Title: Increasing environmental fluctuations can dampen variability of endogenously cycling populations

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Running Title: Environmental Variability Dampens Cycles
Keywords: Long transient | Nonlinear dynamics | Extinction risk | Stability | Resilience
Article Type: Letter
Word Count: Abstract: 177; Main Text: 3990, Boxes: 0
Manuscript elements: References 66; Figures: 6; Tables: 0; Text Boxes: 0.

Author Contributions: JF and JMP conceived of the original idea and secured funding. NK constructed and analyzed models with input and discussion from all authors. NK wrote the original draft, and all authors contributed to revisions.

Data Accessibility Statement: No data were used in this study.

Abstract

Future climate projections predict a more variable world. Understanding how populations respond to increasing variability is a major focus for modelers and natural resource managers attempting to predict extinction risk. Current theory of extinction risk is based on models that assume populations reach a stable, deterministic equilibrium in the absence of environmental variation. The lesson from these models is clear: Increasing environmental variability increases population size variability and extinction risk. Although useful, these models fail to describe a broad class of empirically observed dynamics, namely cycles. We develop new theory for the effects of environmental variability on the dynamics of cycling populations. We find that when populations are driven by endogenous cycles, increasing environmental variability can reduce population size variability, contrary to all prior expectations that populations fluctuate more in more variable environments. This result relies on strong density-dependent feedback and represents a novel mechanism by which stochastic variation interacts with density-dependence. Moreover, these results highlight that theories of extinction and stability must be expanded to include more complex forms of the interactions between density-dependence and stochasticity.

Introduction

One of the most pressing challenges in ecology is understanding how changing climate will impact populations. Most projections of future biodiversity under climate change present a bleak outlook (Bellard et al., 2012; Jetz et al., 2007; Pereira et al., 2010; Thomas et al., 2004; Urban, 2015), although exact causes are uncertain (Cahill et al., 2013). Typically, climate change is believed to cause extinctions because warmer average temperatures are outside the current thermal limits of species (Deutsch et al., 2008; Huey et al., 2009). But other dimensions of the climate will change as well, and the impacts of these other changes have received far less consideration in ecological and population dynamics studies than changes in average condition. Examples include the projected increases in variability of annual temperature (Rahmstorf and Coumou, 2011) and rainfall (Diffenbaugh et al., 2017), as well as the concomitant increased likelihood of extremes events. The few theoretical studies that explicitly account for changing environmental variability show that those changes can be at least as important as changes in average conditions (Vasseur et al., 2014). Overall, theoreticians and practitioners alike are still far from understanding the many ways that increasingly variable environmental conditions impact populations. Of particular interest to conservation biologists and natural resource managers is whether increasingly variable environmental conditions put species at greater risk of extinction.

Theory points to two ways in which environmental variability affects extinction risk. One plausible mechanism is through changes in the long-term growth rate of a population, for it has long been known that long-term population growth is sensitive to the level of environmental variability (Tuljapurkar and Orzack, 1980). One hypothesis is that increasing variability of survival and reproduction (assuming fixed mean values) lowers long-term growth rate (see Real and Ellner 1992 for a graphical application of this idea and Bernhardt et al. 2018 for an experimental demonstration). The second way environmental variability puts species at greater risk of extinction is by causing short-term population fluctuations. To a first approximation, greater fluctuations in population size should enhance extinction risk because populations dip to lower numbers or dip to low numbers more frequently. At low numbers, populations are at heightened risk of extinction from the increased chance of below average survival and reproduction among the remaining individuals (i.e., demographic stochasticity; Lande 1993; Ludwig 1996). To understand how much extinction risk increases with population fluctuations, one first needs to determine how environmental fluctuations influence population fluctuations. But this is no simple task; one needs to understand the demographic effects of environmental variability as well as the influence of environmental variability on density-dependent processes, as the effects of density-dependence are crucial in determining how susceptible species are to extinction (Bonsall and Hastings, 2004; Cushing et al., 2003; Dennis et al., 2001; Drake, 2005; Engen et al., 1998).

Despite recognition that density-dependence may be crucial for predicting extinction, the current paradigm for understanding the effects of environmental variability on population fluctuations is limited by the fact that it is based on models that assume relatively simple forms of density-dependence. These models predict population growth as either declining to extinction, or, alternatively, approaching a carrying capacity and then persisting indefinitely. The logistic model is one such example, but similar dynamical behavior occurs for many commonly used models in ecology, such as the Gompertz, Beverton-Holt, and theta-Ricker models (Anazawa, 2010; Turchin, 2003). The predictions from these models often apply to more complex forms of density-dependence with multiple potential equilibria, such as models used to describe tipping points (Scheffer et al., 2001).

Such extensions are justified by the idea that simple density-dependence describes local dynamics *around* the equilibria (see, for example, (Arnoldi et al., 2018)). A number of tools have been developed to study these local dynamics which predict, for example, how sensitive populations are to variation in their environment, how quickly populations return to carrying capacity following a perturbation, and myriad other properties (see Van Meerbeek et al. 2021 for a recent review). The use of standard models of density-dependence to approximate more complex systems is valuable because they are analytically tractable. As such, much has been learned about population models for single species (Arnoldi et al., 2018; Dennis et al., 2006; Lande, 1993; Ludwig, 1975) and communities (Arnoldi et al., 2019; Ives et al., 2003) characterized by a single equilibrium. That being said, it is still unclear whether this simplified and analytically tractable framework also applies to more complex population dynamics, such as population cycles (Barraquand et al., 2017). Populations cycles are common in nature (Elton and Nicholson, 1942; Krebs et al., 1973; Myers, 1988, 1990; Stenseth, 1999), and any explanation for cycles requires more complex models than those that assume a fixed carrying capacity (Berryman, 2002; Myers, 2018).

We try here to understand how environmental variability influences population fluctuations in cycling populations. To do so, we use a stochastic, time-dependent Ricker model, which is sufficiently flexible to model simple density-dependence and cycling populations in a single framework. We consider two cycling mechanisms: exogenous and endogenous, which represent cycles caused by external factors (e.g., weather; Axelsen et al. 2014) and delayed density feedback loops (e.g., stage structure; Stenseth 1999; Zeng et al. 1998), respectively (Berryman, 2002). The two mechanisms imply different dynamics of density-dependence itself, as illustrated in Fig. 1 a-c. In each of these cases, we add stochastic variation to a deterministic skeleton (illustrated in Figs. 1d-f) and ask how increasing the magnitude of stochastic variation changes the variance in population size over time. In doing so, we evaluate whether the established framework for population stability, which assumes a fixed stable equilibrium, adequately explains the responses of cycling populations to increasing environmental variability. We find that prior theoretical frameworks are inadequate to describe how endogenously cycling populations respond to environmental variability. Remarkably, endogenously cycling populations (characterized by cycles driven by changes in density-dependence along the cycle) may exhibit weaker population fluctuations under scenarios of increasing environmental variability, a result we find in two distinctly different models.

