Dependence

The proximal causes of demographic density dependence are competition, and in special circumstances, predation (including parasitism and disease). Competition is directly density-dependent by definition, being a function of the ratio between population size and resource availability. Thus, competition for living space, for spatial refuges from harsh conditions, for prey refuges, for nutrients and food and for reproductive opportunities all can cause density-dependent responses in demographic rates (Keddy, 1989). **See also**: Competition; Predation (Including Parasites and Disease) and Herbivory

Predators can cause density-dependent mortality of their prey by a variety of mechanisms (Taylor, 1984). First, a numerical response (population growth manifesting total density) of predators may be sufficiently responsive to prey availability to result in more predators when prey are abundant and vice versa. Second, an aggregative response (distributional shift manifesting local density) of predators may congregate predators at high-density patches of prey, and vice versa. Third, a developmental response (individual growth manifesting consumption rate) of predators may increase predator consumption rates to match increases in prey availability. Fourth, a type III functional response, which is a sigmoid individual predator consumption rate as a function of prey density, causes density-dependent prey mortality at lower (but not higher) prey densities (Holling, 1959).

Old Debates Resolved

An old debate in ecology was whether population dynamics are regulated by density-dependent processes or driven by density-independent processes (Kingsland, 1995). Given that each demographic rate is affected by both kinds of processes (**Figure 1c**), such debates were misguided, yet they nonetheless waxed and waned through the years. The last manifestation was the recruitment limitation hypothesis (Doherty, 1981), formulated for demographically open populations whose replenishment relies on external input following settlement of dispersal stages (recruitment), as occurs in many marine species as well as some plants and terrestrial arthropods. This hypothesis asserted that post-settlement mortality was density-independent, so that variations in the external recruitment rate alone drove local population dynamics (Doherty, 1998). The hypothesis was originally proposed for seafloor-associated marine fishes, a group for which most subsequent field experiments have detected density dependence (Hixon and Jones, 2005). The current consensus is that both density-independent and density-dependent processes operate in natural populations, with the latter necessary at some time and place for populations to persist (Hassell, 1986; Turchin, 1995; Hixon *et al*., 2002).

Detecting Density Dependence

Empirical tests for density dependence are often motivated by the question of whether populations are regulated. Although many different types of statistical tests are available, empirical support for density dependence is most easily understood and commonly tested by examining the relationship between the population growth rate and N. Often, population-level data are available as time-series observations of abundance or density (**Figure 3a**). Discrete population growth between two censuses intervals (λ) is calculated as N_{t+1}/N_t, though it is common to use an approximation of the instantaneous population growth rate (r) by taking the natural logarithm of discrete growth values (**Figure 3b**). Direct density dependence in growth rate (**Figure 3b**) implies that when populations deviate from their long-term average, they exhibit a return tendency toward the average trajectory – a necessary but not sufficient condition for population regulation.

