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Bridging the gap between ecology and evolution: integrating density regulation and life-history evolution

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Early demographic models of life-history evolution were formulated in a density-independent framework and saw extrinsic sources of mortality, such as predation, as the primary driving force that shaped the evolution of life-history traits. The evidence for density dependence in nature motivated theoreticians to build models that incorporated population regulation. These later generations of models acknowledge that demographic mechanisms of population regulation and extrinsic mortality interact with one another and predict a wide variety of life-history responses. Such ecologically realistic models require knowledge of the demographic traits and life-stages most affected by density. Despite the vast empirical literature characterizing population regulation, and a wealth of methods to analyze it, such mechanistic understanding is rare. Ecological experiments whereby density is manipulated can be a powerful tool to disentangle the life-history determinants of population regulation. Here we review published density-manipulation experiments and highlight how they can be coupled with existing analytical tools to extract the mechanistic information needed for evolutionary models of life histories.

Keywords: life history; density dependence; population regulation; demography

Introduction

The concept of density dependence was fundamental to Darwin's formulation of evolution by natural selection. It was the impossibility of exponential growth when resources are limited that inspired the idea of the struggle for existence, or the role of biotic interactions, in shaping how organisms adapt to their environment.[1] In the early part of the 20th century, evolutionary biologists set aside the ecological struggle and focused on merging population genetics and the principles of natural selection.[2] At the same time, ecologists began to debate whether density-dependent or density-independent factors governed population numbers.[3–5] Much of the debate centered around defining the necessary and sufficient conditions for regulation to occur.[6] Today population regulation is recognized as the presence of a "long-term stationary probability distribution of population densities,"[6,7] which incorporates earlier arguments regarding the meaning of equilibrium.[7–10]

Despite Darwin's early emphasis on this link between ecology and evolution, we argue here that we have yet to fully and clearly integrate the disciplines of population ecology and evolutionary biology. We can see early phases of such integration in the work of David Lack[11,12] or Reginald Moreau,[13] who were interested in the interaction between population biology, resource availability, and the evolution of clutch size. The early formulation of life-history theory as *r*- and K-selection also attempted a synthesis between population regulation and evolutionary ecology by championing natural selection as density dependent.[14,15] Later development of demographic theory had the opposite effect because it defined fitness as the intrinsic rate of increase (r) in a population that lacked limits to population growth.[16,17] As demographic theory was modified to become more biologically realistic, it once again

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incorporated the "struggle for existence" in the form of density regulation, first by simply substituting r with R_0 (net reproductive rate) as the measure of fitness,[18] then by incorporating specific demographic mechanisms for density regulation.[19]

Density regulation thus has two lives in the scientific literature. One lies in the realm of population ecology, where there has been a vast amount of work done to characterize how natural populations respond to density. The second lies in the realm of evolutionary biology, where population regulation plays a key role in theory for the evolution of life histories. We see little evidence of a connection between the two in the empirical literature despite repeated calls for integration from theoreticians.[18–26] Few empirical investigators have attempted to test evolutionary theory that incorporates density regulation with empirical studies of life-history evolution. Those evolutionary studies that have incorporated density have mostly focused on finding evidence for density-dependent selection.[27–29]

This disconnect comes at a time when ecology and evolutionary biology are at an important crossroads. Ecological factors have long been known to influence the outcome of evolutionary processes. However, because evolutionary processes were thought to occur on longer timescales, short-term evolution was thought either to not influence ecological processes or to have such a small effect that it could safely be ignored. Recent theoretical and empirical work has shown this assumption to be false and that evolution can influence ecological processes.[30–37] Many of these influences are a result of, or are modified by, the effects of density and thus we see density as playing a central role in bridging the gap between ecology and life-history evolution.

In this review, we ask how much of what has been learned by ecologists is useful to evolutionary biologists who seek to use it to understand the evolution of life histories. More specifically, our aims are to (1) briefly outline the way density is incorporated into life-history evolution and integrative ecological-evolutionary (eco-evo) theory and, in doing so, define the types of information on density effects that are needed to advance theory; (2) evaluate the extent to which existing ecological experiments can be applied to these bodies of theory; and (3) suggest how future experiments can be more effectively designed and analyzed to address the interface between ecological and evolutionary processes.

Life-history theory and population regulation

The vast majority of life-history models are optimality models wherein the end points of the evolutionary process are the optimal values of traits that together maximize some measure of fitness.[38–40] At the heart of such evolutionary arguments is the question of how to define fitness.[41] Traditional measures of fitness have been derived from the Euler–Lotka equation, which emphasizes the numerical nature of fitness and its relation to the spread of alleles in population.[25] These equations are the same as those used in population biology to characterize the growth or decline of the population. In population biology, these equations describe the growth of the entire population (all genotypes). However, when used in life-history models, these equations represent a single genotype and do not explicitly include any information on the rest of the population. The measures of fitness (r or R_0) thus represent the intrinsic rate of increase and the net reproductive rate of individual genotypes.

Each metric of fitness makes assumptions about the population dynamic state of the populations. The choice of fitness measure can alter the predicted optimal life history.[42] In general, when the environment is constant and the population is not subject to density-dependent effects on growth, r is the appropriate measure of fitness. If the environment is constant and the population is at or near equilibrium then R_0 is thought to be the appropriate measure.[39,40,42,43] Other traditional measures of fitness modify r or R_0 when the constant environment assumption is violated.[43]

Maximizing one of the fitness measures implies that some set of fitness components must be optimized. Williams[44] developed the idea of partitioning Fisher's[45] reproductive value into current and residual reproductive value. At the heart of Williams' argument was the insight that the total amount of energy available to an organism was fixed and that increased allocation toward one function like reproduction came at the cost of decreasing the energy allocation to others such as survival and maintenance. Thus there exists a trade-off between the reproduction and survival. The exact shape of the trade-off depends on the relationship between the function in question and reproductive effort.

Schaffer[16] showed how by using these functions one could determine the optimum reproductive

effort at each age in a density-independent age-structured population. Charlesworth and Leon[18] extended Schaffer's analysis of life-history optimization in age-structured populations under density-independent and density-dependent conditions. For density-independent populations they assumed that r was the fitness parameter to be maximized and for density-dependent populations R_0 was maximized. They emphasized the importance of density-dependent regulation in determining the predictions of life-history models when they stated that "the effect on our conclusions of including density dependence seems to depend on the precise model of population regulation which is envisaged." As examples, they showed that applying a density-dependent mortality factor to all ages should cause a decrease of reproductive effort with age and if this mortality factor acted only on the juvenile age classes, then this should cause an increase in reproductive effort with age (Fig. 1).

