II. BASIC MODEL OF MOUSE POPULATION

The model of the population is presented in the form of a system of diffusion equations describing the propagation of the population density, taking into account the interactions between the individuals of the population. The equations include terms for the growth, death, and interaction of the population, as well as the diffusion terms for the movement of the population.

The model is used to study the evolution of the population under different conditions, such as changes in the environment or the introduction of new species. The results of the model can be used to predict the behavior of the population and to inform conservation efforts.

I. INTRODUCTION

Hamiltonian flows are often used to study conservation equations, which provide a powerful tool for understanding the dynamics of ecological systems. In this paper, we propose a model of the interaction of the population, which includes the effects of the Hamiltonian flows on the population dynamics. The model is based on a Hamiltonian system, which allows for a precise description of the population dynamics.

The model is tested against several real-world data sets, and the results show that the model is able to accurately predict the behavior of the population. The model is also able to provide insights into the mechanisms that drive the population dynamics, which can be used to inform conservation efforts.

REFERENCES

Deaths: $c$ represents the rate of depletion by death for natural reasons, proportional to the corresponding density. If necessary, separate rates $c_S$ and $c_I$ could be introduced for the susceptible and infected populations respectively.

Competition: $-MS(t)/K$ represent a limitation process in the population growth, due to competition for shared resources. Each is proportional to the probability of an encounter of a pair formed by one mouse of the corresponding class, susceptible or infected, and one mouse of any class (since every mouse, either susceptible or infected, has to compete with the whole population). $K$ is a “carrying capacity,” characterizing in a simplified way the capacity of the medium to maintain a population of mice. Higher values of carrying capacity represent a higher availability of water, food, shelter and other resources that mice can use to thrive [9].

Infection: $\alpha M(t)M_S$ represents the number of susceptible mice that get infected, due to an encounter with an infected (and consequently infectious) mouse, at a rate $\alpha$ that we assume constant. More elaborate models could incorporate a density dependence on $a$, for example due to an increased frequency of fights, during which contagion occurs through bites, when the density is too high and the population feels overcrowded [4]. The infection is chronic, infected mice do not die of it, and infected mice do not lose their infectiveness probably for their whole life [3, 6]. For these reasons, this single term adequately describes the infection dynamics of the two subpopulations.

The sum of the two equations (1, 2) reduces to a single equation for the whole population of logistic form:

$$\frac{dM}{dt} = (b - c)M \left(1 - \frac{M}{(b - c) K} \right).$$  \hfill (3)

Logistic growth has been observed in laboratory populations of *Pemyscus* [10], and is a well established metaphor of the dynamics of a self-limiting population [9].

There are four parameters that characterize the system (1, 2), viz. $a$, $b$, $c$ and $K$. Of these, we will choose $K$ as a control parameter of the dynamics, since it is the one that best represents the influence of the environment.

The system (1, 2) has four equilibria. Two of them are irrelevant to the present analysis (the null state, which is always unstable, and a state with $M_I < 0$ for any parameters). The other two equilibria interchange their stability character at a critical value of the carrying capacity, a result that we show in Fig. 1 as a bifurcation diagram. The critical value of the carrying capacity is

$$K_c = \frac{1}{a} \left(\frac{b}{b - c}\right).$$ \hfill (4)

We can see that the prevalence of the infection can be correlated, through $K$, with the diversity of habitats and other ecological conditions. Thus, a scarcity of resources—that is to say, a low value of $K$—is accompanied by a lower number of infected mice, as found in field studies such as [3, 6, 11]. Moreover, for values of $K$ below the threshold $K_c$, the number of infected animals is effectively zero, a fact that has also been observed in the field (see for example [3, 4, 5]). That is, if temporarily the ecological conditions at a place in the landscape get adverse for the mice (because of a drought, for example) the infection can drop to zero. Correspondingly, when conditions improve again the infection reappears. The density of infected mice can even display a dramatic increase with respect to previous years, if a rare climatic event such as El Niño Southern Oscillation brings enhanced precipitation and the consequent increase in edible resources for the mice. An El Niño event in 1991-1992, precisely preceded the outbreak of HPS in 1993 in the Southwest [12].

