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Seasonally Limited Host Supply Generates Microparasite Population Cycles

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Cycles in biological populations have been shown to arise from enemy–victim systems, delayed density dependence, and maternal effects. In an initial effort to model the year-to-year dynamics of natural populations of entomopathogenic nematodes and their insect hosts, we find that a simple, nonlinear, mechanistic model produces large amplitude, period two population cycles. The cycles are generated by

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seasonal dynamics within semi-isolated populations independently of inter-annual feedback in host population numbers, which differs from previously studied mechanisms. The microparasites compete for a fixed number of host insect larvae. Many nematodes at the beginning of the year quickly eliminate the pool of small hosts, and few nematodes are produced for the subsequent year. Conversely, initially small nematode populations do not over-exploit the host population, so the surviving hosts grow to be large and produce many nematodes that survive to the following year.

1. INTRODUCTION

Recent advances in the theory of nonlinear dynamics provide tools for viewing the complex behavior of biological populations (Hastings *et al*., 1993; Hanski and Korpimaki, 1995; Higgins *et al*., 1997; Grenfell *et al*., 1998; Blasius *et al*., 1999; Kendall *et al*., 1999; Stenseth, 1999; Finkenstadt and Grenfell, 2000; Turchin and Ellner, 2000; Bjørnstad and Grenfell, 2001). Connections between the observed dynamics of natural populations (Higgins *et al*., 1997) and ecological theory are developing rapidly, particularly in the areas of human epidemiology (Earn *et al*., 2000) and the dynamics of small mammals (Stenseth, 1999). Cyclic behavior is perhaps the most well studied aspect of enemy–victim interactions, but the specific mechanisms producing these cycles have been the subject of debate (Kendall *et al*., 1999). We model a common but little studied enemy–victim system where microparasite population cycles are driven by competition for the limited supply of host larvae within a single season.

Entomopathogenic nematodes are common, widespread enemies of soil insects (Hominick, 2002). Natural populations are extremely patchy in space (Stuart and Gaugler, 1994) and can cause extremely high mortalities to natural populations of hosts (Strong, 1999), suggesting local over-exploitation of hosts (Strong, 2002). Local populations of these enemies wax and wane over runs of 3–5 years (D.R.S. and E.L.P., unpublished). Immature, soil dwelling, 'infective juveniles' of the entomopathogenic nematode (*Heterorhabditis marelatus*) search in the soil for host ghost moth (*Hepialus californicus*) caterpillars that feed upon the taproot of bush lupine (*Lupinus arboreus*) (Strong *et al*., 1996). They enter and kill the insect host, reproduce inside, and emerge into the soil in a pulse when the cadaver's resources are exhausted. Small, young hosts yield few infective juveniles, while large old caterpillars yield upward of a million (E.L.P., unpublished data). This means that hosts escaping infection early in the season can grow into a much more productive resource for late season infection by the nematode. Infective juveniles cycle back into the non-reproductive, non-feeding population in the soil when they exit the cadaver. The cycle can repeat several times in the wet soil of a winter growing season. The environment of this interaction is the few liters of soil around the taproot of a lupine rhizosphere. Several meters separate most taproots from one

another in the grassland matrix where lupines grow. Both nematodes and caterpillars remain near the taproot, rates of dispersal are low, and populations are virtually restricted to single rhizospheres within a growing season. In dry summer soil the nematodes are quiescent, and host attack rates decrease to virtually nil.

The nematode is less frequent in space than the host ghost moth, and rhizospheres without the enemy produce most of the ghost moth recruits (Strong *et al*., 1996). Ghost moths are very strong flyers, and can lay eggs many hundreds of meters away from their natal rhizosphere (Wagner, 1985). Their great powers of dispersal supplies rhizospheres with host recruits independently of their local nematode densities.