The Ricker model

Model Description

To understand the effects of environmental variability on cycling populations, we first consider the Ricker model, which is a two-parameter discrete-time model of density-dependent population growth. Let n_t be the density of individuals in the population at time t. For the general Ricker model, the density of individuals the following time step, n_{t+1}, is

$$n_{t+1} = n_t e^{a_t - b n_t}, \tag{1}$$

where a_t is the (possibly time-dependent) density-independent per-capita growth rate and b is a coefficient scaling the effect of density. A typical interpretation is that the species under consideration has discrete generations, and the time t is then in units of generations. Under this interpretation, individuals in generation t produce e^{a_t} offspring and e^{-bn_t} is the fraction that survive.

We use the Ricker model because of its flexibility in modeling qualitatively distinct population dynamics. With this single model, we can model a single, stable equilibrium, exogenous cycles, and endogenous cycles simply by changing the structure of the time-dependent parameter a_t (the parameter b simply scales population density and can be re-scaled out of the model; see SI S3.2 for details). In brief, we model a stable equilibrium by letting a_t be a constant less than 2 for all t, we model exogenous cycles by letting a_t be a time-dependent cyclic function, and model endogenous cycles by letting a_t be a constant slightly larger than 2. What follows are more details about the assumptions about a_t.

When both parameters are constant over time and a is small, the Ricker model is a discrete-time analog to the logistic growth model (supplementary material S1) with carrying capacity given by a/b (dashed line in Fig. 1d). We use this case to model simple density-dependence. Any cyclic, time-dependent function for a_t mimics the action of a cycling environmental factor. For simplicity, we chose the sine wave representation of a_t:

$$a_t = \bar{a} + \sqrt{2\sigma_a^2}\sin(2\pi t/\Omega). \tag{2}$$

In this writing, $\bar{a}$ is the mean value a_t over a full cycle, σ_a^2 is (approximately) the variance of a_t determined by the amplitude of the cycle, and Ω is the cycle duration. The equilibrium point of this model, given by a_t/b, moves in time and is itself a sine wave function (the dashed line in Fig. 1e). To model endogenous cycles, we make use of the fact that the Ricker model is among the simplest models to describe overcompensatory density-dependence that generates a 2-cycles dynamics (May, 1976; Strogatz, 2015) when a_t is a constant between 2 and ≈ 2.526 (dashed line in Fig. 1f).

The different cases represent qualitatively different changes in density-dependence over time. The case of a stable equilibrium (dashed line in Fig. 1d) implies simple density-dependence much like assumptions in previous models used to understand the effects of environmental variability (e.g, (Ferguson and Ponciano, 2015; Ives et al., 2003; Melbourne and Hastings, 2008)). The functionally important assumption of this model (and many allied models, such as the logistic, Gompertz, and Beverton-Holt) is that the per-capita growth rate (i.e., $dn/dt = \ln\{n_{t+1}/n_t\}$) declines linearly with some function of density. The exogenous cycle can be visualized with a single, stable equilibrium point that cycles in time, being driven by changes in the exogenous factor (Fig. 1b). The parameter a_t then represents the ultimate effect of the exogenously cycling factor on birth rates. Correspondingly, the population approximately tracks this cycling equilibrium (see e.g., Turchin and Ostfeld 1997). An endogenous cycle must be represented by a more complex form of density-dependence that includes some time delay (Fig. 1c Stenseth 1999; Zeng et al. 1998). The 2-cycle generated by the Ricker model implicitly includes such time delays via overcompesatory density-dependence.

To model environmental stochasticity, we consider the following stochastic version of the the Ricker model:

$$N_{t+1} = N_t e^{a_t - bN_t + \sigma Z_t}, \tag{3}$$

where N_t is now the random variable of population size in year t, Z_t is a random variable representing stochastic environmental conditions in year t, and σ is a measure of the magnitude of environmental variation over time. Following standard modeling assumptions, we assume that Z_t is an i.i.d. standard normal random variable. As such, $Z_t \sim \mathcal{N}(0,1)$ for all t, and $E[Z_t Z_s] = 0$ for all $t \neq s$. Environmental variation is given by the term σZ_t, which has mean zero and variance σ^2. A biological interpretation of this model is that individual reproduction varies independently each year and follows a log-normal distribution. We simulated the stochastic model Eq. [3] for 2000

time steps and for 1000 sample paths of Z_t ($t = 1, 2, \ldots, T = 2000$). To minimize the time to reach stationarity, we initiated each simulation with a population size near its deterministic attractor.

To describe the magnitude of population fluctuations, we use standard statistical measures of time series. Namely, we calculated the long-run variance in population sizes,

$$\mathrm{Var}(N) = \lim_{T\to\infty} \frac{1}{T} \sum_{t=1}^{T} (N_t - \bar{N})^2, \tag{4}$$

where $\bar{N} = \lim_{T\to\infty} T^{-1} \sum_{t=1}^{T} N_t$ is the long-run average population size. Eq. 4 represents the expected magnitude of fluctuations when the population is in a stochastic stationary state (a stochastic analogue to a traditional point equilibrium).

Relationships between environmental variability and population fluctuations

Prior theory developed from standard models of density-dependence are not suitable for understanding the variation in such endogenous cycles. To see why, consider a small variance approximation to the variability in population size. Small variance approximations are commonly used in studies of population dynamics in variable environments. However, their analytical limitations — which we highlight here — get at the root of limitations of prior theory developed under simple density-dependence. Small variance approximations simplify the shape of density-dependence around a point (typically an equilibrium) and, as such, are only valid in so far as density-dependence is approximately linear.

The approximation for variance in population size in the stochastic Ricker model is

$$\mathrm{Var}(N) \approx \underbrace{\frac{\bar{N}^2}{\bar{a}(2-\bar{a})}\sigma_a^2(1+c)}_{\substack{\text{Variance from}\\ \text{exogenous driver}}} + \underbrace{\frac{\bar{N}^2}{\bar{a}(2-\bar{a})}\sigma^2}_{\substack{\text{Variance from}\\ \text{environmental}\\ \text{stochasticity}}}, \tag{5}$$

where $\bar{N} = \bar{a}/b$ is the average population size, $\bar{a}$ is the average low density growth rate, σ_a^2 is the (approximate) variance when a_t cycles exogenously, σ^2 is the variance in the stochastic component of the environment, and $c = \lim_{T\to\infty} \sum_{u=1}^{T} 2(1-\bar{a})^u \rho_a(u)$ is the effect of autocorrelated environmental variation on population fluctuations ($\rho_a(u)$ is the autocorrelation function of a_t with time lag u; derivation in supplementary material S3). Equation (5) states that the long-run variance in population size can be partitioned into a sum of two components: variability from an exogenous factor (σ_a^2) and from environmental stochasticity (σ^2).

