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VARIATION IN RISK IN SINGLE-SPECIES DISCRETE-TIME MODELS

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Abstract. Simple, discrete-time, population models typically exhibit complex dynamics, like cyclic oscillations and chaos, when the net reproductive rate, R, is large. These traditional models generally do not incorporate variability in juvenile "risk," defined to be a measure of a juvenile's vulnerability to density-dependent mortality. For a broad class of discrete-time models we show that variability in risk across juveniles tends to stabilize the equilibrium. We consider both density-independent and density-dependent risk, and for each, we identify appropriate shapes of the distribution of risk that will stabilize the equilibrium for all values of R. In both cases, it is the *shape* of the distribution of risk and not the amount of variation in risk that is crucial for stability.

1. **Introduction.** Much of Tom Hallam's impact on theoretical ecology can be traced back to his leadership of a series of courses and conferences in the 1980s at the International Center for Theoretical Physics in Trieste, Italy. The first of these included a lucid introduction to theory for population dynamics in a homogeneous environment ([9]). A large part of Hallam's paper focused on simple, single-species models with density-dependence that might seem too simplistic to yield ecological insight, as all natural populations experience spatial and temporal variability in their environment and interact with other species. Yet more recent work ([18, 17]) has demonstrated that many natural populations exhibit population dynamics consistent with the interactions incorporated into these single-species models. In particular, [18] identified many populations with *delayed feedback cycles*, as predicted by many discrete-time, single-species models with parameter values leading to an unstable equilibrium (e.g. [15]). It is therefore important to understand how the variability that is inevitably present in natural populations impacts the stability of equilibria in the simple models. This paper explores one form of such variability.

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We work with the simplest family of single-species discrete-time models, those describing population dynamics of organisms that have one year life cycles, reproduce in a discrete pulse determined by season, and are regulated through a single density-dependent process. We recognize two life stages: reproductively mature adults and "juveniles," a term we use to include all earlier stages. We write the dynamical equation representing year-to-year population changes in the form

$$H_{t+1} = RH_t f(cH_t), \tag{1}$$

where R represents the net reproduction rate, i.e., the geometric rate of increase (or decrease) of a small population, and the function f denotes the fraction of juveniles that escape some additional, density-dependent mortality. The variable H_t denotes the population density for the stage that determines the juvenile survival, f. For example, if the population is regulated by cannibalism of juveniles by adults, then H_t would represent adult density. We discuss how other mechanisms influence the interpretation of H_t in Section 3. The precise form of the function f is determined by calculating the net effect of density-dependent juvenile mortality throughout the juvenile stage ([6]). The parameter c is a measure of the strength of density dependence; it has dimensions (density)$^{-1}$ and characterizes the "risk" faced by an individual due to conspecifics in its environment. We further develop the concept of risk in Section 3.

The effects of variability in risk across individuals have been studied extensively in host-parasitoid models, where "risk" is a measure of the vulnerability of an individual host to parasitoid attacks. The authors of [1] showed that if risk is gamma-distributed, then high variability in the risk of parasitism among individual hosts can stabilize the otherwise unstable equilibrium of the Nicholson-Bailey host-parasitoid model ([19]). In [13] it was proposed that a condition for stability is $CV^2 > 1$, where CV is the coefficient of variation[1] of the distribution of risk. An important restriction is that risk is independent of local host density if the host is nonuniformly distributed in space ([3]). This "$CV^2 > 1$ rule," and earlier results stressing the role of spatially aggregated parasitism (e.g. [16]), stimulated hundreds of studies investigating parasitism patterns in the field. Recently, [22] challenged the generality of the $CV^2 > 1$ rule by showing that the *shape* of the distribution of risk, and not its coefficient of variation, determines the stabilizing effects of variability in risk in host-parasitoid systems.

Perhaps surprisingly, the literature on variability in risk in *single*-species models is much thinner. It is well known for the Ricker model ($f(cH_t) = \exp(-cH_t)$ in (1)) that for specific distributions of risk across juveniles, sufficiently large variation in juvenile risk, i.e., variability in each juvenile's ability to survive to adulthood, can stabilize an otherwise unstable model equilibrium [4, 11]. We here derive results that generalize this stability condition to any arbitrary function f and distribution of risk across juveniles $p(x)$ for cases when risk is density-dependent (i.e., juvenile risk is completely determined by the local juvenile density) and density-independent (i.e., juvenile risk is independent of the local juvenile density). We show that as in host-parasitoid systems, stabilizing effects of variability in risk in single-species models are also tied to the *shape* of the distribution $p(x)$. More specifically, in both cases when juvenile risk is density-independent and density-dependent, the model equilibrium is stabilized for all R if $p(x)/x$ is a non-increasing function. We further show that when risk is density-dependent, the distribution of risk $p(x)$ is

[1] Coefficient of variation is defined as the ratio of the standard deviation to the mean.

related to $a(x)$, the distribution of juveniles across patches, and $p(x)$ will have the appropriate shape (i.e., $p(x)/x$ is a non-increasing function) when $a(x)$ itself is a non-increasing function of x. We emphasize throughout that it is the *shape* of the distribution of risk and *not* the *amount* of variation in risk that is crucial for stability. Indeed, we show that with certain distributions of risk, even arbitrarily large variation in juvenile risk will not stabilize the equilibrium, and in some cases, increasing variability beyond a certain point *destabilizes* the equilibrium.

2. **Preamble: equilibrium and local stability.** We assume that the net reproductive rate $R > 1$ and that f is a monotonically decreasing function with

$$f(0) = 1 \quad \text{and} \quad \lim_{x\to\infty} f(x) = 0. \tag{2}$$

With these assumptions, the unique, positive equilibrium H^* of the discrete-time model (1) is given as the solution of the equation

$$\frac{1}{R} = f(cH^*), \quad R > 1. \tag{3}$$

A number of texts (e.g. [14]) discuss local stability analysis and bifurcations in this model. Typically, the parameter c is recognized as setting the scale of population density and stability is related to the parameter R. For the work to be reported in this paper, it is useful to relate local stability to the *sensitivity* of the equilibrium to changes in R, defined by

$$S = \frac{R}{H^*}\frac{dH^*}{dR} = \frac{d\log(H^*)}{d\log(R)}. \tag{4}$$

In Appendix A, we show that the equilibrium is locally stable if and only if $S > \frac{1}{2}$. This condition in turn implies that the equilibrium is stable, if and only if, $H^*/\sqrt{R}$ *is an increasing function of* R. We further show that the equilibrium is *monotonically stable*, if and only if, $S > 1$, which corresponds to H^*/R being an increasing function of R.