Charlesworth and Leon[18] argued that in the case where populations are limiting in their growth, R_0 was the appropriate measure of fitness because the phenotype that had the highest R_0 should dominate the population. At the same time, however, R_0 for the broader population is assumed to be equal to 1 (not growing). In Charlesworth and Leon and previous models both things can be true because the population is assumed to be at equilibrium. However, there was no explicit formulation of the population dynamics in their models.

Michod[19] extended Charlesworth and Leon's[18] model by explicitly incorporating the effect of the density of the dominant phenotype (resident phenotype) on the reproductive effort and R_0 of an alternative (mutant phenotype) invading at a low density. In doing so, he explicitly modeled both the evolutionary and ecological dynamics such that the ecological dynamics could influence the outcome of the evolutionary model. He examined how the distribution of reproductive effort should change in response to changes in the mortality schedules at various ages in density-independent and density-dependent scenarios. He found that how reproductive effort should change with age depends on how the mortality change is distributed across age groups and the age-group(s) that underlie density-dependent population regulation (Fig. 1).

Demographic mechanisms that bring about density regulation are changes in the age-specific birth and death rates. In each of the above models, density-dependent population regulation is seen to be achieved by adding density-dependent terms to each demographic variable. These terms describe how the demographic rate changes as a function of density and explicitly describe how genotypes interact with the environment to determine performance in that environment. Thus these terms describe the plastic nature of the phenotype. This approach assumes these parameters respond to changes in density in a specific manner and also their relative importance in determining how populations respond

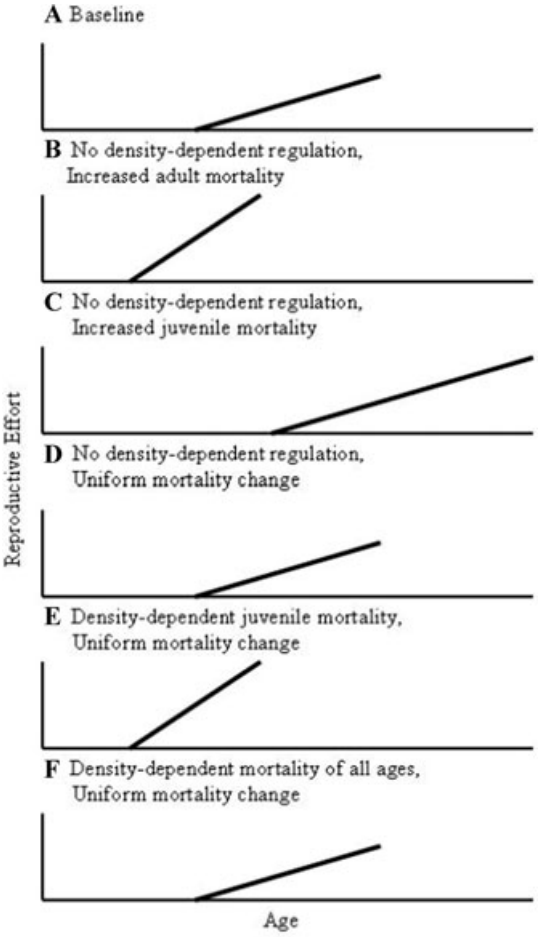

Figure 1. **Predictions of changes in lifetime distribution of reproductive effort in response to changes in age-specific mortality. Panel (A) represents the baseline. Panels (B–D) represent expected changes with no density-dependent regulation. Panels (E–F) represent expected changes when population is regulated via juvenile mortality (E) and via all age classes (F). Predictions are summarized in Charlesworth[25] and figure is redrawn after (data in Ref. 107).**

to density. When this is done, an assumption is made regarding how age-specific vital rates respond to changes in density and their relative importance in determining the demographic density-dependent response. To provide generality, the predictions of the models are evaluated under different conditions, often with different qualitative results. Understanding precisely which demographic factors respond to changes in density is no trivial matter but is critical to understanding the predictions for how life histories will evolve in response to changes in mortality.

Density-dependent life-history models typically assume a single demographic parameter is responsible for demographic compensation. However, it is likely that such density compensation occurs through multiple demographic mechanisms. Many demographic traits can change in response to density, whether independently or linked through trade-offs. Moreover, these can respond differently in different age classes. The challenge is to identify which trait or traits are the most important in leading to changes in the population growth rate.

Demographic compensation mechanisms describe how the population responds to ecological changes but ignore the underlying ecological interactions that determine the response of individuals in the population. For example, predators increase the mortality rate of their prey, but as predators kill prey, they can also reduce prey population density and hence reduce density-dependent mortality. Ecological mechanisms such as resource availability and predation determine the density-dependent response, but is it important which ecological factor is responsible? Such indirect ecological effects have been widely investigated in ecology, however, their influence in determining the outcome of the evolutionary process is not well understood.[24,46]

Abrams and Rowe[24] used an optimality model to investigate the effects of increased nonsize selective predation on size and age at maturity that included both the direct effects of predation and the indirect effects of increased resources. They evaluated the models with age and size at maturity as either flexible or fixed and with a fixed or flexible growth effort. The predictions of the model are highly dependent upon which of the demographic variables are flexible, whether increased growth effort increases susceptibility to predation, and how predator density affects the abundance of resources for the prey. The general conclusion was that indirect effects of changes in mortality can exert strong influences on the size and age at maturity and may, depending on the strength of the indirect effect, override the direct effect of predation.

Incorporating indirect effects into life-history evolution is appealing because it explicitly considers the mechanisms of how mortality and resource variation can interact to produce changes in the life history. Such lower level explanations are likely to yield higher predictive power. Early work on life histories envisioned resources as playing a major role[4,12] in shaping life-history evolution whereas subsequent research emphasized the role of age-specific mortality in shaping life-history evolution. Resources were seen as playing a role in determining the proximate adjustment of vital rates but ultimately were thought to play little role in causing interspecific variation in life-history traits.[47] Models that incorporate such indirect effects add a way for us to understand the ecological processes that determine phenotypic performance and thus nest classic evolutionary demographic models within a broader ecological context.

