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Population Cycles Caused by Selection by Density Dependent Competitive Interactions

LARS WITTING

Greenland Institute of Natural Resources,
P. O. Box 570,
DK-3900 Nuuk, Greenland
E-mail: larsw@natur.gl

Several animal species have cyclic population dynamics with phase-related cycles in life history traits such as body mass, reproductive rate, and pre-reproductive period. Although many mechanisms have been proposed there is no agreement on the cause of these cycles, and no population equation that deduces both the abundance and the life history cycles from basic ecological constraints has been formulated. Here I deduce a population dynamic equation from the selection pressure of density dependent competitive interactions in order to explain the cyclic dynamics in abundance and life history traits. The model can explain cycles by evolutionary changes in the genotype or by plastic responses in the phenotype. It treats the population dynamic growth rate as an initial condition, and its density independent fundament is Fisher's (1930, *The Genetical Theory of Natural Selection*, Oxford: Clarendon) fundamental theorem of natural selection that predicts a hyper-geometrical increase in abundance. The predicted periods coincide with the cyclic dynamics of Lepidoptera, and the Calder hypothesis, which suggests that the period of population cycles is proportional to the $1/4$ power of body mass, follows from first principles of the proposed density dependent ecology.

1. INTRODUCTION

The widespread occurrence of cycles in the abundance and life history characters of animal species has remained an unsolved problem in animal ecology [e.g., Boonstra and Krebs (1979), Lidicker and Ostfeld (1991), Stenseth and Ims (1993), Chitty (1996) and Krebs (1996)]. According to classical population dynamic theory, which is based on density regulation and Malthusian growth for populations in density independent environments, population cycles are the exception that require some sort of delayed density dependence. At present at least 22 mechanisms have been proposed to explain delayed density dependence and the cyclic dynamics (Batzli, 1996), but no clear conclusions have been made. The proposed hypotheses have been based on mechanisms extrinsic or intrinsic to the population, with the most widespread extrinsic hypothesis being predator–prey interactions in their broadest definition that include both host–parasitoid and plant–herbivore interac-

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tions. The most widespread intrinsic hypotheses are those of over-compensation and density dependent changes in individual quality.

Predator–prey interactions may be the most popular hypothesis for population cycles in small mammals and insects [e.g., Akçakaya (1992), Hanski *et al*. (1993), Hanski and Korpimäki (1995), Krebs *et al*. (1995), Norrdahl (1995), Berryman (1996), Jedrzejewski and Jedrzejewski (1996) and Turchin *et al*. (2000)]. Despite the extensive research effort devoted to this hypothesis, Berryman (1996) notes that the best documented cycle in insects lacks a firm predator–prey interaction. And Krebs (1996) concludes that 'Changes in the food supply have never been shown to be necessary or by itself sufficient to cause cycles', and that 'there is not a single field experiment to show that predator removal has any impact on cycles of voles or lemmings.' Especially the phase-related cycles in life history traits are not expected from the extrinsic hypotheses unless, of course, they are induced by another mechanism and follow as a consequence and not a cause of cyclic population dynamics (Lidicker and Ostfeld, 1991; Oksanen and Lundberg, 1995; Oli, 1999).

The intrinsic hypothesis of over-compensatory dynamics has been studied theoretically by May and Oster (1976), Sandefur (1990), and Strogatz (1994). This mechanism can explain periods for up to two generations [e.g., Tuljapurkar *et al*. (1994)], and over-compensatory dynamics have been found in semi natural populations as diverse as ungulates (Grenfell *et al*., 1992) and insects (Hassell *et al*., 1976; Gurney *et al*., 1980; Desharnais and Liu, 1987; Costantino *et al*., 1995). But it is generally agreed that over-compensation cannot explain the general tendency for cyclic dynamics with periods of more than two generations (Turchin, 1990; Witteman *et al*., 1990; Turchin and Taylor, 1992; Ginzburg and Taneyhill, 1994).

It has often been suggested that other intrinsic mechanisms that are based on density dependent changes in individual quality can explain population cycles with periods of more than two generations. These intrinsic hypotheses include changes caused by physiological (Christian, 1950; Boonstra, 1994), selectional (Chitty, 1960, 1967), behavioural (Charnov and Finnerty, 1980), and maternal effect (Wellington, 1965) mechanisms. Compared with the extrinsic hypotheses the intrinsic models may seem superior in the way that they tend to incorporate the phase-related changes in life history traits. But comparing the different intrinsic models Stenseth (1981, 1985, 1995) concludes that they generally fail to explain the occurrence of population cycles and/or that they are burdened by unrealistic assumptions.

It has been argued that the selection mechanism is experimentally rejected because the level of heritable variation seems to be so low that evolutionary changes in the genotype cannot cause cyclic population dynamics (Boonstra and Boag, 1987; Boonstra and Hochachka, 1997). However, the phenotypic response to selection may not only arise from evolutionary changes in the genotype. The phenotype may also change due to inherited environmental effects, where an epigenetic inheritance system transfers a plastic phenotypic response from a parent to an offspring

generation [e.g., Jablonka and Lamb (1998, 1989) and Rossiter (1996)]. Maternal effect is one of many possible inherited environmental effects, and the maternal effect hypothesis for population cycles has recently received much attention [e.g., Rossiter (1991, 1992, 1994), Ginzburg and Taneyhill (1994, 1995), Boonstra and Hochachka (1997), Ginzburg (1998) and Inchausti and Ginzburg (1998)]. Compared with earlier versions of the intrinsic hypotheses, the maternal effect hypothesis is more promising because it is now formulated into a population equation with dynamics that resemble the cycles of Lepidoptera and small mammals (Ginzburg and Taneyhill, 1994, 1995; Ginzburg, 1998; Inchausti and Ginzburg, 1998). Although it has not been shown empirically whether cyclic and density dependent changes in inherited environmental effects are generating population cycles in natural populations, the models of Ginzburg and Taneyhill (1994, 1995) and Inchausti and Ginzburg (1998) show that it is a plausible hypothesis. Their studies, however, do not suggest from what ecological factors the plastic phenotypic response arises. From a mechanistic point of view it is the density dependent factor that induces selection for between-generation changes in life history parameters that is the ultimate factor that may explain cyclic population dynamics by density dependent changes in individual quality. On a longer evolutionary time scale such factors can induce the evolution of an inheritable phenotypic response, especially if the density dependent environment fluctuates with a period that exceeds the generation time of the organism (Lachmann and Jablonka, 1996). And if the potential for an inheritable phenotypic response has evolved by these means, then it is the density dependent changes in the same ecological factor that is most likely to induce a plastic between-generation response.

To fully understand the potential for cyclic dynamics by density dependent changes in individual quality we need first to identify a density dependent factor that selects for cyclic and density dependent changes in individual quality and population abundance. But despite several attempts [e.g., Dekker (1975), Stenseth (1978, 1981), Thue Poulsen (1979) and Hunt (1982)], the original selection hypothesis of Chitty was never formulated into a realistic population dynamic equation (Stenseth, 1981, 1985, 1995). In this paper I show that selection by the density dependent competitive interactions among individuals in populations can be the ultimate ecological factor behind cyclic animal dynamics. This is achieved by a selection model where the across-generation response in population parameters to the selection pressure of density dependent competitive interactions can arise in two ways. The first is a plastic response where an epigenetic inheritance system adjusts the phenotype to the selection pressure of competitive interactions. The second is evolution by natural selection as originally proposed by Chitty (1960). Provided that there is a sufficiently high level of additive genetic variation in population parameters, it is shown that selection by density dependent competitive interactions can generate population cycles in the absence of a plastic phenotypic response. If instead the level of genetic variation is negligible, a plastic response is needed before the cycle may arise.