3. **Incorporation of juvenile risk.** In this section, we flesh out the idea of juvenile "risk," represented by the parameter c in equation (1). We do this by considering two different biological mechanisms that can lead to this model. In both mechanisms H_t represents the population density for the stage that determines the fraction f of juveniles that survive to become the adults for the next year.

3.1. **Adults attack juveniles.** Here, H_t denotes the adult density and RH_t denotes the juvenile density at the *beginning* of the juvenile stage in year t. We assume that juveniles are cannabilized by adults and face a *constant, density-dependent* per capita mortality rate given by $g(cH_t)$ for some parameter c and monotonically increasing function g. If the duration of the juvenile stage is given by T, then the fraction of juveniles that survive at the end of the juvenile stage is given by

$$f(cH_t) := \exp(-g(cH_t)T). \tag{5}$$

Without loss of any generality we take $T = 1$ from now on. These surviving juveniles then become the adults for the next year to give the discrete-time model (1). If adults have a linear functional response with attack rate c, then $g(cH_t) = cH_t$, and hence we have $f(cH_t) = \exp(-cH_t)$ in equation (1), which corresponds to the well-known Ricker model ([21, 6]).

3.2. **Juvenile face intraspecific competition.** We now consider the situation where the population is regulated by intraspecific competition among the juveniles. In this case, the fraction of juveniles that survive is determined by the initial juvenile density. Hence, for this mechanism, H_t represents the *juvenile* density at the *beginning* of the juvenile stage in year t. The density of juveniles that survive and become the adults for the next year is then $H_t f(H_t)$. These adults lay eggs that mature into juveniles, giving a discrete-time model of the form in equation (1).

The rationale is discussed in [8] and [26]. In brief, the form for f is obtained using a semi-discrete approach ([23, 7]) as follows. Let τ denote the time inside the juvenile stage which varies from $0 \leq \tau \leq 1$ (the duration of the juvenile stage is assumed to be 1 time units). Let $H(t,\tau)$ denote the juvenile density in year t at a time τ within the juvenile stage. We assume that the per capita mortality rate at a time τ in the juvenile stage is given by $g(cH(t,\tau))$ for some parameter c and monotonically increasing function g. Then, $H(t,\tau)$ is the solution to the following ordinary differential equation:

$$\frac{dH(t,\tau)}{d\tau} = -H(t,\tau)g(cH(t,\tau)), \quad H(t,0) = H_t. \tag{6}$$

The fraction surviving $f(cH_t) = H(t,1)/H(t,0)$ is given by

$$f(cH_t) = \frac{z(1)}{cH_t}, \tag{7}$$

where $z(\tau)$ is the solution of

$$\frac{dz(\tau)}{d\tau} = -z(\tau)g(z(\tau)), \quad z(0) = cH_t. \tag{8}$$

For example, when we take

$$g(cH(t,\tau)) = (cH(\tau,t))^b \tag{9}$$

for some constant $b > 0$, the above semi-discrete analysis leads to

$$f(cH_t) = \left(\frac{1}{1 + b(cH_t)^b}\right)^{1/b}. \tag{10}$$

With both mechanisms, one can think of the parameter c in model (1) as a property of an individual juvenile, that determines its susceptibility to density-dependent mortality, and can be interpreted as the "risk" experienced by that juvenile.

4. **Variability in risk.** Our model formulation to this point assumes that all juveniles have the same value of c. Variability in risk is introduced by assuming that each juvenile has a *different* value for the parameter c, which is treated as a random variable. We define the distribution of risk (c) across juveniles, $p(x)$, by specifying that the probability of a juvenile having a c value (risk) in the infinitesimally small interval $[x, x + dx]$ is $p(x)dx$. We consider both density-independent and density-dependent risk.

4.1. **Variability in density-independent risk.** We first consider the situation when risk is *independent* of the local juvenile density. This would be the case, for example, when a particular juvenile's risk is determined by its phenotype or by its micro-habitat. As mentioned above, the probability of a juvenile having a c value (risk) in the infinitesimally small interval $[x, x + dx]$ is $p(x)dx$. The probability of a juvenile with risk x surviving the juvenile stage is $f(xH_t)$, and the total fraction

of juveniles that survive is then $\int_{x=0}^{\infty} p(x)f(xH_t)dx$. Thus the population dynamics is described by the discrete-time model

$$H_{t+1} = RH_t \int_{x=0}^{\infty} p(x)f(xH_t)dx. \tag{11}$$

As discussed in Section 3, depending on the biological mechanism that creates the density-dependent mortality, H_t will either denote the adult density or the juvenile density at the beginning of the juvenile stage in the above model. We discuss alternative ways of defining a juvenile's risk in Appendix B. Depending on the form of the function f, these alternative definitions may, or may not, lead to the same form of the model as (11), a point we revisit briefly in the Discussion.