New modeling approaches related to Michod's invasibility models use and extend the analytical tools of evolutionary game theory[48,49] by modeling the ability of a mutant genotype to invade a resident population.[32] While only the resident population is assumed to have demographic effects, successful mutant phenotypes are allowed to invade, become resident, and consequently determine the demography. Such explicit modeling of evolution as an invasion process acknowledges that ecological factors and context affects evolution, but also that evolution changes the ecological context as it proceeds. The bidirectional dependence of fitness on the environmental selection pressures and the phenotype on the environment has been called the eco-evo feedback loop.[32] In these approaches, the environment of the invading phenotype can be extended to include not only the resident phenotype, but also broader interspecific interactions (competitive, mutualistic, parasitic, or predatory) that are affected by the resident.[50] The explicit inclusion of these broader interactions represents a new framework to understand the relative importance of direct and indirect

ecological and evolutionary effects on life-history evolution.[50]

Deriving general predictions from this body of theory is difficult because of the complexity of such interactions. However, as is true for simpler models of life-history evolution, knowing the demographic mechanisms of population regulation is crucial because they determine, in part, the ability of new phenotypes to invade. Density also plays a role in determining the dimension of the feedback loop. If all individuals are equally affected by density, the eco-evo feedback is said to be one dimensional. This is because the demographic environment is then appropriately described by a single parameter, which is the total number of individuals in the population. If genotypes affect and are affected by the environment differently, then more parameters are necessary to describe the eco-evo feedback. Frequency dependence is an example of a multidimensional feedback. In the simplest case, for example, where just two genotypes are considered and each of them affects and is affected differently by density, two parameters (the densities of each genotype) are needed to describe the feedback.[51] Density-dependent selection, where the effect of the environment differs as a function of the genotype, also represents a multidimensional system. If the population is structured (as most are) by age, size, state, etc., then each of these stages may define a different dimension in the feedback loop. Only when the environment feedback loop is taken to be one dimensional does an optimum exists that can maximize r or R_0.[51] Otherwise, the fitness measure that is maximized depends on the specific nature of the environmental effect (i.e., the resident population dynamics).[21,25,32,34,35,51,52]

The development of life-history theory started off as an attempt to understand how the interaction between population biology and evolutionary processes combined to cause variation in the life histories of organisms.[11–13] Because a complete understanding of the scope of these interactions was initially too complex, theoreticians resorted to tackling smaller portions of the theory. In the last half century the field has matured to the point where the original vision of these interactions is within view. A central theme in these advancements has been the incorporation of the limits to population growth into theory. The theory reviewed above points at the sensitivity of life-history predictions to the specifics of density dependence. In particular, it highlights the importance of three key empirical questions: (1) Which life stages and demographic rates are affected by density dependence? (2) What is the dimensionality of the feedback between evolution and the demographic environment? (3) Are the mechanisms of density dependence direct or indirectly expressed? With these three questions in hand, what can we learn from the vast ecological literature on density dependence?

Ecological approaches

Numerous reviews on population regulation have been written through the years. It is beyond the scope of this article to review population regulation as a concept or to evaluate all the empirical evidence for accepting it as an ecological tenet.[53–61] Rather we concentrate on discussing the factors that are important in determining the predictions from life-history theory, namely, which demographic parameters and which age/size classes of the population are the most important in regulating the population. To do so, we briefly discuss how the various methods used by ecologists to answer these questions can provide the information required by evolutionary biologists. We then present the results of a literature search with the goal of determining if the ecological literature provides the types of necessary demographic information. Finally, we highlight a few well studied biological systems that can provide insights into how this ecological information can be used in the evolutionary study of life histories.

Methods of testing for regulation

Methods in the study of population regulation have been designed to answer three general questions. First, do density-dependent processes regulate the population? Stated another way, does the population exhibit a return tendency? Second, since this return tendency must be achieved via changes in the demographic rates of the population, which of these rates are primarily responsible for driving the change in the population growth rate? Third, what are the underlying ecological factors (e.g., competition or predation) responsible for the demographic changes?

Most studies of population regulation are either long-term observational studies, or mid- to short-term manipulative experiments. A review of

studies on population regulation would likely reveal a trade-off between long-term data and mechanistic understanding. Long-term observational studies are well suited to determine if populations exhibit a return tendency over the long term. If individuals are followed through time, then they can answer how the many demographic rates are correlated with changes in population growth rate. Changes in demographic rates that determine population dynamics are likely to be influenced by multiple ecological factors[62] that may act additively or interact. While data from long-term individual-based observational studies can go a long way in evaluating how these alternative ecological factors interact to influence demographic rates,[62] these data sets suffer from two problems. First, they are slow and logistically involved to build. Second, their observational nature and low (if any) replication limits their ability to determine causality.

Long-term manipulative experiments wherein either the environment or the population itself is manipulated have been advocated as being ideal to address these questions.[55] When data on demographic rates are available, these data sets can be analyzed using the statistical techniques that decompose population growth into contributions from individual rates (see below). Manipulations in these experiments generally consist of "press" manipulations wherein the manipulation is routinely imposed on the population. However, data sets such as these are rare and expensive to obtain and are thus unlikely to be good models to answer these questions except in a few rare cases. In addition, the responses seen to multigeneration, repeated imposition of experimental treatments could be attributable to the phenotypic adjustment of the demographic rates and/or the evolution of the population in response to the imposed treatments.[63] It is generally not known if the demographic responses exhibited by these populations represent demographic compensation to altered density or to the evolution of alternative life-histories strategies in response to altered mortality regimes or both.[64,65]

Short-term (one generation or less) density manipulations represent a valuable and underexploited tool in evolutionary biology. If demographic data is collected during the course of the experiment, then methods to calculate and decompose population growth rates into contributions from individual rates are possible. Population size, resources, or predators can be manipulated directly in these experiments to determine causality between demographic changes and ecological factors. Because these experiments can be performed over short time intervals, evolution of the target population can be minimized and the demographic changes will likely be dominated by phenotypic adjustments (at least for surviving individuals). Expense in these experiments is also reduced, which means that many more populations/species can be manipulated and inferences about the generality of the responses can be made. Despite these advantages, the short-term nature of these experiments may not allow fully capture the density-dependent response if delayed responses or cross-generational effects are important. Some of the arguments in favor of using short-term density manipulations have been said elsewhere.[66]