1.1. ***Selection by density dependent competitive interactions.*** The model in this paper is part of a larger theory where the selection pressure of density dependent competitive interactions is used to deduce major life history transitions in mobile organisms (Witting, 1995, 1997, 2000). The theory deals with the evolution of life history characters such as reproductive rate, metabolic rate, body mass, exponents of body mass allometries, senescence, sex ratios, and sexual and eusocial reproduction. In this paper I examine the population dynamics around an evolutionary equilibrium of this theory. This is achieved by considering the effects that selection by density dependent competitive interactions have on traits such as body mass and the population dynamic growth rate. The implications of a theoretical deduction of the exponents of 10 body mass allometries (Witting, 1995, 1998) is also considered in the sense that the Calder hypothesis (Calder, 1983, 1984), which states that the period of cyclic dynamics is proportional to the $1/4$ power of body mass, is deduced from first principles of the proposed density dependent ecology.

Classical life history models [reviewed by Roff (1992), Stearns (1992), Charnov (1993), Bulmer (1994), Charlesworth (1994) and Kozłowski (1999)], including models of density dependent selection [reviewed Mueller (1997)], were developed independently of competitive interactions and frequency dependent selection. Selection by competitive interactions has instead been described under the concepts of game theory and evolutionary stable strategies (ESSs) (Maynard Smith and Price, 1973; Axelrod and Hamilton, 1981; Maynard Smith, 1982; Vincent and Brown, 1988; Vega-Redondo, 1996; Dugatkin and Reeve, 1998; Hofbauer and Sigmund, 1998), with more recent considerations on evolutionary convergence (Eshel, 1983; Taylor, 1989; Christiansen, 1991) and evolutionary branching (Metz *et al*., 1992, 1996; Dieckmann, 1997; Eshel *et al*., 1997; Gertiz *et al*., 1997, 1998; Kisdi, 1999). Like most of this work I deal with the evolution of traits that indicate the competitive ability of the organism.

To understand how selection by density dependent competitive interactions can generate cyclic dynamics consider first selection by the classical life history theory that disregard the effects of competitive interactions. This theory defines fitness as the intrinsic population dynamic growth rate, also known as the intrinsic Malthusian parameter. Dependent upon the density dependent state of the population the classical theory is often understood in terms of either r- or k-selection, where, respectively, the maximal population dynamic growth rate and/or the carrying capacity increases. More generally, however, as predicted by Fisher's fundamental theorem of natural selection (Fisher, 1930; Price, 1972; Witting, 2000), classical life history selection is given by an absolute increase in the intrinsic growth rate (this increase is also known as a partial increase in the absolute growth rate). Thus, as competitive traits tend to be related to the intrinsic growth rate by an energetic trade-off (Witting, 2000), from the classical theory we expect a continuous decline in competitive traits independently of the density dependent ecology. This prediction will not generate cyclic dynamics that require that the direction of the evolutionary changes in the intrinsic growth rate is density dependent.

With density dependent competitive interactions there are instead two opposing forces of selection on the intrinsic growth rate and the competitive traits. The first is classical r- and k-selection that favours the intrinsic growth rate at the cost of the competitive traits. The second is the selection pressure of density dependent competitive interactions that favours the competitive traits at the cost of the intrinsic growth rate. If the population abundance and, thus, the level of interference competition is sufficiently high, the latter force is stronger than the classical force resulting in overall selection for an increase in the competitive traits at the cost of the intrinsic growth rate. If instead the population abundance and the level of interference are sufficiently low, the force of r- and k-selection will dominate and the intrinsic growth rate will increase. At the evolutionarily determined population dynamic equilibrium the population abundance is exactly so high that the two selection forces are balanced against one another generating no overall selection. However, if the population is not at equilibrium the density dependent selection will generate across-generation changes in the population dynamic growth rate. And dependent upon the magnitude of the population's response to selection this form of delayed density dependence may generate cyclic population dynamics.

It has earlier been shown that selection by competitive interactions may induce evolutionarily driven population dynamic cycles for the case of density independent competitive interactions (Maynard Smith and Brown, 1986; Härdling, 1999). This is possible when evolution is mutation limited in the sense that mutations occur so infrequently that a mutant is excluded or spread to equilibrium before a new mutant arises. In this case a large variant may be invaded by extreme small mutants that do not experience the cost of a large size. The small mutant may then spread to fixation so that the size of the organism can increase until a new invasion by a small mutant is possible. These cycles, however, may not be applicable to higher organisms. Invasions by extreme types are generally not possible with sexual reproduction and quantitative inheritance where a unimodal phenotypic distribution tends to be maintained (Matsuda and Abrams, 1994). A period of approximately 4000 generations, as predicted by Härdling (1999), is also far beyond the periods usually considered in connection with cyclic population dynamics. The population cycles predicted in the present paper can have much faster periods and they are applicable to higher organisms in the sense that they operate through evolutionary cycles in the mean of unimodal phenotypic distributions.

2. THE MODEL

In the absence of selection the density dependent dynamics of a species with non-overlapping generations can be described as

$$\tilde{N}_{t+1} = \tilde{N}_t \lambda_m f(\tilde{N}_t) \tag{1}$$

where λ_m is the maximal per generation growth rate, $\tilde{N}$ is the population abundance that is larger than or equal to one for all extant populations, and $f(\tilde{N})$ is the density regulation function that declines monotonically from one to zero as the abundance increases from one to infinity. Instead of representing density regulation by the general model of equation (1), let me focus on the process that is linear at the logarithmic scale of population dynamics. The density regulation function can then be defined as

$$f(\tilde{N}) = \begin{cases} \nu \tilde{N}^{-\gamma} & \text{if } \tilde{N} > \dot{\tilde{N}} \\ 1 & \text{if } \tilde{N} < \dot{\tilde{N}} \end{cases} \tag{2}$$

where $\dot{\tilde{N}} = 1$ is the abundance where the effects of density regulation vanish, γ is the parameter that defines the curvature of the density regulation function, and $\nu = \dot{\tilde{N}}^{\gamma}$. Thus, if the population abundance is scaled as $N = \tilde{N}\nu^{-1/\gamma}$, and if we assume that $\tilde{N} \geq \dot{\tilde{N}}$, which is reasonable as long as we consider only local perturbations of the population equilibrium, we obtain the following population dynamic equation

$$N_{t+1} = N_t \lambda_m N_t^{-\gamma} \tag{3}$$

that forms the basis of my study. Let, for this model, the density regulation parameter γ be defined as $\gamma = \gamma_\alpha + \mu\gamma_\iota$, where γ_α is density regulation by resource exploitation, γ_ι is density dependence in the level of interference competition, and μ is the cost per unit interference.