With the interactions that lead to the Ricker model (see Section 3.1), ($f(xH_t) = \exp(-xH_t)$), the integral in equation (11), $\int_{x=0}^{\infty} p(x)f(xH_t)dx$, is simply the moment-generating function of the distribution $p(x)$ evaluated at $-H_t$. Hence, in this case, if $p(x)$ is a gamma distribution (mean equal to m and coefficient of variation equal to V) we have the well-known model ([10])

$$H_{t+1} = \frac{RH_t}{(1 + mH_tV^2)^{1/V^2}}. \tag{12}$$

The Beverton-Holt model is a special case of this when $V = 1$ ([2]). When $p(x)$ is a uniform distribution (with mean m) given by

$$p(x) = \begin{cases} \frac{1}{2m} & \text{for} \quad 0 \le x \le 2m \\ 0 & \text{for} \quad x > 2m \end{cases} \tag{13}$$

we have the Skellam model ([24])

$$H_{t+1} = R(1 - \exp(-2mH_t))/2m. \tag{14}$$

We investigate the stability of the equilibrium H^* of the general discrete-time model (11) in terms of the distribution of risk. This equilibrium is given as the solution to the equation

$$\int_{x=0}^{\infty} p(x)f(xH^*)dx = \frac{1}{R}. \tag{15}$$

Standard local stability analysis show (see Appendix C for details) that this equilibrium is stable, if and only if,

$$-H^* \int_{x=0}^{\infty} xp(x)f'(xH^*)dx < \frac{2}{R}, \tag{16}$$

where $f'(xH^*)$ denotes the derivative of function f evaluated at xH^*. For a given function f and distribution of risk, the stability region of the model can be determined by numerically solving equations (15) and (16).

We now present results that connect the stability of the model equilibrium with the shape of the distribution of risk, and are *independent* of the function f. Details are in Appendix C where we show that the stability condition (16) can be re-written as

$$\int_{x=0}^{\infty} x^2 \left(\frac{d(p(x)/x)}{dx} \right) f(xH^*)dx < 0. \tag{17}$$

Consequently, a *sufficient* condition for the above equilibrium H^* to be stable *for all* values of $R > 1$ is that the distribution $p(x)$ be such that, $p(x)/x$ is a non-increasing function of x. Moreover, if $p(x)$ is a non-increasing function then the equilibrium will be monotonically stable *for all* values of $R > 1$ (Appendix C).

We now illustrate these conditions with some examples. If $p(x)$ is a gamma distribution with mean m and coefficient of variation V given by

$$p(x) = \frac{x^{(1/V^2-1)} \exp\left(-\frac{x}{mV^2}\right)}{(mV^2)^{1/V^2}\Gamma(1/V^2)}, \tag{18}$$

where Γ denotes the Gamma function, then $p(x)/x$ is a non-increasing function if $V^2 \geq 0.5$. Hence if risk is density-independent and gamma distributed, then the model equilibrium is stable for all $R > 1$ if $V^2 \geq 0.5$ (see Fig. 1 which plots the gamma distribution for this value). If $p(x)$ is a Weibull distribution ([27]) which has the form

$$p(x) = \frac{k}{\lambda}\left(\frac{x}{\lambda}\right)^{k-1} \exp -(x/\lambda)^k \tag{19}$$

for some constants k and λ, then the equilibrium is stable for all $R > 1$ if $k \leq 2$, which corresponds to $V^2 > 0.273$. The above two examples show that even though different distributions might have the right shape to stabilize the equilibrium (i.e., $p(x)/x$ is a non-increasing function), the amount of variability required, represented by the value of V, for stability might be very different. Thus just determining the coefficient of variation in risk will not be enough to conclude stability for all R unless the specific form of the distribution is known.

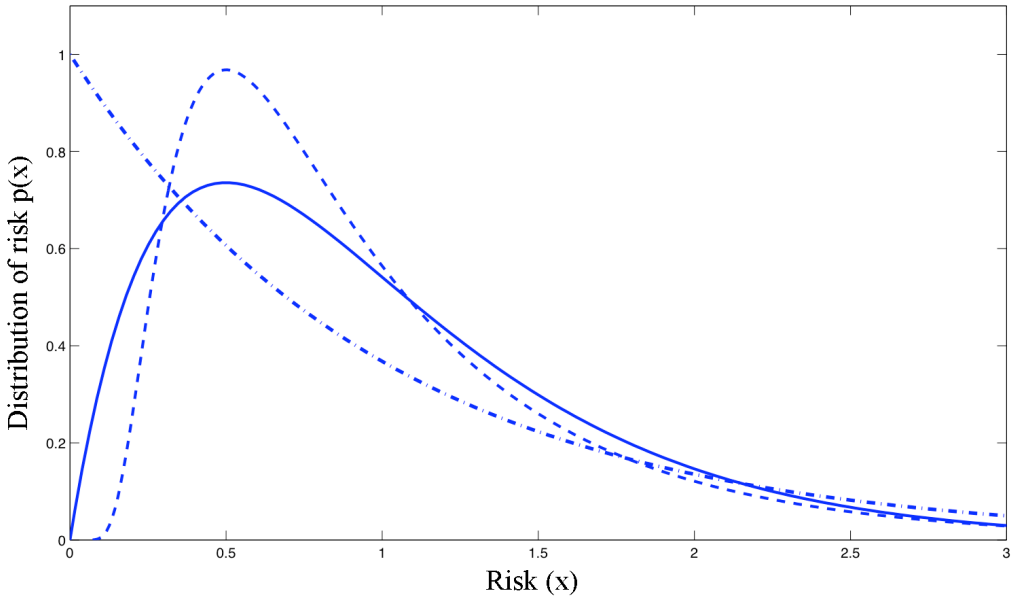

FIGURE 1. Plots of a gamma distribution with mean 1 and $V^2 = 0.5$ (solid line), $V^2 = 1$ (dashed-dot line) and inverse gaussian distribution with mean 1 and $V^2 = 0.5$ (dashed line).

