Chaotic Dynamics Can Select for Long-Term Dormancy*

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abstract: Extended dormancy in a population is evolutionarily costly unless some variance in season-to-season fitness (usually driven by variance in environmental quality) makes bet hedging useful. Consequently, dormancy in a population is usually accepted as evidence of environmental variance. Using a Ricker-type model with heritable variation in dormancy, we show that this need not be so. Intrinsic population dynamics can generate chaotic fluctuations in the absence of environmental variance. Chaotic dynamics increase the frequency of a range of dormant strategists under natural selection, even when mortality during dormancy is relatively high. The buffering effect of dormant individuals then eliminates chaotic dynamics or generates periodic orbits of relatively low amplitude. These stabilized populations harbor a high frequency of dormant individuals that express a range of propensities to enter dormancy.

Keywords: chaos, diapause, dormancy, risk spreading, demographic, bet hedging.

Paradoxes are common in biology. For example, many organisms, such as plants and insects, commonly exhibit long-distance dispersal or dormancy extending for many generations (Hanski 1988). Such strategies are problematic because direct fecundity costs associated with dispersal and the demographic costs incurred by delaying reproduction result in reduction of fitness. Although dispersal and dormancy allow organisms to hedge against highly variable environments (Denno et al. 1991; Holt and McPeek 1996) or temporally variable mortality resulting from predation (Ringel et al. 1998), these tactics can also persist in situations where no variance source is apparent (Denno et al. 1991; Aluja et al. 1998). When the latter is true, it is usual to assume selection from some unmeasured environmental variance-driving factor (Danks 1992; Hopper 1999). A second paradox is that internally generated chaotic dynamics are a feature of many ecological models (Hassell et al. 1976; May 1976), but such dynamics have rarely been demonstrated in nature (Hassell et al. 1976; Turchin and Ellner 2000). It is usual to ascribe this absence of evidence to the difficulties associated with separating the effects of intrinsic and extrinsic factors on population dynamics (Turchin and Ellner 2000).

We describe here a scenario where natural selection in chaotic populations favors the spread of a demographically costly risk-spreading strategy. In this case, we model dormancy evolution, but our results are broadly applicable to dispersal as well. Although it is well known that dormant (or dispersing) individuals can stabilize population dynamics (Bulmer 1984; Van Dooren and Metz 1998; Newman et al. 2002), it is not known whether dormancy can evolve in a chaotic population, given its demographic cost. We demonstrate here that allowing natural selection for dormancy generates very stable dynamics in otherwise chaotic systems while maintaining a high frequency of dormant strategists.

The Model

We begin with a population constrained to a single strategy of nondormancy whose dynamics are described by the familiar Ricker (1954) equation (using the discrete-time logistic produces identical qualitative results; May 1976),

$$N_{t+1} = RN_t e^{-\delta N_t}, \quad (1)$$

where the population at one time step in the future, N_{t+1}, is a function of the replacement rate (R) and the negative influence (competition, cannibalism) of the present population ($-\delta N_t$). This model has the useful characteristic of displaying dynamics that range from extreme stability at low values of R to a series of period-doubling bifurcations leading to chaos as R increases (fig. 1).

We modify this basic model to include dormancy, where

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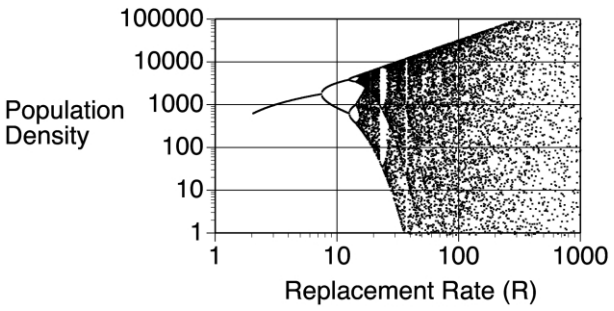

Figure 1: Ricker population dynamics. Bifurcation plot shows the effect of changing the replacement rate (R) on population stability in Ricker-type populations with no dormancy.