The model of equation (3) is based on the assumption that the maximal growth rate (λ_m) is constant. But due to the action of selection we expect that the maximal growth rate is both time and density dependent. To describe the expected changes in λ_m and their implications for population dynamics I partition the phenotype into demographic and competitive traits. Let the demographic traits be lifetime reproduction (R) and the probability that an offspring survives to reproduce (p), which define the population dynamic growth rate $\lambda = pR$. And let the competitive traits be traits that individuals, or variants, can use to dominate other individuals, or variants, during competitive encounters.

A common feature of the competitive traits is that they are connected to the population dynamic growth rate by a trade-off. Let $q = wBT_p$ represent competitive quality as defined by the product between the body mass (w), the metabolic rate per unit body mass (B), and the pre-reproductive period (T_p). The body mass can be a competitive trait because it can be selected so that the individuals with the larger body masses can dominate the smaller individuals during competitive encounters. The same is true for the metabolic rate where the individual with the highest metabolic rate has the potential to allocate the largest amount of energy into the competitive encounter. The pre-reproductive period may also represent competitive quality because it is during this period that the offspring has the time to learn the behaviour that is needed to dominate other individuals. The idea of treating these traits as competitive traits is not to suggest that the traits might not

have other functions in natural organisms. It is only to show that the evolution of these traits, and their association with population dynamics, is relatively easily explained by density dependent competitive interactions.

If we assume that the offspring are reared by the parents the competitive quality q may also be interpreted as the energy that the parents invest per offspring. Actually this energy is better described as $w + q$ because q represents only the energy that the offspring metabolizes during the pre-reproductive period, while the energy invested in an offspring also includes the energy contained in the body mass of that offspring. But, for the sake of simplicity I assume that q resembles the energy invested per offspring. Thus, we expect that

$$\lambda_m = \rho/q \tag{4}$$

where $\rho = p\epsilon$ is the product between the average probability p that an offspring will survive and reproduce and the average amount of energy ϵ that an individual on an unexploited resource allocates to reproduction. With ϵ amounts of energy allocated to reproduction ϵ/q offspring can be produced. Thus, the organism can choose to allocate resource either to the competitive quality q or to the population dynamic growth rate λ. In this paper I assume that the selection pressure on the three traits w, B, and T_p is similar so that I can model the evolutionary changes in the three traits by considering only the evolutionary changes in q. I will also assume that there are no evolutionary changes in p and ϵ so that the evolutionary changes in λ_m can be described by the evolutionary changes in q.

2.1. ***Evolutionary changes.*** To describe the evolutionary changes mathematically note, from equations (3) and (4), that the population dynamic growth rate of the tth generation is

$$\lambda_t = \rho N_t^{-\gamma}/q_t \tag{5}$$

assuming that ρ is constant. The action of density dependent competitive interactions implies that the density regulation parameter γ is different for the different individuals in the population. The differentiation in γ arises because the competitively superior individuals dominate the competitively inferior individuals during competitive encounters. The competitively superior individuals will thus experience a lower cost of interference than the competitively inferior individuals. If we assume that the individuals encounter one another at random, the cost per unit interference (μ) of a given individual can be defined as a function of the competitive quality of that individual relative to the average competitive quality in the population. Hence, the density regulation parameter of the ith variant is $\gamma_i = \gamma_\alpha + \gamma_\iota(\mu + \Delta\mu_i)$, where $\Delta\mu_i = \mu_i - \mu$. If we assume that the cost of interference is a linear function on logarithmic scale it follows that $\Delta\mu_i = \psi(\ln q - \ln q_i)$, where ψ is the within-population slope between the cost of interference competition and ln competitive quality. Thus, the growth rate of the ith variant is

$$\lambda_{i,t} = \rho N_t^{-\gamma_i}/q_{i,t}$$

$$
\begin{aligned}
&= \lambda_t(\rho N_t^{-\gamma_i}/q_{i,t})/(\rho N_t^{-\gamma}/q_t) \\
&= \lambda_t(q_t/q_{i,t})N_t^{\gamma-\gamma_i} \\
&= \lambda_t(q_t/q_{i,t})N_t^{\gamma_i\psi(\ln q_{i,t}-\ln q_t)} \qquad (6)
\end{aligned}
$$

which can be rearranged to

$$\lambda_{i,t} = \lambda_t e^{(\gamma_i\psi \ln N_t - 1)(\ln q_{i,t}-\ln q_t)}. \qquad (7)$$

Differentiating with respect to $\ln q_{i,t}$, and letting $q_{i,t}\to q_t$, the per generation selection gradient on $\ln q$ is

$$\partial\lambda_{i,t}/\partial \ln q_{i,t}|_{q_{i,t}=q_t} = \lambda_t(\gamma_i\psi \ln N_t - 1) \qquad (8)$$

where $|_{q_{i,t}=q_t}$ indicates that the derivative is to be taken at the limit $q_{i,t} = q_t$. Solving for the evolutionary equilibrium we find that $N^{**} = e^{1/\gamma_i\psi}$, with $**$ denoting the evolutionary equilibrium. Combining this abundance with equations (3) and (4) it follows that the evolutionary equilibrium is characterized as

$$
\begin{aligned}
\lambda = \lambda_m^{**} N^{**-\gamma} &= 1 \\
N^{**} &= \sqrt[\gamma]{\lambda_m^{**}} \\
N^{**} &= e^{1/\gamma_i\psi} \\
\lambda_m^{**} &= e^{\gamma/\gamma_i\psi} \\
q^{**} &= \rho e^{-\gamma/\gamma_i\psi}. \qquad (9)
\end{aligned}
$$

To predict the per generation change in the growth rate λ_m, let $\sigma^2 = \hat{\sigma}^2 + \tilde{\sigma}^2$ represents the potential by which $\ln q$ responds to selection, where $\hat{\sigma}^2$ is the additive genetic variance in the Malthusian parameter $r = \ln\lambda_m$ and $\ln q$ (this variance is the same in the model because of the energetic trade-off between r and q) and $\tilde{\sigma}^2$ is a plastic response of inherited environmental effects. Assume also that the plastic response to selection can be modelled as the response of quantitative genetics, which implies that it operates by adjusting the phenotype of the offspring to the selection pressure experienced by the parents. Following Robertson (1968), Charlesworth (1990), Iwasa *et al.* (1991), Taper and Case (1992), Abrams *et al.* (1993) and Taylor (1996), the per generation change in $\ln q$ can then be approximated as

$$
\begin{aligned}
\Delta \ln q_t &= \frac{\sigma^2}{\lambda_t}\frac{\partial\lambda_{i,t}}{\partial \ln q_{i,t}}\bigg|_{q_{i,t}=q_t} \\
&= \sigma^2(\gamma_i\psi \ln N_t - 1). \qquad (10)
\end{aligned}
$$

From equation (4) note that $r = \ln \lambda_m = \ln \rho - \ln q$. Hence, with a constant ρ, σ^2 also denotes the potential response of the growth rate r to natural selection.

