

Theory of possible effects of the Allee phenomenon on the population of an epidemic reservoir

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We investigate possible effects of high-order nonlinearities on the shapes of infection refugia of the reservoir of an infectious disease. We replace Fisher-type equations that have been recently used to describe, among others, the Hantavirus spread in mouse populations by generalizations capable of describing Allee effects that are a consequence of the high-order nonlinearities. After analyzing the equations to calculate steady-state solutions, we study the stability of those solutions and compare to the earlier Fisher-type case. Finally, we consider the spatial modulation of the environment and find that unexpected results appear, including a bifurcation that has not been studied before.

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I. INTRODUCTION

Techniques of nonlinear physics and mathematics are finding much application in recent times in biological and ecological systems, which, in turn, are enriching nonlinear science. One example is the spread of epidemics [1–5], in particular the Hantavirus [6–10]. Hantavirus, which is transmitted to humans by mice, is of great concern to human health in many regions of the world. Interpersonal transmission in humans has not been reported. Different species of mice act as the reservoir of specific strains of virus and the infection among them is only intraspecific. Thus, it is sufficient to study the dynamics of Hantavirus spreading in mice populations alone. This has been recently modeled [11–14] with the help of the Fisher equation [15] with internal states representing the infection or its absence in the mouse population. Inspired by these phenomena particularly related to the Hantavirus spreading in mice populations, we study the consequences of Allee mechanisms (to be described below) in the dynamics of a population of susceptible and infected individuals. As with mice that are the carriers of the Hantavirus, the infection does not affect the survival of the infected individuals. Without these effects, the (Fisher-type) equations are [11,12],

$$\begin{aligned}\frac{\partial M_S}{\partial T} &= bM - cM_S - \frac{M_S M}{K} - aM_S M_I + D \frac{\partial^2 M_S}{\partial X^2}, \\ \frac{\partial M_I}{\partial T} &= -cM_I - \frac{M_I M}{K} + aM_S M_I + D \frac{\partial^2 M_I}{\partial X^2}, \\ \frac{\partial M}{\partial T} &= bM - cM - \frac{M^2}{K} + D \frac{\partial^2 M}{\partial X^2},\end{aligned}\quad (1)$$

where we display a one-dimensional (1D) system for simplicity. Here, M_S and M_I are, respectively, densities of susceptible and infected individuals and $M = M_S + M_I$ is the total population density, a controls transmission of infection on encounter, b and c are rates of birth and death, respectively, and D is the diffusion constant of the individuals. The third

equation in Eq. (1) is obtained simply by adding the first two and is of the standard Fisher form [1]. The first two equations are associated to characteristic features of any population that plays the role of the reservoir of an infectious disease [10]. Individuals are never born infected and they are unaffected in any other way (for instance, they do not die faster) when infected. The logistic reaction term in the last equation in Eq. (1) is made up of a linear growth term $(b-c)M$ and bilinear depletion term $-M^2/K$. Such a description of population dynamics is widespread [1]. The steady-state homogeneous solutions for M are [11] $0, K(b-c)$. The first of these is unstable whereas the second $M = K(b-c)$ is stable.

The Fisher analysis of the dynamics of a given population is based on the use of a logistic reaction term, whereas the investigation we report in the following assumes a Nagumo term which provides an additional zero in the nonlinearity relative to the logistic case. The physical content behind such a term is the *Allee effect*, in the presence of which—unlike in the logistic case—the zero- M solution is stable. If M is small initially, it is attracted to the vanishing value; if large, it is attracted to the nonzero value. The physical origin of the Allee effect is the possible increase in the survival fitness as a function of the population size for low values of the latter. Existence of other members of the species may induce individuals to live longer whereas low densities may, through loneliness, lead to extinction. There is a great deal of evidence for such an effect in nature [16,17] and there have been recent reports [18,19] of theoretical work addressing the effect.

This paper is set out as follows. The Allee effect is described by adding cubic terms to the logistic dependence and the model is displayed in Sec. II: both in dimensioned and dimensionless forms. The former is important to understand the connection of the parameters to quantities observed in nature, while the latter facilitates mathematical analysis. We also define two quantities α and χ important to our later development. The former is central to the classification of regimes of behavior and is directly associated with the Allee phenomenon. We present a linear stability analysis of the steady-state solutions in Sec. III and argue the importance of the threshold $\alpha = 0.5$ on the basis of a Ginzburg-Landau dis-

discussion. Restricting attention to the interesting case when χ exceeds a critical value χ_c , we carry out numerical solutions of the model equations in Sec. IV for the simple case of spatially constant α , and in Sec. V for the richer case of spatially modulated α . Conclusions appear in Sec. VI.