Approximation (5) predicts a linear increase in population variability with environmental stochasticity for $\bar{a} < 2$, which is the domain of simple density-dependence and exogenous cycles. This prediction matches well the observed linear increase in $\mathrm{Var}(N)$ with increasing σ^2 for both models of standard density-dependence (Fig. 2a) and exogenous cycles (Fig. 2b).

However, the small variance approximation is invalid for the endogenous cycle case because eq. (4) predicts negative variance when a_t is a constant greater than 2 (i.e., $\sigma_a = 0$ and $a_t = \bar{a} > 2$ for all t). Note that when $a_t = \bar{a} > 2$, it follows that $\bar{N}^2/[\bar{a}(2-\bar{a})] < 0$ in eq. (4), implying negative

variance from the exogenous driver. Negative variance is neither mathematically valid (variances are, by definition, non-negative) nor is consistent with expectations under small σ^2, where population variance should reflect the asymptotic behavior of the deterministic 2-cycle. Asymptotically, $\mathrm{Var}(n) = \bar{n}^2\Delta^2$ for the deterministic 2-cycle, where $\Delta = |\hat{n} - \bar{n}|/\bar{n}$ is the deviation of the two fixed points from the mean of the cycle, scaled by population size (derivation in supplementary material S3). Thus, standard analytical methods for understanding population fluctuations fail when the population cycles endogenously.

Remarkably, our simulations indicate that upon adding environmental variability to endogenously cycling populations, the variance in population size declines (Fig. 2c). The implication here is that, for some levels of environmental variation (σ^2), increasing environmental variability stabilizes population fluctuations. What explains the reduction in population size variance with greater environmental variability?

A possible explanation emerges from close inspection of the shape of density-dependence. One way to understand density-dependence for the period-2 cycle is to consider the 2-step map. If $F(n_t)$ is the deterministic Ricker map (i.e., $n_{t+1} = F(n_t)$), then the 2-step map is $F_2(n_t) = F(F(n_t))$ and projects population size 2 time-steps in the future. For a 2-cycle, the 2-step map has 3 non-trivial fixed points: 2 stable fixed points corresponding to the the two asymptotic cycling values (the dashed lines in Fig. 3), and an unstable fixed point exactly between the two (Fig. S2). This unstable fixed point is given by the value $\bar{a}/b$, the mean population size (supplementary material S2), and has weak local stability as measured by the local rate of change in the 2-step per-capita growth rate (i.e., the slope of $\ln\{n_{t+2}/n_t\}$ with respect to n_t at each fixed point). Populations that begin near this weak unstable fixed point can take a long time before exhibiting asymptotic dynamics (supplement Fig. S2), a phenomenon termed a long transient dynamic (Hastings et al., 2018). (For our purposes, weak should be understood as a relatively slow rate at which the system moves away from the the unstable fixed point.)

The fact that the system might take a long time to reach the fixed points leads to unequal effects of stochastic variation at different population sizes. Near stable fixed points, stochasticity has relatively minimal effect. Near the weak unstable fixed point, stochastic effects can dominate the weak effect of density-dependence. For example, consider the sample path of the stochastic endogenously cycling model in Fig. 3. Periodically, stochastic variation pushes the population towards the unstable fixed point. The deterministic forces there are weak and so stochasticity dominates. As a consequence, the population gets "stuck" and spends extended time in a transient state. This dynamical phenomenon keeps the population near the long-run average, reducing overall population size variability. The inset of Fig. 3 shows one particularly acute instantiation of this effect.

Testing the mechanism for nonlinear responses to environmental variability

To test whether stability properties of equilibria, and their interaction with stochasticity, can more generally damp population variance, we must turn to other models. The Ricker model, with only two parameters, is not flexible enough to modify the stability properties of the unstable fixed point. Moreover, stability properties are difficult to measure in difference equation models. For these reasons, we tested our understanding using a continuous-time dynamical system, where we can make use of dynamical potentials to understand local and global stability properties.

A Model of Recurrent Outbreaks

The model we used to test the role of alternative equilibrium stability is based on the spruce budworm (*Zeiraphera canadensis*). Budworm larvae are herbivores of conifers, with highly repeatable transitions between states of high ("outbreak") and low ("endemic") larval density. Ludwig et al. (1978) proposed a model that recapitulates this behavior with the characteristic feature being the presence of two alternative stable states: a high density equilibrium and a low density equilibrium. These alternative equilibria arise based on temporal changes in host tree quality, which are caused by changes in budworm density in a time-delayed fashion. To keep the model simple and isolate the effect of alternative equilibria, we model tree quality independently as a delayed response to budworm density rather than as a variable dynamically linked to budworm density as done previously (Ludwig et al., 1978). However, the result is the same; budworms cycle between endemic and outbreak states.

The deterministic outbreak model is the following non-autonomous first-order ordinary differential equation:

$$\frac{dn}{dt} \equiv F(n,t) = \overbrace{-\gamma n(n-\hat{n}_1)(n-\hat{n}_2)(n-\tilde{n})^{1+2k}}^{\text{Multiple Stable States}} + \overbrace{\delta \cdot \cos\left(2\pi t/\Omega\right) n}^{\text{Forcing Between States}} \tag{6}$$

where n is population size, $\hat{n}_1$ and $\hat{n}_2$ are the low and high density equilibria, respectively, $\tilde{n}$ is an unstable equilibrium, γ and δ are parameters controlling the speed of population dynamics, and k ($k = 0, 1, 2, 3, \ldots$) is a parameter controlling the local stability properties of $\tilde{n}$, the unstable equilibrium. This model describes the population growth rate only as a function of population size. Cycles occur because we assume tree quality cycles seasonally with the term $\delta\cos(2\pi t/\Omega)$ in Eq. [6], which is a sine wave function with period Ω and amplitude δ. Many studies would describe this term as an "environmental forcing" term (see e.g., Becker et al. 2020; Ireland et al. 2004; Turchin and Ostfeld 1997; White and Hastings 2020), although we use it within a defined region of parameter space to mimic delayed feedback of host tree quality in response to budworm density. As tree quality increases and peaks, the low density equilibrium is lost and only the high density equilibrium remains. When tree quality declines, the low density equilibrium returns followed by loss of the high density equilibrium. Given that tree quality changes on a lagged time scale of changes in budworm density, the budworm population will continuously oscillate between the two alternative equilibria. The per-capita growth rate ($1/n \cdot dn/dt$) for this model (assuming a constant environmental driver $\delta = 0$) is plotted in Fig. 4a. While phenomenological, our model recapitulates the salient features of the spruce-budworm moth system (Ludwig et al., 1978).