From equation (10) and $\ln q_t = \ln q_{t-1} + \Delta \ln q_{t-1}$, the average quality at time t is

$$q_t = q_{t-1} N_{t-1}^{\gamma_t \psi \sigma^2} e^{-\sigma^2} \tag{11}$$

and from equations (4) and (11), the maximal growth rate in generation t is

$$\begin{aligned} \lambda_{m,t} &= \rho N_{t-1}^{-\gamma_t \psi \sigma^2} e^{\sigma^2} / q_{t-1} \\ &= \lambda_{m,t-1} N_{t-1}^{-\gamma_q} e^{\sigma^2} \end{aligned} \tag{12}$$

with $\gamma_q = \gamma_t \psi \sigma^2$. Then, from equations (3) and (12), the population dynamic equation with density dependent selection on competitive quality is

$$\begin{aligned} \lambda_{m,t} &= \lambda_{m,t-1} N_{t-1}^{-\gamma_q} e^{\sigma^2} \\ N_{t+1} &= N_t \lambda_{m,t} N_t^{-\gamma}. \end{aligned} \tag{13}$$

This model reduces into the classical model of equation (3) when $\sigma^2 = 0$, and it treats the growth rate as an initial condition, which contrasts to classical models where the growth rate is a fixed parameter.

2.1.1. *Density independent environments.* For equation (13) it is the selection pressure of density dependent competitive interactions that generates the evolutionary balance between the competitive traits, the population dynamic growth rate, and the carrying capacity. This balance is lost if density regulation is absent. In this case $\gamma = 0$ and $\gamma_q = 0$ and, thus, from equations (11) and (13) we obtain

$$\begin{aligned} q_t &= q_0 e^{-\sigma^2 t} \\ \lambda_{m,t} &= \lambda_{m,0} e^{\sigma^2 t} \\ N_t &= N_0 \lambda_{m,0}^t e^{\sigma^2 \sum_{\tau=0}^{t} \tau} \end{aligned} \tag{14}$$

a situation with a geometrical decline in competitive quality, a geometrical increase in the maximal growth rate, and a hyper-geometrical increase in the abundance.

To deduce the law behind equation (14) recall that $r = \ln \lambda_m$, where r is the Malthusian parameter. Hence, from equation (14) we obtain

$$\begin{aligned} r_t &= r_0 + \sigma^2 t \\ dr/dt &= \sigma^2. \end{aligned} \tag{15}$$

For the case of a constant environment and $\tilde{\sigma}^2 = 0$, this equation represents Fisher's fundamental theorem of natural selection (Fisher, 1930) that predicts that

the partial increase in the Malthusian parameter due to natural selection is equal to the additive genetic variance in that parameter.

The observation that the fundamental theorem of natural selection may form the basis of population dynamics was probably first noted by Ginzburg (1980). From equations (14) and (15) we note that the fundamental theorem and the law of hyper-geometrical increase reduces into the Malthusian law of geometrical increase (Malthus, 1798) in the absence of both additive genetic variation and inherited environmental effects. A more detailed description of the relationship between the fundamental theorem and the selection pressure of density dependent competitive interactions is given by Witting (2000).

3. MODEL BEHAVIOUR

The model with no selection [equation (3)] returns monotonically to the equilibrium when $0 < \gamma \leq 1$, and it oscillates with a period of two generations when $\gamma > 1$. The oscillation is damped for $1 < \gamma < 2$, stable for $\gamma = 2$, and repelling or exploding for $\gamma > 2$. This contrasts to the model with density dependent selection [equation (13)], where the dynamics generally is cyclic with periods of more than two generations. The ultimate factor behind the cyclic dynamics of equation (13) is the selection pressure of density dependent competitive interactions, but the proximate factor is probably best understood in terms of a cyclic change in population equilibrium. For the model with selection the population equilibrium $N^* = \sqrt[\gamma]{\lambda_m}$ is defined as the equilibrium for a given λ_m and, thus, with cyclic- and selection-induced changes in λ_m there is a cycle in the population equilibrium. The evolutionary population equilibrium is the population equilibrium defined by the maximal growth rate at the evolutionary equilibrium, i.e., $N^{**} = \sqrt[\gamma]{\lambda_m^{**}}$, with double star denoting evolutionary equilibrium.

The connection between the dynamics of the population equilibrium and the dynamics of the abundance is illustrated in Fig. 1. Following a downward perturbation from the evolutionary equilibrium the density regulation is relaxed so that the reproductive rate rises and the abundance increases toward the evolutionary equilibrium. Associated with the relaxed density regulation there is a decline in the level of interference competition and this decline selects for an increase in the population dynamic growth rate at the cost of the competitive traits. Thus, when the abundance has reached the evolutionary equilibrium the population will no longer be in population equilibrium at that equilibrium because the increased growth rate has induced an increase in the population equilibrium. Consequently, the population will increase beyond the evolutionary equilibrium heading toward the new population equilibrium. As illustrated in Fig. 1, the population will never reach this equilibrium because as soon as the abundance increases beyond the evolutionary equilibrium the competitive interactions will select for an increase in the competitive traits at the cost of the population dynamic growth rate. Thus, the population

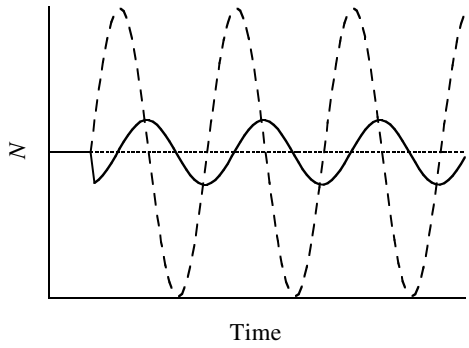

Figure 1. Projection of equation (13). The solid curve is the population abundance, the dashed curve the population equilibrium, and the dotted line the evolutionary equilibrium.

equilibrium evolves downward toward the increasing population.

At some intermediate abundance the downward evolving equilibrium and the upward increasing population will intercept, and the population will be in population equilibrium with no change in abundance. At this equilibrium the abundance is at its peak, and selection for competitive quality is at its strongest. Hence, the population equilibrium will continue to evolve downward with the result that the abundance will decline in order to keep up with the downward evolving equilibrium. In Fig. 1 the abundance cannot keep up with the equilibrium, and consequently the distance between the abundance and the equilibrium increases until the population intercepts the evolutionary equilibrium with the fastest decline in abundance that the population encounters during the cycle. Having crossed the evolutionary equilibrium selection reverses, so that energy is allocated from the competitive traits to the population dynamic growth rate, and the population equilibrium evolves upward toward the declining population. At some intermediate abundance the declining population intercepts the upward evolving equilibrium, the rate of change in abundance changes from negative to positive, and the next period begins.

The dynamics of equation (13) is analysed in Appendix A. The dynamics is generally cyclic with an either stable or unstable equilibrium. The equilibrium is unstable when $\gamma_q \geq \gamma$, or when $\gamma \geq 2$ and $\gamma_q \leq \gamma - 4$. The dynamics associated with the latter of these two criteria is given primarily by the over-compensatory density regulation that induces the oscillatory dynamics of equation (3). The result is oscillatory dynamics with a period of two generations. The dynamics associated with the former criterion is caused primarily by the evolutionary modulation of life history characters. This dynamics is cyclic with a highly variable period. The cycles are damped when $\gamma_q < \gamma$, stable when $\gamma_q = \gamma$, and repelling or exploding when $\gamma_q > \gamma$.