II. MODEL

Our starting set of equations is, instead of Eq. (1),

$$\begin{aligned}\frac{\partial M_S}{\partial T} &= bM^2 - cM_S - \frac{M_S M^2}{K} - aM_S M_I + D \frac{\partial^2 M_S}{\partial X^2}, \\ \frac{\partial M_I}{\partial T} &= -cM_I - \frac{M_I M^2}{K} + aM_S M_I + D \frac{\partial^2 M_I}{\partial X^2}, \\ \frac{\partial M}{\partial T} &= -cM + bM^2 - \frac{M^3}{K} + D \frac{\partial^2 M}{\partial X^2}.\end{aligned}\quad (2)$$

The birth term is quadratic in the population density and the environment population term is cubic. This is in contrast to the Fisher case (1) where these terms are linear and quadratic, respectively. Needless to say, using a quadratic versus linear birth rate has nothing to do with sexual versus asexual reproduction. Surely, the individuals do reproduce sexually. The effective power to be used in the equations is the result of a variety of factors influencing one another including the probability of encounter of mates. The appropriate power of the density can be determined only phenomenologically, after the fact. Similar considerations apply to the quadratic versus cubic environment term. The important distinguishing feature of the Allee case is the possibility of extinction that small enough populations have to face as a result of the nonlinearity, and mathematically speaking, the existence of two (rather than one) stable fixed points.

The simplest way to analyze the steady states of the system is to first look at the solutions of M from the last equation in Eq. (2). There are three possible homogeneous steady-state solutions for M : $M_0=0$, $M_{mx}=(bK+\sqrt{b^2K^2-4cK})/2$, and $M_{mn}=(bK-\sqrt{b^2K^2-4cK})/2$. By performing the linear stability analysis of these solutions, we find that M_0 and M_{mx} are stable while M_{mn} is unstable. However, it is important to note that if $b^2 < 4c/K$, there is only one real solution corresponding to $M=0$. It is convenient to reduce Eq. (2) to dimensionless form by performing the following substitutions:

$$\begin{aligned}x &= X\sqrt{M_{mx}^2/KD}, \\ t &= M_{mx}^2 T/K, \\ m &= M/M_{mx}, \\ m_s &= M_S/M_{mx}, \\ m_i &= M_I/M_{mx}.\end{aligned}\quad (3)$$

We introduce two new quantities that will prove important in the subsequent analysis,

$$\alpha = \frac{M_{mn}}{M_{mx}} = \frac{bK - \sqrt{b^2K^2 - 4cK}}{bK + \sqrt{b^2K^2 - 4cK}}, \quad (4)$$

$$\chi = (1 + \alpha) \frac{a}{b}. \quad (5)$$

Using $b/M_{mx}=(\alpha+1)/K$ and $c/M_{mx}^2=\alpha/K$, we get the following transformed dimensionless equation set:

$$\begin{aligned}\frac{\partial m_s}{\partial t} &= (\alpha + 1)m^2 - \alpha m_s - m_s m^2 - \chi m_s m_i + \frac{\partial^2 m_s}{\partial x^2}, \\ \frac{\partial m_i}{\partial t} &= -\alpha m_i - m_i m^2 + \chi m_s m_i + \frac{\partial^2 m_i}{\partial x^2}, \\ \frac{\partial m}{\partial t} &= m(1 - m)(m - \alpha) + \frac{\partial^2 m}{\partial x^2}.\end{aligned}\quad (6)$$

III. LINEAR STABILITY ANALYSIS

Given that the definition of α in Eq. (5) as the population density ratio excludes consideration of values of α that are not real, the only steady values for m are 0, α , and 1, and there is only one steady stable state for the total population, corresponding to extinction. The solutions $m=0, 1$ are stable while $m=\alpha$ is unstable. The crucial consequence of the Nagumo term (cubic nonlinearity) is that the solution $m=0$ is stable, whereas in the Fisher case it is necessarily unstable. Thus, in the presence of Allee effects, it is possible that the population may vanish completely. Since the system in its steady state adopts the values of the stable solutions, we need to analyze the population density when $m=0$ or $m=1$.

