INTRODUCING A POPULATION INTO A STEADY COMMUNITY: THE CRITICAL CASE, THE CENTER MANIFOLD, AND THE DIRECTION OF BIFURCATION∗

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Abstract. In this paper we study deterministic, finite dimensional, continuous, as well as discrete time population invasion models. The ability of a newly introduced population, either a new species or a reproductively isolated subpopulation of one of the already present species, to settle in the community relies upon the basic reproduction ratio of the invader, $R_0$. When $R_0$ exceeds 1, the invading population meets with success, and when $R_0$ is below 1, the invasion fails. The aim of this paper is to investigate the possible effects of an invasion when the parameters of a model are varied so that $R_0$ of the invading population passes the value 1. We argue that population invasion models, regardless of the biology that underlies them, take a specific form that significantly simplifies the center manifold analysis. We make a uniform study of ecological, adaptive dynamics and disease transmission models and derive a simple formula for the direction of bifurcation from a steady state in which only the resident populations are present. Furthermore, we observe that among those bifurcation parameters that satisfy a certain condition, we acquire the same direction of bifurcation. The obtained mathematical results are used to gain insight into the biology of invasions. The theory is illustrated by several examples.

Key words. population model, physiologically structured population, i-state, p-state, reproductively isolated, population, species, center manifold, transcritical bifurcation, direction of bifurcation, basic reproduction ratio, finitely many states at birth, next generation matrix, invasibility

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1. Introduction. One of the basic questions of population biology is the following. Suppose that a population is introduced into a steady community in which it has not been present before. Under what conditions will this newly introduced population be able to settle in the community, and when will the dynamics lead to its extinction?

The literature that deals with this question is vast and diverse. We could roughly group the biological settings into the following categories:

1. In ecology one is studying an introduction of (i) a population of predators that forages on a resident prey community (i.e., the so-called predator-prey models; see [7], [11], [20], [26], [27], [30], [31], [32], [34] for some examples), or (ii) a population that competes for resources with the resident community (for examples of competition models, see [7], [20], [26], [31], [33]).

While it is common that the newly introduced population is also a new species, i.e., one that is not present in the resident community, there are also examples in which one is interested in the ability of what we shall call a reproductively isolated subpopulation of one species to be able to settle among individuals of another subpopulation of the same species. We shall define the precise meaning of the term reproductive isolation in Appendix B. The reader may at this point have in mind, for example, studying interactions (say, competition for shared resources) among different year classes of

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semelparous species \cite{2}, \cite{8}, \cite{9}, \cite{10} or different morphs in size-structured populations \cite{3}, \cite{4}.

2. In a branch of the theory of evolution called adaptive dynamics one is investigating the ability of a rare mutant phenotype to invade the environment set by the resident community (see \cite{12}, \cite{21}, \cite{25} and the references therein).

3. Epidemiology of infectious diseases is concerned with introductions of infectious pathogens into susceptible populations (see, for example, \cite{13}, \cite{18}, \cite{19}, \cite{22}, \cite{34}, \cite{35}).

When the resident community is at a stable equilibrium and we describe the process of invasion by a deterministic model we can, regardless of the biological background, answer the invasibility question in terms of the basic reproduction ratio \cite{13}, \cite{15} of the invading population, \( R_0 \), as follows: if \( R_0 < 1 \), the invading population will go extinct, and if \( R_0 > 1 \), it will settle in the community. The transition hence occurs when \( R_0 = 1 \).

Now, what happens when \( R_0 \) passes the value 1? The answer can be formulated mathematically or biologically. In mathematical terms one says that a transcritical bifurcation of a steady state and an exchange of stability take place \cite{5}, \cite{6}, \cite{24}, \cite{36}. From a strictly mathematical point of view there is but one generic type of transcritical bifurcation. But when it comes to seeing the results from a biologist’s point of view one must realize that a steady state is meaningful only when all its components are nonnegative, in particular those corresponding to the invading population. In many models the latter requirement is fulfilled only when \( R_0 > 1 \). The bifurcation is then called supercritical or forward or, also, soft or smooth, since the size of the invading population remains small when \( R_0 - 1 \) is positive but small. In some models, however, the positivity requirement is fulfilled only for \( R_0 < 1 \), and one then speaks of a subcritical or backward bifurcation.

While in the case of a supercritical bifurcation the invasion fails when \( R_0 \) of the invading population falls below 1, the invader can meet with success even if \( R_0 < 1 \) (when introduced in sufficiently large quantities) when the bifurcation is backward. Moreover, even when the invader is introduced in small quantities, a small perturbation of \( R_0 \) to a value greater than 1 can in a subcritical case lead to a rather large invader population size. This phenomenon is sometimes called catastrophic transition (see Figure 2.2).

Clearly then, it is important to be able to tell which of the two cases applies in any given situation, and in this paper we provide the reader with a simple criterion to distinguish between the two scenarios.

Of course, the invasibility question is equally meaningful when the resident community resides in a dynamic attractor. This situation is, however, outside the scope of this paper.

Throughout this paper we consider communities whose members differ in a finite number of characteristics. These characteristics are in the context of population models often called i-states \cite{13}, \cite{15}, with \( i \) standing for individual. Ideally, they should capture precisely the features that are relevant for the description of the process one is studying, and are hence to be considered for each problem separately.

In a general setting we assume that the community is divided into \( m + n \) subpopulations, of which \( m \) subpopulations constitute the invading population and the remaining \( n \) make up the resident community. We denote by

\[
\mathcal{Y} = \{(y_1, \ldots, y_m) : y_j \geq 0 \text{ for } j = 1, \ldots, m\} = \mathbb{R}^m_+
\]
the population state space ($p$-state space) of the invading population (i.e., for each $j \in \{1, \ldots, m\}$ we denote by $y_j$ the number (or density) of individuals in the $j$th subpopulation) and by

$$Z = \{(z_1, \ldots, z_n) ; \ z_j \geq 0 \ for \ j = 1, \ldots, n\} = \mathbb{R}_+^n$$

the community state space of the resident community. The $c$-state space of the joint community will be written as $\mathcal{Y} \times Z$.

Now let $(y(t), z(t))$ denote the community state at time $t$, where time is measured from some conveniently chosen point. The dynamics of $(y(t), z(t))$ in time often depends not only on the present community state, but also on a number of parameters, such as per capita death rates, birth rates, etc., and, quite commonly, population models involve more than one parameter.

The aim of this paper is to study the ability of a newly introduced population to invade the existing community in the case when its basic reproduction ratio is near $1$ and to derive a formula for the direction of bifurcation from a steady state in which the invading population is not present. We shall therefore concentrate on one distinguished parameter which we call the bifurcation parameter.

With this in mind we already at this point include only one (real) parameter $\mu$ and assume that the process we study is either a continuous time process described by a parametrized system of ordinary differential equations,

$$\begin{align*}
\dot{y} &= g(y, z, \mu), \\
\dot{z} &= h(y, z, \mu), \quad y \in \mathcal{Y}, z \in Z, \mu \in \mathbb{R},
\end{align*}$$

(1.1a)

or a discrete time process described by a parametrized map,

$$\begin{align*}
y &\mapsto g(y, z, \mu), \\
z &\mapsto h(y, z, \mu), \quad y \in \mathcal{Y}, z \in Z, \mu \in \mathbb{R}.
\end{align*}$$

(1.1b)

If we consider a steady state of (1.1a) (or (1.1b)) in which the invading population is not present (these steady states lie on the boundary of the $c$-state space) and study the effect of perturbations corresponding to an introduction (in small quantities) of the missing population, we find that such an equilibrium is locally asymptotically stable when $R_0$ of the invading population is below $1$, and unstable when $R_0$ exceeds $1$. Moreover, stability can in these two cases be inferred from the linearization of (1.1) around the steady state.

The Perron–Frobenius theory of nonnegative matrices [1], [29], which applies for problems in population dynamics, leads us to the observation that the critical case, i.e., the case when $R_0 = 1$, corresponds to the situation when

(i) the linearization of (1.1a) around the steady state yields a zero eigenvalue,

(ii) the linearization of (1.1b) around the steady state yields an eigenvalue $1$.

In other words, when $R_0 = 1$ we are dealing with nonhyperbolic steady states, and it is well known [24], [36] that the stability of nonhyperbolic equilibria cannot be determined by linearization alone.

Several papers (e.g., [11], [14], [18], [19], [22], [28], [30], [32], [35]) deal with this situation in the context of population models, most of them (with the exception of [14]) treating special cases or restricting their analysis to models describing the spread of infectious diseases.

In the present paper we study the critical case for general (not restricted to any particular biological background) finite dimensional population models. We will
argue that an introduction of either one new species or a reproductively isolated subpopulation of one of the existing species yields a property of (1.1) that significantly simplifies the center manifold analysis. More precisely, (1.1a) will be shown to be of the form

\[ \dot{y} = G(y, z, \mu) y, \]
\[ \dot{z} = h(y, z, \mu), \quad y \in Y, z \in Z, \mu \in \mathbb{R}. \]

A similar decomposition can be obtained for parametrized maps in (1.1b).

This will lead us to the observation that an introduction of a population whose basic reproduction ratio is close to 1 corresponds to a transcritical bifurcation of a steady state of (1.1) in which only the resident populations are present. In order to obtain the direction of bifurcation from such a steady state only the first derivatives of \( G \) and \( h \) are needed. This reduction of the order of the derivatives needed (in general, second order derivatives are needed) is, of course, most useful when one is dealing with large systems.