To understand the dynamics of natural populations of these potent natural enemies, we developed the simplest model consistent with their biology: a continuous time mechanistic model describing the within-year dynamics of *H. marelatus* with ghost moth caterpillars in single lupine rhizospheres. The within-year model is then used to generate a discrete time model of between-year dynamics (Briggs and Godfray, 1996; Roberts and Heesterbeck, 1998; Gamarra and Sole, 2002) for natural populations that exist in a highly seasonal environment. Fenton *et al*. (2000) modeled entomopathogenic nematode dynamics for biological control of glasshouse insect pests with an approach featuring aseasonal dynamics and uniformly sized hosts, finding instability. In a second paper, they focused upon transient dynamics appropriate to short-term aseasonal biological control of host pests, and included host stage structure and a constant nematode development period (Fenton *et al*., 2001). Using parameters derived from field and laboratory data, we model long-term seasonal dynamics of natural populations of nematodes with univoltine hosts, and include effects of host size and host immigration. While our model is based on the interactions between a particular microparasite and its host, it should be broadly applicable to organisms with seasonally limited resources.

2. MODEL

We modeled the population dynamics of nematodes and their hosts within a single rhizosphere. The infective juveniles search randomly through the wet, winter soil of the rhizosphere to find and infect hosts at a rate proportional to the product of the nematode and host densities (Strong *et al*., 1999). In nature, most hosts are killed by a single hermaphroditic infective juvenile; thus, we model single infections of hosts. Infection causes the death of the host within hours, removes a nematode from the soil-dwelling population, and adds an infected host cadaver.

Caterpillars hatch almost simultaneously and grow at similar rates; the host cohort is comprised of similar-sized caterpillars. A time-dependent (within the season) number $\Lambda(t)$ of infective juvenile nematodes emerge simultaneously after a fixed period of time, τ, from the host cadaver. Early in the growing season, hosts are small and produce few infective juveniles. By the end of the season, hosts are

large and can produce as many as a million infective juveniles. We denote the density of free-living, soil-dwelling nematodes by N and the density of hosts by H. Nematodes emerging from a cadaver at time t came from an infection event τ time units earlier; emergence from cadavers increases the nematode density in the soil at the rate $\beta H(t-\tau)N(t-\tau)\Lambda(t-\tau)$, where β is infectivity, the rate at which nematodes find and infect hosts. The dynamics of the hosts are described by two terms: death from nematodes, and density-independent deaths at a rate k_H. The density of soil-dwelling nematodes decrease owing to entrance into hosts and to density-independent deaths of nematodes at a rate k_N. Density of soil-dwelling nematodes increases owing to emergence from hosts. The model for wet, growing season dynamics becomes

$$\begin{aligned} \frac{dH(t)}{dt} &= -\beta H(t)N(t) - k_H H(t) \\ \frac{dN(t)}{dt} &= -\beta H(t)N(t) - k_N N(t) + \beta H(t-\tau)N(t-\tau)\Lambda(t-\tau), \end{aligned} \tag{1}$$

which we solve over the period $t = 0$ to T, where T is the (fixed) length of the wet season. During the dry season, when the nematodes are quiescent and do not infect hosts, we assume that nematodes experience only density-independent mortality. Nematodes that are inside host cadavers at the end of the dry season can remain in the cadaver for extended periods of time, where mortality rates are lower than in the soil (Kaya and Gaugler, 1993). Because of this, over-summer survival probabilities are higher for these nematodes. We assume that all of the nematodes have either emerged from hosts or died by the end of the dry season. A solution of equation (1) yields the density of nematodes outside $N^k(T)$ of host cadavers at the end of wet season. The density of nematodes inside of hosts at the end of the wet season is $N_i^k(T) = \int_{T-\tau}^{T} \beta H(\sigma)N(\sigma)\Lambda(\sigma)d\sigma$. The dry-season submodel then yields the density of the nematodes at the beginning of the next wet season, $N^{k+1}(0) = \lambda_o N^k(T) + \lambda_i N_i^k(T)$, where λ_o and λ_i are, respectively, the survival probabilities of nematodes that began the dry season inside and outside of hosts. Returning to the wet season submodel [equations (1)], we study year-to-year dynamics by taking as initial conditions the resulting number of nematodes after the dry season $N^{k+1}(0)$ and the assumed fixed number of host eggs per bush, $H(0)$. We assume that hosts emerge simultaneously so the initial interval for the hosts required by the delay term is uniformly zero. Because the only delay term is a proportional to the hosts, the initial nematode interval is not needed.