Analytical techniques used for these experiments vary widely. The simplest way to analyze demographic responses to density manipulations is to increase or decrease the population density of some populations and use others as controls, then test for differences in these treatment groups after some time period using conventional inferential statistical techniques such as ANOVA. Although these methods of analysis are common (see below) they yield little information regarding the demographic changes that actually influence population growth.[67] The demographic variables that differ significantly between the control and experimental treatments may in fact have little or no impact on the population growth or individual fitness.[67,68] Because of these issues, there have been efforts to develop analytical techniques that decompose changes in population growth into contributions from each trait. The earliest attempt at such a decomposition was key-factor analysis.[69] This method was previously popular but because of several methodological problems is rarely used.[70,71] Modern analytic techniques, such as matrix life-table analysis[67,72,73] and structured accounting of the variance of demographic change (SDA)[74,75] decompose population growth rates into contributions from individual demographic rates. The SDA approach is similar to the matrix life-table analysis approach with two exceptions. First, matrix population models assume that the population age structure is in equilibrium, whereas the SDA approach does not. Second, the matrix life-table analysis approach is only a linear

approximation of the decomposition of variation in population growth, whereas the SDA approach provides an exact decomposition. Despite these differences, however, matrix life-table analysis and SDA approaches seem to provide similar results.[74] To illustrate how these analytical techniques can be used, we discuss only matrix life-table analyses, though SDA could be used as well.

Two types of matrix life-table analyses are available that can detail how changes in trait values influence population growth: prospective and retrospective analysis.[67,73,76,77] Prospective analyses use life-table data to look forward and determine which demographic variables can potentially contribute the most to population growth. Prospective analyses yield sensitivity and elasticity measures for each trait that can be interpreted as the sensitivity of population growth rate to absolute changes (sensitivity) and proportional changes (elasticity) in trait values (Table 1). However, these methods measure only what the potential impact of changing the vital rates would have been in the absence of any covariation between traits[73,74] and, thus, do not measure the actual influence of traits to actual changes in population growth.

In contrast, retrospective analyses or life-table response experiments (LTRE) look backwards at life tables that describe trait values under different population growth conditions and ask which vital rates contributed most to changes in observed population growth rate after an observed change in population growth. If life tables are constructed for populations that have had their densities manipulated, then they can yield the necessary details to characterize the demographic responses that underlie density regulation. The result of LTRE analysis is a matrix of contribution values for each age-specific vital rate. These values are a first-order linear approximation of decomposition of the change in the population growth rate (λ) between control and manipulated population (Table 1). Values with larger absolute values contributed more to changes in population growth rate than changes with smaller absolute values. This method allows for the identification of the age-specific demographic traits that respond to the perturbation and permits evaluation of each trait's relative impact on population growth rates (Table 1). This information, in turn, can be incorporated into life-history models that explicitly include density-dependent regulatory mechanisms. For example, if density perturbation experiments reveal that changes in density mostly affect juvenile survival, then researchers could use this information to derive specific predictions regarding the evolution of life histories in their particular system. These methods are not restricted to using lambda as a measure of population growth and other measures have been used such as R_0[78] and the stochastic growth rate.[79] Methods for calculating standard errors and hypothesis testing are available.[73]

It has been emphasized that estimates of demographic parameters and hence the sensitivities of population growth to changes in demographic parameters are sensitive to the duration of the life stage.[80,81] Integral projection models (IPM) have been developed that describe the changes in demographic rates as continuous functions rather than discrete life stages to remedy this problem.[82,83] Using these methods, one does not obtain sensitivities for each stage, but rather a surface of sensitivities to each demographic rate. Although, an explicit link between these methods and LTRE analysis has not been covered in the literature, the institution of these IPM's in LTRE analysis should be straightforward.

Sensitivities are typically estimated as the partial derivative of population growth rate (λ) with respect to the demographic parameter of interest. Because they are partial derivatives, they represent the effect of changing that parameter while holding other parameters constant. However, because demographic parameters are linked to each other through lower-level parameters and physiological or genetic tradeoffs, change in one demographic parameter often causes change in other parameters. This covariance among demographic traits is well studied in life-history theory[38–40] and can potentially lead to incorrect sensitivities and resulting LTRE contributions if not incorporated into the analysis. To remedy this, van Tienderen[84] introduced the concept of integrated sensitivities, which conceptually are a simple extension and allow one to examine the direct of changes in a trait on population growth plus the added indirect effect of changes in that parameter on other parameters. Mathematically, this is equivalent to calculating the sensitivity as the ordinary derivative of population growth with respect to the demographic parameter.[84]

Table 1. Life tables, leslie matrices, sensitivity matrices, elasticity matrices, and contributions from LTRE analyses calculated for density manipulation of hypothetical population