If $p(x)/x$ is an increasing function near $x = 0$, then depending on the form of the function f, the corresponding equilibrium may be unstable for sufficiently large values of R. Note form (2) and (15) that as $R \to \infty$ the equilibrium $H^* \to \infty$. Thus, depending on whether the stability condition (17) holds (does not hold) for sufficiently large H^*, we have a stable (unstable) equilibrium for sufficiently large

R. For example, if $f(cH^*) = 1/(1 + cH^*)$ (the Beverton-Holt model), (17) always holds, and hence a stable equilibrium for all R irrespective of whether $p(x)/x$ is increasing or decreasing near $x = 0$. However, for $f(cH^*) = \exp(-cH^*)$ (the Ricker model) if $p(x)/x$ is an increasing function near $x = 0$, then the corresponding equilibrium will be unstable for sufficiently large R (see Appendix D). This critical value of the reproduction rate above which you get an unstable equilibrium can only be determined numerically by solving the stability conditions (15) and (17). For example, let the distribution of risk be an inverse gaussian distribution with mean m and coefficient of variation V given by

$$p(x) = \left(\sqrt{\frac{\lambda}{2\pi x^3}}\right) \exp\left(\frac{-\lambda(x-m)^2}{2m^2 x}\right), \quad \lambda = \frac{m}{V^2}, \tag{20}$$

for which $p(x)/x$ is an increasing function near $x = 0$ (see Fig. 1 for a plot of the inverse gaussian distribution). For $m = 1$ and $V^2 = 0.5$, numerical analysis of the stability conditions with the Ricker survival function ($f(xH_t) = \exp(-xH_t)$) show that the corresponding discrete-time model is stable for $1 < R < 16.9$ and unstable otherwise. For comparison purposes, the Ricker model with no variability has a stable equilibrium for $1 < R < 7.39$ ([17]). This example illustrates that even though the distribution of risk might not stabilize the equilibrium for all values of R, it can still, in some cases, significantly increase the stability region and push complex dynamics to large values of R (in this case $R > 16.9$), which may be biologically irrelevant.

It is important to point out that increasing variation in risk does not always correspond to an increase in the stability region of the model. In fact in some cases, increasing variability beyond a certain point *destabilizes* the equilibrium. Consider the distribution of risk given by

$$p(x) = \begin{cases} 0 & \text{for} \quad 0 \le x \le c^* \\ g(x - c^*) & \text{for} \quad x > c^* \end{cases} \tag{21}$$

for some function $g(x)$ and $c^* > 0$, which can be thought of as a function $g(x)$ shifted to the right by c^*. Such a distribution of risk explicitly excludes a refuge and implies that all juveniles have a minimum nonzero risk c^*. The corresponding discrete-time model is now given by

$$H_{t+1} = RH_t \int_{x=c^*}^{\infty} g(x - c^*) f(xH_t) dx. \tag{22}$$

If the function f is chosen such that the equilibrium of the model with no variability in risk (i.e., model (1)) is unstable for large enough R, then the model equilibrium of (22) will also be unstable for large enough R, irrespective of the function $g(x)$ (see Appendix E for details). Moreover, in this case, the corresponding stability region is maximized at intermediate levels of variability in risk (see Fig. 2). To illustrate this we take $f(xH_t) = \exp(-xH_t)$ (Ricker model) and let the function $g(x)$ in (22) have the form of a gamma distribution with mean $\bar{c}$ and coefficient of variation V_g. This implies from (21), a mean risk of $\bar{c} + c^*$ and coefficient of variation in risk

$V = \bar{c}V_g/(\bar{c} + c^*)$. In this case the model (22) becomes

$$H_{t+1} = RH_t \int_{x=c^*}^{\infty} g(x - c^*) \exp(-xH_t)dx = RH_t \exp(-c^* H_t) \int_{x=0}^{\infty} g(x) \exp(-xH_t)dx \tag{23}$$

$$= \frac{RH_t \exp(-c^* H_t)}{(1 + \bar{c}{V_g}^2 H_t)^{1/{V_g}^2}}. \tag{24}$$

Note that in the limit $V_g \to 0$ we recover the Ricker model

$$H_{t+1} = RH_t \exp(-(\bar{c} + c^*)H_t). \tag{25}$$

As we now increase V_g (and hence increase V) the stability region of the model (24) first increases (see Fig. 2), however at large values of V_g, increasing V_g decreases the stability region. The latter effect is because for sufficiently large V_g we have

$$H_{t+1} = \frac{RH_t \exp(-c^* H_t)}{(1 + c{V_g}^2 H_t)^{1/{V_g}^2}} \approx RH_t \exp(-c^* H_t), \tag{26}$$

and hence, in the limit $V_g \to \infty$ the stability region shrinks back to the stability region of the Ricker model. In summary, for functions f chosen as above, when all juveniles have minimum nonzero risk then the discrete-time model is stabilized for small values of R and the corresponding stability region is maximized for *intermediate* levels of variability in risk.

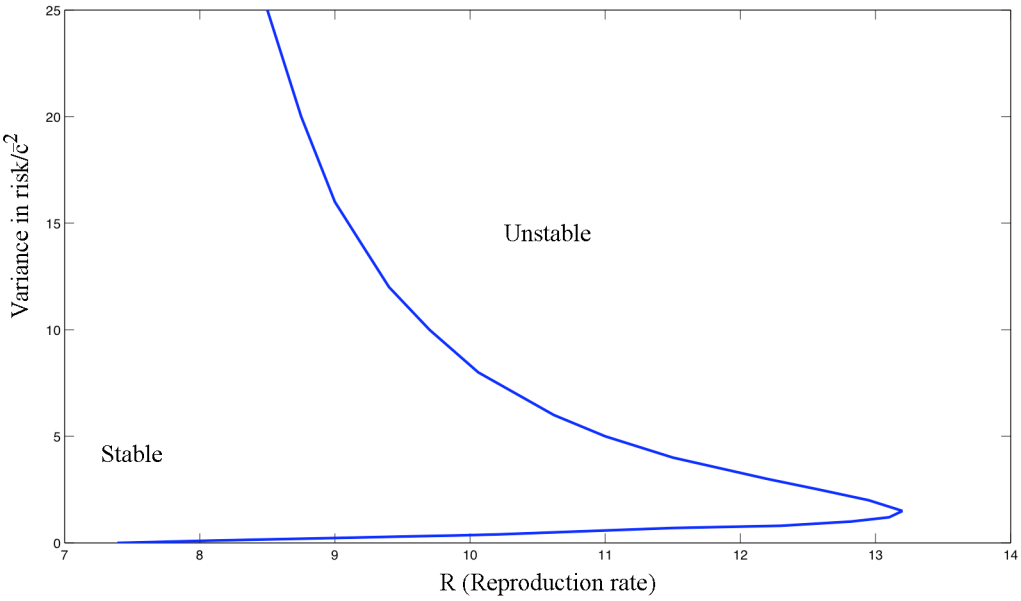

FIGURE 2. The stability region for the model (24) in terms of ${V_g}^2$ (Variance in risk/$\bar{c}^2$) and R when $\bar{c} = 1$ and $c^* = 0.5$. In this case the stability region is maximized when ${V_g}^2 \approx 1.5$.