We will also see that among those bifurcation parameters for which

\[ \begin{cases} 
\mu < 0 \iff R_0 < 1, \\
\mu = 0 \iff R_0 = 1 
\end{cases} \]

holds on some neighborhood of \( \mu = 0 \) and the crossing of the point \( R_0 = 1 \) occurs at a nonzero “speed,” we obtain the same direction of bifurcation.

Moreover, we will show how \( G \) in (1.2) can be obtained by only considering the basic modeling ingredients, such as birth, growth, and survival rates—an approach that might be of interest to more biologically inclined readers.

The paper is structured as follows. In section 2 we study continuous time population invasion models described by (1.2). Section 3 is devoted to justifying the use of this particular form of models. We argue that this form is characteristic of population invasion models. It appears in all biological scenarios mentioned at the beginning of this Introduction and hence allows us to make a uniform study of ecological, adaptive dynamics and disease transmission models. We also show how \( G \) is obtained from basic modeling ingredients. Population models in discrete time are the theme of section 4. Section 5 provides some interpretation of the assumptions made in previous sections and draws attention to the link between continuous and discrete time population models. In section 6 we give some examples to illustrate the theory of the preceding sections. And lastly, in appendices at the end of the paper we collect some basic definitions and results regarding physiologically structured population models and put the notions of a population, species, and reproductively isolated subpopulation into a more mathematical setting.

2. Population invasion models in continuous time. We begin our study of continuous time population models by recalling the decomposition of the community state space

\[ Y \times Z = \mathbb{R}_+^m \times \mathbb{R}_+^n, \]

where \( Y = \mathbb{R}_+^m \) denotes the population state space of the invading population and \( Z = \mathbb{R}_+^n \) the community state space of the resident community. The processes we study in this section are continuous time processes described by

\[ \dot{y} = G(y, z, \mu) y, \]
\[ \dot{z} = h(y, z, \mu), \quad y \in Y, z \in Z, \mu \in \mathbb{R}, \tag{2.1} \]
where we shall furthermore assume that $G \in M_{m \times m}(C^1(\mathbb{R}^m \times \mathbb{R}^n \times \mathbb{R}, \mathbb{R}))$ and $h \in C^1(\mathbb{R}^m \times \mathbb{R}^n \times \mathbb{R}, \mathbb{R}^n)$.

The form (2.1) is characteristic of continuous time population invasion models and one with which an experienced modeler may already be familiar. Those not familiar with it who feel perplexed at this point are referred to section 3, where we shall, in both mathematical and biological terms, explain why and how this form is obtained.

By writing
\[ x = \begin{bmatrix} y \\ z \end{bmatrix}, \quad f = \begin{bmatrix} G y \\ h \end{bmatrix}, \]

we shall write (2.1) as $\dot{x} = f(x)$ and also use (1.1a) whenever this notation will be more convenient.

Consider now an equilibrium of (2.1) of the form $e = (0, z_0)$ for some $z_0 \in \mathbb{Z}$, i.e., a steady state in which only the resident populations are present. In general (2.1) can have more than one steady state of this form, and these steady states may also depend on $\mu$. We therefore write $e(\mu) = (0, z_0(\mu))$ with $z_0(\mu) \in \mathbb{Z}$.

To study the linearized stability of $e(\mu)$ we write
\[ Df((0, z_0(\mu)), \mu) = \begin{bmatrix} G(e(\mu), \mu) & 0 \\ h_y(e(\mu), \mu) & h_z(e(\mu), \mu) \end{bmatrix}, \]

where
\[ h_y(y, z, \mu) = \frac{\partial h(y, z, \mu)}{\partial y} \quad \text{and} \quad h_z(y, z, \mu) = \frac{\partial h(y, z, \mu)}{\partial z}. \]

Hence,
\[ \sigma(Df(e(\mu), \mu)) = \sigma(G(e(\mu), \mu)) \cup \sigma(h_z(e(\mu), \mu)). \]

The next assumption that we shall make is that the equilibrium $e(\mu) = (0, z_0(\mu))$ is internally asymptotically stable; that is, it is asymptotically stable under perturbations within the invariant subspace $\{0\}^m \times \mathbb{Z}$. In other words, as long as no new population is introduced, the steady state of the resident community, $z_0(\mu)$, is locally asymptotically stable. We shall make the slightly stronger assumption that the stability can be inferred from the linearization. In mathematical terms this means that we assume

$A_1$. If $\lambda \in \sigma(h_z(e(\mu)))$, then $Re(\lambda) < 0$.

The spectrum of $G(e(\mu), \mu)$ thus completely determines the linearized stability of the steady state $e(\mu)$.

For the existence and uniqueness assertions that follow we need only “internal hyperbolicity,” i.e., that $Re(\lambda) \neq 0$ for any $\lambda \in \sigma(h_z(e(\mu), \mu))$. Assumption $A_1$ will allow us to make more detailed stability assertions, which are known as the principle of the exchange of stability [5], [6], [24].

Now we would like to know whether the invading population, after being introduced into the community, is able to settle in that community. As mentioned in the Introduction, the answer is no when the basic reproduction ratio of the newly introduced population is below 1 and yes when $R_0$ of the invading population exceeds 1.

The basic reproduction ratio is, by definition, the spectral radius of the next generation matrix.$^1$ All the modeling ingredients needed to write down the next

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$^1$See Appendix A for more on the next generation matrix and $R_0$. 
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The generation matrix in the context of the model (see section 5) given by (2.1) are contained in \( G \) (remember that \( R_0 \) of the invading population is the one we need), and it is known (see [13], [35] for the proof) that \( R_0 \) of the invading population relates to the spectral bound of \( G(e(\mu), \mu) \) in the following way:

\[
\begin{align*}
\text{s}(G(e(\mu), \mu)) < 0 & \iff R_0 < 1, \\
\text{s}(G(e(\mu), \mu)) = 0 & \iff R_0 = 1,
\end{align*}
\]

where \( s(. ) \) denotes the spectral bound

\[
s(A) = \max\{Re(\lambda) ; \lambda \in \sigma(A)\}.
\]

Since the next generation matrix is a nonnegative matrix we can apply the Perron–Frobenius theory [1] to conclude that \( R_0 \) is an eigenvalue with a corresponding nonnegative eigenvector. The dominant eigenvalue is often called the transversal eigenvalue, and if it exceeds 1 we say that the newly introduced population is able to invade successfully. If \( R_0 \) is below 1, the invasion of the newly introduced population is doomed to fail.

The interesting situation to consider is hence the situation when the parameter \( \mu \) is such that \( s(G(e(\mu), \mu)) = 0 \), the case where linearization around the steady state does not yet answer the question of invasibility.

In many models the computation of the basic reproduction ratio \( R_0 \) and certainly the spectral bound \( s(G(e(\mu), \mu)) \) yields complicated functions of parameters that we may not be able to express explicitly. We therefore choose a bifurcation parameter \( \mu \) with the following properties:

\[
A_2. \quad \begin{cases}
\mu < 0 & \iff s(G(e(\mu), \mu)) < 0 \iff R_0 < 1, \\
\mu = 0 & \iff s(G(e(\mu), \mu)) = 0 \iff R_0 = 1, \\
\mu > 0 & \iff s(G(e(\mu), \mu)) > 0 \iff R_0 > 1.
\end{cases}
\]

The results that follow are based on local information only. It therefore suffices that \( A_2 \) holds on some neighborhood of \( \mu = 0 \).

Assumption \( A_2 \) means that the function \( \mu \mapsto s(G(e(\mu), \mu)) \) crosses the origin. We shall furthermore assume that this crossing occurs at a nonzero speed, i.e.,

\[
A_3. \quad \frac{d}{d\mu} s(G(e(\mu), \mu)) \big|_{\mu=0} > 0.
\]

We now denote by \( e \) an equilibrium that corresponds to \( R_0 = 1 \), i.e., \( e = e(0) \); denote \( e' = e'(0) \); and also shorten the notation by defining

\[
H_y = h_y(e, 0), \quad H_z = h_z(e, 0), \quad G_0 = G(e, 0).
\]

Denoting by \( E^c \) the center subspace of \( G_0 \), we shall furthermore assume the following.

\[
A_4. \quad \dim E^c = 1.
\]

We have already given the interpretation behind the first three assumptions. We shall return to this last assumption in section 5 and explain in more detail which biological requirements are sufficient in order for \( A_4 \) to hold. Let us remark only that, in systems that arise from modeling population dynamics, the matrix \( G_0 \) will be a matrix with nonnegative off-diagonal entries, and hence the Perron–Frobenius theory guarantees that \( A_4 \) is satisfied when \( G_0 \) is irreducible.

Before stating the main result we make the following observation, which will be useful later on.
LEMMA 2.1. Let $\mu \mapsto G(e(\mu), \mu) \in C^1(\mathbb{R}, \mathbb{R}^{m \times m})$, assume $A_2$ and $A_4$, and let $w$ and $v$ denote, respectively, the left and the right eigenvector of $G_0$ corresponding to eigenvalue zero, normalized so that $v \cdot w = 1$. Then

$$\left. \frac{d}{d\mu} s(G(e(\mu), \mu)) \right|_{\mu=0} = w \cdot \left( D_x G(e,0)e' + D_\mu G(e,0) \right)v. \quad (2.5)$$

Proof. According to the implicit function theorem there exists a neighborhood of $\mu = 0$, say $U$, on which a branch of eigenvalues of $G(e(\mu), \mu)$ is defined. That is,

$$G(e(\mu), \mu)v(\mu) = \lambda(\mu)v(\mu) \quad (2.6)$$

for $\mu \in U$, and since $\mu \mapsto G(e(\mu), \mu) \in C^1(\mathbb{R}, \mathbb{R}^{m \times m})$ we have $\mu \mapsto \lambda(\mu) \in C^1(U, \mathbb{R})$. Moreover, $\mu \mapsto v(\mu) \in C^1(U, \mathbb{R}^m)$. Differentiation of (2.6) with respect to $\mu$ yields

$$\left( \frac{\partial G}{\partial e} e'(\mu) + \frac{\partial G}{\partial \mu} \right)v(\mu) + Gv'(\mu) = \lambda'(\mu)v(\mu) + \lambda(\mu)v'(\mu). \quad (2.7)$$

Since zero is also the spectral bound of $G_0$ and since the spectral bound $s(G(e(\mu), \mu))$ is a continuous function of $\mu$ we have that $\lambda(0) = s(G(e(\mu), \mu))$ in some neighborhood of $\mu = 0$. By taking $\mu = 0$ in (2.7) and taking into account that $\lambda(0) = 0$, we obtain

$$\left. \frac{d}{d\mu} s(G(e(\mu), \mu)) \right|_{\mu=0} = w \cdot \left( D_x G(e,0)e' + D_\mu G(e,0) \right)v,$$

which brings us, after premultiplication by $w$ on both sides, to (2.5). \qed

We can now prove the following result.

