THE EFFECT OF BIODIVERSITY ON THE HANTAVIRUS EPIZOOTIC

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Abstract. We analyze a mathematical model of the epizootic of Hantavirus in mice populations, including the effect of species that compete with the host. We show that the existence of the second species has an important consequence for the prevalence of the infectious agent in the host. When the two mice species survive in the ecosystem, the competitive pressure of the second species may lead to reduction or complete elimination of the prevalence of infection. The transition between the disappearance of the infection and its presence occurs at a critical value of the competitor’s population, resembling a second-order phase transition in a statistical system. The results provide a rigorous framework for the study of the impact of biodiversity in the propagation of infectious diseases, and further lends itself to future experimental verification.

Key words: biodiversity; epizootic; epidemic; Hantavirus.

INTRODUCTION: THE ROLE OF BIODIVERSITY

In 1993, an outbreak of a severe disease now known as Hantavirus pulmonary syndrome (HPS) struck the region of the Four Corners, in the North American Southwest, with a mortality in excess of 50% of those affected. Shortly afterwards, Sin Nombre Virus (Bunyaviridae: Hantavirus, SNV), the first Hantavirus to be discovered in the Americas, was identified as the infectious agent responsible for HPS. The main host of the virus was identified as one of the most common mammals in North America, the deer mouse, Peromyscus maniculatus. Since then, continuous efforts in different areas of science have been made to understand this epizootic, with the ultimate goal of correctly assessing the risk to humans. Across all of the Americas, new discoveries of Hantavirus (half of them being human pathogens and some of them responsible for a high mortality [Mills and Childs 1998]) have led to a renewed interest in the natural history of the host rodents.

A simple mathematical model for the spatiotemporal patterns in the spread of this epizootic has been proposed and analyzed by the second author and others (Abramson and Kenkre 2002, Abramson et al. 2003). The model takes into account several peculiarities of the Hantavirus–rodent association. An example being the role of El Niño on the prevalence of the SNV infection. Here, we plot the populations on a logarithmic scale to emphasize the dynamics for low-density values. The line which has been found in field studies (Yates et al. 2002), has also been taken into account. We refer the reader to Abramson and Kenkre (2002) and Abramson et al. (2003) for details. That model was able to successfully explain several field observations as environmentally controlled phase transitions, thus providing an analytical support to biological hypotheses such as the trophic cascade discussed in Yates et al. (2003). Among the consequences of the mathematical model that have correlations with the field observations, we mention the sporadic disappearance of the infection and the formation of “refugia,” from which the infection spreads—when conditions change—in the form of waves.

The model of Abramson and Kenkre (henceforth the AK model) predicts a critical transition from a state with a positive prevalence of the infection in the population (an infected phase), to a state without infection. This latter state occurs when a parameter characterizing the environment and controlling the population size drops below a certain threshold value, which we call the “critical carrying capacity.” The population value associated with this critical carrying capacity, $m_c$, is a population threshold for the system: the total population, $m$, needs to be greater than $m_c$ to be able to sustain infection at all. In the terminology of the basic reproduction number, $R_0$, this critical condition corresponds to $R_0 = 1$. Above the critical carrying capacity, the infection is able to sustain itself (equivalent to the condition $R_0 > 1$). This threshold phenomenon has been documented in the Four Corners region, as shown in Fig. 1. These population densities have been published by Yates et al. (2002), where they discuss the role of El Niño on the prevalence of the SNV infection.