We end this section by noting that stability induced due to variation in density-independent risk is generally accompanied by an increase in equilibrium H^* from the equilibrium value when there is no variability. This is illustrated in Fig. 3, which plots H^* for the Ricker model when risk is gamma distributed (discrete-time model

(12)). Note from the figure that the Ricker model is now stable for $1 < R < 9.32$ when $V^2 = 0.1$, $1 < R < 16$ when $V^2 = 0.25$ and stable for all $R > 1$ when $V^2 = 0.5$.

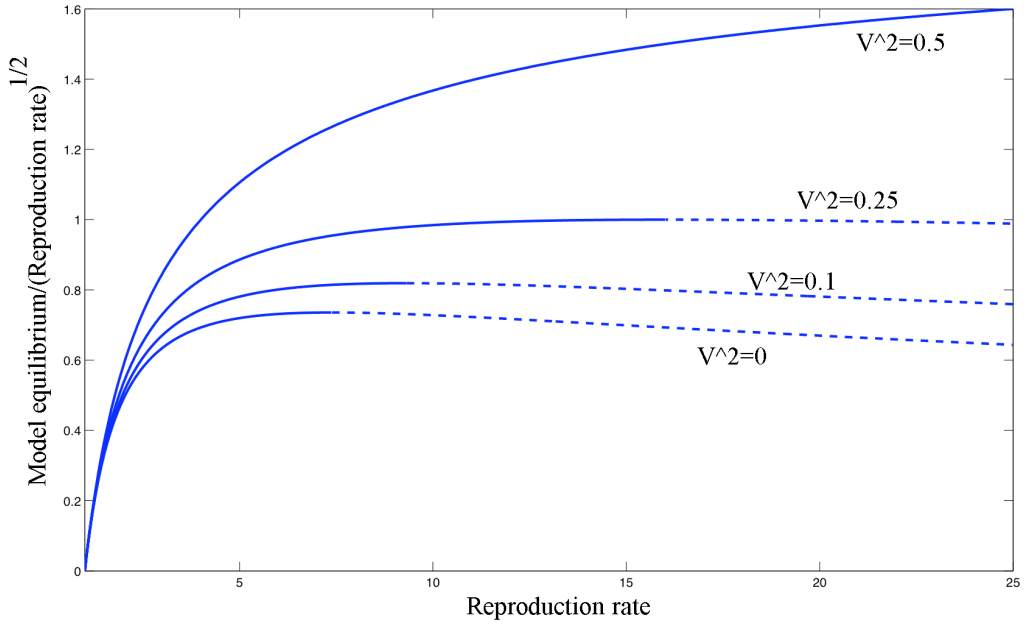

FIGURE 3. Plots of $H^*/\sqrt{R}$ for different values of the coefficient of variation (V) where H^* is equilibrium of the model (12). $V^2 = 0$ corresponds to the Ricker model with no variation in risk. Solid line corresponds to a stable equilibrium while a dashed line corresponds to an unstable equilibrium. Recall that the model equilibrium is stable iff $H^*/\sqrt{R}$ is an increasing function of R.

4.2. **Variability in density-dependent risk.** We now consider the scenario where a juvenile's risk is completely determined by the local juvenile density, i.e., the number of juveniles in the neighborhood. If we envision a situation where juveniles are spread non-uniformly in space across patches with finite resources, then it is natural that patches with higher juvenile density face stiffer intraspecific competition compared to patches with lower juvenile density. Hence, in this case a juvenile's risk in a particular patch is a function of the number of juveniles in that patch. The distribution of risk across juveniles is now determined by the distribution of juveniles across patches, which we denote by $a(x)$. Let H_t denote the average juvenile density *per patch* at the beginning of the juvenile stage. Then the probability of a patch having juvenile density in the infinitesimally small interval $[H_t x, H_t x + H_t dx]$ at the beginning of the juvenile stage is given by $a(x)dx$. Note that the way the distribution $a(x)$ is now defined it automatically has a mean of one, i.e., $\int_{x=0}^{\infty} xa(x)dx = 1$. If the function f represents the fraction of initial juvenile that survive the juvenile stage, then for a patch with initial juvenile density $H_t x$, the density at the end of the juvenile stage will be $H_t x f(H_t x)$. Hence, the average density of surviving

juveniles per patch is $\int_{x=0}^{\infty} H_t x a(x) f(xH_t) dx$, and we have the discrete-time model

$$H_{t+1} = RH_t \int_{x=0}^{\infty} x a(x) f(xH_t) dx = RH_t \int_{x=0}^{\infty} p(x) f(xH_t) dx, \tag{27}$$

where now $p(x) = xa(x)$ represents the distribution of risk across juveniles. The same form of model can be derived for the adult-attack-juvenile mechanism (Section 3.1) by assuming that juveniles distribute themselves across patches according to distribution $a(x)$ and the adult density on a given patch (and hence the mortality rate of the juveniles) is strongly correlated with the number of juveniles in the patch. Such a situation might arise, for example, for fish that that remain close to places where they have laid their eggs. Note that the discrete-time model (27) not only has spatial density dependence but also contains explicit temporal density dependence, i.e., the average mortality rate across patches increase when average density of juveniles across patches increases. Such temporal density dependence is crucial for stabilizing the above discrete-time model as spatial density-dependence on its own is generally not stabilizing ([25]).