THEOREM 2.2. Consider a population model described by (2.1), and let $e(\mu) = (0, z_0(\mu))$ be a steady state of (2.1). Assume that $A_1$, $A_2$, $A_3$, and $A_4$ hold. Furthermore, assume that $\mu \mapsto e(\mu) \in C^1(\mathbb{R}, \mathbb{R}^{n+m})$, and denote by $e$ the steady state that corresponds to $R_0 = 1$, i.e., $e = e(0)$ and by $e' = e'(0)$. Let $G_0, H_y$, and $H_z$ be as in (2.4), and let $w$ and $v$ denote, respectively, the left and the right eigenvector of $G_0$ corresponding to eigenvalue zero, normalized so that $v \cdot w = 1$. Let

$$M = \sum_{i,j,k=1,\ldots,m} w_i \left( \frac{\partial G_{ij}(e,0)}{\partial y_k} + \frac{\partial G_{ik}(e,0)}{\partial y_j} \right) v_j v_k - 2 \sum_{i,j=1,\ldots,m} w_i \frac{\partial G_{ij}(e,0)}{\partial z_k} v_j (H_z^{-1} H_y v)_k. \quad (2.8)$$

There exists a $\delta > 0$ such that

(i) if $M < 0$, there is a branch $\mu \mapsto (y(\mu), z(\mu))$, defined for $\mu \in (0, \delta)$, of positive, locally asymptotically stable steady states of (2.1);

(ii) if $M > 0$, there is a branch $\mu \mapsto (y(\mu), z(\mu))$, defined for $\mu \in (-\delta, 0)$, of positive, unstable steady states of (2.1).

In other words, there exists, in a neighborhood of $\mu = 0$, a branch of nontrivial, positive (and hence biologically meaningful) steady states of (2.1), and $M$ tells us about its initial slope. The former case, case (i), is often referred to as a supercritical bifurcation and the latter, case (ii), as a subcritical or backward bifurcation.

At this point the following remarks regarding the terminology are in order.

Remark 1. As already mentioned in the Introduction, the resulting bifurcations are the so-called transcritical bifurcations. They correspond to an intersection of two
Fig. 2.1. Supercritical (on the left) and subcritical (on the right) bifurcation. The branch of nonnegative steady states is denoted by a solid line. A dashed line represents steady states with negative components. Stability of equilibria is indicated by $s$ (for stable) and $u$ (for unstable).

branches of equilibria, the trivial and the nontrivial, at $\mu = 0$, where the branches exchange stability (see Figure 2.1). In contrast with the purely mathematical point of view where these two transitions are qualitatively the same, we need to distinguish between the two in the biological context, since in that case only the nonnegative equilibria are of any relevance.

**Remark 2.** Note that only the first order derivatives of $G$ and $h$ are needed to determine the direction of bifurcation from $e$. Moreover, the expression $M$ for the direction of bifurcation is independent of the bifurcation parameter except for the restrictions $A_2$ and $A_3$. In other words, provided that $A_2$ and $A_3$ are satisfied, we obtain the same direction of bifurcation for any choice of the bifurcation parameter.

The principle of the exchange of stability guarantees that the biologically meaningful, nontrivial bifurcating branch consists of stable equilibria in the supercritical case and of unstable equilibria in the subcritical case. The stable manifold of an unstable equilibrium then serves as a separatrix between the domains of attraction of the "residents only" steady state and some other attractor (frequently the same branch bent forward in a saddle node bifurcation).

Suppose now that an invader is introduced in small quantities into the resident community. In both the supercritical and the subcritical cases, this invasion will fail if the basic reproduction ratio of the invader is below 1.

In the supercritical case, the invader will be successful when its basic reproduction ratio exceeds 1, but its population size will be small when $R_0 - 1$ is small. Because of this smooth transition, one sometimes calls this bifurcation soft or smooth.

In the subcritical case, on the other hand, a small introduction of the invading population for $R_0 - 1$ small but positive leads to a large invader population size. Accordingly one also calls this bifurcation hard or catastrophic. Moreover, the invader can meet with success, despite $R_0 < 1$, if it is introduced in sufficiently large quantities. Catastrophic transition is illustrated in Figure 2.2, where unstable equilibria are denoted by a dashed line, stable by a solid line.

To restate Theorem 2.2 in biological terms we could say the following. When a new population, an invader, is successfully introduced into the community we observe one of the following:

(i) a smooth change to a positive but small invader population size or
(ii) a sudden, catastrophic transition to a rather large invader population size.

When all the assumptions of Theorem 2.2 are met, the sign of $M$ in (2.8) determines which of the two scenarios we will observe in a concrete situation.

We now prove Theorem 2.2.

**Proof.** We have $G_0v = 0$, $w^rG_0 = 0$, and $v \cdot w = 1$. By $A_1$, the matrix $H_z$ is
invertible. The left and the right zero eigenvectors of $Df(e)$, denoted by $W$ and $V$, are then of the form

\begin{equation}
W = \begin{bmatrix} w \\ 0 \end{bmatrix}, \quad V = \begin{bmatrix} v \\ -H^{-1}_y v \end{bmatrix}.
\end{equation}

Moreover, $V \cdot W = 1$. By $A_1$ and $A_4$, the dimension of the center linear subspace equals 1, and the subspace is spanned by $V$.

We take the (generalized) right eigenvectors of $Df(e)$ for the basis of $\mathbb{R}^{m+n}$. It is known that the right (generalized) eigenvectors of $Df(e)$ that correspond to nonzero eigenvalues are orthogonal to $W$.

The center manifold theory [24], [36] states that the center manifold of the equilibrium $e$, denoted by $\mathcal{M}^c(e)$, can be (locally) parametrized by $\mu$ and a real variable $u$ as

\[
\mathcal{M}^c(e) = \{(x, \mu); \; x = e(\mu) + uV + \Phi(u, \mu)\},
\]

where $\Phi(.)$ is defined on some neighborhood of the origin. Moreover, $\Phi(0, 0) = D\Phi(0, 0) = 0$ and $W \cdot \Phi(u, \mu) = 0$ for every $u$ and $\mu$.

The center manifold is also invariant under (2.1); that is,

\[
\dot{x} = \dot{u}V + \dot{\Phi}(u, \mu) = f(x, \mu) = f(e(\mu) + uV + \Phi(u, \mu), \mu).
\]

Since $W \cdot \frac{d}{d\mu}(\Phi(u, \mu)) = 0$ and $V \cdot W = 1$ the inner product with $W$ yields

\[
\dot{u} = W \cdot f(e(\mu) + uV + \Phi(u, \mu), \mu) = w \cdot g(e(\mu) + uV + \Phi(u, \mu), \mu),
\]

where we have used (2.9) in the last equality. Using the Taylor series expansion around $(e, 0)$, we can continue as follows:

\[
\dot{u} = w \cdot g(e, 0) + w \cdot D_{\mu}g(e, 0)(e(\mu) - e + uV + \Phi(u, \mu)) + w \cdot D_{\mu\mu}g(e, 0)\mu + O(3),
\]

where $O(3)$ contains the terms of third and higher order in $u$ and $\mu$.

Now, since $e$ is an equilibrium of (2.1) the first term equals zero. So does the second because $w^T G_0 = 0$ and $D_{\mu}g(e, 0) = 0$. Since $g = Gy$ the third and the fourth terms also equal zero. Hence

\[
\dot{u} = \frac{1}{2} w \cdot D_{xx}g(e, 0)(e(\mu) - e + uV + \Phi(u, \mu))^2
\]

\[
+ w \cdot D_{\mu x}g(e, 0)(e(\mu) - e + uV + \Phi(u, \mu))\mu + O(3).
\]
By writing \( e(\mu) = e + e'(0)\mu + \mathcal{O}(2) \) and taking into account that \( \Phi \) has no constant and linear terms in \( u \) and \( \mu \), we can continue with

\[
\dot{u} = w \cdot \left( \frac{1}{2} D_{xx} g(e,0)e' + D_{xx} g(e,0)e' \right) \mu^2
+ w \cdot \left( D_{xx} g(e,0)e' V + D_{xx} g(e,0)V \right) \mu u
+ \frac{1}{2} w \cdot D_{xx} g(e,0)u^2 V^2 + \mathcal{O}(3)
\]

\[
= w \cdot \left( D_{xx} g(e,0)e' V + D_{xx} g(e,0)V \right) \mu u
+ \frac{1}{2} w \cdot D_{xx} g(e,0)u^2 V^2 + \mathcal{O}(3),
\]

where we have in the last equality taken into account that the first \( m \) components of \( e(\mu) \) equal zero and the fact that \( g = G_y \) implies \( D_{zz} g(e,0) = D_{z\mu} g(e,0) = 0 \).