The case $m=0$ is trivial. When $m=1$, we have only two valid solutions for (m_s, m_i) , $(1, 0)$, and $[(\alpha+1)/\chi, (\chi-1-\alpha)/\chi]$. We can observe, by performing a linear stability analysis, that when the second solution adopts negative values, it is unstable while the first one is stable. When $\chi=1+\alpha$, there is a transition: the first solution becoming unstable and the second becoming positive definite and stable. Thus, $1+\alpha$ represents the critical value of χ ,

$$\chi_c = 1 + \alpha.$$

The results can be summarized as follows:

$$(m_s, m_i) = \begin{cases} (0, 0), (1, 0) & \text{if } \alpha > \chi - 1 \\ (0, 0), \\ [(\alpha + 1)/\chi, (\chi - 1 - \alpha)/\chi] & \text{if } \alpha \leq \chi - 1. \end{cases} \quad (7)$$

These comments about the fixed points shed light on the main issue of interest in this paper, viz., the steady-state *spatial density profiles* of the population. Here, we would like to stress that, in the presence of Allee effects, the infected population density vanishes for $a < b$. In their absence on the other hand, when the Fisher equation is appropriate [11], the condition involves not only a and b but also c and K . As χ crosses the value χ_c , there is a change in the stable character

of the solutions. The system shows an *imperfect pitchfork bifurcation*: the number of fixed points is two for $\chi < \chi_c$ and three for $\chi > \chi_c$. The bifurcation is imperfect as the system under study is not symmetric under reflection, i.e., $m_s \rightarrow -m_s$ and $m_i \rightarrow -m_i$. By contrast, the Fisher modeling of the epidemics shows a *transcritical bifurcation* [11].

Our study of this system shows that $\alpha=0.5$ marks an interesting threshold that separates two regimes corresponding to larger or smaller values of α . To see this, recast Eq. (6) in the Ginzburg-Landau form,

$$\frac{\partial m}{\partial t} = - \frac{\delta \mathcal{F}}{\delta m},$$

where the functional representing the free-energy density is given by

$$\mathcal{F}(x,t) = \int dx \left[\frac{1}{2} \left(\frac{\partial m}{\partial x} \right)^2 + F(m) \right], \quad (8)$$

$$F(m) = - \int_0^m f(m') dm'. \quad (9)$$

The dynamics is determined by \mathcal{F} in that the system evolves such that $\frac{d\mathcal{F}}{dt} \leq 0$. The expression for \mathcal{F} contains two terms. The first term involving the derivative tries to minimize \mathcal{F} by minimizing the fluctuations in the density. The minima of second term, i.e., $F(m)$ are governed by the reaction term $f(m) = m(1-m)(m-\alpha)$, and so we have

$$F(m) = m^4/4 + \alpha m^2/2 - (1 + \alpha)m^3/3.$$

The significance of the threshold value $\alpha=0.5$ is that $F(0) = F(1)$, i.e., solutions corresponding to both $m=0$ and $m=1$ are *equally* stable. When $\alpha < 0.5$, the solution $m=1$ is relatively more stable than the solution $m=0$. The situation is reversed for $\alpha > 0.5$.

IV. HOMOGENEOUS ENVIRONMENT (CONSTANT α)

Because infected population density is always zero irrespective of values of α in the subcritical regime $\chi < \chi_c$, we focus our attention below only on steady-state population density profiles for the other more interesting supercritical regime $\chi > \chi_c$. In this case, there is a possibility of getting nonzero m_i and the steady state depends on the parameter α as well. In order to find the spatial pattern, we solve Eq. (6) numerically with a given initial spatial distribution of susceptible and infected population density.

We consider a bounded domain where $\chi=2$ everywhere (supercritical case since $\alpha < 1$) and use reflective boundary conditions. We analyze various values of α and spatially varying initial densities. Except for $\alpha=0.5$, we find that the system evolves toward a homogeneous steady state, with values corresponding to the steady solutions. If we take $\alpha < 0.5$ and $m_s(x) = m_i(x) = A|\cos(2\pi\omega x)|$ —a periodically modulated initial condition for the initial population—we observe that the system evolves to a steady state characterized by a homogeneous solution, with $m=1$ and a nonzero value for the infected population density $m_i = (\chi-1-\alpha)/\chi$, in the

entire spatial domain. In other words, the system evolves toward the stable state corresponding to $m=1$ and *not* toward the other stable state for $m=0$. Such a possibility of choice between attractors is a feature associated with the Allee effect. It does not arise in the earlier description of Hantavirus spread [11] via Fisher-type equations because there is, in that description, only one attractor. If, on the contrary, $\alpha > 0.5$, the total population density m vanishes completely in the whole spatial domain. This is, in fact, an Allee effect. The observed effect is in contrast to the result obtained using Fisher-type equation where population can never go to zero because that would correspond to an unstable state.