Stability conditions for the discrete-time model (27) in terms of the distribution of risk $p(x)$ are identical to those derived in Section 4.1 for the discrete-time model (11), as both the models have the same form irrespective of whether risk is density-dependent or density-independent. We now formulate the stability condition for model (27) in terms of the distribution of juveniles across patches, $a(x)$, by substituting $xa(x)$ for $p(x)$ in the stability conditions of model (11) (Section 4.1). Thus we have from (17) that the model equilibrium H^* of (27) is stable, if and only if,

$$\int_{x=0}^{\infty} x^2 a'(x) f(xH^*) dx < 0. \tag{28}$$

A *sufficient* condition for the model equilibrium of (27) to be stable *for all* values of R is that the distribution $a(x)$ be a non-increasing function, i.e., the distribution has a mode at zero. Also note that if $a(x)$ is an increasing function near $x = 0$ (i.e., the distribution does not have a mode at zero) and function f is such that inequality (28) does not holds for sufficiently large H^* (for example, this would be true for the Ricker model), then the model equilibrium of (27) will be unstable for sufficiently large R.

We now illustrate these conditions through examples. If $a(x)$ is a uniform distribution given by

$$a(x) = \begin{cases} \frac{1}{2} & \text{for} \quad 0 \le x \le 2 \\ 0 & \text{for} \quad x > 2, \end{cases} \tag{29}$$

which is a non-increasing function, we have a stable equilibrium for all $R > 1$. If $a(x)$ is a gamma distribution given by (18), then when $V^2 \ge 1$ it is a non-increasing function, and hence, the model equilibrium of (27) is stable for all $R > 1$ (see Fig. 1 for a plot of the gamma distribution for this value). [4] and others ([11, 12]) derived this particular result, for a special case of the model (27) where $f(xH_t) = \exp(-cH_t)$ and $a(x)$ was taken as the gamma distribution, in which case the model (27) becomes

$$H_{t+1} = \frac{RH_t}{(1 + V^2 H_t)^{(1+1/V^2)}}. \tag{30}$$

Thus the main result presented in this section (stability for all $R > 1$ when $a(x)$ is non-increasing) is a generalization of the results of [4, 11], to any distribution

of juveniles across patches $a(x)$ and any form of density-dependent mortality (i.e., function f).

Fig. 4 plots the model equilibrium of (30) as a function of R for different values of V and illustrates that stability due to variation in density-dependent risk comes at a cost of a decrease in model equilibrium (when R is small) and an increase in model equilibrium (when R is large) from the model equilibrium value when there is no variability in risk.

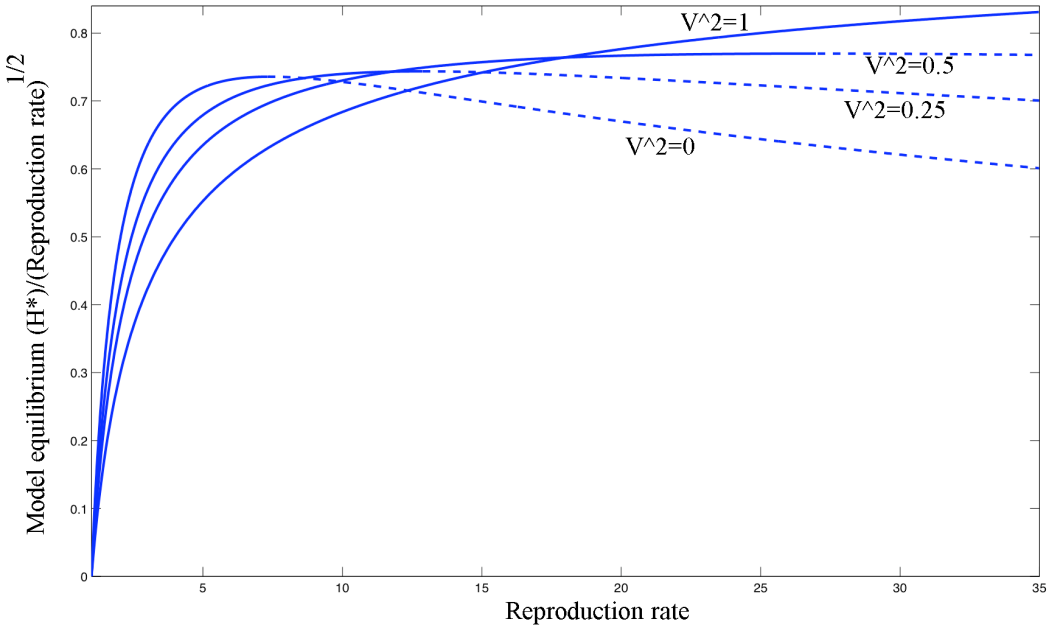

FIGURE 4. Plots of $H^*/\sqrt{R}$ for different values of the coefficient of variation (V) where H^* is equilibrium of the model (30). $V^2 = 0$ corresponds to the Ricker model with no variation in risk. Solid line corresponds to a stable equilibrium while a dashed line corresponds to an unstable equilibrium.

5. **Discussion.** Theory presented in this paper shows that variation in risk of mortality among juveniles can stabilize the model equilibrium of a general class of discrete-time models. We investigated both density-independent and density-dependent risk and identified the distribution of risk and distribution of juveniles across patches (when risk is density dependent) that will stabilize the model equilibrium for all values of the reproduction rate R. For certain models such as the Ricker model, we identified distributions that inevitably lead to instability, and probably to complex dynamics, for large values of R. We showed that stability due to variation in density-independent risk is generally associated with an increased model equilibrium when compared with the model equilibrium with constant risk.

Our results connecting shape of the risk distribution to stability depend critically on how risk enters the function f, which defines the probability that a juvenile will survive to become an adult. For our results to hold, a juvenile's risk x has to be defined in such a way that it appears in the function $f(xH_t)$ multiplicatively with

H_t. We discussed various biological mechanisms where this would be true, as well as noting exceptions (see Appendix B). Numerical stability analysis of some models where this assumption is not true shows that variability in risk can still stabilize the corresponding equilibrium of the model. However, in this case the shape of distribution required for stability may depend on the way the risk x enters the function f.