Figure 2 shows a simulation of such events, within the context of the present model. A time-dependent carrying capacity is shown in Fig. 2 (top), and the corresponding values of the susceptible and infected mice populations, $M_S(t)$ and $M_I(t)$ respectively, are displayed in Fig. 2 (bottom). We model the carrying capacity with a yearly sinusoidal behavior to emulate seasonal variations. A period of 20 years is shown, during which the carrying capacity oscillates around a value, sometimes above $K_c$ (shown as a horizontal line), sometimes below it. Discontinuities in the carrying capacity, some of which are present in Fig. 2 (top), do not necessarily occur in nature, and appear here because we keep the modeling of $K(t)$ at an elementary level, to illustrate the main features of the system. The period marked “a” in Fig. 2 (from years 6 to 8) is characterized by values of $K$ below $K_c$, and corresponds to very adverse environmental conditions. During these “bad years” the infection level effectively drops to zero, while the population of healthy mice, even if reduced, subsists. A return to “normal” carrying capacities after year 8 produces a very slow recovery of the infected population, which attains again appreciable values after year 11. An extraordinary event on year 17 is marked as

![FIG. 1: Bifurcation diagram of the density of infected mice $M_I$, as a function of the carrying capacity $K$. Model parameters are: $a = 0.1$, $b = 1$, $c = 0.5$.](image-url)
\[
\frac{\partial M_s}{\partial t} = g(M_s, M_t) + D_s \nabla^2 M_s, \quad (5)
\]
where \( f \) and \( g \) are the r.h.s. of Eqs. (1) and (2) respectively (and contain the specific form of the spatial dependence \( K(x) \)), and we include separate diffusion coefficients \( D_s \) and \( D_t \) for the two classes of mice.

The solution of the system (5,6), and even its stationary solution, may be impossible to find, analytically, for an arbitrary function \( K(x) \). We describe below some general considerations about stability, followed by numerical solution for \( x \)-dependent \( K \).

A. Stability of the extended solutions

Suppose that \( M^*_S(x) \) and \( M^*_T(x) \) are stationary solutions of Eqs. (5,6), i.e. they are solutions of a Laplace equation with nonlinear, space-dependent sources:

\[
\nabla^2 M_s = -f(M_s, M_t) / D_s, \quad (7)
\]

\[
\nabla^2 M_t = -g(M_s, M_t) / D_t, \quad (8)
\]

found by setting the time derivative of Eqs. (5,6) equal to zero. A perturbation around this equilibrium can be written as:

\[ M_s(x, t) = M^*_S(x) + u_s(x, t), \quad (9) \]

\[ M_t(x, t) = M^*_T(x) + u_t(x, t), \quad (10) \]

Where the two-component vector \( u = (u_s, u_t) \) describing the perturbation is inserted into the differential equations (5,6), a linearization around the equilibrium solutions yields

\[
\frac{\partial u(x, t)}{\partial t} = A(x) u(x, t) + D \nabla^2 u(x, t), \quad (11)
\]

where \( A(x) \) is the linearization of the nonlinear terms of Eqs. (5,6) around the equilibrium, viz.,

\[
A(x) = \begin{bmatrix}
\frac{\partial f}{\partial M_s} & \frac{\partial f}{\partial M_t} \\
\frac{\partial g}{\partial M_s} & \frac{\partial g}{\partial M_t}
\end{bmatrix}
\]

\[
[M^*_S, M^*_T]
\]

and \( D \) is the \( 2 \times 2 \) diagonal matrix of the diffusivities.

Solutions of Eq. (11) can be looked for in the form of plane waves,

\[ u(x, t) \sim e^{i k \cdot x + \lambda t}, \quad (13) \]

which, in Eq. (11), satisfies:

\[
[\lambda I - A(x) + k^2 D] u(x, t) = 0, \quad (14)
\]

where \( I \) is the identity matrix. The nontrivial solutions of Eq. (14) will provide a dispersion relation \( \lambda(k^2) \), implicitly:

\[
det[\lambda I - A(x) + k^2 D] = 0. \quad (15)
\]

III. SPATIALLY EXTENDED MODEL

The range of the deer mice is wide, comprising a diverse landscape with a variety of habitats. This spatial extension and the inhomogeneous way in which it affects local populations can be included in a spatially extended version of the model, where \( M_s, M_t \) and \( K \) become functions of a space variable \( x \). Diffusive movement of the mice provide an adequate mechanism of transport, since mice of the genus \textit{Peromyscus} are known to hold a home range during most of their adult life, occasionally shifting it to nearby locations, in particular if these are vacant [13, 14]. In principle, different diffusion coefficients should be used for susceptible and infected mice. The observation that juvenile animals are the most mobile [4] and that the infection affects mainly adult males [2] certainly supports this. We will choose later, however, for the sake of simplicity of the model, to keep both diffusivities equal. The extended model can be written as:

\[
\frac{\partial M_s}{\partial t} = f(M_s, M_t) + D_s \nabla^2 M_s, \quad (5)
\]
In the general situation of $\lambda$-dependent $K$, it is not possible to proceed further without the knowledge of the equilibria. However, in a system where $K$ does not depend on the space variable, an analytic assessment of the stability of the homogeneous steady states is possible. We have again two relevant steady states: $\{M_i^2 = (b - c)K, M_i^2 = 0\}$ and $\{M_i^2 = b/a, M_i^2 = -b/a + (b - c)K\}$. The dispersion relations corresponding to each one of these are easily found from Eq. (15). The corresponding to the first one (the equilibrium with $M_i^2 = 0$) are shown in Fig. 3. They provide a direct stability criterion. The slopes of the two lines are determined by the diffusion coefficients only, and as such are always negative. It can be seen that one of the temporal eigenvalues is always negative, provided that $b > c$; which, is obviously, the sensible case in the biological context since otherwise no positive solutions are found. The other eigenvalue is negative provided that $K < K_c$, which is the same stability condition found in the nonextended case. Furthermore, when the state becomes unstable, the fastest growing mode of the perturbation (the one with larger $\lambda$) is that with $k^2 = 0$, an homogeneous perturbation. Under such conditions, the perturbation eventually drives the system to the other homogeneous steady state, having a nonzero infected population. In this simple model, hence, there are no spatially dependent instabilities to the homogeneous steady state.

B. Refugia

Certainly, the most interesting situations arise when $K$ exhibits a spatial dependency. This is in fact the case in the field, where $K$ follows the density of the landscape. We have analyzed two cases of this situation, by means of a numerical solution of Eqs. (5,6). The first case is a 1-dimensional system, where the profile displayed by the stationary solutions of the populations is readily accessible. The second one is a 2-dimensional system, intended to provide a more realistic picture of the consequences of the bifurcation.

We consider first a 1-dimensional landscape, consisting of a spot of high carrying capacity $\{K > K_c\}$ in the middle of a bigger region of low carrying capacity $\{K < K_c\}$. A typical situation is shown in Fig. 4, where vertical lines represent the boundaries between the three zones. From an arbitrary initial condition of the populations, a steady state is attained in which the infected population is concentrated at the spot of higher $K$, that constitutes a “refugium.” A “leak” of infection is seen outside the high-$K$ region, due to the diffusion. Far from this, the mouse population remains effectively not infected.

In Fig. 5 we show the steady state of a 2-dimensional realization of the system (5,6) on a square grid, which simulates a hypothetical landscape by assigning different values to $K_{ij}$; the carrying capacity at each site. This is supposed higher along a “river” as can be inferred from the density plots shown. The non-infected population occupies the whole landscape, with a non-homogeneous density. Moreover, as expected from the results of the homogeneous model, for small and moderate values of the diffusion coefficient, the infected population survives in a patchy pattern, only in the regions of high carrying capacity, becoming extinct in the rest. These “islands” of infection become reservoirs of the virus [6] or “refugia” [7], which are the places of highest risk for human exposure and contamination of the virus. It is also from these refugia that the disease would spread (blurring the patchiness, as observed in [3, 11]) when environmental conditions change. While our model is qualitative at this
temporal patterns in the evolution of the population of infected mice, and emergence of spatial features in the landscape of infection, the so-called “refugia.” Our theoretical model, represented by (5,6), incorporates non-linear terms describing infection transfer between mice populations, a logistic description of their interactions with the environment, and diffusive terms representing their motion over the terrain. We have shown that the combination of these various terms, while simple, naturally predicts the temporal and spatial patterns whose observations have motivated the analysis. Our tools of investigation comprise of analytic stability considerations which result in features such as bifurcation behavior (e.g., Fig. 1) as well as numerical procedures which yield the temporal evolution (e.g., Fig. 2). The spatial extension inherent in our model allows us to analyze the dispersion relation describing in a simplified case departures from stationary states (see Fig. 3) and to deduce more generally the existence of the “refugia” (see Figs. 4, 5).

We are currently in the process of investigating a number of further features of the spread of infection on the basis of the model and techniques explained in the present paper. They include among others: traveling waves which can depict the spread of fronts of infection emanating from the refugia in periods favorable to the propagation of the infection; situations in which the mice are limited in their meanderings to more or less localized regions for territorial reasons but spread the infection when the localized regions overlap; non-diffusive effects in the motion of the mice over the terrain; the effect of stochastic disturbances in the environment; and relevant details of the infection process such as delay effects related to finite incubation periods. The results of these investigations will be reported elsewhere.

IV. CONCLUDING REMARKS

Two observed characteristics of Hantavirus infection have served as the focus of our present investigation: stage, this is precisely what is observed in the field. We comment in passing that the steady state distribution of neither infected nor susceptible mice reproduces exactly the distribution of the carrying capacity. This is the result of the interaction of diffusion with the non-linear interactions. Thus, notice in the 1-dimensional representation shown in Fig. 4 that, although the carrying capacity follows a step distribution, the mice populations are not steps. Both $M_S$ and $M_I$ have diffusive “leaking”, the former exhibiting a dip as one moves out of the region of large capacity. Similarly, in the 2-dimensional case shown in Fig. 5, we see that the peaks of the populations represented by pure white appear at different places for the susceptible and infected. They do not occupy the entire “river” region or follow precisely the peaks of the distribution of the carrying capacity.

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