The endemic and outbreak states (labelled $\hat{n}_1$ and $\hat{n}_2$, respectively) are separated by an unstable equilibrium point, which we label $\tilde{n}$. Our model is valuable for testing the stability properties of equilibria because we can control the local stability properties of $\tilde{n}$ independently of the stability properties of $\hat{n}_1$ and $\hat{n}_2$ with a parameter k ($k = 0, 1, 2, ...$). As k increases, the strength of density-dependence around $\tilde{n}$ declines, as illustrated when plotting the per-capita growth rate as a function of density (Fig. 4a).

The relative effects of density-dependence can be visualized with a dynamical potential, $U(n)$, which is a dynamical systems analogy to potential energy in physical systems (Nolting and Abbott, 2016). Dynamical potentials are defined as $\frac{dn}{dt} = -\frac{dU}{dn}$, which envisions the population size, n, as a ball rolling downhill following the contours of U. Stable equilibria are represented by troughs of U and unstable equilibria are represented as peaks of U. Such a representation is useful

in understanding the effects of stochasticity in dynamical systems because it provides a natural measure in which to compare the relative effects of stochasticity across the system (Nolting and Abbott, 2016); stochasticity has less of an effect on observed dynamics for steeper contours of U. As the parameter k increases, the ridge separating the two stable equilibria becomes flatter (Fig. 4b), representing weaker density-dependence pushing the population to return to either of the stable equilibria. We thus expect stochastic forces to be more influential with larger k, and reduce population size fluctuations when the population oscillates between the two alternative equilibria.

To model environmental stochasticity, we use the following stochastic differential equation (SDE) model

$$dN = \underbrace{F(N,t)dt}_{\substack{\text{Deterministic}\\ \text{dynamics}}} + \underbrace{\sigma N dW}_{\substack{\text{Environmental}\\ \text{stochasticity}}} . \tag{7}$$

where W is a Weiner process. The Weiner process is characterized by the fact that time increments (dW) are distributed as standard normal random variables in one unit of time, i.e., $W_{t+1} - W_t \sim \mathcal{N}(0,1)$, and any two (non-overlapping) time increments are independent, i.e., $\mathrm{E}[(W_{t_2} - W_{t_1})(W_{t_1} - W_{t_0})] = 0$ for $t_2 > t_1 > t_0$. The Wiener process is thus the continuous-time analogue to i.i.d. sampling of standard normal distributions in discrete-time. Moreover, environmental stochasticity scales with population size in eqn. [7], and so is interpretable as generating additive variation in per-capita growth, just as σZ_t generates additive variation in the discrete-time per-capita growth rate. Thus, the effect of environmental variability is functionally equivalent in the discrete and continuous-time models. Methods for simulation this model are found in the supplementary material S6.

Responses to increasing environmental stochasticity

The outbreak model (eq. 6-7), which could embody a fairly large class of systems other than the budworm example (including, for example, disease outbreaks), exhibits the same variance scaling relationships found in the Ricker model under different values of k. As in the Ricker model with simple density-dependence and exogenous cycles, $\mathrm{Var}(N)$ strictly increases with stochastic variation (σ^2) when the unstable equilibrium is strongly repelling and deterministic transients are short ($k = 0$) (purple line; Fig. 5). However, $\mathrm{Var}(N)$ declines when the strength of the repellor is weakened and deterministic transients are long (teal and green lines; Fig. 5), reflecting the fact that the population can get stuck at points in between the two cycle endpoints. In the case of an extremely weak repellor ($k = 2$), populations fluctuate less for a broad range of stochasticity than in the corresponding deterministic model (i.e., when $\sigma = 0$) .

Measuring interactions between environmental variability and density-dependence

The prevailing conception of the effects of environmental variability on population dynamics is that the variability generates "noise" around a deterministic process. In effect, this perspective implies that the relevant biological processes are described by a deterministic skeleton (see Cushing et al. 2003, chap. 2 and Dennis et al. 2001), which is valuable for its relative simplicity both conceptually and mathematically. One can view environmental variation as perturbations to a well-understood

process. When viewed this way, greater variation in the environment generates larger perturbations to a system, with greater fluctuations away from a deterministic path. Mathematically, the noise perspective implies that population dynamics (on some scale) should vary at any give time symmetrically about the deterministic path, akin to residual variation with zero mean in regression models. Much like the principle of superposition in physics (Oppenheim, 1967), the traditional perspective of environmental fluctuations is that they add to deterministic fluctuations, with some modulation of fluctuation intensity. Despite this simplicity, a failure of this perspective is that it disallows for interactions between environmental stochasticity on deterministic dynamics, such as those illustrated here. A first step in understanding interactions between deterministic and stochastic dynamics more generally is developing a way to measure them, which we do here using the concept of variance partitioning.

To measure how much population size variance deviates from the simple conceptual perspective, we partitioned population size fluctuations as follows:

$$\mathrm{Var}(N) = \sigma^2_{\mathrm{Det}} + \sigma^2_{\mathrm{Stoc}} + \sigma_{\mathrm{Int}} \tag{8}$$

(derivation in supplementary material S7). Equation [8] writes the variance in population size as a sum of three components: σ^2_{Det}, the variance in population size in a purely deterministic model, σ^2_{Stoc}, the contribution of stochastic variation on top of the deterministic variation assuming a "noise" based perspective, and σ_{Int}, the remaining variance in population size. Of most relevance to the understanding of interactions between deterministic and stochastic dynamics is that the magnitude of σ_{Int} measures the deviation from a principle of superposition. The principle of superposition holds when $\sigma_{\mathrm{Int}} = 0$, in which case variation in population size can be thought of as additive effects of deterministic (σ^2_{Det}) and stochastic variation ($\sigma^2_{\mathrm{Stoch}}$). However, if environmental stochasticity amplifies deterministic variation, $\sigma_{\mathrm{Int}} > 0$. If environmental stochasticity diminishes deterministic variation, $\sigma_{\mathrm{Int}} < 0$.