The mechanism whereby an unstable equilibrium becomes a stable equilibrium in the presence of variation in risk is similar to that of a fractional refuge. It is well known from previous work that the presence of a fractional refuge, i.e., a fraction of juveniles survive with probability one, can stabilize the equilibrium of a discrete-time model ([17]). Variation in risk similarly creates a situation where some juveniles have a high probability of survival. The existence of these juveniles buffers the population from perturbations and contributes to the stability of the model. The importance of these low-risk juveniles was illustrated here in the case when we explicitly excluded a refuge and all juveniles had a non-zero, minimum risk. In such a situation the corresponding discrete-time model could never be stabilized for certain range of parameters, irrespective of the form of the distribution of risk.

The primary limitation of our work is the assumption that risk can be defined so that its effects operate multiplicatively with some measure of population density. Typically derivation of the survival function requires the "semi-discrete" approach (e.g. [20, 23]). One problem is that the resulting model may take the form

$$H_{t+1} = RH_t f(R, cH_t) \tag{31}$$

rather than equation (1). Then, our results relating stability to the variation of H^* with R would not necessarily hold.

In summary, we have developed theory on the effects of variation in risk in single-species models that generalizes previous work, notably that of [11]. Our findings on the importance of the distribution of risk parallel similar recent findings with host-parasitoid models ([22]). Our most important findings relate to the presence of individuals that experience very low risk. This presents serious challenges for empiricists, as identifying such individuals is much more challenging than obtaining some measure of variability such as the coefficient of variation.

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Appendix A. **Stability analysis for the discrete-time model** (1)**.** Consider the discrete-time model

$$H_{t+1} = RH_t f(cH_t). \tag{32}$$

The equilibrium H^* of this model is given by

$$f(cH^*) = \frac{1}{R} \tag{33}$$

and is a monotonically increasing function of R. Denoting small fluctuation around the equilibrium by $h_t := H_t - H^*$, one obtains after linearization the following linear discrete system

$$h_{t+1} = \lambda h_t, \quad \lambda = 1 + RcH^* f'(cH^*) < 1 \tag{34}$$

where $f'(cH^*) < 0$ (f is a monotonically decreasing fucntion) denotes the derivative of function f evaluated at cH^*. The equilibrium H^* is stable, if and only if, $1 >$

$\lambda > -1$ ([5]), which implies,

$$-cH^* f'(cH^*) < \frac{2}{R}. \tag{35}$$

Differentiating (33) with respect to R we have

$$-cf'(cH^*)\frac{dH^*}{dR} = \frac{1}{R^2}. \tag{36}$$

Substituting in (35) we have the stability condition

$$\frac{R}{H^*}\frac{dH^*}{dR} = \frac{d\log H^*}{d\log R} > \frac{1}{2}. \tag{37}$$

The above stability condition can be re-written as

$$\frac{d\log\left(H^*/\sqrt{R}\right)}{d\log R} > 0, \tag{38}$$

which implies that the equilibrium H^* is stable, if and only if,

$$\frac{d\left(H^*/\sqrt{R}\right)}{dR} > 0, \tag{39}$$

i.e., $H^*/\sqrt{R}$ is an increasing function of R.

The equilibrium H^* is monotonically stable, if and only if, $1 > \lambda > 0$ ([5]), which implies,

$$-cH^* f'(cH^*) < \frac{1}{R}. \tag{40}$$

Doing the same analysis as above we can conclude that the equilibrium is monotonically stable, if and only if,

$$\frac{R}{H^*}\frac{dH^*}{dR} > 1, \tag{41}$$

which is equivalent to H^*/R being an increasing function of R.

Appendix B. **Different interpretation of risk.** In Section 3 we explicitly defined a juvenile's risk in terms c which in turn defined the mortality rate faced by the juveniles from adults or competing juveniles. Risk can also be interpreted in other terms. For example, an alternative way to define a juvenile's risk is by the duration of time it is exposed to these mortality rates. We illustrate this with the adult-attack-juvenile mechanism, where if a group of juveniles are exposed to attacks from the adults for duration T, then the fraction of these juveniles that survive is given by (see Section 3.1)

$$\exp(-Tg(cH_t)) := f(cH_t)^T, \tag{42}$$

assuming that all juveniles face the same mortality rate (i.e., have the same value of c). Thus, if now $p(x)$ represents the distribution of T (risk) across juveniles we have the discrete-time model

$$H_{t+1} = RH_t \int_{x=0}^{\infty} p(x) f(cH_t)^x dx. \tag{43}$$

Note that for the Ricker model ($f(cH_t)^T = \exp(-cTH_t)$), model (43) reduces to

$$H_{t+1} = RH_t \int_{x=0}^{\infty} p(x)\exp(-cxH_t)dx, \tag{44}$$

which is essentially similar to (11). As for the Ricker model both c and T appear together, defining a juvenile's risk in terms of c or T or even as a product cT will lead to the same form of the discrete-time model (11), give or take some constants.

Appendix C. **Stability analysis for variability in density-independent risk.** This equilibrium H^* is given as the solution to the equation

$$\int_{x=0}^{\infty} p(x)f(xH^*)dx = \frac{1}{R}. \tag{45}$$

Denoting small fluctuation around the equilibrium H^* by h_t, one obtains using linearization the following linear discrete system

$$h_{t+1} = \lambda h_t, \quad \lambda = 1 + RH^* \int_{x=0}^{\infty} xp(x)f'(xH^*)dx < 1. \tag{46}$$

The equilibrium H^* is stable if and only if $1 > \lambda > -1$, which implies,

$$-H^* \int_{x=0}^{\infty} xp(x)f'(xH^*)dx < \frac{2}{R}. \tag{47}$$

If $p(x)$ is a differentiable function, then using integration by parts and the fact that for a probability density function $p(x)$, $\lim_{x\to 0} xp(x) = \lim_{x\to\infty} xp(x) = 0$, we have

$$\int_{x=0}^{\infty} (xp'(x) + p(x))f(xH^*)dx < \frac{2}{R}, \tag{48}$$

which using (45) reduces the stability condition to

$$\int_{x=0}^{\infty} (xp'(x) - p(x))f(xH^*)dx < 0. \tag{49}$$

This stability condition can be re-written as

$$\int_{x=0}^{\infty} x^2 \left(\frac{d(p(x)/x)}{dx}\right) f(xH^*)dx < 0. \tag{50}$$