When the cyclic dynamics generated by density dependent selection is stable, i.e., when $\gamma_q = \gamma$, the period is determined by the γ parameter. In this case the period declines from an infinite number of generations for $\gamma = 0$ to two generations

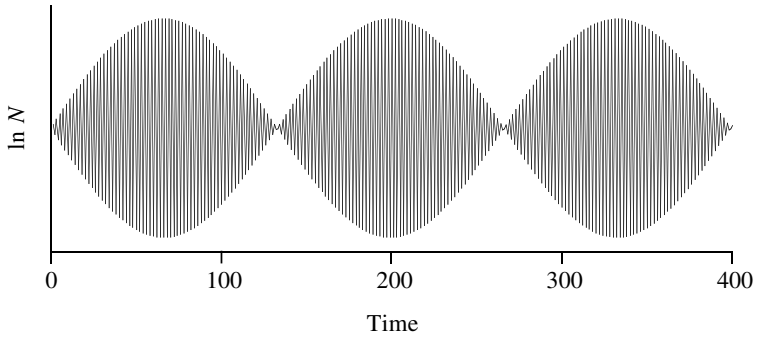

Figure 2. A projection of equation (13) showing the occurrence of a cycle in the amplitude of the population cycle. In the figure, the population cycle has a period of ≈2 generations, and the amplitude has a period of ≈133 generations.

for $\gamma = 4$. From equation (12) we have that $\gamma_q = \gamma_\iota \psi \sigma^2$. Thus, the population goes through faster cycles: (i) when the density dependence (γ and γ_q) increases; (ii) when the slope between the cost of interference and ln competitive quality (ψ) increases, and (iii) when the additive genetic variance ($\hat{\sigma}^2$) and/or the plastic response ($\tilde{\sigma}^2$) to selection increase.

In the case where $\gamma_q = \gamma$ and $3.5 \leq \gamma \leq 4$ there is a cycle in the amplitude of the population cycle, as illustrated in Fig. 2. On the continuum from $\gamma = 3.5$ to $\gamma = 4$ the population cycle is dominated increasingly by the cyclic change in amplitude. As $\gamma \to 4$ the period of the amplitude cycle continues to increase, from ≈10 generations for $\gamma = 3.9$ to an infinite number of generations at the limit $\gamma = 4$. Beyond $\gamma = 4$ the population period is two generations, while the amplitude increases geometrically.

Associated with the cyclic dynamics of equation (13) there are cyclic changes in both the maximal population dynamic growth rate and the competitive traits. These changes are described by equations (11) and (13), which predict that the maximal growth rate increases whenever the population abundance is below the abundance of the evolutionary equilibrium, and that the growth rate declines when the abundance is above the abundance at equilibrium. The reverse is true for competitive traits such as the body mass, the metabolic rate, and the pre-reproductive period, which are treated collectively by the competitive quality q. During the population cycle the competitive traits will have their maximal values when the abundance is crossing the population equilibrium from above, and they will take their minimal values when the abundance is crossing the equilibrium from below.

4. DISCUSSION

Even though the deduction presented here is a bit simplified, it clearly shows that density dependent competitive interactions select for density dependent changes in

individual quality and for cyclic population dynamics; a deduction that has been missing since the first selection hypothesis was proposed by Chitty (1960). In the presented model the population dynamic growth rate is treated as an initial condition and not as a parameter, as is usually the case in population dynamic equations. And the density independent fundament is Fisher's (1930) fundamental theorem of natural selection, which predicts a hyper-geometrical increase in the abundance instead of the geometrical increase of the Malthusian law (Malthus, 1798). The model may thus be seen as a variant of the inertia growth initially proposed by Ginzburg (1980, 1986) [see also related discussions in Clark (1971) and Innis (1972)]. Where Ginzburg (1998) has more recently developed inertia growth into the hypothesis of maternal effects, my study suggests that it may instead be understood by a general law of selection by density dependent competitive interactions.

It is often argued that a successful theory of cyclic population dynamics needs to explain at set of observations [e.g., Krebs and Myers (1974) and Stenseth (1985)]. These observations include: (i) that cyclic dynamics is more common and that it has larger amplitudes toward arctic regions; (ii) that the period of cyclic dynamics is more fixed than the amplitude; (iii) the actual periods of the cyclic dynamics; and (iv) the co-occurrence of cyclic population dynamics and cyclic life histories. As described in the following three sub-sections these observations can be explained by the mechanism of selection by density dependent competitive interactions. The resemblance between the predicted dynamics and the dynamics of animal species does not imply that the proposed mechanism necessarily applies to natural populations. In line with other recent studies [e.g., Ginzburg and Taneyhill (1994) and Inchausti and Ginzburg (1998)], the resemblance shows only that the hypothesis is a plausible mechanism. To determine the actual causes in natural populations we need empirical evidence, but empirical studies have so far failed to determine the true cause for cyclic population dynamics [e.g., Krebs (1996)].

4.1. ***Neutral stability.*** Like the dynamics of many other models, the cycles of equation (13) are neutrally stable in the sense that their amplitude is given by initial conditions even though the period is fixed and given by the particular values of the density regulation parameters. This implies that the amplitude of the cycle will increase on a cline from a stable to a fluctuating environment. One such cline is from temperate areas toward the polar regions where an increased frequency and severity of unfavourable climatic conditions occurs. On this cline it is generally observed that population cycles tend to vanish toward the south while they are more pronounced with progressively larger amplitudes toward arctic regions [e.g., Howell (1923), Hansson (1971, 1987), Hansson and Henttonen (1985), Stenseth *et al*. (1985), Hanski *et al*. (1991), Akçakaya (1992) and Bjørnstad *et al*. (1995)].

As the amplitude of neutrally stable dynamics depends on the magnitude of the environmental perturbation a special type of dynamics can arise from extreme perturbations of equilibria with strongly damped dynamics. In Fig. 3 I have used

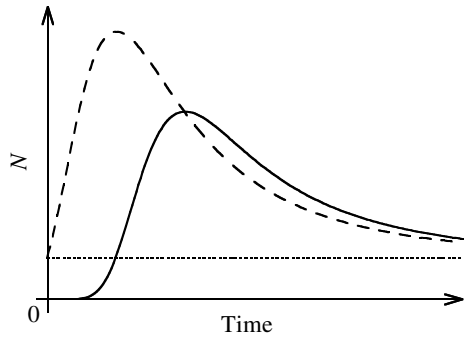

Figure 3. A projection of equation (13) illustrating the dynamics of an introduced species with strongly damped dynamics ($\gamma = 0.07$ and $\gamma_q = 0.01\gamma$). At time zero the species is introduced at an abundance that is 10^{-7} times smaller than the abundance at the evolutionary equilibrium. The solid curve is the population abundance, the dashed curve the population equilibrium, and the dotted line the evolutionary equilibrium.

equation (13) to simulate the dynamics following a perturbation to an extraordinarily low abundance. At this abundance interference competition is almost absent and there is strong selection for an increase in the population dynamic growth rate. The result is a lack phase with only a marginal increase before the evolutionary increase in population dynamic growth rate causes the population to explode to an extreme abundance. The peak abundance is associated with high levels of interference competition where energy is selected from demographic traits into competitive traits causing the population to decline slowly to a normal and stable abundance at the evolutionary equilibrium. This type of dynamics with a single, or a few, over-shoots is often observed when humans release exotic species into new areas [e.g., Elton (1927) and Adam *et al*. (1993)].