3. METHODS

We numerically integrated the wet-season delay differential equations using Euler's method, calculated the dry-season dynamics, and recorded the number of

Table 1. Estimates of infectivity, β, in different sized arenas from published sources and our data, measured in (rhizosphere volume/day $= 20 \times \pi 50^2$ cm^3/day).

Nematode species	Host species	Soil vol. (cc)	Mean β (rhiz. vol/day)	Reference
Heterorhabditis spp.	*G. mellonella*	25	1.25×10^{-5}	Westerman (1998)
Heterorhabditis spp.	*Otiorhynchus sulcatus*	25	2.91×10^{-6}	Westerman (1998)
H. bacteriophora	*G. mellonella*	50	1.26×10^{-6}	Campbell *et al*. (1999)
Heterorhabditis spp.	*G. mellonella*	15	1.39×10^{-4}	Koppenhöfer *et al*. (1996)
H. marelatus	*G. mellonella*	10	2.10×10^{-6}	Strong (unpublished data)
H. marelatus	*G. mellonella*	100	7.33×10^{-6}	Strong (unpublished data)
H. marelatus	*G. mellonella*	1000	2.50×10^{-6}	Strong (unpublished data)

hosts killed during each wet season. We tested the stability of the numerical method by decreasing the time step size. To understand the full range of the dynamics, we computed a bifurcation diagram numerically, by varying the infectivity, β, iterating the return map for 1000 generations to eliminate transients, and then plotting the next 100 points to describe the attractor as a function of the parameters. The complexity of the year-to-year map makes any analytic treatment of bifurcation impossible, but we numerically generated the time one return map from the continuous, within-year dynamics in the regions of parameter space where the dynamics are qualitatively different. We used several different initial conditions to assure that all attractors had been found. We used β as the bifurcation parameter, because the qualitative dynamics are most sensitive to this parameter (see Discussion).

To focus on the most relevant dynamical behavior, we estimated infectivity, β, from laboratory experiments (Table 1). We exactly solved equations (1), for β in terms of the initial density of nematodes N_0, the number of successful infecting nematodes I, the time in days of the experiment T, and V_e/V_r, the ratio of the experimental volume to the rhizosphere volume ($V_e \approx 20 \times \pi 50^2$ cm^3). We solved the equations for short periods too brief for either nematode reproduction or significant nematode mortality, $k_N = 0$ and $I = N_0 - N$. Nematode mortality is low in the pasteurized soil of experiments (Koppenhöfer *et al*., 1996). Although multiple infections are quite rare in nature, multiple infections are common in the laboratory experiments for estimation of β; thus, the number of hosts available for infection, H, is constant. The resulting equation for infectivity, β, is

$$\beta = \frac{1}{HT} \cdot \ln\left(\frac{N_0}{N_0 - I}\right) \cdot \frac{V_e}{V_r}. \tag{2}$$

Our estimates based on equation (2) and previous estimates of β are presented with the appropriate units in Table 1. However, we believe these are overestimates of natural values of this parameter (see the Discussion). Nematode mortality k_N, and host productivity growth rate r are taken from the literature Strong (2002),

Wagner (1985). The maximum productivity of nematodes from hosts, Λ_{max}, equaled the mean productivity of host larvae near pupation. To determine the influence of within-season growth of hosts on the dynamics, we compared unvarying Λ with a saturating function in which productivity of nematodes increased as hosts grew in size, i.e. $\Lambda(t) = \min(Ce^{rt}, \Lambda_{max})$. This function closely matches data on host growth (Wagner, 1985). To test the robustness of our results, several solutions were calculated with small variations in the parameters. A saturating type II functional response for nematode attack was used as well, but did not qualitatively change results.