	Control					Increased density					Decreased density				
x	s_x	l_x	m_x	b_x	$l_x m_x$	s_x	l_x	b_x	m_x	$l_x m_x$	s_x	l_x	b_x	m_x	$l_x m_x$
Life tables															
0	0.50	1.00			0.00	0.33	1.00		0	0.00	0.68	1.00		0	0.00
1	0.50	0.50	0	0.00	0.00	0.32	0.33	0.00	0	0.00	0.67	0.68	0.00	0	0.00
2	0.47	0.25	2	1.00	0.50	0.31	0.10	0.33	1	0.10	0.64	0.45	2.03	3	1.36
3	0.40	0.12	3	1.50	0.35	0.26	0.03	0.65	2	0.06	0.54	0.29	2.70	4	1.15
4	0.27	0.05	3	1.50	0.14	0.17	0.01	0.65	2	0.02	0.36	0.16	2.03	3	0.47
5	0.04	0.01	2	1.00	0.03	0.03	0.00	0.65	2	0.00	0.06	0.06	1.35	2	0.11
6	0.00	0.00	1	0.50	0.00	0.00	0.00	0.33	1	0.00	0.00	0.00	0.68	1	0.00
R_0					1.01					0.19					3.09
r					0.00					−0.61					0.43
λ					1.00					0.55					1.53
Leslie matrices															
	0.00	1.00	1.50	1.50	1.00	0.50	0.00	0.33	0.65	0.65	0.65	0.33	0.0	2.03	2.70	2.03	1.35	0.68
	0.50	0	0	0	0	0	0.32	0	0	0	0	0	0.67	0	0	0	0	0
	0	0.47	0	0	0	0	0	0.31	0	0	0	0	0	0.64	0	0	0	0
	0	0	0.40	0	0	0	0	0	0.26	0	0	0	0	0	0.54	0	0	0
	0	0	0	0.27	0	0	0	0	0	0.17	0	0	0	0	0	0.36	0	0
	0	0	0	0	0.04	0	0	0	0	0	0.03	0	0	0	0	0	0.06	0
Sensitivity matrices																		
	0.000	0.183	0.086	0.034	0.009	0.000	0.000	0.200	0.112	0.054	0.017	0.001	0.000	0.173	0.072	0.025	0.006	0.000
	0.751	0	0	0	0	0	**0.571**	0	0	0	0	0	**0.901**	0	0	0	0	0
	0	**0.402**	0	0	0	0	0	**0.389**	0	0	0	0	0	**0.399**	0	0	0	0
	0	0	0.151	0	0	0	0	0	0.177	0	0	0	0	0	0.110	0	0	0
	0	0	0	0.035	0	0	0	0	0	0.066	0	0	0	0	0	0.023	0	0
	0	0	0	0	0.005	0	0	0	0	0	0.010	0	0	0	0	0	0.003	0
Elasticity matrices																		
	0.000	0.182	0.128	0.051	0.009	0.000	0.000	0.119	0.134	0.064	0.020	0.001	0.000	**0.229**	0.127	0.034	0.005	0.000
	0.371	0	0	0	0	0	**0.338**	0	0	0	0	0	**0.395**	0	0	0	0	0
	0	**0.189**	0	0	0	0	0	**0.219**	0	0	0	0	0	0.166	0	0	0	0
	0	0	0.060	0	0	0	0	0	0.085	0	0	0	0	0	0.039	0	0	0
	0	0	0	0.009	0	0	0	0	0	0.021	0	0	0	0	0	0.005	0	0
	0	0	0	0	0.000	0	0	0	0	0	0.001	0	0	0	0	0	0.000	0
LTRE matrices								Increase vs. control							Decrease vs. control			
						0.000	**−0.128**	−0.080	−0.034	−0.004	0.000		0.000	**0.182**	0.093	0.015	0.002	0.000
						−0.119	0	0	0	0	0		**0.146**	0	0	0	0	0
						0	−0.066	0	0	0	0		0	0.066	0	0	0	0
						0	0	−0.022	0	0	0		0	0	0.018	0	0	0
						0	0	0	−0.004	0	0		0	0	0	0.003	0	0
						0	0	0	0	0.000	0		0	0	0	0	0.000	0

Notes: x represents the age class, s_i is the probability of surviving from age class s_x to s_{x+1}, m_x is the fecundity at age x, b_x is the fecundity at age x times the survival at age 0. Values in bold are sensitivity, elasticity, and LTRE contribution matrices that represent the life-table values with the two highest values.

What ecological studies have revealed about demographic mechanisms

To characterize how the ecological study of population regulation can inform evolutionary study of life histories, we surveyed the ecological literature for empirical studies that performed density-perturbation experiments and evaluated if these studies provided the necessary information on demographic responses to changes in density. We also included studies that indirectly altered density by altering resource availability to test for bottom-up or top-down control of population size. We were particularly interested in studies wherein the density manipulation was conducted on one or multiple ages/sizes in the population and where the demographic response variables could be broken up into age/size classes. Additionally, we restricted our literature search to studies of vertebrate populations.

Some selective review of the literature was necessary because the literature is so large. Additionally, since much of the support for classic demographic life-history theory has come from comparative studies of vertebrate populations[85–96] understanding how regulation is attained in this taxa would integrate well with the empirical literature on life-history evolution. We began our survey in the ISI Zoological Record database with the keywords *population regulation, density dependence, density manipulation, density perturbation,* and various suffixes of these words in the keywords, abstracts, or titles. We supplemented this search by including studies from the references cited sections of these papers that fit the criteria of our search, but they may have been too old to be included in the ISI database. These relatively *ad hoc* search criteria yielded 38 studies in which density manipulation experiments were used to evaluate demographic regulatory mechanisms.

Our literature survey revealed that despite the abundance of empirical studies that examine the demographic mechanisms of density regulation, most (36 of 38) used standard tests of statistical differences (e.g., ANOVA, regression, *t*-tests, goodness of fit) to test for significant differences of individual demographic traits between treatment and controls or multiple treatment groups (Table 2). Only two density perturbation experiments used the LTRE approach to investigate the response of demographic rates to density perturbations, and none used SDA. The rarity of these analyses in these studies may be a combined result of the studies being conducted before published accounts of the methods and incomplete data on the full spectrum of life-table variables. Of the studies that did not use life-table methods 77.8% (28 of 36) were published after the original publication of the details of the LTRE methods.[67] Of these 28 studies, only 21.4% (6 of 28) collected complete life-table data to conduct these analyses. Of these six studies, one study was from reptiles, two were from passerine birds, and two and one were from small and large mammals, respectively. Studies of amphibians and coral reef fishes were well represented, but none contained the complete data from a single study to evaluate which demographic parameters were responsible for adjustment of population growth rate. Notably missing from these studies were characterizations of the adult demography including survival and fecundity (Table 2), due to adults and juveniles in these taxa occupying vastly different habitats. The parameters necessary to apply these methods differ slightly between taxonomic groups. For example, many organisms can only reproduce at discrete age units because of the seasonal nature of reproduction. Also, somatic growth may be important in organisms with indeterminate growth, but less so in those that cease or significantly slow growth after maturation.

Of the 36 studies that did not use an LTRE framework, 97.2% (35 of 36) found a significant effect of the density manipulation on one demographic trait. Of the studies that measured changes in more than one demographic variable, 72.4% (21 of 29) found significant differences in more than one variable. For amphibians, 100.0% (9 of 9) studies found significant effect on somatic growth and 80% (8 of 10) found significant effects on age or size at maturity or metamorphosis. For coral reef fishes, only two vital rate categories had enough studies to calculate percentages. There were significant effects on juvenile survival in 83.3% (5 of 6) and significant effects on growth in 25% (1 of 4). For small mammals, fecundity or reproductive success showed significant effects in 100.0% (4 of 4) of the studies, while juvenile and adult survival showed significant effects in only 66.7% (4 of 6) of studies. Taken together, age or size at maturity or metamorphosis was the most likely trait to show a significant effect (85.7%, Table 2). This was followed by fecundity or reproductive success (83.3%, Table 2) and somatic growth (76.2%, Table 2). These studies generally conclude that increased density has a negative effect on traits that contribute to population growth, which leads to regulation of the population. However, understanding how changes in these traits actually contribute to changes in population growth rate cannot generally be evaluated. There was an attempt to rank traits according to their influence on population growth rate in only three of these studies. In only one of these three studies did there appear to be complete demographic data to adequately weigh the contributions of each trait to changes in population growth. The information that can be gleaned from these studies is that density does act to significantly change demographic traits and that it often affects more than one demographic trait. However, we have learned little about the relative importance of different traits in contributing to population regulation.