a parent's offspring show a range of dormancies with a mean that we allowed to range from zero to five generations. Actual dormancies across individuals are assigned according to a Poisson distribution, to a maximum of 13 generations of dormancy, with the very small fraction of dormancies that would have been assigned values greater than the maximum set to that maximum value. The population of active individuals at time $t + 1$, N_{t+1}, is now given by

$$N_{t+1} = \sum_{j=0}^{\infty} \left[RN_t e^{-\delta N_t}(p_{0j})\right] + d_{1_t}(1 - m). \qquad (2)$$

Here, p_{0j} is the proportion of the population that is composed of individuals of genotype j that express no dormancy, and d_{1_t} is the element of vector **d** that contains the number of individuals at time t that have broken dormancy at time $t + 1$ and have escaped density-independent mortality (m). Additionally, all individuals in the dormancy vector **d** that survive one season of dormancy are moved to the next lowest element (e.g., d_5 to d_4) each generation. Finally, each element of **d** at time t is also updated by direct recruitment of the dormant offspring of active individuals of genotype j. Thus,

$$d_{i_t} = \sum_{j=0}^{\infty} \left[RN_t e^{-\delta N_t}(p_{ij})\right] + d_{i+1_{t-1}}(1 - m). \qquad (3)$$

One convenient aspect of this approach is that it allowed us to assign dormancy "fates" directly to individuals such that the duration of each individual's term of dormancy was defined at birth to be of a specific duration. This avoids problems associated with simply assigning Markov probabilities to the processes of entering and leaving the dormant pool. The latter approach makes for analytically tractable models. However, in numerical models, this practice has the effect of creating individuals that can experience very long dormancies because the likelihood that any individual in the dormant pool will ultimately break dormancy is independent of the length of time that the individual has been dormant.

Sensitivity Analyses for Evolving Dormancy

We started our population with no dormant strategists and allowed dormancy to appear by mutation after 1,900 generations and for the next 100 generations. To do this, we introduced a mutation rate of 0.002/individual that could result in a spontaneous change in the maternal strategy with respect to dormancy. Depending on the magnitude of the replacement rate (R), this meant that over 100 generations, anywhere from 200 to 2,000 mutants were introduced into the population. Natural populations with dormancy typically express a range of dormancy durations (Menu et al. 2000). We assume here that this variation is a genotypic strategy, and we have all mutant strategists play a mixed game wherein progeny display a range of dormancies with a genotypically specific mean value rather than a dormancy of fixed duration.

For computational purposes, we discretized this range into value increments of 0.5. When a mutation occurred, the offspring of the individual with the mutation all carried the same resulting genotype (asexual inheritance). After the population was seeded with dormant strategists, mutation was terminated, and the model population was allowed to evolve for a further 3,000 generations. At the end of this time, the population size during the last 50 generations was recorded, along with the distribution of strategists within the population. All combinations of parameters were replicated 10 times.

The model setup did not lend itself to classical analysis for determining evolutionary stable states. However, we did test the robustness of our results by a perturbation analysis as a complement to each of the replicate runs described above. Our results below are robust with respect to variation in the density-dependent mortality term (δ) in the Ricker model.

Our model produces several striking results. First, the unstable dynamics displayed by the original Ricker model essentially disappear with the invasion of dormancy. Parameter combinations that would otherwise produce a period doubling to chaos (fig. 1; Hassell et al. 1976; Newman et al. 2002) instead produce stable equilibria, simple two-phase cycles, or periodic orbits of relatively narrow amplitude (fig. 2). Second, we find that populations support dormant strategists above the threshold replacement rate that would normally see the beginning of the period-doubling process in the unmodified Ricker model. Most interestingly, we see that a range of dormancy strategies, rather than a single "optimal" dormancy strategy, is maintained (fig. 3). This effect is most pronounced at zero or