Such a partitioning of the variance in population size reveals potential interactions between deterministic and stochastic processes. The principle of superposition implies that $\sigma_{\mathrm{Int}} = 0$ because, in that case, stochastic variation generates "noise" around the deterministic attractor. Hence, the variance in population size reflects additive components of deterministic and residual stochastic variation.

Using this measure, it is clear that the Ricker model follows the principle of superposition under simple density-dependence and exogenous cycles, but deviates substantially from superposition for endogenous cycling (Fig. 6). The value of σ_{Int} is no different from zero under exogenous cycling and simple density-dependence, but σ_{Int} is a large negative value for endogenous cycling, which represents a strong effect of complex density-dependence damping environmental variability. Similar results hold for the outbreak model, with σ_{Int} being negative with larger magnitude as k increases (not shown).

These findings reinforce our understanding that the reduction in the variance of population abundance with environmental variability results from interactions between environmental stochasticity and density-dependence. Most importantly, most models of population growth used to understand the effects of increasing climate variability do not allow for interactions between environmental stochasticity and density-dependence. This fact should be a point of concern/consideration for population dynamics modeling in an era of climate change research, as we are possibly working with a toolkit with limited ability to describe plausible outcomes.

Discussion

As the climate continues to change, one expectation is that conditions will become more variable over time. With such increasing environmental variability, a common worry is that many populations will fluctuate more wildly in abundance, with the increased probability that they drop to low enough numbers where they are at risk of extinction. Such a perspective derives from models assuming populations fluctuate around a steady state (Arnoldi et al., 2018, 2019; Cuddington and Yodzis, 1999; Ives et al., 2003). Here, we considered the response of non-equilibrium dynamics, namely cycles, to increasing environmental variability. We found that the mechanism of population cycling is a crucial factor determining population responses to environmental variability. Exogenous cycles (i.e., those caused by a dynamically unlinked cycling factor) admit greater population fluctuations in response to environmental variability in much the same way as standard models assuming a carrying capacity do. Endogenous cycles, however, respond to environmental variability in a qualitatively different way; environmental variability can actually diminish population fluctuations, reflecting more stable dynamics. With less variable population size, exogenous cycling populations may be less vulnerable to global change than might otherwise be predicted.

Why does environmental variability reduce population size variation for some cycles and increase it for others? We found that the complexity of density-dependence in shaping the geometry of population dynamics (as visualized with dynamical potentials like those in Fig. 1 and 4b) plays an important role. In our exogenously cycling model, density-dependence is similar to that in traditional models with a single equilibrium, but where the single equilibrium moves with the value of the cycling factor. Importantly, the shape of density-dependence around the moving equilibrium is no different than if the equilibrium were fixed in time. Thus, stochastic environmental variability acts in much the same way in this case as it does in the case of simple density-dependence (Fig. 2a-b), albeit with some variability generated by the cycle itself. Endogenous cycles, however, have complex (i.e., highly nonlinear) geometry affecting population dynamics (Fig. 1c), and such complexity allows for non-trivial ways by which it interacts with environmental variability.

The mechanism of population variance damping reflects an interaction between stochastic variation and density-dependence that relies on the presence of long-transients in the deterministic system. A long transient is a dynamic that is not represented in the asymptotic behavior of a dynamical system, but lasts for multiple generations and therefore could be relevant on the timescales of typical ecological observations (Hastings et al., 2018). We found a long transient around the unstable equilibrium bisecting the alternative stable states of the deterministic 2-cycle and assumed it played a role in variance damping. To test this idea, we constructed a phenomenological model of insect outbreaks where we could control the length of deterministic transients (Fig. 4). We found clear evidence that variance damping in the stochastic system occurs when transients in the corresponding deterministic system are long, and variance damping in the stochastic system does not occur when transients in the corresponding deterministic system are short (Fig. 5). Furthermore, we developed a scheme to quantify interactions between stochastic and deterministic processes (def. in [8]). Using this scheme, we illustrated the fact that a reduction in population size variance with increasing environmental variability can be measured and is negative for models of complex density-dependence (Fig. 6).

Others have noted that environmental variability can interact in complex ways with deterministic dynamics. However, these studies almost always emphasize an interaction where system variability amplifies environmental variability (i.e., a positive interaction in [8]). For example, en-

vironmental variability can push a system currently in one stable state to another (Hastings et al., 2021) or "stochastically flicker" between alternative stable states (Gardner et al., 2000; Touboul et al., 2018). In addition to stochastic flickering, stochastic resonance (Gammaitoni et al., 1998) and stochastic amplification (Alonso et al., 2007) both describe interactions between stochastic variation and deterministic dynamics that enhance system variability. Here, we present a phenomenon where increases in stochastic variation reduce overall system variability. This appears to be most similar to inverse stochastic resonance, where oscillatory dynamics collapse to a fixed point with stochastic variation (Touboul et al., 2018), although we note that our model still expresses cycles, but they are weaker in amplitude, on average.

One question worthy of further investigation is whether the conditions are present in nature to give rise to the variance damping phenomenon. Given the difficulty that comes with fitting complex, nonlinear functions and the relatively long time series required to estimate the strength of density-dependence (much less its shape), we expect it to be challenging to identify variance damping in nature. Nonetheless, laboratory model systems—in which these issues are less constraining—have proven extraordinarily helpful in understanding nonlinear dynamical systems in the past (Cushing et al., 2003) and recent studies have estimated complex dynamics using microbial populations (Chang et al., 2021). Determining the shape of density-dependence in empirical systems will likely require highly flexible model fitting approaches such as a generalized additive models (Wood, 2006) or model-free forecasting (Perretti et al., 2013).

That said, even if there is limited evidence (or power to detect) strong interactions between environmental stochasticity and density-dependence, there is still much value in recognizing its presence in models, for models are the main inferential and prediction tools in population and conservation biology (Murray and Sandercock, 2020; Sutherland et al., 2020). Understanding what behaviors any given model can produce is a pre-requisite for the task of doing proper inference. The Ricker model is a workhorse for the study of population dynamics, being one of the simplest models used to fit observed population time-series and flexible enough to encompass simple and highly nonlinear dynamics, as illustrated here. Recent analyses suggest that the prevalence of nonlinear dynamics is much more common than previously appreciated (Clark and Luis, 2020; Rogers et al., 2022), so understanding the behavior of the Ricker model is paramount to making sound inferences about the future of these populations. We hope that these results motivate further development of models to deal with complex dynamics. As it currently stands, our baseline understanding of environmental variability is constrained by the simplicity models we use to describe populations. What we show here is that even elementary modifications to simple models may reveal unexpected and non-intuitive, yet plausible and game changing, results for extinction risk studies. Simple population models with environmental stochasticity remain widely understudied mathematical microscopes (sensu Cohen (2004)) of natural systems.