4. Results

The qualitative result of our model is a series of large amplitude, period-two cycles of the nematode population, yielding high and very low numbers for a large range of parameter values (Fig. 1). The wide range over which cycles occur critically depends on the assumption of host growth during the season. Were hosts not to grow in size (i.e., constant Λ) the high-low cycles would be restricted to a much smaller range of parameters. The cycles will persist for the low immigration rates [corresponding to results for equilibria of difference equations in Karlin and McGregor (1972)] that we have observed within seasons in the field. Host mortality in the model tracks the two-cycle nematode population dynamics, with almost 100% mortality for rhizospheres in which the initial nematode population is high at the beginning of the growing season and much lower mortality for rhizospheres in which the nematode population is initially low.

For very low values of nematode infectivity β, the nematodes become extinct. As β increases there is a nematode population steady state that quickly undergoes a series period doubling bifurcation, leading to chaos (Fig. 2). These initial bifurcations are explained by classic work dating back to May and Oster (1976). They showed that these dynamics are 'generic to any curve with a hump whose steepness can be parametrically tuned'. Integrating the within year dynamics leads to a year-to-year map for the nematodes that has a single hump. The parameter β plays the role of the reproduction parameter in the Ricker and logistic maps by controling the steepness (see Figs. 3 and 4). The complex dynamics quickly lock in on a period two cycle, because the hump in the discrete map moves to nematode population numbers below one. As β increases further the system returns to an equilibrium. This equilibrium persists, but approaches zero.

5. Discussion

Competition for hosts is the indirect mechanism of density dependence leading to cycles in nematode numbers in our model. Because hosts grow larger during the season, density dependence is stronger; elimination of small, relatively unproductive hosts early in the season reduces the availability of large, highly productive

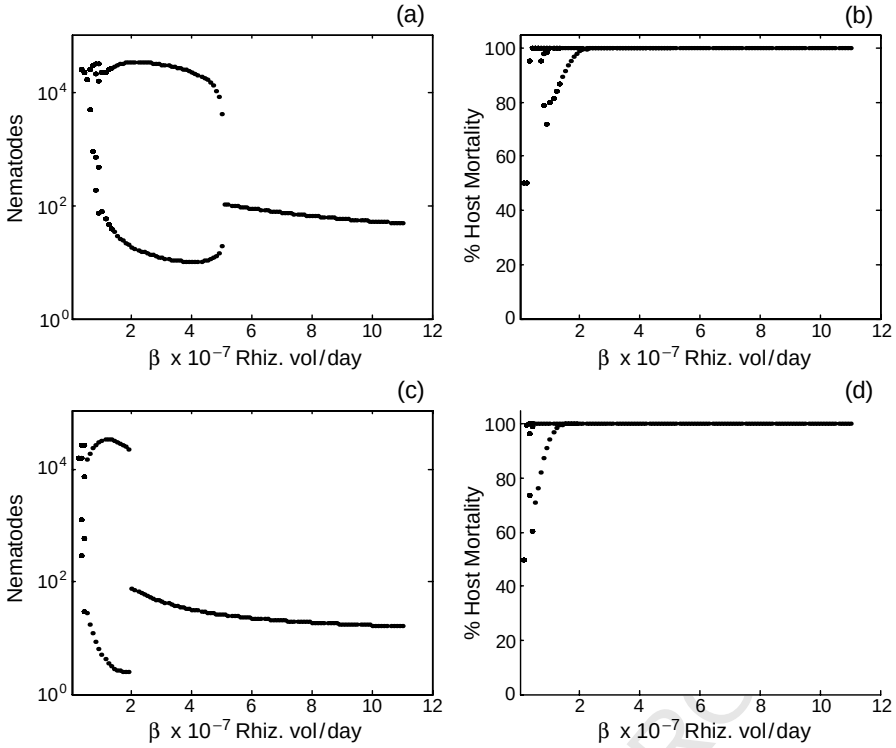

Figure 1. Model results showing dependence of nematode population and annual host mortality on nematode infectivity, β, and host growth. Panels (a) and (b) show nematode population at the start of the wet season, and total annual host mortality with host size included. Panels (c) and (d) are analogous, but with no host size effect. For both simulations host mortality $k_H = 0.0001$/days, nematode mortality $k_N = 0.063$/days, and nematode generation time $\tau = 35$ days. The length of the wet season was 160 days, and dry-season survivorship for soil-dwelling nematodes was 10^{-6} and 10^{-3} for nematodes inside cadavers. In (a) and (b) maximal host productivity was $\Lambda_{\max} = 800\,000$ nematodes per host, $r = 0.09$/days, $C = 10000$, and in (c) and (d) host productivity was held constant at $\Lambda = 800\,000$. Notice that the cycles exist for a much smaller range of parameters.