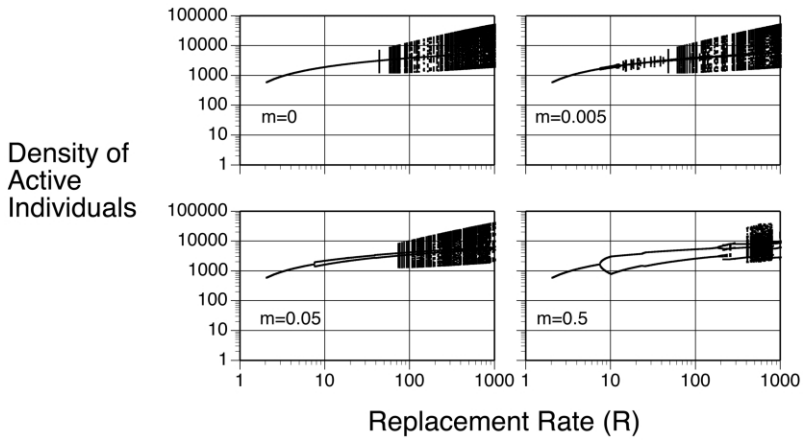

Figure 2: Dynamics of Ricker systems with dormancy allowed to invade. Plots show the effect of increasing mortality (m) during dormancy on system dynamics. Active individuals (Y-axis) are those individuals in the population that are not dormant at any given time; they may be either nondormant or dormant strategists.

low mortality levels and is much less evident with strong mortality during the dormant phase (fig. 3). This production of a range of dormancy strategies is robust in a qualitative way. Replicate runs produce similar but slightly different ranges of dormancy strategies under identical parameters and show a strong tendency to return to similar states after perturbation (results not shown). Finally, we find that higher replacement rates tend to increase population fluctuations, in this case manifested by the system entering periodic orbits of increasing amplitude. These orbits are driven by temporal fluctuation of strategist frequencies, in a manner similar to that of Doebeli's (1993, 1995) intermittent chaos and laboratory predator-prey systems (Yoshida et al. 2003).

The first two results, system stability and invasion and maintenance of dormancy, compare very closely with findings of other studies on the evolution of demographically costly strategies. It has been amply demonstrated, at least theoretically, that dispersal and dormancy can be maintained in variable environments (e.g., Hopper 1999). For example, Menu et al. (2000) showed that a pure risk-spreading strategy can, despite its demographic cost, perform very well when environmentally determined mortality varies greatly over time. Furthermore, Doebeli (1995) demonstrated that chaotic dynamics can be ameliorated by introduction of demographically costly strategies, although he did not invoke specific biological mechanisms. In a similar way, Newman et al. (2002) showed that a dormant pool can stabilize Ricker-type dynamics over a range of parameters, but they did not evaluate the fitness of dormant strategies. Finally, Holt and McPeek (1996) showed that dispersal strategies are favored under natural selection in chaotic systems, which in turn affect system dynamics.

Maintenance of a range of genotypes within a model system suggests that there is no unique evolutionarily stable strategy (ESS) for many sets of parameters. This type of result can be obtained in randomly fluctuating (Sasaki and Ellner 1995) or cyclical systems where strategists differ markedly in functional response to resource availability (Armstrong and McGehee 1976; Abrams and Holt 2002). However, our model includes none of these features. Instead, we see a mixture of dormancy strategy "genotypes" under parameter sets that ultimately result in strong population stability once dormancy invades (fig. 2). This result may have been obtained when mortality during dormancy was set to zero because selection against increased mean time to reproduction goes to zero in equilibrium populations (Karban 1997; Van Dooren and Metz 1998). In this light, adding mortality during dormancy should have two effects. First, because mortality imposes a demographic cost on all dormant strategists, high mortality should select for the shortest dormancy duration that stabilizes system dynamics. We find exactly this. The average duration of dormancies present in the population is much more constrained when $m = 0.50$ than when $m = 0.05$, 0.005, or 0 (fig. 3). Second, the demographic cost that mortality imposes on dormancy should produce variation in strategy fitness at system equilibrium. Thus, if absence of mortality is the sole factor allowing a wide range of dormancy strategists to coexist, any mortality should select for a single optimum genotype. This does not happen. However, our results suggest that increasing mortality does force these systems to approach such a single optimum. We find that