Moreover, this special form of \( g \) then gives us

\[
\dot{u} = \mu w \cdot \left( D_x g(e,0)e' + D_{x\mu} g(e,0) \right) v
+ \frac{1}{2} w \cdot D_x g(e,0)u^2 V^2 + \mathcal{O}(3),
\]

which, by denoting

\[
N = w \cdot \left( D_x g(e,0)e' + D_{x\mu} g(e,0) \right) v,
\]

using (2.8), (2.9) and the fact that the first \( m \) components of \( e' \) equal zero, becomes

(2.10)

\[
\dot{u} = \mu Nu + \frac{1}{2} Mu^2 + \mathcal{O}(3).
\]

Note that, according to the Lemma 2.1, \( N = \frac{d}{d\mu} \Phi(G(e(\mu),\mu)) \big|_{\mu=0} \), and so by assumption \( A_3, N \neq 0 \).

Now, the center manifold theory also states that the stability of the steady state under the initial system is determined by its stability under the restriction of the system to the center manifold. This restriction is now given in (2.10).

For \( u \) and \( \mu \) close to zero we can neglect the higher order terms that are collected in \( \mathcal{O}(3) \). The nontrivial steady state solutions of (2.10) that are near the origin are then close to the line \( u = -\frac{\mu}{2 \mu}N M^{-1} \), assuming, of course, that \( M \neq 0 \). By assumption, \( M \) is nonzero.

Our assumptions were that the steady state \( e \) is locally stable for \( \mu < 0 \) and unstable when \( \mu > 0 \). This steady state corresponds to \( u = 0 \). The local stability analysis shows that the nontrivial steady states are locally stable when \( \mu > 0 \) and unstable when \( \mu < 0 \). We shall see in the following section that we can choose the eigenvectors \( v \) and \( w \) so that all their components are nonnegative. Hence, the steady states of (2.1) that correspond to nontrivial equilibria of (2.10) can be biologically meaningful only when either \( M < 0 \) and \( \mu > 0 \) or \( M > 0 \) and \( \mu < 0 \).

Of course, when \( M \) is zero, higher order terms of the Taylor expansion need to be taken into account in order to obtain some information about the nontrivial equilibria of (2.10).

Remark 3. One situation in which the expression for the direction of bifurcation can be further simplified is when (2.1) describes the spread of an infectious
disease. Introduction of an infectious agent to the community of hosts results in a redistribution of hosts to new compartments, such as, for example, latent or infectious individuals. A quite common assumption is that the population of hosts has reached an invariant attracting affine set (the reader can find two such examples in section 6), which means that we can eliminate one of the variables. In the case when the population of susceptible hosts is homogeneous (i.e., \( n = 1 \)) we can, by choosing to eliminate the variable corresponding to the susceptible subpopulation \( z \), redefine \( G \) (which is now a function of \( y \) only and will be denoted by \( \hat{G} \)) and arrive at

\[
M = \sum_{i,j,k=1,\ldots,m} w_i \left( \frac{\partial \hat{G}_{ij}(e, 0)}{\partial y_k} + \frac{\partial \hat{G}_{ik}(e, 0)}{\partial y_j} \right) v_j v_k.
\]

**Remark 4.** Another circumstance that allows for simplification of (2.8) is when the newly introduced population is homogeneous, i.e., \( m = 1 \). We can then choose \( v = w = 1 \), and the expression for the direction of bifurcation becomes

\[
\frac{1}{2} M = \frac{\partial G(e, 0)}{\partial y} - \sum_{k=1,\ldots,n} \frac{\partial G(e, 0)}{\partial z_k} (H^{-1} z H)_k.
\]

The reader can find two examples in this spirit in section 6.

3. **On the characteristic form of population invasion models.** The derivation of \( G \). The purpose of this section in twofold. We first fulfill the promise made in section 6 and show that assuming that population invasion models in continuous time have the form (2.1) did not confine our study to a certain subclass of population invasion models. We will see that population invasion models, regardless of the biology that underlies them, indeed have a distinctive form of which the continuous time version is given in (2.1).

Once this part is established we shall provide the reader with a way of obtaining \( G \) by only considering the basic modeling ingredients, such as birth, survival, and reproduction rates.

So let us suppose that the process of invasion is described by the more general system (1.1) with \( g \in C^2(\mathbb{R}^m \times \mathbb{R}^n \times \mathbb{R}, \mathbb{R}^m) \) and \( h \in C^1(\mathbb{R}^m \times \mathbb{R}^n \times \mathbb{R}, \mathbb{R}^n) \).

Now, in ecology and adaptive dynamics we consider invasions of either one new species or a reproductively isolated subpopulation of one of the already present species, and we have by the very definition of reproductive isolation (see Appendix B for this definition) that \( g(0, z, \mu) = 0 \) for every \( z \in \mathcal{Z}, \mu \in \mathbb{R} \) and also \( h(y, 0, \mu) = 0 \) for every \( y \in \mathcal{Y}, \mu \in \mathbb{R} \).

On the other hand, when (1.1a) (or (1.1b)) describes the spread of an infectious disease into a population of susceptible hosts, a slight modification of terminology is needed. Namely, when an infectious agent is introduced into a population of susceptible hosts we indeed introduce another species, the pathogen. However, from that point on we are, on a population level, interested in how this agent spreads among the population of hosts. In this case, therefore, \( \mathcal{Y} \) captures the subpopulations of hosts (i.e., members of the resident community) that carry the agent (i.e., the invading species). Since susceptible individuals don’t have infected offspring we have that \( g(0, z, \mu) = 0 \) for every \( z \in \mathcal{Z}, \mu \in \mathbb{R} \). But since infected individuals (that belong to \( \mathcal{Y} \)) may become susceptible again (i.e., enter \( \mathcal{Z} \)) once they get rid of the infection or they might have susceptible offspring, the subspace of the invading community, \( \mathcal{Y} \times \{0\}^n \), may not be invariant under (1.1).
In any case we can say the following: since individuals in $Z$ don’t have offspring in $Y$ the subspace of the resident community, $\{0\}^m \times Z$, remains invariant under (1.1). In other words,\\n
$$g(0, z, \mu) = 0 \quad \text{for every } z \in Z, \mu \in \mathbb{R}.$$\\n
Hence the following known result, due to Hadamard, can be used.

**Lemma 3.1.** Let $f = (f_1, \ldots, f_k)^T \in C^r(\mathbb{R}^m \times \mathbb{R}^n, \mathbb{R}^k)$ for some $r \in \mathbb{N}$ be such that $f(0, y) = 0$ for every $y \in \mathbb{R}^n$. There exists $F \in C^{r-1}(\mathbb{R}^m \times \mathbb{R}^n, \mathbb{R}^{k \times m})$ such that\\n
$$f(x, y) = F(x, y)x, \quad x = (x_1, \ldots, x_m)^T.$$\\n
The proof of this result can be found in [17].

The property (3.1) therefore yields a matrix $G \in M_{m \times m}(C^1(\mathbb{R}^m \times \mathbb{R}^n \times \mathbb{R}, \mathbb{R}))$ such that $g$ in (1.1) can be written as\\n
$$g(y, z, \mu) = G(y, z, \mu)y, \quad y = (y_1, \ldots, y_m)^T.$$\\n
Note that this decomposition is in general not unique, as the following simple example shows.

**Example 1.** Take $y = (y_1, y_2)$ and $g = y_1y_2$. Then $G = [y_2, 0]$ and $G = [0, y_1]$ are two possible decompositions.

This nonuniqueness will, however, not affect our results—for our purposes, any correct decomposition will do. We shall nevertheless now point out one way, more interpretation motivated (and hence perhaps mainly of use to more biologically inclined readers), of obtaining $G$ in (3.2).

The key to this decomposition is the so-called environmental condition [15], [16].

The defining property of an environmental condition is that individuals are independent of one another (and hence the equations are linear) when this condition is prescribed as a function of time. We then view (1.1) as a linear system together with feedback equations that tell us how, in turn, the environmental condition is influenced by the population size and composition. In general, the environment is set by all subpopulations involved. The environmental condition will hence be a function of $x = (y, z)$. Readers that are not familiar with the notion of an environmental condition and find this general definition a bit unclear are encouraged to take a look at Appendix A, where we explain the notion of an environmental condition by way of a simple example.

In order to arrive directly at the desired decomposition of $g$ in (1.1) we first separate the reproduction in $Y$ from all other processes.

Since individuals in $Z$ don’t have offspring in $Y$ the invading population completely determines the reproduction in $Y$. To describe it we define the following matrix:

$$P_{ij}(x) := \text{the rate with which individuals with birth state } i \text{ are born to an individual with state } j, \text{ given a constant environmental condition } x \in X.$$\\n
What remains is to describe other processes, namely maturation and survival.

We consider the dynamics of an individual’s state after birth as a Markov process on the set of $i$-states, where the probabilities of changing a state are again determined by the environmental condition $x \in Y \times Z$. We define the matrix $Q$ by

$$Q_{ij}(x) = \begin{cases} \text{the rate of leaving state } j \text{ to go to state } i, & i \neq j, \\ -(\text{the rate of leaving state } j), & i = j. \end{cases}$$
given an environmental condition $x$.

Hence, the off-diagonal elements of $Q$ describe the changes of states as long as the individual remains alive and does not move to $Z$, and the diagonal elements denote the rate of leaving the state, either by leaving to another state in $Y$, to $Z$, or by death.