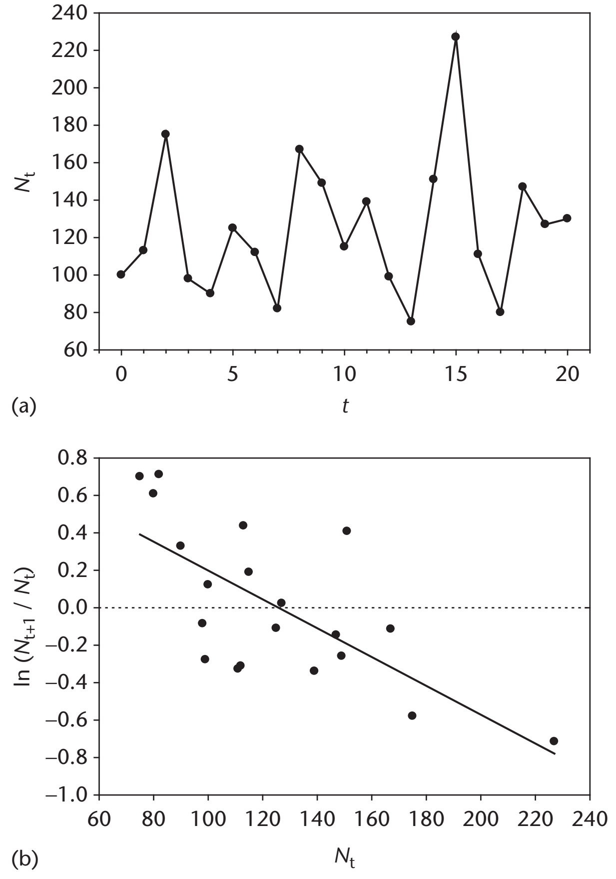

Figure 3 An example of observational data from a population that is regulated by density dependence. Panel A illustrates variation in abundance (N_t) through time (t). Note that the observed values of abundance tend to exhibit bounded fluctuations about a long-term average. Panel B examines the relationship between density and an estimate of the instantaneous population growth rate during the subsequent time interval, which yields a decreasing pattern, indicating direct density dependence.

One should exercise caution when using differences in successive measures of population size to estimate population growth rate. For a multiplicatively growing population, it is clear that growth rate theoretically relates to differences in successive population sizes. In the discrete case, $N_t = \lambda^t N_0$, and therefore $\lambda^t = N_t/N_0$. In the continuous case, $N_t = N_0 e^{rt}$, and therefore $r = \ln(N_t/N_0)/t$. However, exact measurements of population size are rarely available. Rather, population sizes are usually estimated with some degree of imprecision. In such cases, population size estimates represent the 'true' population size ($\hat{N}_t$) plus a positive or negative deviation due to estimation error (ε). When testing for density dependence, the relationship between estimated initial population size ($\hat{N}_1 + \epsilon_1$) and estimated growth rate ($(\hat{N}_2 + \epsilon_2)/(\hat{N}_1 + \epsilon_1)$) is examined. A problem with this approach is that when the initial population is overestimated (i.e. when $\varepsilon_1 > 0$), N_t appears large, and the growth rate (proportional to $1/\varepsilon_1$) appears smaller than it actually is. Likewise, when initial population size is underestimated (i.e. when $\varepsilon_1 < 0$), N_t appears small, and the growth rate appears larger than it actually is. Thus, as the same estimation error has opposite effects on opposite sides of the equation testing for density dependence, estimation errors can cause a spurious, negative correlation between N_t and λ, which may be falsely interpreted as evidence for density dependence. In practice, this problem can often be overcome by comparing population sizes at every other interval (e.g. by estimating the effects of N_t on growth between N_{t-1} and N_{t+1}) or by resorting to more sophisticated statistical tests (Pollard *et al*., 1987; Dennis and Taper, 1994).

Observational approaches to detect density dependence

Early approaches to test for density dependence in time-series of observations concentrated on testing null hypotheses. These methods were designed to determine the probability that a density-independent null model could generate a relationship between density and growth rate as or more extreme than the observed data (Bulmer, 1975; Pollard *et al*., 1987; Dennis and Taper, 1994). Though such tests are very useful, they are restricted to evaluating a single null model, and have been criticized because they tend to reduce the analysis to a dichotomy of whether a density-independent model is rejected or not. Moreover, for many empirical tests of density dependence, particularly those with few observations, the inability to confidently reject a null (density-independent) hypothesis may be largely due to a lack of statistical power, rather than weak or absent density dependence. As a result, hypothesis tests based on short time-series are likely to conclude spuriously that dynamics are strictly density-independent. Indeed, empirical evaluations of such effects do indicate that longer time-series tend to detect density-dependent dynamics more often (Woiwod and Hanski, 1992; Holyoak, 1993; Wolda and Dennis, 1993).

Although the drawbacks of few observations can be ameliorated by including assessments of statistical power and type II errors as part of the hypothesis testing framework, more recent studies have gravitated toward using a model-selection approach to evaluate the support of multiple working hypotheses to explain observed population dynamics (Burnham and Anderson, 2002). This approach typically involves selecting a set of density-independent and density-dependent models beforehand and using a likelihood-based criterion to simultaneously quantify the relative fit to each model. Conclusions about underlying dynamics are based on a continuous measure of the relative ability of density-independent and density-dependent models to explain the observed data, rather than a binary assessment of whether a strictly density-independent model is rejected with 95% confidence. As such, model-selection approaches are less sensitive, though not immune, to problems caused by relatively few observations and low power.