4.2. ***The period.*** Forest insects are known for their pronounced population cycles. In the literature I found nine species, mainly Lepidoptera, with yearly non-overlapping generations, cyclic dynamics, and yearly abundance estimates for periods longer than 10 years. Assuming that the cycles are stable, i.e., that $\gamma_q = \gamma$, the parameters of equation (13) can be estimated from such time series by regression equation (B4) in Appendix B. Table 1 lists the estimated models. The estimated evolutionary equilibria (N^{**}) resemble the geometric mean of the abundance in the time series ($\bar{N}$), a result that supports the assumption of linearity on logarithmic scale.

As mentioned in Section 2, the period in the dynamics of equation (13) is given by the γ parameter, and in Fig. 4 this relationship is shown by the curve while the diamonds represent the nine species. Probably the best documented cycle is found in the larch budmoth that 'goes through 10 000-fold changes in density during its very regular [8.24 ± 0.27 (SE) years] cycle in the Alps' (Berryman, 1996). The data on one population of this species are shown by the diamonds in Fig. 5, where

Table 1. The parameter estimates for equation (13) for nine insect species (assuming $\gamma_q = \gamma$). n is the number of years with abundance estimates, $\bar{N}$ the geometric mean in the data, P the average period in the data, c the correlation coefficient of the regression [equation (B4)] that was used to estimate the two parameters γ and σ^2, and N^{**} the estimated population equilibrium. The estimation procedure is described in Appendix B.

Species	n	$\bar{N}$	P	c	$\gamma \pm$ SE	$\sigma^2 \pm$ SE	N^{**}
Douglas-fir tussock moth	10	4.1	9.0	0.96	0.52 ± 0.17	0.77 ± 0.41	4.39
Fall webworm	22	10.2	7.0	0.82	0.69 ± 0.12	1.54 ± 0.44	9.83
Larch budmoth	38	2.2	9.3	0.96	0.53 ± 0.07	0.51 ± 0.28	2.62
Larch cone fly	10	945	4.5	0.32	1.68 ± 0.09	11.5 ± 0.88	950
Nun moth	42	2600	4.1	0.55	1.28 ± 0.05	10.0 ± 0.61	2630
Pine looper moth	50	0.66	4.9	0.62	1.30 ± 0.14	-0.48 ± 0.19	0.69
Southern pine beetle	30	700	7.7	0.94	0.42 ± 0.04	2.67 ± 0.39	605
Spruce budworm	28	1.5	21	0.94	0.28 ± 0.13	0.16 ± 0.31	1.76
Wasp spp.	25	10.2	2.2	-0.57	2.89 ± 0.31	6.59 ± 1.05	9.78

Data from Morris (1964), Southwood (1967), Royama (1984), Baltensweiler and Fischlin (1988), Bejer (1988), Roques (1988), Barbour (1990), Dahlsten *et al*. (1990) and Turchin *et al*. (1991).

the curve represents a projection of the model in equation (13). The resemblance between theory and data in Figs 4 and 5 is reasonable, suggesting that selection by density dependent competitive interactions may play a role in the cyclic dynamics of forest insects. But other hypotheses, like the maternal effect hypothesis (Ginzburg and Taneyhill, 1994, 1995; Ginzburg, 1998), have produced comparable fits and the resemblance may be a more general result of second-order population dynamic equations.

4.3. ***Life history cycles.*** Selection by density dependent competitive interactions suggests that population cycles are associated with cyclic changes in life history parameters such as body mass, metabolic rate, and age of reproduction. Such changes have often been found in small rodents with cyclic dynamics (Krebs and Myers, 1974; Krebs, 1978; Boonstra and Krebs, 1979; Stenseth, 1982; Lidicker and Ostfeld, 1991; Stenseth and Ims, 1993). It is observed that rodents are small, non-aggressive, and that they have a high rate of reproduction when the abundance is low and increasing. When instead the abundance is high and declining the rodents are aggressive, 20–30% larger than at low abundance, and they have delayed reproduction and a low reproductive rate.

The cyclic changes in life history characters are also found in other species. Murdoch and McCauley (1985) observed cyclic dynamics in a *Daphnia* population, and Fig. 6 shows the average body mass and the population abundance against time. It is apparent that the body mass cycles, and that this cycle is lagging behind the cycle in abundance, as predicted by the dynamics of equation (13). From equation (11)

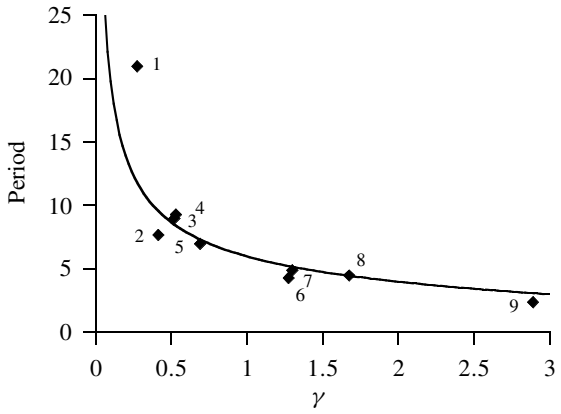

Figure 4. The period of the population cycle in generations against the γ parameter (assuming $\gamma_q = \gamma$). The curve is defined by equation (13), and the numbered diamonds represent the following species: (1) spruce budworm, (2) southern pine beetle, (3) douglas-fir tussock moth, (4) larch budmoth, (5) fall webworm, (6) nun moth, (7) pine looper moth, (8) larch cone fly, (9) wasp spp.

the predicted relation between body mass (w) and population abundance is

$$\ln w_t - \ln w_{t-1} = \gamma_q \ln N_{t-1} + c \tag{16}$$

where c is a constant. This relation is significantly present in the *Daphnia* population [correlation coefficient = 0.49, $n = 26$, $\gamma_q = 1.7 \pm 0.5$ (SE), and $c = -0.55 \pm 0.36$].

Some studies have reported no correlation between the body mass and the phase of the abundance cycle (Myllymäki, 1977; Ferns, 1979), but the widespread observation of phase-related life history cycles has been seen as essential for a mechanistic understanding of cyclic population dynamics [e.g., Chitty (1987, 1996) and Krebs (1996)]. As the body mass tends to be largest at the peak abundance, the life history cycles cannot be explained as simple reflections of density dependent changes in the environment. The hypothesis of density dependent competitive interactions incorporates the cyclic changes in life history parameters as part of the causal agent that is responsible for the delayed density dependence that is so essential for the generation of cyclic dynamics.