Acknowledgements

Many thanks to Bob Holt and the Holt lab group for comments on previous versions of this manuscript. Funding was provided by the National Science Foundation grant NSF-DMS-2052372 to JF and JMP.

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Figures

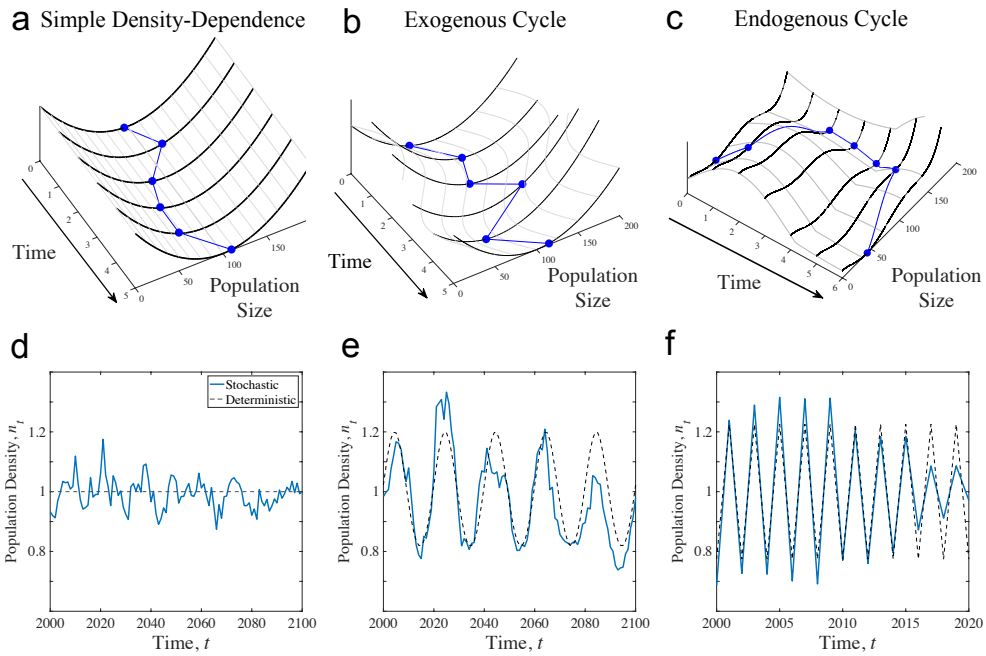

Figure 1: Conceptualization of environmental variability on population dynamics in different models. Under standard models of density-dependence (a), population fluctuations reflect a ball (blue lines) bouncing in a bowl (heavy black lines), where the depth of the bowl (z-axis in all plots) reflects the action of density-dependence. Environmental variation drives random perturbations of the ball away from the bottom of the bowl, which is the carrying capacity in this framework. A conception of exogenous cycles (b) is that the position of the bottom of the bowl moves with the exogenous driver, but the shape of the bowl is unchanging. A conception of endogenous cycles (c) is that the shape and action of density-dependence (black lines) changes over time, meaning that when the population is at low density, the equilibrium moves to higher density, but at high density, the equilibrium moves again to lower density. The topography of the landscape of population change – the bowl itself – is thus changing with the state of the population. Clearly, the conceptual and mathematical frameworks of standard density-dependent models do not immediately extend to the case of endogenous cycles. (d) - (f) Examples of the three types of dynamics in the Ricker model, as considered here.

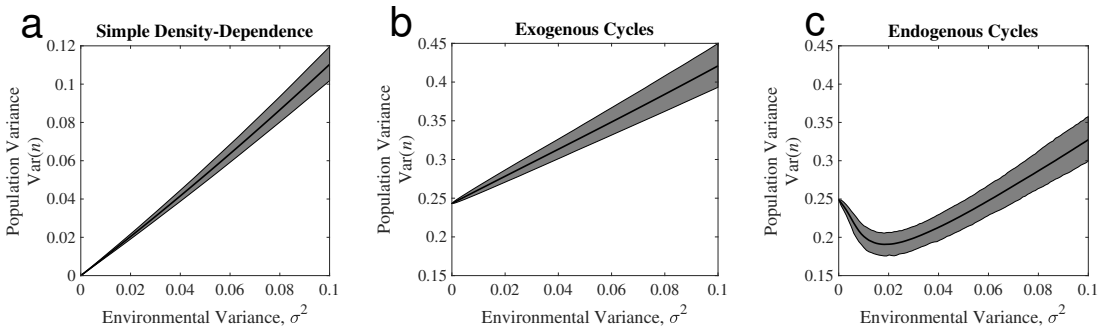

Figure 2: Effect of changing environmental variability for three qualitatively different kinds of dynamics. Each plot shows the average variance of population size over the time series across 1000 replicates (solid black line) as well as the the 95% upper and lower quantiles across replicates. Parameters: In (a), $\bar{a} = 1$, $\sigma_a^2 = 0$, and $b = 1$. In (b), $\bar{a} = 1$, $\sigma_a^2 = 0.25$, and $b = 1$. In (c), $\bar{a} = 2\ln\{3\} \approx 2.2$, $\sigma_a^2 = 0$, and $b = 2\ln\{3\}$. The value of a in (c) is chosen so that $\mathrm{Var}(N)$ in the deterministic cycling models are approximately the same, thereby facilitating comparisons of the two different mechanisms of cycles.