Recently, the compilation of time-series abundance data into large databases (e.g. the global population dynamics database; NERC, 1999) has facilitated the examination of density dependence in unprecedented depth. For example, a recent study by Brook and Bradshaw (2006) compared the fit of density-independent and density-dependent models to time-series observations for approximately 1200 species. This synthesis strongly supported several important hypotheses suggested by evidence from previous, though less comprehensive, studies within the field of population dynamics. Specifically, Brook and Bradshaw (2006) concluded that: (1) density dependence is a pervasive feature of population dynamics for most species, (2) evidence of density dependence increases with study duration (and greater statistical power), (3) density dependence is equally prevalent among major taxonomic groups and (4) model-selection methods detect density dependence more frequently than null-hypothesis tests.

It is now well accepted that most populations experience some level of density dependence and that the dynamics of most populations are regulated, albeit to different degrees. It is also recognized that the form of density dependence can be complex, involving features such as time lags (Turchin, 1990, 2003) and nonlinear responses (Sibly *et al.*, 2005). Such complexities underscore the importance of understanding the mechanisms generating density-dependent regulation. Although observational studies may provide large-scale, population-level data appropriate for testing for density dependence, understanding mechanisms requires more detailed, experimental approaches.

Experimental approaches to detect density dependence

Experimental field studies, in which population density is manipulated while other variables are controlled or at least randomly distributed across experimental treatments, provide the most powerful evidence of density dependence. A common approach to test for density dependence is similar to the method outlined in **Figure 3b**, except that population density is a manipulated variable. The general approach would be the same for testing the effects of density on individual demographic rates, rather than the population growth rate. Note that the direction of directly density-dependent responses will vary among demographic rates (i.e. decreasing for gain rates and increasing for loss rates; see **Figure 1a** and **b**). Historically, experimental tests focused on whether the value of the slope is different from zero, with a significant deviation indicating the presence of direct (or inverse) density dependence. However, in recent years, many experimental studies have shifted toward estimating both the density-independent and density-dependent components of dynamics (e.g. fishes: Schmitt *et al.*, 1999; marine algae: Wright and Steinberg, 2001; terrestrial plants: Poulsen *et al.*, 2007). This progression parallels the shift toward the use of model selection in evaluating how well both density-independent and density-dependent models explain the dynamics of time-series observations of abundance. Estimates of both density-independent and density-dependent components of demographic rates may be easily constructed from a regression-based analysis (**Figure 1c**), though similar estimates can also be obtained by fitting nonlinear models to experimental data.

In addition to providing rigorous tests of density-dependent dynamics, experimental studies are invaluable for identifying the mechanisms that generate and/or modify density dependence. For example, competition and predation may both contribute to density dependence in demographic rates, though the effects of these factors may depend on other, ecologically important variables (e.g. resource availability, habitat complexity, abiotic conditions, etc.). Understanding how a particular population is regulated requires that the underlying mechanism(s) of density dependence and any mediating factors are identified. An informative experimental approach is to simultaneously manipulate population density within different levels of a potentially modifying variable (e.g. presence or absence of predators, nutrient level, etc.). Patterns of density dependence or independence within each treatment can identify mechanisms of density dependence and factors causing variability in density dependence (**Figure 4**). Indeed, as observations of long-term dynamics of populations have become increasingly available, and as approaches to modelling populations have become more sophisticated, it is increasingly apparent that dynamics may be governed by a suite of density-independent and density-dependent factors that may interact in complex ways (Sinclair and Pech, 1996; Kendall *et al.*, 1999). Experimental studies that identify and quantify mechanisms generating and modifying density dependence will be integral to a broader synthesis seeking to understand and predict the complex dynamics of biological populations (Cappuccino and Price, 1995; Hixon and Jones, 2005).