By taking into account all the processes, we can now write the matrix $G$ as

$$G = P + Q.$$  

(3.3)

This decomposition has, apart from offering biological interpretation, another advantage. Since the off-diagonal elements of $G$ in (3.3) are nonnegative we can apply the theory of nonnegative matrices [1] to see that we can indeed choose the (left and right) eigenvector of $G_0$ in (2.4) corresponding to eigenvalue zero to be nonnegative.

If, for example, the off-diagonal elements of $G_0$ are strictly positive, the eigenvectors can be chosen to be strictly positive. In many cases this observation makes it a lot easier to determine the sign of $M$ in (2.8).

Note that one could make a similar “per capita” description for the resident populations. However, for our purposes, this description is irrelevant since we are interested in only the $c$-states of the resident community that are not close to zero. It may help, though, when one wants to find $z_0(\mu)$ (see [14]).

4. Population invasion models in discrete time. Sometimes the nature of the problem, the available data, or some other reason makes it more convenient to formulate a discrete time population model. Since the linearization theorem of Hartman and Grobman (see [24], [36]) and the center manifold theory apply for discrete time dynamical systems generated by maps as well as for flows generated by vector fields we can reformulate the problem and the results so that they hold for population invasion models in discrete time.

In the same way as before we decompose the population state space

$$\mathcal{Y} \times Z = \mathbb{R}_+^m \times \mathbb{R}_+^n$$

so that $\mathcal{Y}$ denotes the population state space of the newly introduced population and $Z$ the community state space of the resident community.

We shall now study processes described by a parametrized map

$$y \mapsto G(y, z, \mu)y,$$

$$z \mapsto h(y, z, \mu), \quad y \in \mathcal{Y}, z \in Z, \mu \in \mathbb{R},$$

(4.1)

where we shall furthermore assume that $G \in M_{m \times m}(C^1(\mathbb{R}_+^m \times \mathbb{R}_+^n \times \mathbb{R}, \mathbb{R}))$ and $h \in C^1(\mathbb{R}_+^m \times \mathbb{R}_+^n \times \mathbb{R}, \mathbb{R})$.

We shall use the notation in (1.1b) or in (2.2) to write the map (4.1) as $x \mapsto f(x)$ whenever this notation will be more convenient.

Suppose that we have a steady state, that is, a fixed point of (4.1) of the form $e(\mu) = (0, z_0(\mu))$ for some $z_0(\mu) \in Z$. The associated linear map is then given by

$$Df(e(\mu)) = \begin{bmatrix} G(e(\mu), \mu) & 0 \\ h_y(e(\mu), \mu) & h_z(e(\mu), \mu) \end{bmatrix}.$$

Hence

$$\sigma(Df(e(\mu), \mu)) = \sigma(G(e(\mu), \mu)) \cup \sigma(h_z(e(\mu), \mu)).$$
We shall again assume that the steady state $e(\mu)$ is internally asymptotically stable, i.e., that it is asymptotically stable under perturbations within the invariant subspace \( \{0\}^m \times \mathcal{Z} \) and that this can be inferred from the linearization. In the discrete time setting this means that we assume

\[ B_1. \text{ If } \lambda \in \sigma(h_z(e(\mu), \mu)), \text{ then } |\lambda| < 1. \]

The spectrum of $G(e(\mu), \mu)$ hence determines the linearized stability of $e(\mu)$. Again, the theory of nonnegative matrices tells us that the spectral radius of $G$ is an eigenvalue. The interesting case to consider is therefore when the parameter $\mu$ is such that $G(e(\mu), \mu)$ has an eigenvalue one, a situation where the linearization alone does not tell us whether the newly introduced population is able to settle in the community.

We now again take for a bifurcation parameter some $\mu$ such that the fixed points of (4.1) of the form $e(\mu) = (0, z_0(\mu))$ are linearly stable for $\mu < 0$ and unstable when $\mu > 0$. Thus,

\[ B_2. \begin{cases} 
\mu < 0 & \iff r(G(e(\mu), \mu)) \iff R_0 < 1, \\
\mu = 0 & \iff r(G(e(\mu), \mu)) \iff R_0 = 1, \\
\mu > 0 & \iff r(G(e(\mu), \mu)) \iff R_0 > 1, 
\end{cases} \]

where $r(.)$ denotes the spectral radius. Let us note that here $R_0$ refers to the basic reproduction ratio in the context of the model, and we refer to [7], [29] for the justification of the equivalence between $r(.)$ and $R_0$.

Since the results that follow rely upon local information only, it suffices that $B_2$ holds in some neighborhood of $\mu = 0$.

Assumption $B_2$ tells us that the function $\mu \mapsto r(G(e(\mu), \mu))$ crosses the point $(\mu, r(.)) = (0, 1)$. We shall again assume that this crossing occurs at nonzero speed, i.e.,

\[ B_3. \quad \frac{d}{d\mu} r(G(e(\mu), \mu)) \bigg|_{\mu=0} > 0. \]

Let $e$ denote the equilibrium that corresponds to $R_0 = 1$, i.e., $e = e(\mu = 0)$, and let $e' = e(0)$. We shall also use the notation introduced in (2.4). Denoting by $E^c$ the center subspace of $G_0$, we shall furthermore assume that

\[ B_4. \quad \text{dim } E^c = 1 \]

and refer the reader to the next section for the biological interpretation of this assumption.

We can now prove the discrete time analogue of Theorem 2.2.

**Theorem 4.1.** Consider a population model described by (4.1), and let $e(\mu) = (0, z_0(\mu))$ be a steady state of (4.1). Assume that $B_1$, $B_2$, $B_3$, and $B_4$ hold. Furthermore assume that $\mu \mapsto e(\mu) \in C^1(\mathbb{R}, \mathbb{R}^{n+m})$, and denote by $e$ the steady state that corresponds to $R_0 = 1$, i.e., $e = e(0)$ and by $e' = e'(0)$. Let $G_0, H_y$, and $H_z$ be as in (2.4), and let $w$ and $v$ denote, respectively, the left and the right eigenvectors of $G_0$ corresponding to eigenvalue one, normalized so that $v \cdot w = 1$. Denote

\[ M = \sum_{i,j,k=1,\ldots,m} w_i \left( \frac{\partial G_{ij}(e,0)}{\partial y_k} + \frac{\partial G_{ik}(e,0)}{\partial y_j} \right) v_j v_k \]

\[ -2 \sum_{i,j=1,\ldots,m, k=1,\ldots,m} w_i \frac{\partial G_{ij}(e,0)}{\partial z_k} v_j ((I - H_z)^{-1} H_y v)_k. \]

(4.2)

There exists a $\delta > 0$ such that
(i) if $M < 0$, there is a branch $\mu \mapsto (y(\mu), z(\mu))$, defined for $\mu \in (0, \delta)$, of positive, locally asymptotically stable steady states of (4.1). In other words, the bifurcation is supercritical.

(ii) if $M > 0$, there is a branch $\mu \mapsto (y(\mu), z(\mu))$, defined for $\mu \in (-\delta, 0)$, of positive, unstable steady states of (4.1). That is, the bifurcation is subcritical.

Remark 5. We again see that only the first order derivatives of $G$ and $h$ are needed to determine the direction of bifurcation from $e$. Moreover, the expression for the direction of bifurcation is independent of the bifurcation parameter except for the restrictions $B_2$ and $B_3$. In other words, provided that all the assumptions of Theorem 4.1 are satisfied, we obtain the same direction of bifurcation for any bifurcation parameter.

Remark 6. For some further remarks on the terminology and on the interpretation of the results of Theorem 4.1 in biological terms we refer the reader to the remarks made after Theorem 2.2.

Proof. We have $G_0v = v, w^T G_0 = w^T$, and $w \cdot v = 1$. By $B_1$ the matrix $I - H_z$ is invertible. We can then calculate the left and the right eigenvectors of $Df(e)$ corresponding to eigenvalue one, denote them by $W$ and $V$, and find that

$$W = \begin{bmatrix} w \\ 0 \end{bmatrix}, \quad V = \begin{bmatrix} v \\ (I - H_z)^{-1} H_y v \end{bmatrix}.$$

Moreover, $W \cdot V = 1$.

By $B_1$ and $B_4$, the dimension of the center linear subspace equals 1, and the subspace is spanned by $V$. We take for the basis of $\mathbb{R}^{n+m}$ (generalized) eigenvectors of $Df(e)$. The eigenvectors of $Df(e)$ that correspond to eigenvalues different from 1 are orthogonal to $W$.

The center manifold theory states that there exists a center manifold of the equilibrium $e$, denoted by $\mathcal{M}^c(e)$, that can be locally parametrized by $\mu$ and a real variable $u$ as

$$\mathcal{M}^c(e) = \{(x, \mu); \ x = e(\mu) + uV + \Phi(u, \mu)\},$$

where $\Phi$ is defined on some neighborhood of the origin.

Moreover, $\Phi(0, 0) = D\Phi(0, 0) = 0$ and $W \cdot \Phi(u, \mu) = 0$ for every $u$ and $\mu$. Since the center manifold is also invariant under (4.1) we have

$$x(k+1) = e(\mu) + u(k+1)V + \Phi(u(k+1), \mu) = f(x(k), \mu) = f(e(\mu) + u(k)V + \Phi(u(k), \mu), \mu).$$

We calculate the inner product with $W$, take into account that $W \cdot e(\cdot) = 0$, $W \cdot V = 1$, and $W \cdot \Phi(\cdot) = 0$, and obtain

$$u(k+1) = w \cdot g(e(\mu) + u(k)V + \Phi(u(k), \mu), \mu).$$

Written differently, the restriction of (4.1) to the center manifold is given by a map

$$u \mapsto w \cdot g(e(\mu) + uV + \Phi(u, \mu), \mu).$$

Using the Taylor series, we can now write

$$u \mapsto w \cdot g(e, 0) + w \cdot D_x g(e, 0)(e(\mu) - e + uV + \Phi(u, \mu)) + w \cdot D_{\mu} g(e, 0)\mu + \frac{1}{2} w \cdot D_{\mu\mu} g(e, 0)\mu^2 + \frac{1}{2} w \cdot D_{xx} g(e, 0)(e(\mu) - e + uV + \Phi(u, \mu))^2 + w \cdot D_{\mu x} g(e, 0)(e(\mu) - e + uV + \Phi(u, \mu))\mu + O(3),$$
where \( \mathcal{O}(3) \) denotes third and higher order terms in \( u \) and \( \mu \).