hosts late in the season (Briggs *et al.*, 1999). Timing of infections plays a role in the density dependence, because the earlier an infection occurs, the longer the infective juveniles produced from the infection are exposed to high mortality in the soil. This is why cycles can occur when there is no host growth or nearly 100% host mortality every year. Cycles found in previous models for hosts with non-overlapping generations depend on an inter-annual feedback between host and enemy populations (Mills and Getz, 1996). The cycles we find in the model are different, because host recruitment in the following year is independent of local interaction between enemy and victim. In our model we have assumed the time for nematodes to emerge from an infected host, τ, is constant. However, this time delay is shorter for smaller hosts (E.L.P., unpublished data). A large nematode population early in the wet season will thus more quickly reduce the host population,

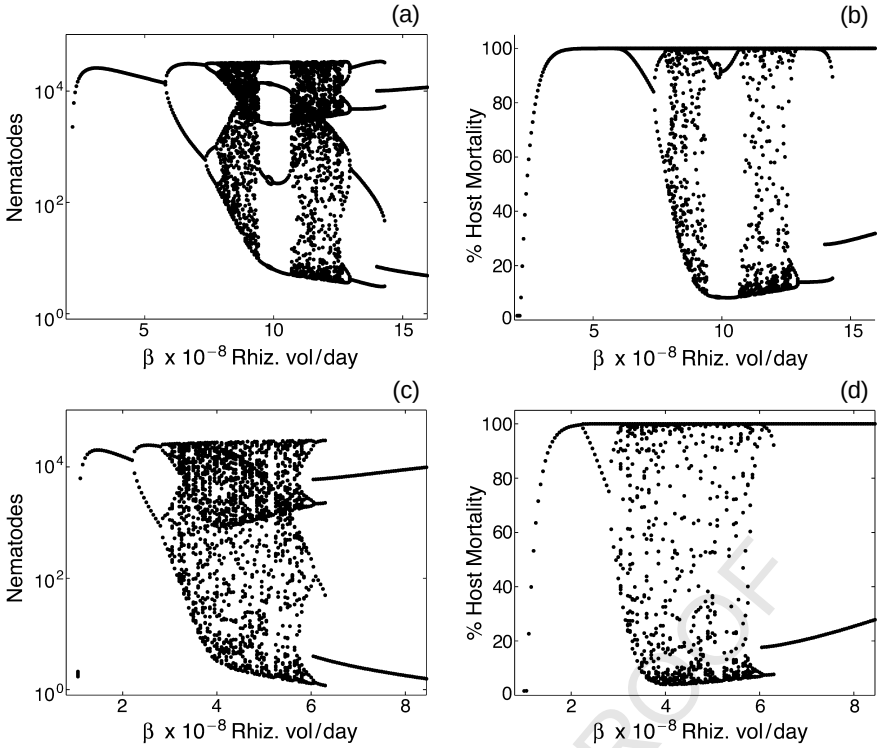

Figure 2. Detail of model results showing dependence of nematode population and annual host mortality on nematode infectivity (β) and host growth for very small values of infectivity. Panels (a) and (b) show nematode population at the start of the wet season, and total annual host mortality with host size included. Panels (c) and (d) are analogous, but with no host size effect. Parameters are the same as in Fig. 1.

leading to a greater likelihood of cycles. These dynamics should apply to many enemy–victim systems in which subsidies are important to dynamics, such as those in which the feedback of predation to prey reproduction occurs on multiple scales; hosts are locally depleted while new hosts arrive from a larger population that is not exposed to the enemy (Polis and Strong, 1996).