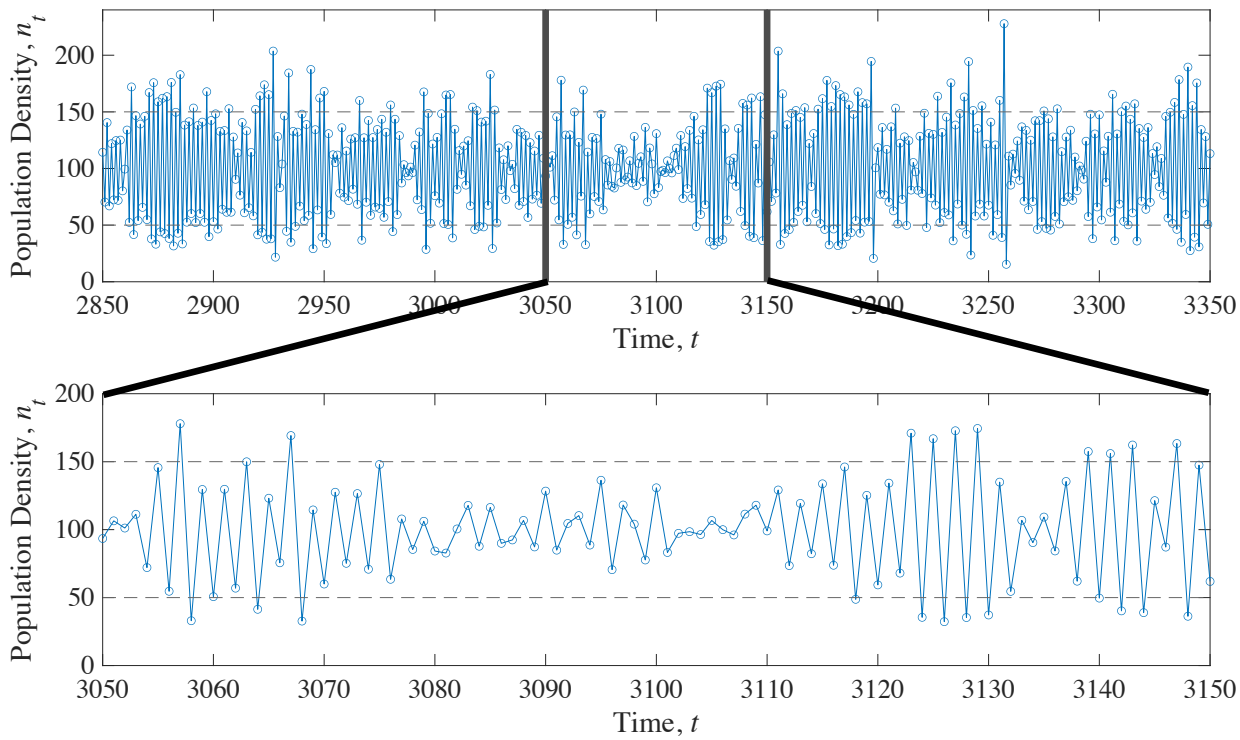

Figure 3: Sample path for the stochastic Ricker model in the case of endogenous cycles showing the origin of stochastic variance induced population fluctuations reduction. Periodically, the population gets "stuck" near the unstable equilibrium separating the two points of the deterministic cycle. In this example, the deterministic model cycles between the two fixed points $n_t = 50$ and $n_t = 150$ (dashed lines). The mean population density (here $\bar{n} = 100$) is an unstable fixed point. In this example $a = 2\ln\{3\}$, $b = a/100$, and $\sigma^2 = 0.02$.

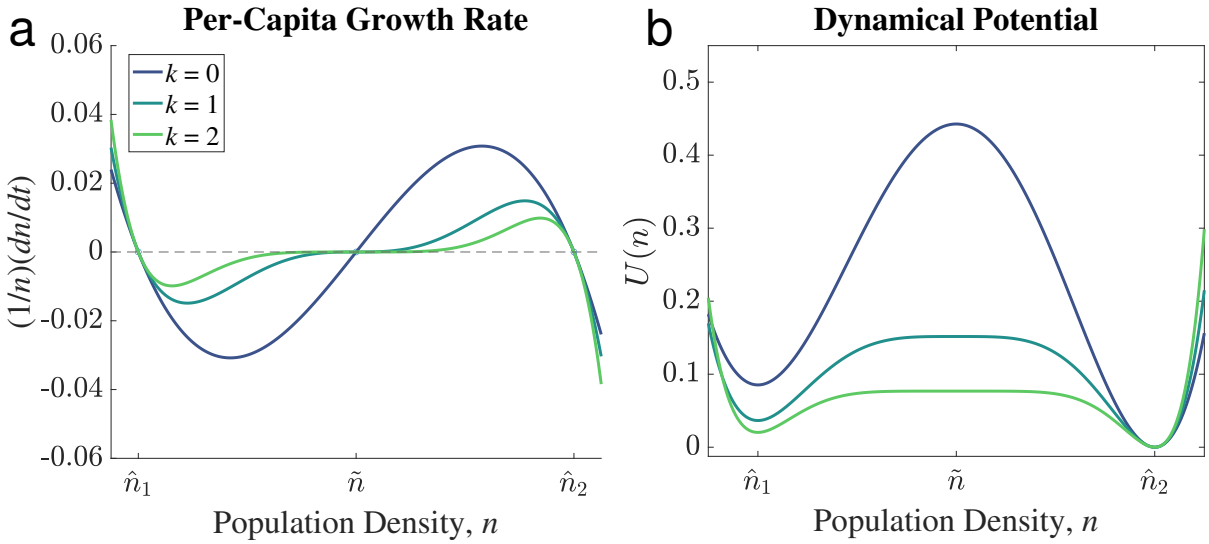

Figure 4: Features of the phenomenological model with alternative stable states without seasonal forcing. (a) The per-capita growth rate function (i.e., $1/n \cdot dn/dt$) when there is no seasonal forcing ($c_2 = 0$) and no environmental stochasticity ($\sigma = 0$). The model has two stable equilibria ($\hat{n}_1$ and $\hat{n}_2$) separated by an unstable equilibrium ($\tilde{n}$). The unstable equilibrium becomes nearly neutral as k increases. (b) The perspective of the model as a physical potential, where it is apparent that deterministic forces pushing a population towards stable equilibria (i.e., the troughs of U) weaken for larger values of k. Parameter are chosen so that the local stability properties of $\hat{n}_1$ and $\hat{n}_2$ are constant for all values of k.