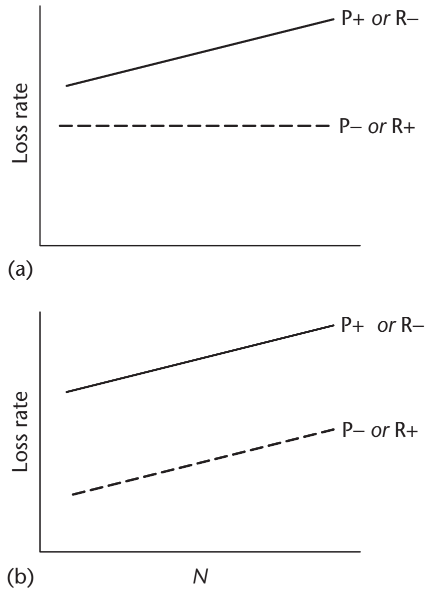

Figure 4 Examples of experimental designs that help to identify factors causing and/or modifying density dependence of a demographic loss rate. Manipulations of population density (*N*) are crossed with manipulations of putative causal agents (e.g. the presence or absence of predators: P+ or P−) or modifying factors (e.g. addition or removal of some resource: R+ or R−). Panel A indicates that density dependence only occurs in the presence of predators or when resources are in short supply. If only predator presence was the manipulated variable, then this pattern would indicate predation as the proximate mechanism of density dependence. If only the resource was manipulated, then this pattern would indicate that low resource availability causes density dependence via competition. In panel B, the presence of predators or removal of the resource has no effect on density dependence, though these manipulations impose a density-independent increase in the loss rate. If only predator presence was the manipulated variable, then this pattern would indicate that predation was not the source of density dependence. If only the resource was manipulated, then this pattern would indicate that removal of that resource increased loss rate, but only in a density-independent fashion. See Hixon and Jones (2005) for a more complete list of possible experimental outcomes and interpretations.

Density dependence in metapopulations

Many species exist within metapopulations, defined as groups of spatially separated, demographically open, local populations that are connected via movement of individuals. Although, at large enough spatial scales all populations are closed, important questions of ecological, conservation and management relevance often focus on the scale of the metapopulation. Understanding density dependence and regulation within the context of a metapopulation faces some major empirical challenges. For many species that disperse during early life stages (e.g. as seeds or larvae) but remain within the local population as adults, local reproduction is decoupled from local population input. The dynamics of local populations are described as open because these populations can be highly influenced by recruitment from external sources and local population size does not grow or decline as a multiplicative function of previous population size (Caley *et al.*, 1996). In such cases, conventional density dependence tests that compare the effects of local population size on local population growth will not be informative. Understanding the regulation of metapopulations requires a two-step approach. First, one must identify factors that regulate local populations, especially given recruitment variability. Importantly, sources of local density dependence must be evaluated with respect to their effects on local population size and reproductive output. Second, one must estimate the proportion of local reproduction that survives and seeds other sites, as well as the proportion of local reproduction that feeds back into the local population. Only by measuring both the local regulation and the connectivity among populations, will there be sufficient information to empirically evaluate density dependence and regulation of the scale of the metapopulation (Hanski, 1999).

Future Directions

The study of density dependence and population dynamics remains a very active field of research. In addition to the practical aspect of understanding population dynamics in terms of species management and/or conservation, many ecologists are fascinated by the complex dynamics exhibited by natural populations. Although most agree that density dependence plays an important role in the dynamics of most populations, it is also clear that populations may be influenced by a suite of density-independent and density-dependent factors that may act independently or interactively, including inverse density dependence. Understanding such complexities requires a combination of both observational and experimental studies.

Recommendations for future research fall into three categories. First, there is need for more data on the ecology of natural populations. The study of population dynamics has made major advances with the compilation of long-term data sets describing temporal patterns of abundance within populations. Many data sets are now available but more are needed to fully describe the range of dynamics exhibited among species that vary in their life histories and ecologies. Second, there is need for models of population dynamics that can adequately describe the complex dynamics of natural populations. Particularly desired are more models that incorporate realistic biological mechanisms as the sources of density dependence and models that can be used to predict dynamics of populations accurately. Third, there is need for more experimental studies that identify causes of density dependence, as well as experiments that investigate factors that modify density dependence. Importantly, these needs are not completely separate endeavours. Experiments are necessary to identify mechanisms that cause variation in demographic rates and population growth. Once these mechanisms are described, they can be incorporated into biologically realistic models of population dynamics, which will be most appropriately tested with, and applied to, long-term data on population abundance.

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