Now, the first term equals zero since \( e \) is a fixed point of \( f \), and therefore \( g(e, 0) = 0 \). The second term equals \( u \) since \( w^T G_0 = w^T \), \( W \cdot e(\mu) = W \cdot e = 0 \), \( W \cdot V = 1 \), and \( W \cdot \Phi(.) = 0 \). Since \( g = G \dot{y} \), the third and fourth terms are also equal to zero. Furthermore, by writing \( e(\mu) = e(0) + e'(0)\mu + \mathcal{O}(2) \), taking into account that \( \Phi \) has no constant and no linear terms in \( u \) and \( \mu \), and noting that \( W^T = (w, 0)^T \), we are left with

\[
\begin{align*}
    u &\mapsto u + w \cdot \left( \frac{1}{2} D_{xx} g(e, 0) e' + D_{ux} g(e, 0) e' \right) \mu^2 \\
    &\quad + w \cdot \left( D_{xx} g(e, 0) e' V + D_{ux} g(e, 0) V \right) \mu u \\
    &\quad + \frac{1}{2} w \cdot D_{xx} g(e, 0) u^2 V^2 + \mathcal{O}(3),
\end{align*}
\]

which, by writing

\[
N = w \cdot \left( D_x G(e, 0) e' + D_\mu G(e, 0) \right) v,
\]

taking into account that \( g = G \dot{y} \), (4.2), and the fact that the first \( m \) components of \( e \) equal zero, becomes

\[
(4.4) \quad u \mapsto u + \frac{1}{2} M u^2 + \mu Nu + \mathcal{O}(3).
\]

Similar reasoning as in Lemma 2.1 establishes that assumptions B\(_2\) and B\(_4\) lead to

\[
(4.5) \quad \frac{d}{d\mu} r(G(e(\mu), \mu)) \big|_{\mu=0} = w \cdot \left( D_x G(e, 0) e' + D_\mu G(e, 0) \right) v,
\]

and so by (4.5) and assumption B\(_4\), \( N \neq 0 \).

Now, for \( u \) and \( \mu \) close to zero we can neglect the higher order terms that are collected in \( \mathcal{O}(3) \) and look for fixed points of \( u \mapsto u + \frac{1}{2} M u^2 + \mu Nu + \mathcal{O}(3) \). Nonzero fixed points are then near the line given by \( u = -2\mu NM^{-1} \).

Our assumptions were that the steady state \( e \) is locally stable for \( \mu < 0 \) and unstable when \( \mu > 0 \). This steady state corresponds to \( u = 0 \). The local stability analysis yields that the nontrivial steady states are locally stable when \( \mu > 0 \) and unstable when \( \mu < 0 \). As we have seen in section 3, we can choose the eigenvectors \( v \) and \( w \) so that all their components are nonnegative. Hence, the steady states of (4.1) that correspond to nontrivial equilibria of (4.4) can be biologically meaningful only when either \( M < 0 \) and \( \mu > 0 \) or \( M > 0 \) and \( \mu < 0 \). If \( M \) is zero, then higher order terms of the Taylor expansion need to be taken into account in order to obtain some information about the nontrivial equilibria of (4.4).

All the situations mentioned at the end of section 3 that lead to a simplified formula for the direction of bifurcation occur, of course, also in the discrete time setting. Modifying the obtained formulas for \( M \) to apply for discrete time models is a rather straightforward matter, and we therefore leave it to the reader.

5. On the basic reproduction ratio in the context of a model. The case \( R_0 = 1 \). The aim of this section is to offer some interpretation of the assumptions made in previous sections. In order to do this we shall state some known results and refer the interested reader to the literature for their proofs. Two basic notions, one of the next generation matrix and the other of \( R_0 \), are also defined in Appendix A.
The basic reproduction ratio $R_0$ is defined as the expected number of offspring an “average” individual has in all of its life and is mathematically expressed as the spectral radius of the next generation operator (see [13], [15], [35]).

The key to the calculation of $R_0$ of the newly introduced population in the context of the model is to decompose $g$ in a way that separates reproduction in $Y$ from other transitions (such as, for example, new infections from progressions of the disease to another stage), as was already done in section 3.

We then write

$$g(y, z) = (P(y, z) + Q(y, z))y,$$

where $P$ and $Q$ are as in section 3.

Now, if we denote by $e = (0, z_0)$ a steady state of the system and define $P = P(e)$ and $Q = Q(e)$, then $P$ is a nonnegative matrix and $Q$ is a nonsingular $M$-matrix [1], [35]. Hence, $Q$ is invertible and $-Q^{-1}$ is nonnegative. Moreover, the elements of $Q^{-1}$ have the following interpretation: the element $-Q^{-1}_{jk}$ equals the time that an individual that was born with $i$-state $k$ is expected to spend in state $j$ [1], [13], [35]. In other words, the matrix $-Q^{-1}$ describes an individual’s $i$-state dynamics. The matrix $P$ describes the reproduction, and so the matrix $-PQ^{-1}$ is the next generation matrix. By definition, $R_0$ equals its spectral radius.

One can also prove [1], [13], [35] that the following holds:

$$R_0 = r(-PQ^{-1}) < 1 \iff s(P + Q) = s(G_0) < 0,$$

$$R_0 = r(-PQ^{-1}) = 1 \iff s(P + Q) = s(G_0) = 0,$$

where $r$ denotes the spectral radius and $s$ the spectral bound.

It is reasonable to assume irreducibility of the next generation matrix. This guarantees that the spectral radius is an algebraically simple eigenvalue and that we can choose a strictly positive corresponding eigenvector [1]. In biological terms the assumption of irreducibility of the next generation matrix means that the populations are well mixed; that is, for every pair of $i$-states $j$ and $k$, the individuals of the $j$th subpopulation will eventually have offspring in the $k$th subpopulation.

Under a more strict condition, namely the primitivity [1] of the next generation matrix, the modulus of the spectral radius is strictly greater than the modulus of all other eigenvalues of $-PQ^{-1}$. In biological terms the assumption of primitivity means that we require that, from some generation on, individuals with birth state $j$ can have offspring with birth state $k$ for any two conceivable $i$-states at birth, $j$ and $k$.

We have assumed in sections 2 and 4 that the dimension of the center subspace of $G_0$ equals 1. Now, in the discrete time setting the matrix $G_0$ is a nonnegative matrix. Its primitivity therefore guarantees that the assumption $B_4$ is satisfied.

In contrast with the discrete time setting we know that $G_0$ in the continuous time case is a nonnegative off-diagonal matrix. The Perron–Frobenius theory then tells us that already the assumption of an irreducible $G_0$ guarantees that $A_4$ holds; i.e., a zero eigenvalue is an algebraically simple eigenvalue, and all other eigenvalues have strictly negative real parts. In biological terms an irreducible $G_0$ means that for every pair of $i$-states $j$ and $k$ ($j \neq k$) we will eventually observe an inflow of individuals of the $j$th subpopulation to the $k$th subpopulation.

The linearization theorem of Hartman and Grobman and the center manifold theory run for discrete time dynamical systems generated by maps parallel to the one for flows generated by vector fields, which allows us to formulate the results for both
settings. The basic reproduction ratio, $R_0$, provides, due to relation (5.1), a further connection and links the continuous time results directly to corresponding discrete time results.

6. Examples. In this section we present four examples to illustrate the theory presented in previous sections.

In the first example we study a continuous time model describing the dynamics in a community in which the predator selectively forages on a stage structured prey. This example is motivated by the work of de Roos, Persson, and Thieme [11] and hopefully demonstrates how little effort is needed to study the occurrence of subcritical equilibria for a class of models, in this particular case models obtained by varying the preference of the predators.

The second example is a discrete time model describing the life cycle of biennials. This example is inspired by the work of Davydova and coworkers [9], [10] and demonstrates how the theory can also be applied to studying reproductively isolated subpopulations of the same species to see whether one missing year class is, after being introduced, able to settle among the existing year classes.

The last two examples are simple continuous time epidemic models related to the author’s other work, namely, modeling the spread of infectious agents that can reside in several different parts of the host’s body. Though very simple in the first place, they illustrate how the determination of the direction of bifurcation can be further simplified by assuming that the total population size has reached an equilibrium and the fact that the eigenvectors in question can be chosen to be nonnegative (see Remark 3).

After determining the direction of bifurcation from a “residents only” steady state, we shall in all of these examples write some interpretation of the results for the problem at hand. We have, however, already in the remarks after Theorem 2.2 described in biological terms what can in general be said about an invasion, given that we know the direction of bifurcation. We shall therefore not repeat these general facts in the examples and rather refer the reader to section 2.

Example 2. In this first example we study a continuous time model describing interactions in a community that consists of a stage structured prey population and a population of predators that preys exclusively on one of the prey stages.