Selection by density dependent competitive interactions suggests that selection is strongest when the abundance is at peak and bottom densities, while selection can be absent at intermediate densities. For a rodent cycle Krebs *et al*. (1973) found that selection was strongest at the peak abundance where the frequency of the LAPs allele changed from $\approx$0.70 to $\approx$0.40 in less than one generation. And for the oak leafroller moth Simchuk *et al*. (1999) found that trends at the Est-4 and Pts-4 loci were directly related to the population dynamics. They also found density dependent changes in the selection pressure on body mass, with larger females

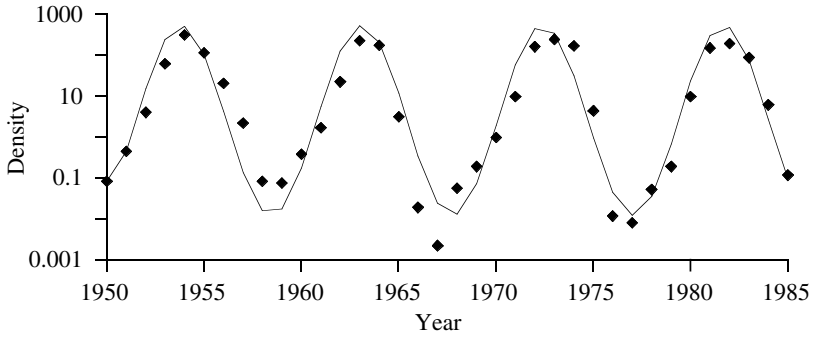

Figure 5. The curve is a projection of equation (13) and the diamonds the yearly abundance of the larch budmoth in the Upper Engadine valley from 1950–1985. The γ and σ^2 parameters of equation (13) are adjusted from those given in Table 1 to $\gamma = 0.44$ and $\sigma^2 = 0.42$ so that the equilibrium is the same and the period resembles the observed period. Data from Baltensweiler and Fischlin (1988).

being selected prior to the population decline, and smaller females being selected during the outbreak phase of the cycle.

Although density dependent selection may be responsible for the cyclic changes in body mass in some cases, in other situations cyclic body masses may be a consequence of cyclic dynamics and not necessarily part of the cause that drives the cycle (Lidicker and Ostfeld, 1991). This can be the case if changes in foraging time and reproductive effort cause phase-related changes in body mass (Oksanen and Lundberg, 1995), or if phase-related changes in body mass are induced by dynamic energy allocation in fluctuating environments, with fluctuating environments including population dynamic cycles (Oli, 1999).

4.4. ***Allometries of population dynamics.*** The allometric relationship between body mass and, respectively, the population abundance and the period of the cyclic dynamics are other relations that a successful population dynamic theory needs to explain. For species outside competitive guilds, the population abundance is approximately proportional to the negative $3/4$ power of body mass (Damuth, 1981, 1987; Nee *et al*., 1991), and the Calder hypothesis suggests that the period of population cycles scale to the $1/4$ power of body mass (Calder, 1983, 1984; Peters, 1983; Peterson *et al*., 1984; Krukonis and Schaffer, 1991). The population abundance allometry has been shown to apply to a variety of taxa, and the Calder hypothesis is confirmed for terrestrial homoiotherms.

The exponents of the abundance and population cycle allometries follow from first principles of the proposed density dependent ecology. The model of equation (13) is part of a larger theory where the across-species exponents of the lifespan and population abundance allometries are deduced from the density dependent constraints that competitive interactions and foraging self inhibition places on the foraging process in mobile organisms (Witting, 1995, 1998). Lifespan (T) is pre-

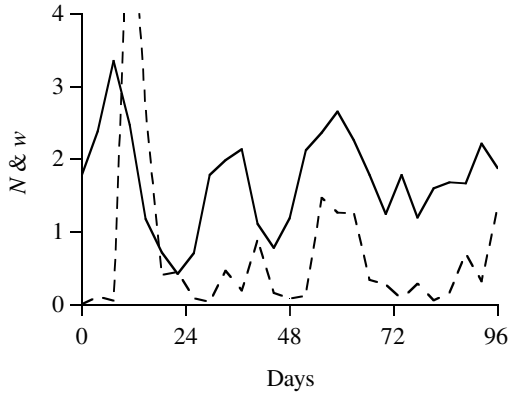

Figure 6. The dynamics in the abundance (N) and body mass (w) of a *Daphnia* population against time. The solid curve is the population abundance (10^5 *Daphnia* per m^{-3}) and the dashed curve the body mass (μg dry weight). Data from Fig. 3 in Murdoch and McCauley (1985).

dicted to scale as $T \propto w^{1/2d}$, and the abundance as $N^* \propto w^{(1-2d)/2d}$, where w is body mass and d the number of dimensions in which the organism forages. Thus, for terrestrial organisms, which are likely to forage in two dimensions, lifespan is expected to scale to the $1/4$ power of body mass and the population abundance to the negative $3/4$ power of body mass. As the period of equation (13) is given in generations it follows that the period in astronomical time is proportional to the $1/4$ power of body mass, as suggested empirically by the Calder hypothesis.

For organisms that forage in three dimensions the predicted exponents are $1/6$ and $5/6$ instead of $1/4$ and $3/4$. The dependence of the allometric exponents upon the dimensionality of the foraging behaviour is empirically confirmed for the lifespan exponent, which is 0.25 ± 0.04 (SE) among 195 species of terrestrial mammals while it is 0.16 ± 0.02 (SE) among 40 species of pelagic mammals expected to forage in three spatial dimension (Witting, 1995, 1998). But whether the exponents of the abundance and population cycle allometries for pelagic species are $5/6$ and $1/6$ instead of $3/4$ and $1/4$ remain to be shown.

APPENDIX A: STABILITY AND DYNAMIC BEHAVIOUR

The stability of the evolutionary equilibrium and the dynamics that follow from a perturbation of that equilibrium is analysed in this section. Let $r = \ln \lambda_m$ and $n = \ln N$. Take the natural logarithm to equation (13) and obtain $r_t = G(r_{t-1}, n_{t-1})$ and $n_{t+1} = F(r_t, n_t)$ where the two functions G and F are defined as

$$G = r_{t-1} - \gamma_q n_{t-1} + \sigma^2$$
$$F = r_t + (1 - \gamma) n_t. \qquad \text{(A1)}$$

Following the procedure in Bulmer (1994), the stability of the evolutionary equilibrium is given by the eigenvalues of the Jacobian matrix

$$\begin{bmatrix} \partial G/\partial r & \partial G/\partial n \\ \partial F/\partial r & \partial F/\partial n \end{bmatrix} \tag{A2}$$

where the equilibrium is unstable when the absolute value of the dominant eigenvalue is larger than or equal to one. For the two-dimensional matrix equation (A2) the eigenvalues are

$$\left(T \pm \sqrt{T^2 - 4D}\right)/2 \tag{A3}$$

where D is the determinant and T the trace of that matrix. To determine D and T we have that $D = (\partial G/\partial r)(\partial F/\partial n) - (\partial F/\partial r)(\partial G/\partial n)$, that $T = \partial G/\partial r + \partial F/\partial n$, and that $\partial G/\partial r = 1$, $\partial G/\partial n = -\gamma_q$, $\partial F/\partial r = 1$, and $\partial F/\partial n = 1 - \gamma$ for equation (A1) for all r and n including the equilibrium. Hence