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with circles shows the total *P. maniculatus* density (mean density of two nearby sites of the field study), which persists during the seven years of observation beginning in December 1994. The line without symbols shows the density of mice infected with SNV. The dashed line represents the critical population able to sustain a positive prevalence of infection, as predicted by the AK model, by using approximate parameters obtained from the time series (details of the calculation can be found in Abramson [2004]). Let us briefly analyze the implications of this graph. At time $t < 15$, the population is above the critical threshold level and, correspondingly, there is a positive infected phase. Within this regime, it is conceivable that the environmental carrying capacity has some time dependence, but that it remains above the critical value. Around $t = 15$, the carrying capacity then drops to some value below criticality, and consequently the total population $m(t)$ drops as well, approaching an equilibrium which is now below the critical value $m_c$. The drop is not monotonic: there are some discrete steps of decreasing population. Concomitantly, as $m$ begins to decrease (the indication that the carrying capacity has gone subcritical), the infected population $m_i$ begins to disappear sporadically. A few infected mice may be entering by migration, but it is clear that the infection is disappearing from the site. After $t = 30$, there begins a steady population explosion, indicating that the carrying capacity has increased, and it is observed that the population grows beyond $m_c$, indicating that the system is again above the critical level. The prevalence of infection is expected to recover. This process takes time, however, just as in the AK model (see Fig. 2 in Abramson and Kenkre 2002), and not before $t = 40$ do we see a positive $m_i$ again. After this, the population remains above critical, and the infection persists. A brief excursion of $m$ below $m_c$ (at $t \approx 70$) might be the beginning of a new extinction event, and indeed $m_i$ reaches its lowest values since those at $t = 40$. But shortly after this, the time series ends and the analysis can not be carried further. Observe that the drop in $m$ takes place in 2001, a year that was particularly dry in the North American Southwest, as can be seen in the precipitation data of Fig. 2 in Abramson (2004).

A threshold in the animal density necessary to sustain a positive seroprevalence has directly been observed in another Hantavirus system, the Puumala Virus in association with the red bank vole in Belgium (Escuente- aire et al. 2000). It is conceivable, then, that other processes that limit the population size would play a similar role in the control of the infection. In real ecosystems, mice share the environment with many others species, competing for limited resources with some, and being preyed on by others. Indeed, competition and predation stand out as the main interspecific relations for the species under consideration.

The effect of a predator is certainly akin to that of a competitor: both tend to reduce the population under study. The role of predation on the prevalence of a disease has been analyzed by Ostfeld and Holt (2004), and previously by Packer et al. (2003). As recognized in the former, other phenomena besides predation (such as limited food supply) may be the principal regulators of rodent populations. However, under the hypothesis that predation is the main factor, they find that the incidence of the disease decreases with an increasing number of predators. An important peculiarity of that model is that the number of predators is not affected by the population of prey. As a consequence, the number of predators can be used as a control parameter in the analysis of the dynamics. This assumption is reasonable for generalist predators, which are sustained by several prey species. A competitor species, on the other hand, will necessarily be affected if the shared resources are limited. The mathematical model, in such a situation, requires a feedback of each of the populations into the dynamics of the other. Various other details of the model of Ostfeld and Holt (2004) and of Packer et al. (2003) do not apply to the problem under consideration in the present paper. Among these we might mention the recovery from infection, the increased mortality of infected animals, and the increased predation of infected animals. Certainly, the model could be tailored for specific situations, and a study of the effect of predation for Hantavirus will be a necessary complement to the competition analysis that we propose in the present contribution.

Another interesting phenomenon (also relying upon the biodiversity of an ecosystem and affecting the dynamics of zoonoses) has received recent attention, as has its relevance to human health. This is the case of Lyme disease, analyzed by means of empirically based computer simulations by Van Buskirk and Ostfeld.
(1995) and by Schmidt and Ostfeld (2001). A conceptual model of the phenomenon, called the dilution effect, was presented by Ostfeld and Keeling (2000a) and further developed in Ostfeld and Keeling (2000b). The dilution effect consists in the fact that the prevalence of an infection, in the vector, of an infectious agent (ixodid ticks and a spirochete bacterium, respectively, in the case of Lyme disease) is reduced if the population of their hosts contains a diversity of species. Ticks (in their juvenile stages, which are the most relevant for the transmission to humans) are nonspecific in their feeding habits, and therefore if only one species of possible hosts acts as a competent reservoir for the transmission of the infectious agent, the existence of other hosts (which are incompetent reservoirs, being unable to transmit the bacterium to a tick feeding on them), reduces the probability of infection of susceptible ticks. As a result of this “dilution,” while the total population of ticks is not affected by the biodiversity of their hosts, the prevalence of the infection is reduced. The mechanism by which this reduction takes place in the tick population is, then, different from the one we propose in the present contribution. The prevalence of the infection in the Hantavirus host is reduced by the presence of competitors at its own trophic level, the species with which the host shares resources. These competitors exert a pressure on the host population as a whole, and the infected subpopulation may be reduced and even led to extinction (even though the total population persists, as we will show below). In other words, with the dilution effect there is a reduction in the infection by means of a reduction of the exposure, that is, of the chance of encounter between the infectious agent and a susceptible host. What it is shown in the present work is a different phenomenon. Even within the host population, the relative weight of the pathogen is reduced. We stress the ecological importance of this feature, rather than its mathematical justification.