Suppose that the prey is divided into three stages, juveniles, subadults, and adults, and let their densities be denoted, respectively, by $J$, $S$, and $A$. The density of the predators will be denoted by $P$. We describe the dynamics of the predator population that forages exclusively on the adult stages of prey by the following differential equation:

$$\frac{dP}{dt} = (\phi f(A) - \nu) P. \tag{6.1}$$

Here, $\phi$ indicates the conversion efficiency of prey biomass into newborn predators, $\nu$ denotes the per capita death rate of the predators, and $f(.)$ stands for the predator functional response (for example, Holling type 2 or Holling type 3 response). In what follows, the function $f$ will not be specified; we shall assume only that it is an increasing function of the adult prey density.

We shall take a closer look at a situation in which the regulation of the prey population takes place within the subadult stage. We describe the dynamics of the
prey population by the following system of differential equations:

\[
\begin{align*}
\frac{dJ}{dt} &= \beta A - \rho J - \mu_J J, \\
\frac{dS}{dt} &= \rho J - \pi(S)S - \mu_S(S)S, \\
\frac{dA}{dt} &= \pi(S)S - \mu_A A - f(A)P.
\end{align*}
\] (6.2)

Here, the parameters have the following meaning: \( \beta \) denotes the adult fecundity, \( \rho \) the maturation rate from the juvenile to the subadult stage, and \( \mu_J \) the per capita death rate of the juveniles. Functions \( \pi(S) \) and \( \mu_S(S) \) denote, respectively, the (possibly density dependent) maturation rate of subadults into adults and the per capita death rate of the subadults. The per capita death rate of the adult prey in the absence of predators is denoted by \( \mu_A \).

Regulation of the subadult prey population through maturation and/or mortality can occur if the maturation rate \( \pi(.) \) decreases and/or the mortality rate \( \mu_S(.) \) increases with an increase in the density of subadults. We shall therefore assume that \( \pi(.) \) is a nonincreasing and \( \mu_S(.) \) a nondecreasing function of \( S \), and we exclude the situation in which the derivatives of both vanish in some point, since the population is not regulated at all in that case.

We now first calculate the steady states of (6.2) in the absence of the predators. We obtain (the steady state values are denoted by \( * \))

\[
J^* = \frac{\beta A^*}{\mu_J + \rho} = \frac{\beta \pi(S^*)S^*}{\mu_A(\mu_J + \rho)},
\]

\[
A^* = \frac{\pi(S^*)S^*}{\mu_A}
\]

as the steady state densities of the juvenile and adult prey, and the following equilibrium equation for the (nontrivial) steady state density of the subadult prey:

\[
\frac{\rho \beta \pi(S^*)}{\mu_A(\mu_J + \rho)} = \pi(S^*) + \mu_S(S^*).
\] (6.3)

Now, in our previous notation we would have \( y = P \) and \( (z_1, z_2, z_3) = (J, S, A) \) and so \( G = \phi f(A) - \nu \). Since the predator population is homogeneous, we can take \( w = v = 1 \). Furthermore, we consider the case when the basic reproduction ratio of the predators equals one, i.e.,

\[
\mathcal{R}_0 = \frac{\phi f(A^*)}{\nu} = 1,
\]

and so \( f(A^*) = \frac{\nu}{\phi} \).

Now, since \( G \) is a function of \( A \) only we have

\[
M = -2G'(A^*)(H_y^{-1}H_y)_3,
\] (6.4)

where

\[
H_y = \begin{bmatrix}
0 \\
0 \\
-f(A^*)
\end{bmatrix} = \begin{bmatrix}
0 \\
0 \\
-\frac{\nu}{\phi}
\end{bmatrix}
\]
and

\[
H_z = \begin{bmatrix}
-(\rho + \mu_J) & 0 & \beta \\
\rho & -(\pi(S)S + \mu_S(S)S)'|_{S=S^*} & 0 \\
0 & (\pi(S)S)'|_{S=S^*} & -\mu_A
\end{bmatrix}.
\]

Now, since only the last component of \(H_y\) is nonzero and we need only the third component of \(H_z^{-1}H_y\) it suffices to calculate \((H_z^{-1})_{33}\). We have

\[
(H_z^{-1})_{33} = \frac{1}{\det H_z} (\rho + \mu_J)(\pi(S)S + \mu_S(S)S)'|_{S=S^*},
\]

and we can now rewrite (6.4) as

\[
M = 2\nu(\rho + \mu_J)f'(A^*)(\det H_z)^{-1}(\pi(S)S + \mu_S(S)S)'|_{S=S^*}.
\]

Now, \(\nu\) and \((\rho + \mu_J)\) are positive. According to our assumptions, so is \(f'(A^*)\). Using (6.3), we can furthermore see that

\[
\det H_z = \frac{\beta \rho S^*(\pi'(S^*)\mu_S(S^*) - \pi(S^*)\mu'_S(S^*))}{\pi(S^*) + \mu_S(S^*)},
\]

which is, by our assumptions on \(\pi(\cdot)\) and \(\mu_S(\cdot)\), strictly negative. We have therefore arrived at the fact that

\[
\text{sign } M = -\text{sign } (\pi(S)S + \mu_S(S)S)'|_{S=S^*},
\]

as was also found in [11].

We have assumed that \(\mu_S\) is a nondecreasing function of the subadult density. The function \(\mu_S S\) is therefore an increasing function. In more biological terms we could therefore interpret the condition required for the subcritical bifurcation to occur (i.e., \(M > 0\)) in the following way: an emergent Allee effect (i.e., \(M > 0\)) is to occur in the predator population if and only if an overcompensation in the total maturation rate \(\pi(S)S\) takes place, i.e., for certain values of \(S\), an increase in the subadult density actually decreases the total maturation rate, and that this overcompensation is sufficiently strong.

In [11] the authors also studied the cases when the predator forages exclusively on either the juvenile or the subadult prey and found that the emergent Allee effect can occur (with a suitable overcompensation in the regulation) when the predators forage on one of the nonregulating stages of the prey population and can never occur when they forage on the regulating stage.

Hopefully, this example shows how little effort it would take, with the tools that we have developed in the previous sections, to consider these and also many other situations of interest.

**Example 3.** In this example we consider a community of strict biennials, that is, a community that consists of two age classes, with only the oldest class reproducing. Time will in this case be measured in years. We shall label the two classes by indices 0 and 1, the 0 denoting the subpopulation of individuals that have not reached age one and 1 the subpopulation of one-year-old individuals. If individuals survive till the end of their second year, they reproduce and die.
Survival and reproduction rates are described in terms of an environmental condition \( I \), which will be taken to be the weighed sum of the two populations. More precisely, if \( x_j(t) \) denotes the number (or the density) of \( j \)-year-old individuals \( (j = 0, 1) \) at time \( t \), we take

\[
I(t) = c_0 x_0(t) + c_1 x_1(t).
\]

The weights \( c_0 \) and \( c_1 \) are also called the impacts of the corresponding age classes. Now let us denote by \( F_0(I(t)) \) the probability of surviving the first year and by \( F_1(I(t)) \) the reproduction rate of individuals that survive till the end of their second year. Since increasing the \( I \) means worsening the conditions for both classes, the functions \( F_0 \) and \( F_1 \) are decreasing functions. We shall also assume that they are differentiable at least once.

We can now formulate the following discrete time model:

\[
\begin{align*}
x_0(t + 1) &= F_1(I(t)) x_1(t), \\
x_1(t + 1) &= F_0(I(t)) x_0(t).
\end{align*}
\]

The functions \( F_i \) are also called sensitivities to the environment, and the index specifies how this sensitivity depends on age. Typical examples of sensitivity functions are the so-called

(i) **Ricker family**, where \( F_i(I) = a_i e^{-b_i I} \);

(ii) **Beverton–Holt family**, where \( F_i(I) = a_i (1 + b_i I)^{-1} \).

In order to illustrate the theory on this example we first compute the full life cycle map; that is, we apply the map

\[
\begin{bmatrix}
x_0(t) \\
x_1(t)
\end{bmatrix} \mapsto \begin{bmatrix}
0 & F_1(I(t)) \\
F_0(I(t)) & 0
\end{bmatrix} \begin{bmatrix}
x_0(t) \\
x_1(t)
\end{bmatrix}
\]

twice to obtain the community state after a two year time interval. We have

\[
x_0(t + 2) = F_1(I_1(t)) x_1(t + 1)
\]

\[
= F_1 \left( c_0 F_1(I(t)) x_1(t) + c_1 F_0(I(t)) x_0(t) \right) F_0(I(t)) x_0(t)
\]

\[
= F_1(I_1(t)) F_0(I(t)) x_0(t),
\]

where

\[
I_1(t) := c_0 F_1(I(t)) x_1(t) + c_1 F_0(I(t)) x_0(t)
\]

denotes the environmental condition in the second year.