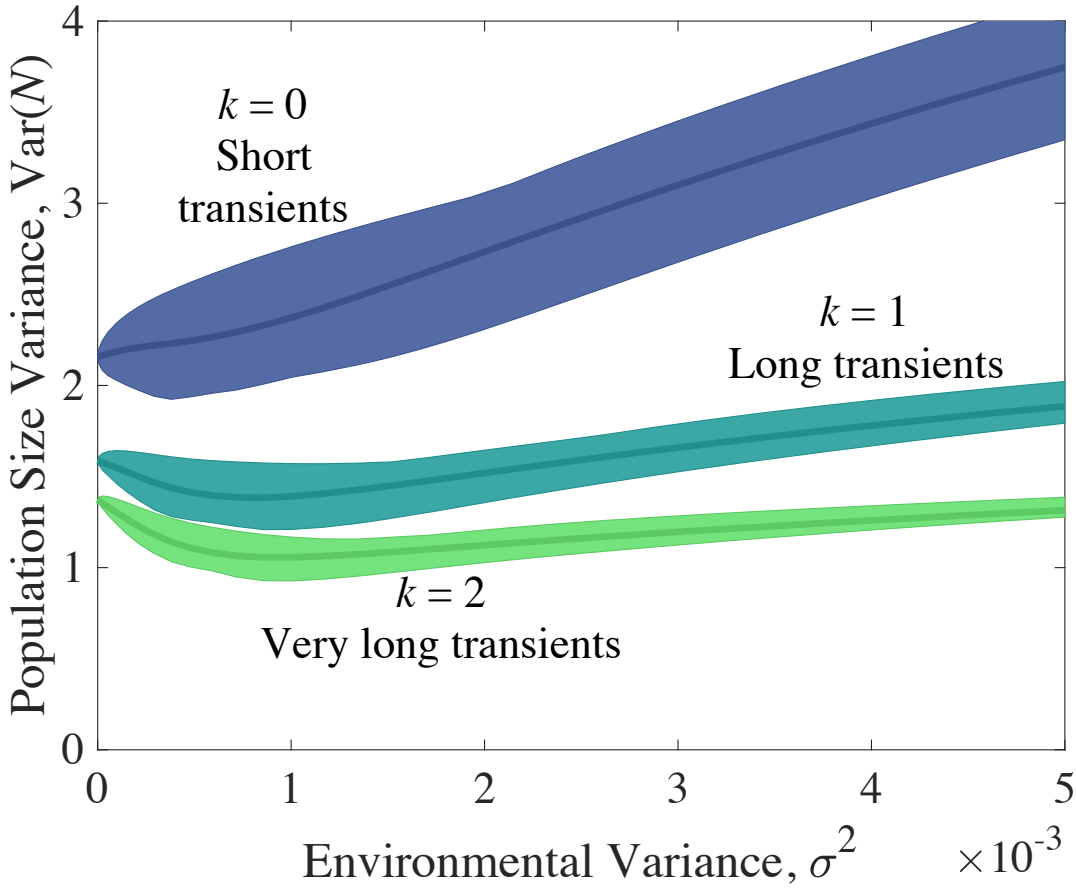

Figure 5: Population variance responses to environmental stochasticity in the outbreak model. Each band of color represents a different value of k, representing the local contours of density-dependence around the unstable equilibrium. Larger values of k correspond to weaker stability of the equilibrium, as measured by the local slope of the dynamical potential U, and longer transient time to reach either alternative state. Each band shows the mean (heavy line) and the range of $\mathrm{Var}(N)$ corresponding to 95% of the population size variance across 20 replicate sample paths of the stochastic model. Population size variance is calculated over 6 population cycles. Parameters: $\Delta t = 0.005$, $\Omega = 500$, $\hat{n}_1 = 10$, $\hat{n}_2 = 12$, $\tilde{n} = 11$, $\gamma = 0.001/(\hat{n}_2 - \hat{n}_1)^{2k}$, and $\delta = 0.005$.

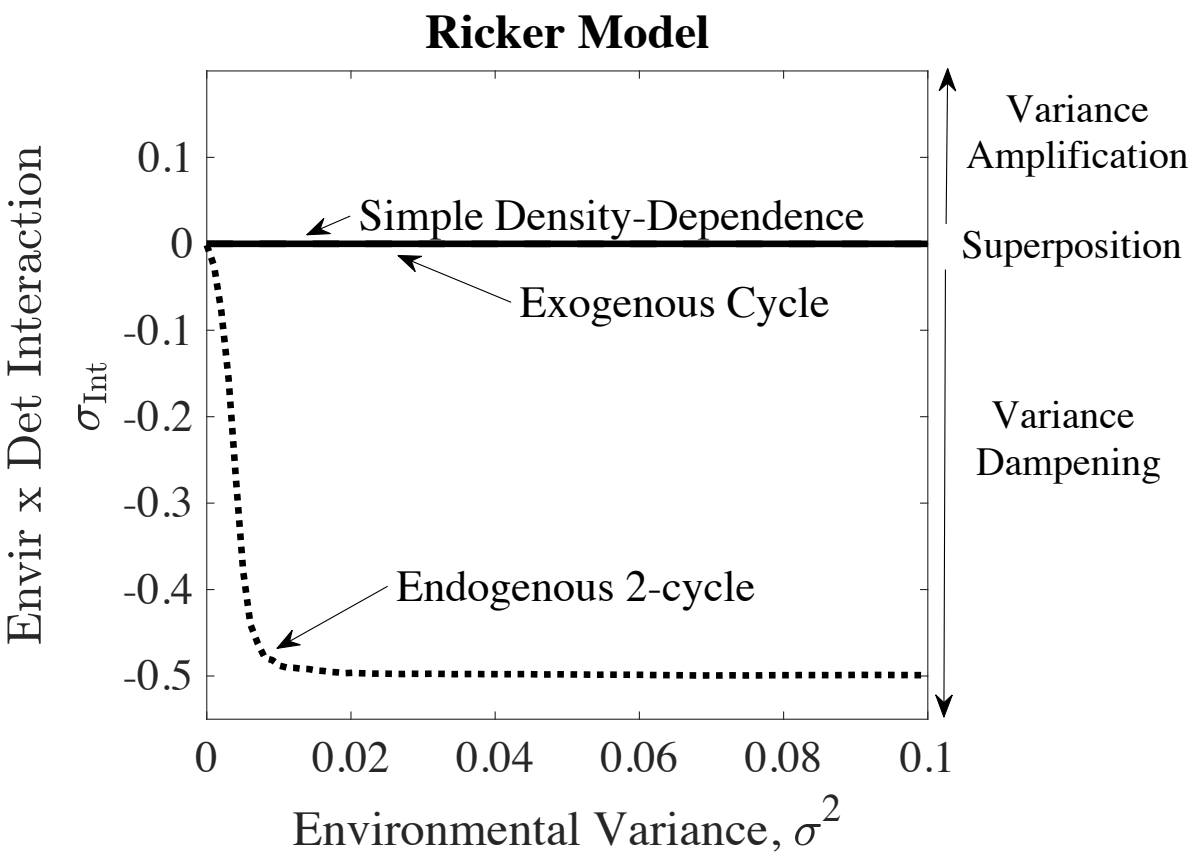

Figure 6: Level of interaction between environmental variability and deterministic processes as measured by σ_{Int} (eq. [8]). Quantifying the interaction is done by numerically estimating σ_{Int} under the three scenarios in Fig. 2. The lines for simple density-dependence and the exogenous cycle lie exactly on top of each other on this scale and so it appears as if there is only a single line.