$$\begin{aligned} D &= 1 - \gamma + \gamma_q \\ T &= 2 - \gamma \end{aligned} \tag{A4}$$

and, thus, for equation (13) on a logarithmic scale, the eigenvalues are

$$\left(2 - \gamma \pm \sqrt{\gamma^2 - 4\gamma_q}\right)/2. \tag{A5}$$

The eigenvalues are real when $\gamma^2 \geq 4\gamma_q$ and complex when $\gamma^2 < 4\gamma_q$. For the situation $\gamma^2 \geq 4\gamma_q$ with real eigenvalues the equilibrium is unstable when $\gamma > 4$ or when $\gamma_q \leq 2\gamma - 4$ and $2 \leq \gamma \leq 4$ while it is otherwise stable. When additive genetic variation and a plastic phenotypic response are absent, i.e., when $\sigma^2 = 0$, the instability criterion $\gamma_q \leq 2\gamma - 4$ reduces to $\gamma \geq 2$ because $\gamma_q = 0$ at the limit where additive genetic variation is absent. This instability is generated by over-compensatory density regulation. This is in contrast to the instability criterion associated with the complex eigenvalues, where the instability arises from the evolutionary modulation of the growth rate. For the latter situation $\gamma^2 < 4\gamma_q$ and the absolute value of the eigenvalues is $\sqrt{1 - \gamma + \gamma_q}$ so that the evolutionary equilibrium is unstable when $\gamma \leq \gamma_q$. This latter instability criterion is impossible when $\sigma^2 = 0$, and this is because $\sigma^2 = 0$ imposes the constraint $\gamma_q = 0$ so that the inequality $\gamma \leq \gamma_q$ is false because γ is positive for natural situations.

To examine the conditions associated with the instability criterion $\gamma_q \geq \gamma$ recall that $\gamma_q = \gamma_\iota \psi \sigma^2$ and that $\gamma = \gamma_\alpha + \mu\gamma_\iota$. Therefore, the condition $\gamma_q \geq \gamma$ resembles

$$\gamma_\alpha/\gamma_\iota \leq \psi\sigma^2 - \mu. \tag{A6}$$

This implies that the equilibrium becomes more stable when the resource regulation (γ_α) or the average cost of interference (μ) are increased. Moreover, the equilibrium becomes more unstable when the density dependence in the level of interference (γ_ι), the potential response to selection (σ^2), and the intra-population differentiation in interference regulation (ψ) are increased.

Let us now turn to the dynamics that follow from a perturbation of the evolutionary equilibrium of equation (13). When there is a response to selection ($\sigma^2 > 0$) the long-term dynamics is given by the eigenvalues of the Jacobian matrix equation (A2). If these eigenvalues are real and their absolute values are smaller than one the perturbation will decline geometrically toward the equilibrium, while it will increase geometrically if the absolute value of the dominant eigenvalue is larger than one. Hence, for equation (13) the perturbation will increase geometrically when $\gamma > 4$ or when $\gamma_q \leq 2\gamma - 4$ and $2 \leq \gamma \leq 4$, while the dynamics is damped otherwise. Moreover, if the sign of the dominant eigenvalue is negative the long-term dynamics is oscillatory with a period of two generations, while it is monotonic if the value is positive. Hence, the long-term dynamics of equation (13) is oscillatory when $\gamma > 2$ while it is monotonic for $\gamma < 2$. Numerical simulations though do show that the dynamics following from a perturbation of the equilibrium is often oscillatory even when $\gamma < 2$.

If instead the eigenvalues are complex they will induce a cycle with a highly variable period. To describe this cycle, let a complex eigenvalue be given by the polar co-ordinates in the Argand diagram, i.e., let x be its absolute value and θ its argument. Then the period of the cycle is $P = 2\pi/\theta$ while the amplitude is proportional both to the initial perturbation and to x. This amplitude is stable if $x = 1$, it declines geometrically if $x < 1$, and it increases geometrically if $x > 1$. That is, $x < 1$ corresponds to a damped cycle, $x = 1$ to a stable, and $x > 1$ to a repelling cycle. For equation (13) we find that

$$\begin{aligned} x &= \sqrt{1 - \gamma + \gamma_q} \\ P &= 2\pi / \arctan \left| \frac{\sqrt{4\gamma_q - \gamma^2}}{2 - \gamma} \right|. \end{aligned} \tag{A7}$$

This implies that the cycle caused by the evolutionary modulation of the growth rate is damped when $\gamma < \gamma_q$, stable when $\gamma = \gamma_q$, and repelling when $\gamma > \gamma_q$. Also, when the cycle is stable the period has a minimum of four generations at $\gamma = 2$, while the period increases monotonically to infinity as γ goes toward either 0 or 4. For $\gamma < 0$ the system is not biologically defined, and at the limit of, and beyond, $\gamma = 4$ the eigenvalues are real with the dominant eigenvalue being smaller than -1 so that an initial perturbation increases geometrically and oscillatory without limits.

Let the period of the population cycle be the period between two neighbouring events where the population abundance crosses the equilibrium in the same direc-

tion. Numerical simulations show that the population period has a close resemblance to the period defined by equation (A7) when $\gamma_q \approx \gamma < 2$. When instead $\gamma_q \approx \gamma \geq 2$ and $\gamma_q \approx \gamma \to 4$ the population period declines monotonically from four to two generations, while the increasing period given by equation (A7) resembles a period that turns up in the amplitude of the population cycle. An example of the period in the amplitude is shown in Fig. 2, and the relation between the γ parameter and the population period is shown in Fig. 4 together with data from forest insects.

APPENDIX B: PARAMETER ESTIMATION

Given that the population cycle is stable the parameters for the population model equation (13) are easily estimated from successive estimates of the population abundance: a stable cycle implies that $\gamma_q = \gamma$ and, thus, that equation (13) reduces to

$$\begin{aligned} \lambda_{m,t} &= \lambda_{m,t-1} N_{t-1}^{-\gamma} e^{\sigma^2} \\ N_{t+1} &= N_t \lambda_{m,t} N_t^{-\gamma}. \end{aligned} \tag{B1}$$

To estimate the parameters γ and σ^2, from the bottom equation of equation (B1), we have

$$\lambda_{m,t} = N_{t+1} N_t^{\gamma-1}. \tag{B2}$$

If we then insert $\lambda_{m,t-1}$ and $\lambda_{m,t}$ from equation (B2) into the top equation of equation (B1) we obtain

$$N_{t+1} N_t^{\gamma-1} = N_t N_{t-1}^{\gamma-1-\gamma} e^{\sigma^2}. \tag{B3}$$

This equation can be rearranged so that we can estimate γ and σ^2 by the linear regression

$$\ln N_{t+1} + \ln N_{t-1} = (2-\gamma) \ln N_t + \sigma^2. \tag{B4}$$

From $\lambda_m^{**} = \lambda_m^{**} N^{**-\gamma} e^{\sigma^2}$ [equation (B1)] the abundance at the equilibrium is $N^{**} = e^{\sigma^2/\gamma}$, which combined with $N^{**} = N^{**} \lambda_m^{**} N^{**-\gamma}$ [equation (B1)] gives us the estimate $\lambda_m^{**} = e^{\sigma^2}$.

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