With the purpose of gaining insight into the system, we aim to understand the role of the different processes separately, while keeping the model analytically manageable. For this reason, we have chosen to make a number of simplifications. As mentioned above, we restrict our discussion to competition interactions only, which arise among rodent species for a number of reasons. Territory, feeding habits, behaviors, and so forth all contribute to competitive behavior. In general, a particular Hantavirus has a specific host, a single species that acts as a reservoir. This host remains infected and maintains the infection in the population by horizontal transmission only. It is known that, in some cases, there may be “alternative host species,” that are able to host the virus, but for some reason are unable to transmit it successfully. Other mammals that come into contact with an infected individual of the host species may become infected. However, nonhost animals normally represent a dead end for the virus, which is eliminated by the immune response of the animal (Peters et al. 1999). In summary, these species do not play a direct role in the spreading of the disease in the wild, but they exercise, nevertheless, an ecological pressure on the host, that eventually may affect the dynamics of the zoonosis.

In the following section, we present a generalization of the AK model in the simplest case, incorporating just one competitive species that cannot be infected, and studying its effect on the dynamics of the infection. The theoretical insight obtained may suggest ways for controlling the disease agent and, therefore, its incidence. Our results show that competition reduces, in a specific way, the prevalence of the infection. Interestingly, this is in agreement with (and provides theoretical support to) a hypothesis that has been recently put to experimental test in populations of Z. brevicauda, the host of the Calabazo Hantavirus, in field studies in Panama (Suzán 2005; G. Suzán et al., unpublished manuscript; G. Suzán, personal communication). In that work, only the host was left at specific sites, removing the competing species, with the purpose of observing the change in the infection prevalence with respect to control sites, where all the competitors coexist.) Similarly, also in Panama, it has been proposed that the maintenance of competitive populations may serve to reduce the risk to human populations exposed to O. fulvescens infected with the Choclo Hantavirus (F. Koster, personal communication). The proposal, which has been called a “moat,” consists of an area surrounding human habitation maintaining a diversity of innocuous species, competing with the hosts of the Hantavirus.

As in the AK model, we study the properties of the system as a function of the parameters that characterize the (eventually changing) environmental conditions. These parameters are proportional to the carrying capacities of the system (one for each species), and therefore provide a natural choice to analyze the dependence of the prevalence of the infection as controlled by limiting resources, as suggested by the trophic cascade proposed by Yates et al. (2002). The role of environmental factors that affect rodent density, such as plant biomass and coverage, as determinants in the seroprevalence of Hantavirus, has also been suggested by Biggs et al. (2000). The potential impact of this relationship with the human risk of HPS has been successfully put to test by the use of remote sensing techniques and GIS, as reported by Boone et al. (2000).

**Intra- and interspecific competition**

We consider two species of mice, one of which is a reservoir of Hantavirus (the host species), and another which is not (the alien species). The host is subdivided into susceptible and infected populations. Let us consider that both species interact by competing for common resources. A usual description of this situation, as discussed for example by May (1981), is the following
model for the population dynamics of the total host, \( m \), and alien, \( z \), populations:

\[
\frac{dm}{dt} = (b - c)m - \frac{m}{K}(m + qz) \quad (1a)
\]

\[
\frac{dz}{dt} = (\beta - \gamma)z - \frac{z}{\kappa}(z + \varepsilon m) \quad (1b)
\]

where, for the host species, \( b \) is the birth rate, \( c \) is the death rate, \( K \) is the carrying capacity in the absence of an alien population (\( z = 0 \)), and \( q \) is the influence of the alien population; for the alien species, the analogous parameters are \( \beta, \gamma, \kappa, \) and \( \varepsilon \), respectively. We consider that the interaction is not necessarily symmetric, i.e., \( q \neq \varepsilon \). One of the species can take resources from the other in a quantity greater than what it is losing.