One study mentioned above that contained complete demographic data, but was not analyzed

Table 2. Summary of literature review of studies that conducted density manipulation experiments on vertebrate populations

Taxonomic category or summary statistic	Species	Setting	Manipulation type		Response variables					Rank	Ecological factor	Analysis type	Ref
			Pop/ Res	Age-class	JS	AS	GR	AMAT/ SMAT/ SMET	F or RS				
Amphibians													
	Ambystoma laterale	nat	P	J	1*	–	1*	–		N	–	A	111
	Ambystoma opacum	nat	P	J	2*	–	2*	1,1*	–	N	–	A	112,113
	Ambystoma talpoideum	meso	P	J	1*	–	1*	1*	–	N	–	A	114
	Bufo americanus	meso	P	J	1	–	1*	1*	–	Y (GR, SMET)	R	R, A	115
	Hyla femoralis	meso	P	J	1*	–	–	1*	–	N	–	A	116
	Hyla gratiosa	meso	P	J	1	–	–	1*	–	N	–	A	116
	Notophthalmus viridescens	meso	P/R	A	–	–	1*	1	1*	N	R	A	117
	Notophthalmus viridescens	nat	P	A	–	1*	1*	–	–	N	–	A	118
	Pseudacris triseriata	nat	P/R	J	1*	–	–	1*	–	N	R	A	119
	Rana lessonae	meso	P	J	1	–	1*	–	–	N	R	A	120
	Rana sylvatica	lab	R	J	–	–	1*	1*	–	N	R	G-tests	121
	Rana temporaria	nat	P	J	1*	–	–	1*	–	Y (JS, SMAT)	–	A	122
Total studies					10	1	9	10	1				
Significant effects					7	1	9	8	1				
Percent significant effects					70.0	100.0	100.0	80.0	100.0				
	Coral reef fishes												
	Coryphopterus glaucofraenum	nat	P	A	–	2*	1	–	–	N	P(2),R	A	123,124
	Coryphopterus nicholsii	nat	P	J	1*	–	–	–	–	N	P	R	125
	Dascyllus aruanus	meso	P/R	J	1*	–	1*	1*	–	N	R	A	126
	Gramma loreto	nat	P	A	1*	–	1	–	–	N	P,R	A	127
	Lythrypnus dalli,	nat	P	J	1*	–	–	–	–	N	P	R	125
	Pomacentru amboinensus	nat	P	B	1	–	1	1*	–	N	R	A	128
	Thalassoma hardwicke	nat	P	J	1*	–	–	–	–	N	–	RA	129
Total studies					6	2	4	2	–				
Significant effects					5	2	1	2	–				
Percent significant effects				83.3	100.0	5	2	–					
Small mammals													
	Microtus pennsylvanicus	nat	P	J	2	1	1*	1*	1*	N	R	A	130,131
	Oryctolagus cuniculus	nat	P	J	1*	1*	–	–	–	N	–	A	132
	Spermophilus armatus	nat	P	B	2*	2*	–	–	2*	N[99] Y (F, JS,AS)[68]	–	A[99] S, LTRE[68]	68,99
	Spermophilus parryii	nat	R	–	–	1*	–	–	1*	N	R	A	133
	Spermophilus columbianus	nat	R	–	1,1*	1,1*	–	1*	2*	Y (RS, JS, AS)[97] Y (F, AGE-MAT)[98]	R	M[97] S, LTRE[98]	97,98
	Tamiasciurus hudsonicus	nat	R	–	1*	1	1	–	–	N	R	A	134

Continued

Table 2. *Continued*

Taxonomic category or summary statistic	Species	Setting	Manipulation type: Pop/Res	Manipulation type: Age-class	Response variables: JS	AS	GR	AMAT/SMAT/SMET	F or RS	Rank	Ecological factor	Analysis type	Ref
Standard analyses													
Total studies					6	6	2	1	4				
Significant effects					4	4	1	1	4				
Percent significant effects					66.7	66.7	50.0	100.0	100.0				
LTRE analyses													
Total studies					2	2	–	1	2				
Major contributions					1	1	–	1	2				
Percent significant effects					50.0	50.0	–	100.0	100.0				
Stream fishes													
	Salmo trutta	nat	P	A	–	1	1*	–	–	N	R	A	135
Passerine birds													
	Dendroica caerulescens	nat	P	A	1	1	–	–	2*	N	R	A	136,137
	Ficedula albicollis	nat	P	A	–	–	–	–	1*	N	R	t-tests	138
	Parus major	nat	P	A	1*	1*	1*	–	1	N	–	A	139
Reptiles													
	Lacerta vivipara	nat	P	B	1*	1	1*	–	1*	N	–	A	140
	Urosausus ornatus	nat	P	A	–	–	1*	–	–	N	R	A	141
Large mammals													
	Equus asinus	nat	P	B	1*	1	1*	1*	1	N	R	A	142
	Odocoileus hemionus	nat	P	B	1*	–	1*	–	–	N	R	A	143
	Ovis canadensis	nat	P	A	–	1	–	–	1*	N	–	A	144
Overall Standard Analyses													
Total studies					26	15	21	14	12				
Significant effects					19	8	16	12	10				
Percent significant effects					73.1	53.3	76.2	85.7	83.3				
Overall LTRE analyses													
Total studies					2	2		1	2				
Major contributions				1	1		1	2					
Percent significant effects				50.0	50.0		100.0	100.0					