Assume that $p(x)/x$ is a non-increasing function, i.e.,

$$\frac{d(p(x)/x)}{dx} \leq 0. \tag{51}$$

The distribution $p(x)$ goes to zero as x approaches infinity, and hence, there must exists an interval $[x_1, x_2]$ for which

$$\frac{d(p(x)/x)}{dx} < 0, \quad \forall x \in [x_1, x_2]. \tag{52}$$

Hence we have from (51) and (52) that

$$\int_{x=0}^{\infty} x^2 \left(\frac{d(p(x)/x)}{dx}\right) f(xH^*)dx = \int_{x=0}^{x_1} x^2 \left(\frac{d(p(x)/x)}{dx}\right) f(xH^*)dx \tag{53}$$

$$+ \int_{x=x_1}^{x_2} x^2 \left(\frac{d(p(x)/x)}{dx}\right) f(xH^*)dx + \int_{x=x_2}^{\infty} x^2 \left(\frac{d(p(x)/x)}{dx}\right) f(xH^*)dx < 0 \tag{54}$$

which from (50) implies a stable equilibrium for all values of $R > 1$. For monotonic stability one needs $0 < \lambda < 1$ where λ is defined in (46). This corresponds to

$$\int_{x=0}^{\infty} (xp'(x) + p(x))f(xH^*)dx < \frac{1}{R}, \tag{55}$$

which implies from (45)

$$\int_{x=0}^{\infty} xp'(x)f(xH^*)dx < 0. \tag{56}$$

Using the same argument as above we can say that if $p(x)$ is a non-increasing function then (56) will always hold, and hence a monotonically stable equilibrium for all values of $R > 1$.

Appendix D. **Stability for large values of R for the Ricker model.** For simplicity, we consider distributions of risks that can be expressed as

$$p(x) = \sum_{i=0}^{\infty} a_{n_i} x^{n_i} \tag{57}$$

for all positive x, where $n_i < n_{i+1}$, $n_i > -1$ and a_{n_i} are non-zero real numbers. Hence $p(x)$ behaves as $a_{n_0}x^{n_0}$ near $x = 0$ and $a_{n_0} > 0$. We assume that $p(x)/x$ is an increasing function near $x = 0$. This is only possible if $n_0 > 1$ or when $n_0 = 1$ then $n_1 > 1$, $a_{n_1} > 0$. Using

$$\int_{x=0}^{\infty} x^{n_i} \exp(-xH^*)dx = \frac{\Gamma[n_i + 1]}{{H^*}^{n_i+1}}, \tag{58}$$

where $\Gamma[n_i + 1]$ is the Euler gamma function we have

$$\int_{x=0}^{\infty} x^2 \left(\frac{d(p(x)/x)}{dx}\right) \exp(-xH^*)dx = \sum_{i=0}^{\infty} a_{n_i}(n_i - 1)\frac{\Gamma[n_i + 1]}{{H^*}^{n_i+1}}. \tag{59}$$

For sufficiently large R, and hence sufficiently large H^*, we have

$$\int_{x=0}^{\infty} x^2 \left(\frac{d(p(x)/x)}{dx}\right) \exp(-xH^*)dx \approx a_{n_0}(n_0 - 1)\frac{\Gamma[n_0 + 1]}{{H^*}^{n_0+1}} > 0 \tag{60}$$

when $n_0 > 1$ or

$$\int_{x=0}^{\infty} x^2 \left(\frac{d(p(x)/x)}{dx}\right) \exp(-xH^*)dx \approx a_{n_1}(n_1 - 1)\frac{\Gamma[n_1 + 1]}{{H^*}^{n_1+1}} > 0 \tag{61}$$

if $n_0 = 1$ and $n_1 > 1$, $a_{n_1} > 0$. As the stability condition (17) does not hold for sufficiently large H^*, we have an unstable equilibrium for sufficiently large R.

Appendix E. **When all juveniles have a minimum non-zero risk.** We assume that the function f has a form such that the equilibrium of model (1) is unstable for sufficiently large R. Thus from Appendix A, it implies that $f^{-1}(1/R)/\sqrt{R}$ is a decreasing function of R, for sufficiently large R where f^{-1} is the inverse function of f. We further assume that

$$\lim_{R\to\infty} \frac{f^{-1}(1/R)}{\sqrt{R}} = 0. \tag{62}$$

Note that this above condition will be true for $f(cH_t) = \exp(-cH_t)$ or if the function $f(cH_t)$ behaves as $1/(cH_t)^{\alpha}$, $\alpha > 2$, for sufficiently large H_t. With the distribution risk given as (21) we have the discrete-time model

$$H_{t+1} = RH_t \int_{x=c^*}^{\infty} g(x - c^*)f(xH_t)dx = RH_t \int_{x=0}^{\infty} g(x)f(xH_t + c^*H_t)dx. \tag{63}$$

The equilibrium H^* for this model is given by

$$\frac{1}{R} = \int_{x=0}^{\infty} g(x) f(xH^* + c^*H^*) dx. \tag{64}$$

As $f(xH^* + c^*H^*) < f(c^*H^*)$ (f is a monotonically decreasing function) and $\int_{x=0}^{\infty} g(x)dx = 1$ (this is because $\int_{x=0}^{\infty} p(x)dx = 1$) we have

$$\frac{f^{-1}\left(\frac{1}{R}\right)}{\sqrt{R}c^*} > \frac{H^*}{\sqrt{R}}. \tag{65}$$

From (62) and (65) we have that

$$\lim_{R\to\infty} \frac{H^*}{\sqrt{R}} = 0, \tag{66}$$

and hence, $H^*/\sqrt{R}$ is a decreasing function of R for sufficiently large R. This implies from Appendix A, that H^* is unstable for large enough R, irrespective of the form of the function $g(x)$.

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