The case $\alpha=0.5$ presents a much richer and complex behavior of the system. This situation is, however, not of biological interest because it corresponds to a set of parameters of measure zero. Although we have obtained some interesting results for that case, we do not display them in detail because of their lack of relevance to observational matters and comment on them only in passing in Sec. VI.

V. INHOMOGENEOUS ENVIRONMENT (SPATIALLY VARYING α)

Noteworthy effects appear when Allee effects combine with a spatially varying α . To study them, we maintain the relationship $\alpha \leq \chi-1$ throughout the extension of the whole domain—so that we have a supercritical solution—but assume that the parameters that go into the making of α , viz., the birth parameter b , the death rate c , and the environment parameter K [see Eq. (5)], are spatially varying and consequently introduce a corresponding modulation into α .

The modulations or inhomogeneities of the environment are a central point in the study of ecological communities and population dynamics. Indeed, the concept of *ecological corridor* has been often used in the biological literature. It means a thin strip of vegetation used by wildlife potentially allowing the movement of biotic factors between two areas. In a more general way, the modulations provide a linkage between the resource habitats of a species consisting of a landscape structure that is different from the matrix surrounding it. In the last years, the analysis of the effect of corridors on mice populations has attracted the attention of biologists [20–22].

To understand transparently the physical meaning of a postulated modulation of α as we take below, it is helpful to consider—only for illustration purposes—the case when the quantity $4c/b^2K$ is small and to expand the α expression in Eq. (5) in powers of that quantity. Then we get

$$\alpha \approx \frac{4c}{b^2K}. \quad (10)$$

Any inhomogeneity in the environment represented by a spatial variation of c , b , or K would be reflected in a spatial modulation of α . A larger death rate, a smaller environmental parameter, or a smaller birth rate would cause a decrease in α .

We have analyzed the effect of such a spatial variation in two forms: first by considering a sinusoidal modulation with a characteristic wave number and then by considering a less

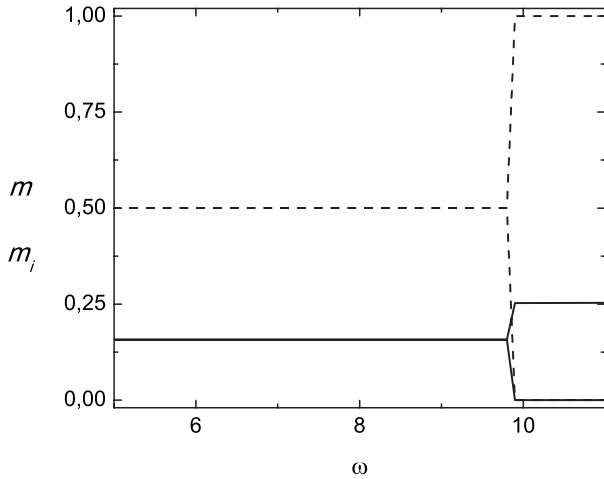


FIG. 1. Mean values of the total (solid) and infected (dashed) populations as a function of ω ; the wave number that describes the modulation of $\alpha(x)$.

regular behavior of α . In both cases, we performed the numerical integration of Eq. (6) using a Crank-Nicolson scheme, expanding the nonlinear terms as shown in [23]. The space step Δx was 100 times smaller than the smallest considered period of the oscillating $\alpha(x)$. First we take $\alpha(x) = 0.5 + B \sin(2\pi\omega x)$, with $B < 0.5$ so we stay within the region where $\chi > \chi_c$ and avoid $\alpha < 0$. Unexpected behavior emerges. The system undergoes a transition as the wave number ω of the modulation of α crosses a given critical value $\omega_c \approx 10$. When $\omega < \omega_c$, the modulation of α induces a modulation in the population. This occurs around 0.5 for the total population and around $\frac{\chi-1-\alpha}{2\chi}$ for the infected population. But as ω exceeds ω_c , despite the modulation of α , the total population adopts a homogeneous profile with two different values. There are two branches, each one corresponding to the steady solutions shown in Eq. (7) for $\omega > \omega_c$. Together with the absence of modulation in the total population, we observe that the infected population survives throughout the whole domain. While the total population is homogeneous, both the infected and susceptible populations are oscillatory in space and take values different from those previously calculated.