There are four significant equilibria for the system defined by Eqs. 1a and 1b; the others involve negative populations. Two of them consist if one species surviving over the extinction of the other. Another is the extinct phase where all populations are zero. Finally, there is a coexistence state where both species are present, and in this we will focus our interest. When only one species persists, the equilibrium population is either

\[
m^* = K(b - c) \quad (2)
\]

or

\[
z^* = \kappa (\beta - \gamma) \quad (3)
\]

where the asterisk denotes equilibrium states. If the two species coexist, then

\[
m^* = \frac{K(b - c) - q\kappa (\beta - \gamma)}{1 - q\varepsilon} \quad (4a)
\]

\[
z^* = \frac{\kappa (\beta - \gamma) - \varepsilon K(b - c)}{1 - q\varepsilon}. \quad (4b)
\]

The stability of these equilibria is immediate with the use of linear stability analysis (see, e.g., Murray 1993, Guckenheimer and Holmes 1983). The conclusion of such analysis is as follows. If the intensity of the interacting competition is not very high, \( q < 1 \) and \( \varepsilon < 1 \), then the coexistence state given by Eq. 4 is stable. On the other hand, if the competition is strong, \( q > 1 \) and \( \varepsilon > 1 \), bistability occurs: the final state depends on the initial conditions. Finally, if \( q > 1 \) and \( \varepsilon < 1 \) (or \( q < 1 \) and \( \varepsilon > 1 \)), only the strong competitor survives. For a more complete survey, see May (1981) or Murray (1993).

With the same considerations made in the formulation of the AK model, we introduce an internal classification of the subpopulation. The internal states consist of the infected subpopulation, \( m_i \), and the susceptible subpopulation, \( m_s \). It is clear that these states are mutually exclusive, and that their sum recovers the total host population, \( m = m_i + m_s \). The evolution equations for these are modeled, also as in AK, according to the following field observations: infected individuals are generated by pairwise interactions between susceptible and infected, there is no vertical transmission (from infected mother to offspring), infected individuals die with the same rate as susceptible ones (indeed, the virus does not appear to affect any physical or behavioral parameter of the infected individuals). It is worth mentioning that the lack of vertical transmission is, in the AK model, crucial for the character of the phase transition controlled by the—environmentally dependent—carrying capacity. If it is relaxed, even infinitesimally, the threshold of the carrying capacity becomes zero, and there is a positive prevalence for all values of \( K \).

The resulting model is now

\[
\frac{dm_i}{dt} = bm - cm_i - \frac{m_i}{K}(m + qz) - am_s m_i \quad (5a)
\]

\[
\frac{dm_s}{dt} = -cm_i - \frac{m_i}{K}(m + qz) + am_s m_i \quad (5b)
\]

\[
\frac{dz}{dt} = (\beta - \gamma)z - \frac{z}{\kappa}(z + \varepsilon m) \quad (5c)
\]

where \( a \) is the contagion rate. The equation for \( m = m_i + m_s \) is the same as in Eq. 1b. The AK model is recovered by setting \( q = 0 \), and ignoring the equation for \( z \). The existence of the two subpopulations of \( m \) makes each of the equilibria corresponding to \( m \neq 0 \) split into two, one with a positive prevalence (an infected phase) and one without infection. The former, for which the three populations are positive, is

\[
m^*_s = \frac{b}{a} \quad (6)
\]

\[
m^*_i = \frac{K(b - c) - q\kappa (\beta - \gamma) - b/a}{1 - q\varepsilon} \quad (7)
\]

\[
z^* = \frac{\kappa (\beta - \gamma) - \varepsilon K(b - c)}{1 - q\varepsilon}. \quad (8)
\]

Due to the fact that the coexistence equilibrium of Eq. 4 can be expressed as

\[
m^* = K(b - c) - qz^* \quad (9)
\]

we can cast the fraction of infected mice, \( \chi^* = m^*_i/m^* \), as

\[
\chi^* = 1 - \frac{b/a}{K(b - c) - qz^*} = 1 - \frac{K^c}{K^c - Kq/(b - c)} \quad (10)
\]

where \( K^c = b/a(b - c) \) is the critical carrying capacity of the AK model, below which the infected phase disappears. This last result coincides with the AK model when \( z = 0 \), so that if \( K > K^c_{AK} \), and then \( \chi^* > 0 \), the infected phase is stable, and if \( K < K^c_{AK} \) it is unstable. In the presence of the alien population \( z > 0 \), we can generalize the critical parameter as

\[
K^c = K^c_{AK} + \frac{q}{b - c}. \quad (11)
\]