Our model produces microparasite cycles over a range of values of infectivity, β, that is slightly lower than our and other estimates of this parameter. However, we believe with Fenton *et al*. (2000) that previous estimates are too high for natural settings. First, the natural host ghost moths are not as vulnerable to the nematodes as the wax worms (*Galleria mellonella*) used in most estimates of β (Strong *et al*., 1996). Second, experiments have been conducted in small enclosed arenas of homogeneous soil that concentrate the waste gasses produced by hosts that nematodes use to find hosts. Consequently, more nematodes infect hosts, leading to artificially high estimates of β. Our preliminary work outside of containers suggests that realistic field estimates of β will prove to be lower than current laboratory estimates. Additionally, mathematical results suggest that stochasticity and

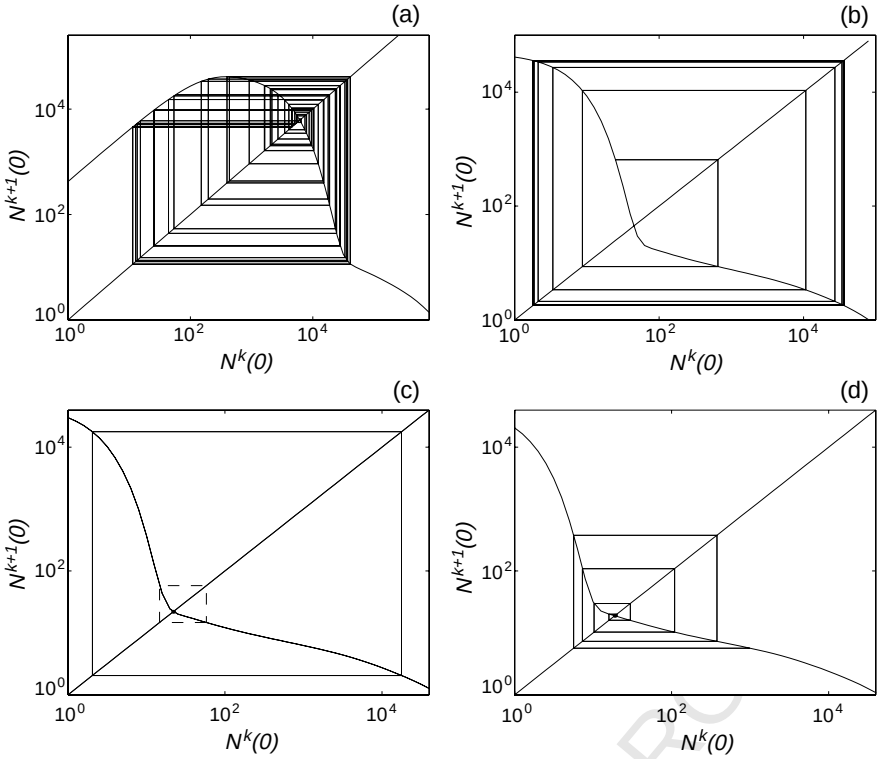

Figure 3. Map that takes the nematode population size at the start of the wet season to the nematode population size at the start of the wet season the following year, with varying host size. Maps are plotted on a log–log scale for clarity; therefore, the origin is excluded. Panel (a) shows the chaotic dynamics in nematode population with $\beta = 8\times10^{-8}$. Panel (b) shows the period two dynamics of map when $\beta = 4.5\times10^{-7}$. The chaotic dynamics are no longer present because the hump has moved to the range where the nematode population is less than one. Panel (c) shows the coexistence of a stable equilibrium and a stable two cycle (solid line); the unstable cycle is indicated with a dashed line, $\beta = 4.9\times10^{-7}$. These dynamics exist for a very narrow region parameter space when the $N^{k+1}(0) = N^k(0)$ line crosses through the region of the map where the slope quickly changes from being greater in absolute value than 1 to being less than 1 in absolute value. Panel (d) shows the stable equilibrium dynamics with $\beta = 6\times10^{-7}$.

a lack of spatial homogeneity extend the range of parameters over which cycles occur [e.g. Billings and Schwartz (2002)].