Figure 1 displays graphically the results discussed above. The mean value of the total and infected population is plotted as a function ω ; the wave number that describes the modulation of $\alpha(x)$. A bifurcation is seen at $\omega_c \approx 10$. The system undergoes a sudden change in the behavior. The total population adopts a homogeneous profile while the infected population now survives even in regions where $\alpha(x) > 0.5$. This can be observed in the two plots of Fig. 2, where the spatial profile of both the total and infected populations are shown for values of ω above and below the value ω_c .

Next we provide an example of a more realistic behavior of the environment that allows us to show that this effect survives even when the modulation is not regular provided regions exist with oscillations of wave number over the critical value. In Fig. 3 we plot a situation presenting an irregular profile for α (also displayed in the figure) and the solutions for both branches.

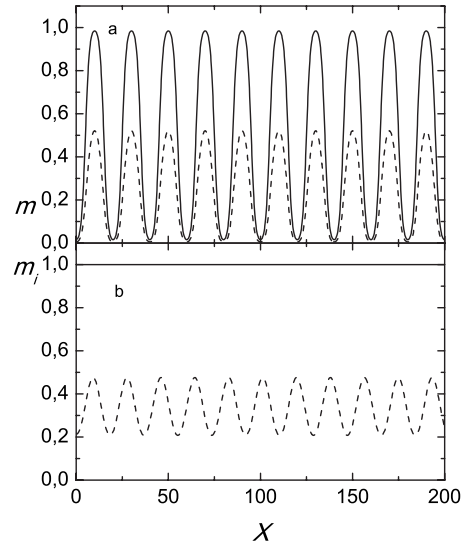


FIG. 2. Spatial profile of the total (solid) and infected (dashed) populations for (a) $\omega < \omega_c$ and (b) $\omega > \omega_c$ (only the non-null branch is shown). Space is plotted on the x axis in arbitrary units.

VI. CONCLUSIONS

In summary, we have studied what consequences the possible presence of an Allee phenomenon might have in a population of individuals involved in the spread of an infectious disease. The Allee features have been incorporated through a Nagumo, i.e., cubic, term in the nonlinearity. We have analytically solved and examined the fixed points of the problem without diffusion and numerically investigated the steady-state profiles in the presence of diffusion. Our findings are that, first, as expected in the presence of the Allee phenomenon, the population can vanish completely. This is not possible when the underlying equation is of the type used

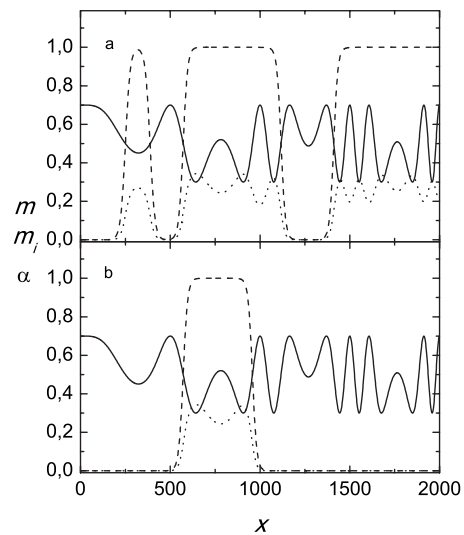


FIG. 3. Spatial behavior of the total and infected populations as α (also displayed) varies in space. Each plot corresponds to a different branch. Depicted are α (solid line), the total population m (dashed line), and the infected population m_i (dotted line). Space is plotted on the x axis in arbitrary units.

earlier [11]. We also observe a dependence on initial conditions that are not present in the earlier analysis. These effects stem from the existence of two rather than one stable solutions. Formally stated, under the effect of the Allee phenomenon, the system exhibits an *imperfect pitchfork bifurcation* instead of the transcritical bifurcation observed in the earlier Fisher case. The most relevant result is the one obtained by introducing environmental spatial inhomogeneities. The observed effect is better appreciated in the most abstract case, when the spatial modulation is sinusoidal. We have found that there is a bifurcation in the behavior of the system. The nature of this bifurcation is more evident when calculating the mean value of the population densities. We have shown the existence of a critical value of the spatial modulation wave number ω at which the behavior of the systems completely changes, displaying bistable behavior that depends on the initial conditions. Later, by taking a hypothetic general situation, we have shown how this effect operates.