Notes: Studies were found using a literature search in ISI Web of Science but were supplemented by references to previous work in these studies that were prior to the date that ISI searches. Setting indicates if the experiment was conducted on natural populations (nat), in mesocosms (meso), or in the laboratory (lab). Two broad types of experiments were included: direct manipulations of density and indirect manipulations that altered resource availability. Population/Resources refers to studies where resources (R) were manipulated and (P) to where one or more of the classes of the population were directly manipulated. Age-class refers to populations where the juvenile (J), adult (A), or both (B) ages were manipulated. Studies varied in the demographic response variables measured: juvenile survival (JS), adult survival (AS), growth (GR), age/size at maturity or metamorphosis (AMAT/SMAT/SMET.), fecundity or reproductive success (F or RS). Numbers indicate the number of studies reporting measurement of demographic variable. All studies used hypothesis testing to test the null that demographic parameters did not differ between manipulated and unmanipulated populations. Asterisks indicate where significant differences were detected. All differences were in the direction of negative density dependence. Ranking indicates whether studies attempted to assign importance to parameters that influence population growth rate, with the order of importance in parentheses. Responsible ecological interaction indicates where studies were able to ascribe demographic responses to either predation (P) or resources (R). Analysis type represents the analytic techniques used to evaluate the treatment effects: A refers to general inferential statistical techniques (e.g., ANOVA/ANCOVA); R refers to regression and correlation analyses; S refers to prospective demographic analyses, including sensitivity and elasticity analyses of static life tables; M refers to a mixture of standard inferential statistical techniques; and LTRE refers to retrospective demographic analyses. Summary statistics for standard analyses and LTRE are given for taxonomic categories with more than five studies. Overall summary statistics are given for all taxonomic categories combined.

using LTRE techniques[97] was reanalyzed using these methods.[98] Dobson manipulated food resources in populations of Columbian ground squirrels (*Spermophilus columbianus*) to test the hypothesis that food resource availability was responsible for regulating population size. Populations with food manipulations were followed for 3 years during the treatment and 3 years posttreatment and subsequently compared to geographically paired populations that did not receive supplemental food. The original study found that populations of ground squirrels that received supplemental food dramatically increased in numbers, then the population declined to the original numbers when the food was removed. Demographic factors that were thought to have the largest effect on the growth of the population were based on effect sizes and included the number and survival of offspring followed by a smaller effect of resident adult survival.

Dobson and Oli[98] reanalyzed this previous study using LTRE techniques and, in addition, contrasted the results with results obtained from static life-table data from these populations. Thus, the authors used both prospective (sensitivity and elasticity) and retrospective (LTRE) analyses to examine which demographic potentially and actually had the greatest effect on population growth. Prospective analyses of the sensitivity of population growth rate to changes in demographic variables suggested and LTRE analysis confirmed that increases and decreases in population size were mostly caused by changes in fertility rate of females and age at maturity. In the original and later analyses increases in population growth were primarily caused by changes in female fecundity. Traits that yielded secondary and tertiary effects differed between the analyses—juvenile and adult survival had the next largest effects in the original analysis whereas age at maturity made the next largest contribution in the LTRE analysis. The LTRE analysis thus yields different answers from the more traditional analyses of variance.

The only other paper that used LTRE methods to identify traits that contributed to differences in population growth was also a reanalysis of an earlier study. Oli *et al.*[68] reanalyzed a previously published study on a density manipulation of Uinta ground squirrels (*Spermophilus armatus*) by Slade and Balph[99] using LTRE analytic techniques. The original analysis of the data, using standard inferential statistical techniques, showed that juvenile survival and dispersal likely played a key role in the regulation of the populations. However, subsequent LTRE analysis revealed that changes in fertility contributed most to changes in population growth rate after the density manipulation. This result was obtained even though the original analysis failed to find statistically significant differences in fertility between treatments. This example highlights that demographic traits that are important to changes in population growth may not always exhibit statistically significant differences when analyzed using traditional statistical techniques, although the results of the LTRE analysis demonstrate that the differences are biologically significant. Furthermore relying on traditional statistical techniques may give the wrong answer to the question of which traits are important to the regulation of populations. Several of the studies in our review were conducted before matrix population analyses such as LTRE were available 21.5% (8 of 38). However, the efforts of Oli and Dobson and Oli *et al*. argue for the careful interpretation of these data and that a reanalysis of these data sets would be useful.

If we consider what is necessary for studies of density regulation to be of value to evolutionary biologists, we are left with a very small subset of the empirical literature on density regulation. What we can infer from our review is that demographic rates of populations do respond to externally imposed changes in density. Further, in those studies that measure more than one demographic trait, it is usually the case that more than one demographic rate is influenced by the manipulation. However, our review of the literature suggests that there is little data from which we can decompose actual changes in population growth into contributions from individual traits. Thus our understanding of the demographic mechanisms (i.e., traits) that are the most influential to changes in population growth is limited, at least for the vertebrate studies reviewed here. Consequentially, at the present time there is no bridge between the empirical literature on population regulation and evolutionary life-history theory.

How to build a bridge between ecology and evolution

Dobzhansky is famously quoted as saying "Nothing in biology makes sense except in the light of evolution."[100] However, as our brief review of

life-history theory should illustrate, nothing in evolution makes sense except in the context of ecology. With this in mind, building a bridge between ecological and evolutionary theory will require integrative studies that synergistically integrate traditional studies of population dynamics with evolutionary studies.

The importance of understanding how these two fields interact can be illustrated by our own studies of life-history evolution in the Trinidadian guppies (*Poecilia reticulata*). Our work on guppies has focused on understanding the ecological factors (e.g., predation) that have shaped the evolution of the life history and has not addressed the population dynamics of the populations. Guppies inhabit two distinct types of communities in the streams that drain the slopes of the northern mountain range in Trinidad, West Indies. Lower order headwater streams contain guppies and killifish (*Rivulus hartii*) and higher order, lowland streams contain the same species as headwater streams but also contain fish species such as the pike cichlid (*Crenicichla alta*, Cichlidae), blue acaras (*Aequidens pulcher,* Cichlidae), characins (*Astyanax bimaculatus, Hemibrycon dentatum*), and the wolffish (*Hoplias malabaricus*) that may be predators of guppies.[91,101,102] Between these extremes sometimes lies a gradient of communities with a reduced number of predator species.[101] Predators from higher order streams are prevented from invading headwater streams by barrier waterfalls. Comparative studies between high and low predation sites across multiple, independent drainages in both field and common garden experiments have demonstrated that guppies from these two community types demonstrate genetically based, repeatable patterns of variation in life-history traits.[90,91] Introduction experiments, wherein guppies from high predation locations are transplanted to low predation areas that previously lacked guppies have shown that the evolution of the low predation genotype evolves very rapidly (4 years or less for some traits).[92]