Let us analyze the relevance of Eqs. 10 and 11. Eq. 10 states that the fraction of infected mice in the host population is reduced by the presence of alien mice, the host’s competitors. A graphical representation of this
result is shown in Fig. 2, where we plot $\chi^*$ as a function of the alien population, for several values of the carrying capacity $K$. Fig. 2 contains one of the fundamental results of the present work, clearly showing that the competitive pressure exerted by the alien population has the effect of reducing the prevalence of infection in the host. In fact, from Eq. 10 it can be seen that the prevalence is maximum when no alien population is present, $\chi^*(z > 0) < \chi^*(z = 0)$, and that increasing the amount of aliens will always reduce the prevalence, since $\alpha yz < 0$. A further important result of the present calculation is the existence of a critical amount of aliens, a threshold level in the population of competitors, that drives the system completely to a noninfected state. This value is given by

$$z_c = \frac{aK(b - c) - b}{aq}.$$  

(12)

When the environmental parameter $K$ has a value greater than the critical $K_c$, the system has a positive prevalence of infection. On the other hand, when $K < K_c$, the competitor population results greater than the minimum necessary to force the infected subpopulation to extinction. The point $K = K_c$ constitutes a critical point for the system, separating two behaviors that qualitatively differ in the stability of the equilibrium of the infected population. Observe that time-varying environmental conditions, as well as a heterogeneous landscape, may provide a framework where a wealth of spatiotemporal patterns of infection would occur.

In Fig. 3 we show a phase diagram that provides a complementary picture to that given by Fig. 2. The shaded contour plot shows the prevalence (as scales of gray) in the parameter space defined by the two carrying capacities, $K$ and $\kappa$. The white region, which includes the origin, is free of infection, where the state with $n_i^* = 0$ is stable. The first contour defines the transition line, beyond which a positive and stable prevalence is to be found, in a potentially infinite region in parameter space. Two sections of the phase plot are also shown, along the lines perpendicular to the axes seen in the contour plot. Each of the sections represents a projection of the prevalence along one of the carrying capacities. Both of them are in the form of a transcritical bifurcation, in which two equilibria of different character exchange their stability. The section shown at the top of the figure represents the transition as a function of the carrying capacity of the host, $K$. There is a threshold above which the population shows a positive prevalence. A similar picture characterized the AK model (as a function of $K$ only; see Fig. 1 in Abramson and Kenkre 2002). The presence of the competitor reduces the seroprevalence in such a way that the bifurcation is not a linear relation between the equilibrium solution and the control parameter. The section shown on the right panel represents the effect of the carrying capacity of the competitor, $\kappa$. In this case, a greater carrying capacity inhibits the infection, because it allows a greater population of competitors.

It is important to note that the “strength” of the competition with the alien population, $q$, is the same for both susceptible and infected hosts (Eq. 5). Indeed, both subpopulations are reduced as a result of the competition, but the infected population suffers the consequences in a stronger manner, becoming extinct in a critical way. This phenomenon, analogous to a second-order phase transition in a physical system, occurs at a finite value of the density of alien mice, $z_c$ as shown in Eq. 12.

The origin of the differential effect by which susceptible and infected hosts feel the competitive pressure differently, deserves some discussion. This effect is caused by an important difference between susceptible and infected mice that—even though it was already mentioned when the classification was introduced—until now did not have its true relevance exposed. The difference resides in the ways in which new members of each type are incorporated in the population. While the susceptible are born, the infected appear only by contagion of already born (and susceptible) mice. In biological terms, there is no vertical transmission, a fact that produces a strong asymmetry between susceptible and infected. The lack of a differential death rate between susceptible and infected animals is also a peculiarity of this system, which is uncommon in other epizootics. These two ingredients, a difference and a similitude between the two classes of host animals, are responsible for the differential effect.