For the sake of completeness, we describe briefly the analysis for the physically (observationally) unimportant but mathematically interesting case when α exactly equals 0.5. The system behaves in a rather complex way. Again, we start with initial conditions of the form $m_s(x) = m_i(x) = A|\cos(2\pi\omega x)|$. The interesting feature is that now both steady states are equally stable. The basin of attraction of each state is such that the amplitude of the initial modulated condition plays a relevant role. There are three different regimes characterized by (1) the system reaching the homogeneous state $m=1$ and $m_i=(\chi-1-\alpha)/\chi$ in the whole spatial domain, (2) the total density m vanishing completely in the whole spatial domain, and (3) a steady state being characterized by spatial periodic patterns with m oscillating periodically between 0 and 1 (see Fig. 4). We also observe a similar kind of oscillation for infected population density between $m_i=0$ and $m_i=(\chi-1-\alpha)/\chi$. The two first cases are like those encountered already in our analysis above for $\alpha > 0.5$, while the third is exclusive to $\alpha=0.5$. As A , the amplitude of the initial condition, decreases the system goes from case 1 to case 2 but with an intermediate regime corresponding to case 3. For $\alpha=0.5$, we note the existence of two critical values of initial conditions at which we observe a transition from oscillating periodic structures to homogeneous structures. The first critical value corresponds to a transition from the oscillating structure to a homogeneous pattern with $m=0$. The second critical value corresponds to transition from the oscillating pattern to the homogeneous one with $m=1$. Thus, in the presence of Allee effects, the population density depends not only on system parameters but also on the *initial distribution*; a dependence that is quite impossible in Fisher equation treatments [11].

Whether rodents of the kind we are describing do or do not exhibit these various Allee effects we have described is a

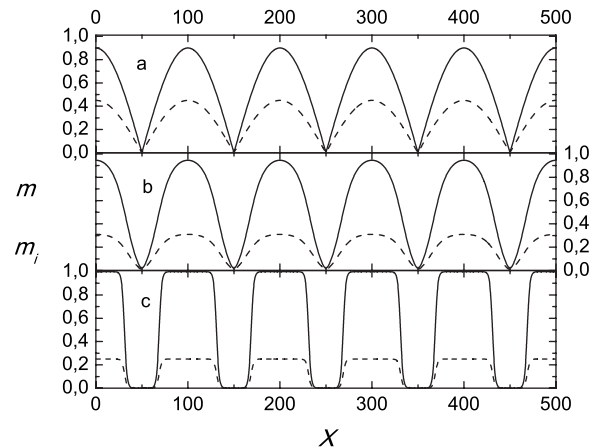


FIG. 4. The evolution of the initial density profile (a) for m (dashed line) and m_i (solid line) toward the steady state (c) for $\alpha=0.5$ and $\alpha=0.45$. Transient patterns are shown in (b). Here x axis is the space and y axis corresponds to density. Units are arbitrary.

question for the field biologist to pursue. We hope that the interesting consequences that we have predicted theoretically in this paper will stimulate observational work in this direction. Work related to the present study (but quite different in spirit as well as detail) may be found in recent papers by Kenkre and Kuperman [18] on bacteria in a Petri dish and more recently in the extensive studies of Clerc *et al.* [19] who reported a number of insights into the Allee effects on pattern formation. Some of the features of the present work that distinguish it from the others is the existence of infected and susceptible subclasses in the population: from the mathematical point of view, the corresponding bifurcations that occur in the system, and from the physical point of view the relevance of the study to the spread of epidemics. Our analysis has been carried out in the present paper in a 1D system only for simplicity. Extensions to a two-dimensional landscape are straightforward. We have not shown them here because they do not introduce any new feature of special interest. The diffusion constant considered is independent of space for the same reasons of simplicity. In other contexts [13,24], Fokker-Planck equations have been used to describe the confined diffusion but such an analysis is beyond the scope of the present paper.

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