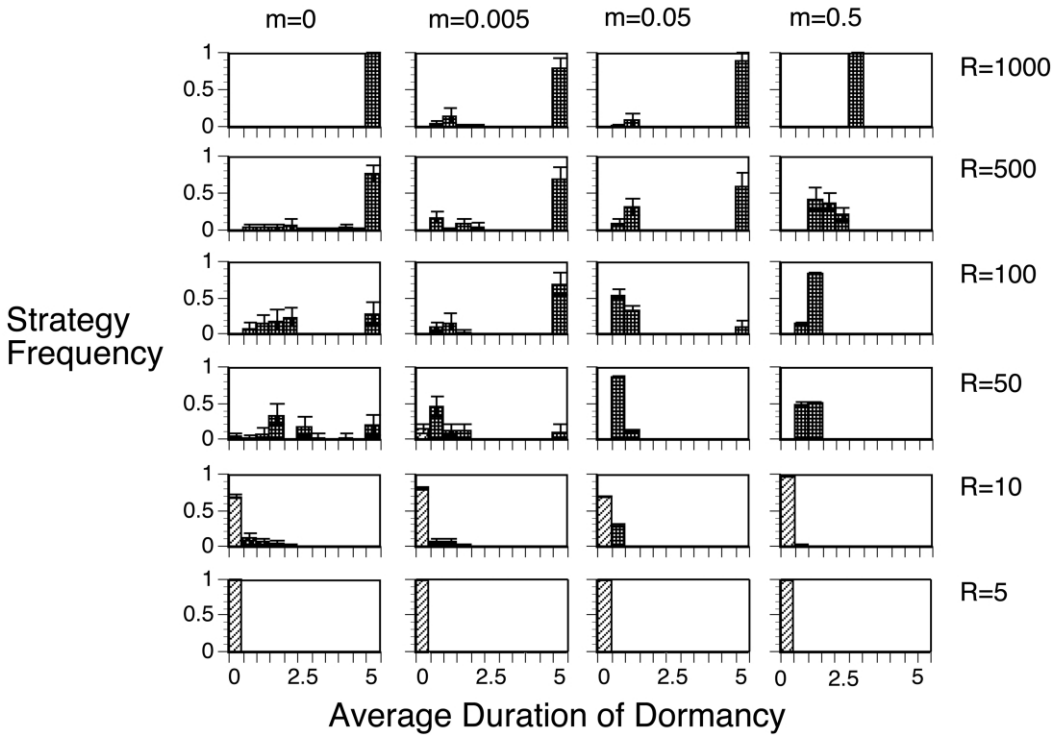

Figure 3: Frequency distribution at the end of simulations of dormancy genotypes as affected by replacement rate and mortality during dormancy. Distributions show the effect of varying mortality (*columns*) and replacement rate (*rows*) on the distribution of dormancy genotypes after 3,000 generations. Each run was replicated 10 times, and distributions represent the mean of all trials. Light hatched bars represent proportion of the population that is composed of nondormant genotypes. Dark stippled bars represent dormancy genotypes. Error bars denote 1 standard error.

moderate to low mortalities result in a range of dormancy strategies being maintained in the population, whereas very strong mortality ($m = 0.5$) produces the lowest variation among dormancy strategists (fig. 3). Finally, an ESS analysis of a simpler model may shed some light on whether coexistence of multiple dormancy strategies is actually a mixed ESS or represents an intermediate stage along a very slow march to a monomorphic ESS.

Both Doebeli's (1995) and Holt and McPeek's (1996) models readily generated intermittent chaos that was driven by negative frequency-dependent selection. Our model never generates intermittent chaos but does generate periodicity at higher replacement rates when very long average dormancies are selected or at lower replacement rates with mortality during dormancy added to the mix. The periodicity obtained at high replacement rates in our model probably occurs because no dormancy genotype within the range allowed can fully stabilize these systems, even when there is no mortality during dormancy (fig. 2). Mortality during dormancy further destabilizes our system. This occurs because mortality increases the demographic cost of dormancy when the system is stable and thus reduces the fitness of dormant strategists. This reduces their frequency and causes system fluctuation, which in turn increases dormant strategist fitness (and thus strategist frequency). This temporal fluctuation of strategist frequency drives system oscillations that take the form of periodic orbits at low to moderate mortalities and strong two- and four-phase cycles at high mortalities.