**Conclusions**

We have studied a model of the epizootic of Hantavirus in a system composed of two rodent
populations: a host of the virus, which remains chronically infected by horizontal transmission, and a competitor species that does not host the virus. This situation is paradigmatic of many Hantavirus–rodent systems, in which a single species hosts the virus in a competent way, and several other species compete with the host. The model generalizes a previous model by Abramson and Kenkre (2002) (also Abramson et al. 2003), that described the dynamics of the host only. The interaction between the two rodent species, since it is a competition for limited resources, produces a mutual limitation of the populations. In particular, the second species exerts a regulatory effect on the host. This competitive pressure is the same for both susceptible and infected hosts (as it should be, since the infection with Hantavirus does not affect any biological parameters in the hosts). Nevertheless, a reduction of the seroprevalence in the host species is observed, up to a point where it completely disappears from the system. This phenomenon can be described as a critical transition between an infected phase and a phase free of infection, controlled by the environmentally determined—carrying capacities of the two species. In the space defined by these carrying capacities, spatially or temporally varying environmental conditions would be described by a path, which might cross the transition line, thus driving a corresponding change in infection phase. This is analogous to the results discussed by Abramson and Kenkre (2002) and Abramson et al. (2003), as well as, conceptually, by Yates et al. (2002). Furthermore, it is interesting that a change in the carrying capacity of the competitor only (a vertical path in the phase space of Fig. 3) is enough to drive the infection to extinction. In a real-world situation, where the carrying capacities exhibit temporal as well as spatial heterogeneity, a wealth of dynamical transitions are to be expected.

A key question in any epidemic situation is, given the values of the parameters and the initial populations, whether an initial infection will spread or not. The answer can be found through the analysis of the basic reproductive rate $R_0$ of the infection, defined as the number of secondary infections produced by one primary infection in a wholly susceptible population (see, e.g., Murray 1993, chapter 19, or Anderson and May 1992, chapter 2). Thus, if $R_0 < 1$, any initial infection dies out, and an epidemic cannot occur. On the other hand, if $R_0 > 1$, an initial infected population increases and spreads the infection. In the absence of the logistic term (as in a standard susceptible–infected [SI] or susceptible–infected–removed [SIR] model) we would have $R_0 = am(0)/c$, since $a$ is the contagion rate and $c$ is the removal rate (observe that $c^{-1}$ is the infection period, which equals the lifespan of the animals). In the present model, the competition terms in the dynamics of the host act as additional death terms, modifying the removal rate (or, equivalently, the life span). From Eq. 5a–c and the usual condition for the

![Fig. 3. Phase diagram of the infection prevalence, in the space defined by the host carrying capacity $K$ and the alien carrying capacity $\kappa$. The contour plot shows the prevalence as shades of gray, with the darkest shades corresponding to higher prevalence. The white region is the region of zero infection. The crossing lines indicate sections of the plot, shown in the upper and right side plots, where the prevalence is shown as a function of the relevant control parameter. The right panel represents the extinction of the infection controlled by the alien carrying capacity. The parameters, in this example, are: $b=1, c=0.6, q=0.2, \beta=1, \gamma=0.5, \varepsilon=0.1, a=0.1$; where $q$ is the influence of the alien population, $b$ is the birth rate, and $c$ is the death rate; $\beta, \gamma,$ and $\varepsilon$ are the analogous parameters for the alien species; and $a$ is the contagion rate.](image-url)
onset of an epidemic, \( dn_i/dt > 0 \), it is easy to arrive at the following expression for \( R_0 \):

\[
R_0 = \frac{am_i(0)}{c + \frac{m(0)}{K}}
\]

(13)

where the populations take their initial values, at time \( t = 0 \). We can see, in Eq. 13, that any alien population \( z(0) \) tends to reduce \( R_0 \), thus reducing the ability of the infection to spread out. Its effect is opposed by the carrying capacity \( K \), which tends to increase \( R_0 \) and favor the spread of the infection. In addition, the threshold condition \( R_0 = 1 \) allows us to derive an equivalent threshold in the alien population. As a result, the alien population necessary to suppress the initial spread of the infection is

\[
z(0) > \frac{K[am_i(0) - c] - m(0)}{q}.
\]

(14)

Given an initial state of a healthy population of hosts, Eq. 14 defines a minimum population of competitors that would inhibit the spread of a small outbreak of infection.

Our model has been intentionally kept simple to allow a direct analysis of the consequences by analytical means. This thus provides a manageable framework to study a necessary step towards the modeling of realistic systems, which certainly contain several species of rodents and small mammals in competitive interaction, as well as other species in the trophic web. The study of similar systems, for example, when competing species are organized in a hierarchical way (Tilman [1984]; also see Hess [1996] for its use in the context of epidemic dynamics), the role of fluctuations when populations are small, etc., are currently under study and will be presented in forthcoming contributions.

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