We had previously assumed that guppies from lowland streams experienced higher adult mortality due to the presence of the *C. alta* and guppies from headwater streams experienced higher juvenile mortality due to the presence of *R. hartii*. We made these assumptions based on results from behavioral choice experiments on *C. alta* and *R. hartii* that show when given the choice they prey on larger and smaller size guppies, respectively.[102,103] These differences in preference provided the differences in age/size specific mortality required by density-independent theory to cause evolutionary shifts in demographic traits (Fig. 1). However, more recent empirical mark-recapture[93] and laboratory assessment[104] of size-specific mortality between these population types has revealed that the size-specific nature of mortality rates are not what was previously assumed. Specifically, populations that occur with a larger suite of predators exhibit an overall increase in mortality across all size classes. Under density-independent demographic life-history theory, such a change in mortality between populations should not result in evolutionary changes in the demography observed between these two population types[23,24,52,105,106] (Fig. 1). However, these traits do evolve, so we can at least conclude that our understanding of how mortality shapes the evolution of these traits is incomplete.[93,107]

Demographic adjustment of vital rates due to density-dependent population regulation, indirect ecological effects of predators, and eco-evo feedbacks may play a role in shaping how the life history evolves between these two population types. For example, if guppies were unregulated, a uniform increase in mortality across age classes is predicted to result in no evolution of age at maturity or reproductive effort (Fig. 1D). However, if guppy populations are regulated through changes in juvenile survival or adult fecundity, a uniform change in mortality across age classes is predicted to decrease age at maturity and increase reproductive effort (Fig. 1E).[19,25]

Ecological studies such as the experimental manipulations of Columbian ground squirrels[97,98] discussed earlier can provide the answers about the demographic variables. In this study, food resources of two populations across an elevation gradient were manipulated. Populations were found to be regulated by food resources and demographic mechanisms of regulation were identified. Interestingly, the control groups of these two population types exhibited differences in adult and juvenile survival probabilities. The low elevation populations had lower survival probabilities for both adults and juveniles. This pattern of mortality differences between populations is similar to what we find for guppies.

In both cases, classic demographic models of life-history evolution, which use the intrinsic rate of increase as a measure of fitness, predict no changes in the age at maturity or reproductive effort. However, in squirrels, like guppies, the controls for lower elevation populations exhibited younger age at maturity and higher fertility rates compared to the high-elevation populations. These two population types displayed relatively stable populations during the course of the study ($\lambda \approx 1$).

The parallels between ground squirrels and guppies offers insights into how the studies of population regulation and life-history evolution can be integrated. Demographic theory following Michod[19] shows that when density regulation is integrated into the life-history models the changes in the ground squirrels can be understood (Fig. 1). In this case fecundity was the demographic parameter that showed the largest contribution, indicating that regulation was mostly caused by changes in fecundity. Currently it is unclear if regulation can explain this discrepancy, but there is some indication that regulation via changes in fecundity should be the same as regulation through juvenile survival.[52] If so, then this pattern of demographic adjustment of the vital rates could explain the differences in life histories among these populations. For guppies, currently we do not know if and how guppy populations are regulated, though preliminary density manipulations suggest that they indeed are regulated (Bassar and Reznick, unpublished data).

Alternatively, these data can inform how the indirect effects of predation influence the evolutionary processes. We have shown that guppy populations from low predation environments have high population densities, measured as biomass per unit area or volume of water[108] and that their populations are dominated by older, larger fish, relative to populations from high predation localities. These differences can be attributed to the lower birth and mortality rates in low predation environments, which means that they are the product of a combination of the evolved differences in life histories and the differences in mortality risk.[109] The guppies from low predation environments also have lower somatic growth rates, which possibly reflects lower per capita food availability.[108] This pattern of responses suggests that predators may have shaped guppy life-history evolution in part via their direct effect on guppy mortality rates and in part by their indirect effect on guppy population density. We have a parallel research program on *Rivulus hartii* that makes a strong argument for the evolutionary consequences of such indirect effects of predators on the evolution of life histories in *R. hartii*.[110]

Conclusions

Density regulation has two distinct lives in the scientific literature. Its first life was to be one side of one of the oldest debates in ecology, which addresses how the abundance of organisms in nature are regulated. Density regulation attained a second life when it was incorporated into evolutionary theory, largely to address the factors that shape life-history evolution. Density regulation was incorporated into evolutionary theory to make it more realistic; it is more realistic to assume that populations cannot grow forever without bound. Adding density regulation to evolutionary theory meant changing our definition of fitness and explicitly modeling both the ecological and evolutionary dynamics. Theory reveals that the demographic mechanisms that underlay density regulation play a critical role in defining the life history that should evolve. This necessity is apparent whether one is interested in defining how the life history should evolve in either a strictly evolutionary optimality model or in an eco-evo feedback model. Thus, regardless of the methodology, knowledge of the regulatory mechanisms in a population is critical to predicting the life history that should evolve. A direct effect of predators is to kill prey, but they also reduce the density of the prey, and the resources of the remaining prey are indirectly influenced by predation. Traditional approaches address the direct action of the predator but ignore other ecological changes that occur in conjuction with the change in mortality regime. Incorporating density regulation into theory incorporates the demographic effect of these indirect ecological effects. However, how the ecological origin of these changes (predation or resource availability) influences the evolution of the life history is relatively unknown.[23,24] These indirect ecological effects are rarely studied in evolutionary biology but comprise a vast literature in ecology.

Ultimately, our ability to tackle each of these questions depends on our knowledge of how variation in population density affects traits that underlay the vital rates of all individuals in the population. This

question has traditionally been addressed by population ecologists who were interested in whether population regulation is density dependent or independent. Though much has been learned through theoretical and empirical research we have found that there is actually very little experimental data that describes how individual life stages respond demographically to variation in density in natural populations. This is perhaps because few study systems offer the opportunity to manipulate and study such demographic changes simultaneously in all life stages in a single experiment. However, if we are to make progress in bridging the gap between empirical ecology and evolutionary theory we must advance past simple studies of manipulating only a subset of the population and using *F*-tests to assign statistical significance. We must instead focus on studies that inform us about how variation in the density of the population influences the demography of the entire population. Ecological studies of population dynamics and evolutionary studies of the life histories are related but have proceeded largely in ignorance of each other. Future collaborations between population ecologists and evolutionary biologists on the same study systems could provide the cross-fostering needed to integrate these two fields.

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Conflicts of interest

The authors declare no conflicts of interest.

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