We return to the paradox. Dormancy should not exist in stable populations, and we find that this holds true here. At low replacement rates, dormancy never invades when rare and tends to disappear if added in large frequencies as a perturbation. Chaotic dynamics selects for dormancy because the bet-hedging strategy gives higher geometric mean fitness to dormant individuals than to nondormant strategists. The resultant high frequency of dormancy then eliminates the chaotic dynamics, and the system persists in a much more stable condition.

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Literature Cited

Abrams, P. A., and R. D. Holt. 2002. The impact of consumer-resource cycles on the coexistence of competing consumers. Theoretical Population Biology 62:281–295.

Aluja, M., M. López, and J. Sivinski. 1998. Ecological evidence for diapause in four native and one exotic species of larval-pupal fruit fly (Diptera: Tephritidae) parasitoids in tropical environments. Annals of the Entomological Society of America 91:821–833.

Armstrong, R. A., and R. McGehee. 1976. Coexistence of two competitors on one resource. Theoretical Population Biology 56:499–502.

Bulmer, M. G. 1984. Delayed germination of seeds: Cohen's model revisited. Theoretical Population Biology 26:367–377.

Danks, H. V. 1992. Long life cycles in insects. Canadian Entomologist 124:167–187.

Denno, R. F., G. K. Roderick, K. L. Olmstead, and H. G. Dobel. 1991. Density-related migration in planthoppers (Homoptera: Delphacidae): the role of habitat persistence. American Naturalist 138: 1513–1541.

Doebeli, M. 1993. The evolutionary advantage of controlled chaos. Proceedings of the Royal Society of London B 254:281–285.

———. 1995. Updating Gillespie with controlled chaos. American Naturalist 146:479–487.

Hanski, I. 1988. Four kinds of extra long diapause in insects: a review of theory and observations. Annales Zoologici Fennici 25:37–53.

Hassell, M. P., J. H. Lawton, and R. M. May. 1976. Patterns of dynamic behaviour in single-species populations. Journal of Animal Ecology 45:471–486.

Holt, R. D., and M. A. McPeek. 1996. Chaotic population dynamics favors the evolution of dispersal. American Naturalist 148:709–718.

Hopper, K. R. 1999. Risk-spreading and bet-hedging in insect population biology. Annual Review of Entomology 44:535–560.

Karban, R. 1997. Evolution of prolonged development: a life table analysis for periodical cicadas. American Naturalist 150:446–461.

May, R. M. 1976. Simple mathematical models with very complicated dynamics. Nature 261:459–467.

Menu, F., J. P. Roebuck, and M. Viala. 2000. Bet-hedging diapause strategies in stochastic environments. American Naturalist 155: 724–734.

Newman, T. J., J. Antonovics, and H. M. Wilbur. 2002. Population dynamics with a refuge: fractal basins and the suppression of chaos. Theoretical Population Biology 62:121–128.

Ricker, W. E. 1954. Effects of compensatory mortality upon population abundance. Journal of Wildlife Management 18:45–51.

Ringel, M. S., M. Rees, and H. C. J. Godfray. 1998. The evolution of diapause in a coupled host-parasitoid system. Journal of Theoretical Biology 194:195–204.

Sasaki, A., and S. P. Ellner. 1995. The evolutionarily stable phenotype distribution in a random environment. Evolution 49:337–350.

Turchin, P., and S. P. Ellner. 2000. Living on the edge of chaos: population dynamics of Fennoscandian voles. Ecology 81:3099–3116.

Van Dooren, T. J. M., and J. A. J. Metz. 1998. Delayed maturation in temporally structured populations with non-linear dynamics. Journal of Evolutionary Biology 11:41–62.

Yoshida, T., L. E. Jones, S. P. Ellner, G. F. Fussmann, and N. G. Hairston. 2003. Rapid evolution drives ecological dynamics in a predator-prey system. Nature 424:303–306.

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