Entomopathogenic nematodes persist in nature, while published models find their dynamics unstable. We present the simplest mechanistic model consistent with their biology in an attempt to understand the dynamics of the interaction with hosts of these widespread, important, but little known natural enemies. The extreme cycles that we found in the model appear in previous modeling of these enemies, which addressed their use in biological control of mushroom pests (Fenton *et al.*, 2000). These cycles bode instability, and suggest that there may be high rates of local extinction in nature. The model cycles have directed our attention to mea-

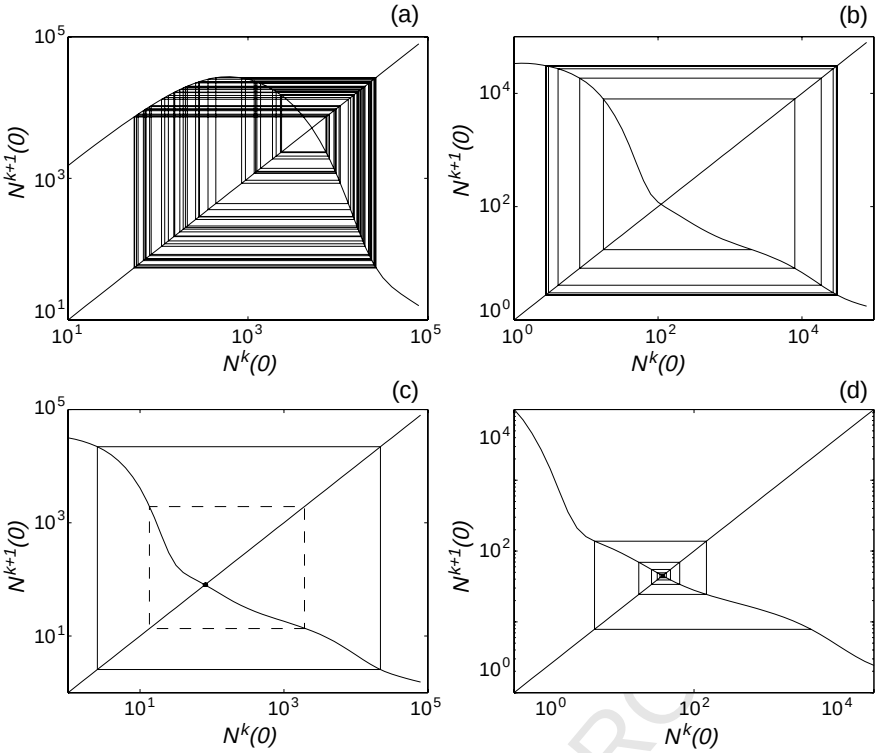

Figure 4. Map that takes the nematode population size at the start of the wet season to the nematode population size at the start of the wet season the following year, with no host size effect. Maps are plotted on a log–log scale for clarity; therefore, the origin is excluded. Panel (a) shows the chaotic dynamics in nematode population with $\beta = 3.5 \times 10^{-8}$. Panel (b) shows the period two dynamics of map when $\beta = 1.5 \times 10^{-7}$. The chaotic dynamics are no longer present because the hump has moved to the range where the nematode population is less than 1. Panel (c) shows the coexistence of a stable equilibrium and a stable two cycle (solid line); the unstable cycle is indicated with a dashed line, $\beta = 1.9 \times 10^{-7}$. These dynamics exist for a very narrow region parameter space when the $N^{k+1}(0) = N^k(0)$ line crosses through the region of the map where the slope quickly transitions from being greater in absolute value than 1 to being less than 1 in absolute value. Panel (d) shows the stable equilibrium dynamics with $\beta = 3 \times 10^{-7}$.

suring time series of infective juvenile densities in the soil. We have documented colonization of new lupine rhizospheres, and suggest the reasonable hypothesis that low rates of nematode immigration counter local extinctions, as first suggested for host–parasitoid interactions by Nichlson and Bailey (1935) and as in classical metapopulation theory [e.g. Hastings and Harrison (1994), Hanski (1999)].

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