The full life cycle map is then given by

\[
\begin{align*}
x_0(t + 2) &= F_1(I_1(t)) F_0(I(t)) x_0(t), \\
x_1(t + 2) &= F_0(I_1(t)) F_1(I(t)) x_1(t).
\end{align*}
\]

Now let us assume that only the individuals with label zero are present in every second year and that the population is in a steady state, say \( x_0^* \). This means that

\[
F_1(c_1 F_0(c_0 x_0^*) x_0^*) F_0(c_0 x_0^*) = 1.
\]
Furthermore, the assumption that the basic reproduction ratio of individuals with label 1 equals 1 translates into

\[ F_0(c_1 F_0(c_0 x_0^*) x_0^*) F_1(c_0 x_0^*) = 1. \]

Now, in our previous notation we have

\[ G = F_0(I_1(t)) F_1(I(t)), \]
\[ h = F_1(I_1(t)) F_0(I(t)) x_0(t). \]

Let us now compute the required derivatives for the case where both sensitivity functions belong to the Ricker family. We obtain

\[
\frac{\partial G}{\partial x_1} = F_0(I_1) F_1(I) \left( b_0 b_1 c_0 c_1 F_1(I) x_1 + b_0^2 c_1^2 F_0(I) x_0 - b_0 c_0 F_1(I) - b_1 c_1 \right),
\]
\[
\frac{\partial G}{\partial x_0} = F_0(I_1) F_1(I) \left( b_0 b_1 c_0^2 F_1(I) x_1 + b_0^2 c_0 c_1 F_0(I) x_0 - b_0 c_1 F_0(I) - b_1 c_0 \right),
\]
\[
\frac{\partial h}{\partial x_1} = F_0(I_1) F_1(I) x_0 \left( b_1^2 c_0 c_1 F_1(I) x_1 + b_0 b_1 c_1^2 F_0(I) x_0 - b_1 c_0 F_1(I) - b_1 c_1 \right),
\]
\[
\frac{\partial h}{\partial x_0} = F_0(I_1) F_1(I) \left( x_0 (b_1^2 c_0^2 F_1(I) x_1 + b_0 b_1 c_0 c_1 F_0(I) x_0 - b_1 c_0 F_0(I) - b_0 c_0) + 1 \right).\]

We evaluate these derivatives in \( x_0 = x_0^*, x_1 = 0 \); take (6.6) and (6.7) into account; and denote the results, respectively, by \( \mathcal{G}_1, \mathcal{G}_0, \mathcal{H}_1, \) and \( \mathcal{H}_0 \). We arrive at

\[
\mathcal{G}_1 = b_0^2 c_1^2 F_0(c_0 x_0^*) x_0^* - b_0 c_0 F_1(c_0 x_0^*) - b_1 c_1,
\]
\[
\mathcal{G}_0 = b_0^2 c_0 c_1 F_0(c_0 x_0^*) x_0^* - b_0 c_1 F_0(c_0 x_0^*) - b_1 c_0,
\]
\[
\mathcal{H}_1 = x_0^* \left( b_0 b_1 c_0^2 F_0(c_0 x_0^*) x_0^* - b_1 c_0 F_1(c_0 x_0^*) - b_0 c_1 \right),
\]
\[
\mathcal{H}_0 = x_0^* \left( b_0 b_1 c_0 c_1 F_0(c_0 x_0^*) x_0^* - b_1 c_1 F_0(c_0 x_0^*) - b_0 c_0 \right) + 1.
\]

Now, equalities (6.6) and (6.7) in the Ricker case imply that

\[ b_0 = b_1 \quad \text{or} \quad \left( F_0(c_0 x_0^*) = \frac{c_0}{c_1} \quad \text{and} \quad F_1(c_0 x_0^*) = \frac{c_1}{c_0} \right). \]

Moreover, we can take \( v = w = 1 \) in (4.2). The expression for the direction of bifurcation then translates into

\[ M = 2 \mathcal{G}_1 - 2 \mathcal{G}_0 \mathcal{H}_1 (\mathcal{H}_0 - 1)^{-1}, \]

and one can quickly see, by taking (6.8) into account, that the bifurcation is vertical (i.e., \( M = 0 \), as was also found in [9].

In [9], [10] it was actually shown that the bifurcation is vertical in the stronger sense that a family of period two points exists for exactly the critical parameter combination.

**Example 4.** Consider an infectious disease that spreads in a population of hosts that are susceptible to this infection, and assume that there are two parts of the body (the same two parts for all individuals) that can become infected. We shall assume that one of these two parts, part one, is necessarily the part where an individual’s first infection occurs. Once infected at part one, the infection can spread by endogenous transmission to part two. We shall use the following notation and assumptions:
1. $\beta_1$ denotes the rate with which one individual that is infected at part one infects a susceptible individual, $\beta_{12}$ the rate with which one individual that is infected at both parts infects a susceptible individual.

2. $\alpha$ denotes the rate of endogenous transmission of an individual’s infection from part one to part two.

3. Infected individuals become infectious at the moment of infection.

4. Infected individuals retain their infection(s) until death.

5. The death rate is the same for all individuals and is denoted by $\mu$.

6. The population birth rate is denoted by $\lambda$.

7. All newborns are susceptible.

Now let $S$ denote the number of susceptible individuals, $I_1$ the number of those infected at part one, and $I_{12}$ the number of individuals infected at both parts. If the sizes of all subpopulations are large, we can write the following system of differential equations to describe the dynamics:

\[
\begin{align*}
ds \frac{dt}{dt} &= \lambda - \beta_1 I_1 S - \beta_{12} I_{12} S - \mu S, \\
\frac{dI_1}{dt} &= \beta_1 I_1 S + \beta_{12} I_{12} S - (\alpha + \mu) I_1, \\
\frac{dI_{12}}{dt} &= \alpha I_1 - \mu I_{12}.
\end{align*}
\] (6.9)

Put into our previous notation we have

\[ y = (I_1, I_{12}), \quad z = S, \]

and the disease-free steady state is $e = (0, 0, \frac{\lambda}{\mu})$.

Now we have only one $i$-state at birth in this case—all individuals are born (from an epidemiological point of view) by acquiring the infection at part one. Each individual that is infected at part one is expected to retain (only) this infection for time $1/(\mu + \alpha)$. In this time it is expected to infect $\beta_1 \frac{\lambda}{\mu}$ individuals. With probability $\alpha/(\alpha + \mu)$ an individual is also expected to become infected at part two. It is then expected to remain as such for time $1/\mu$ and in that time infects on average $\beta_{12} \frac{\lambda}{\mu}$ susceptibles.

The basic reproduction ratio (i.e., the expected number of new infections caused by an infected individual that is introduced into a completely susceptible population, in all of its infectious period) hence equals

\[ R_0 = \frac{\lambda \beta_1}{\mu(\mu + \alpha)} + \frac{\lambda \alpha \beta_{12}}{\mu^2(\mu + \alpha)}. \]

The elaboration of the direction of the bifurcation can be further simplified in this case if we assume that the total population size has reached an equilibrium. The size of the whole population is then $\lambda/\mu$, and we can eliminate one of the equations in (6.9). By choosing to eliminate the first and replacing $S$ by $\lambda/\mu - I_1 - I_{12}$ in the other two equations, we see that we don’t need to compute $H_y$ and $H_z$ in (2.8).

To compute the direction of bifurcation we write

\[ G = \begin{bmatrix} \beta_1 S - \alpha - \mu & \beta_{12} S \\ \alpha & -\mu \end{bmatrix} \]

with $S = \frac{\lambda}{\mu} - I_1 - I_{12}$. Then

\[ \frac{\partial G_{2i}}{\partial y_j} = 0 \quad \text{for } i, j = 1, 2 \]
and

\[ \frac{\partial G_{11}(e, 0)}{\partial y_1} = -\beta_1, \quad \frac{\partial G_{11}(e, 0)}{\partial y_2} = -\beta_1, \]
\[ \frac{\partial G_{12}(e, 0)}{\partial y_1} = -\beta_{12}, \quad \frac{\partial G_{12}(e, 0)}{\partial y_2} = -\beta_{12}. \]

Hence

\[ M = w_1 \left( -2\beta_1 v_1^2 - 2(\beta_1 + \beta_{12})v_1 v_2 - 2\beta_{12} v_2^2 \right). \]

Since the off-diagonal elements of \( G_0 \) are strictly positive we can choose \( w \) to be strictly positive. Since \( v \) can always be chosen to be nonnegative we see that \( M \) is negative and the bifurcation is supercritical. In other words, control measures with which we will decrease the value of \( R_0 \) below 1 will allow us to eradicate the disease, while the infection will spread further as long as \( R_0 \) stays above 1.

The attentive reader must have noticed that we have not specified the bifurcation parameter. As explained in section 2, we obtain the same direction of bifurcation for all bifurcation parameters, assuming that the assumptions of Theorem 2.2 are satisfied. That they indeed hold in this case can easily be verified, and we leave the details to the reader.

Example 5. Consider again an infectious agent that spreads in the population of susceptibles, and suppose again that there are two different parts of the body (the same for all individuals) at which a susceptible can become infected. These are the additional assumptions and the notation:

1. Susceptibility and infectivity of an individual have independent influences on the rate of transmission. Susceptibility to infection does not change if one is already infected at the other part of the body. That way we can write the rate with which someone, who is already infected at \( J \subseteq \{1, 2\} \), infects someone at part \( j \) as \( b_j B_J \).
2. Once infected at one of the parts, individuals can obtain another infection only by another cross transmission.
3. Infected individuals become infectious at the moment of infection.
4. Infected individuals retain their infection(s) until death.
5. The death rate is the same for all individuals and is denoted by \( \mu \).
6. The population birth rate is denoted by \( \lambda \).
7. All newborns are susceptible.

Let \( S \) denote the number of susceptibles and \( I_1, I_2, I_{12} \) the number of infected individuals that carry an infection at, respectively, the first, second, or both parts of the body.

If we assume that the sizes of all subpopulations are large, we can describe the dynamics by the following system of differential equations:

\[ \frac{dS}{dt} = \lambda - \left( \mu + (b_1 + b_2)B_1 I_1 + (b_1 + b_2)B_2 I_2 + (b_1 + b_2)B_{12} I_{12} \right) S, \]
\[ \frac{dI_1}{dt} = b_1 S(B_1 I_1 + B_2 I_2 + B_{12} I_{12}) - b_2 I_1(B_1 I_1 + B_2 I_2 + B_{12} I_{12}) - \mu I_1, \]
\[ \frac{dI_2}{dt} = b_2 S(B_1 I_1 + B_2 I_2 + B_{12} I_{12}) - b_1 I_2(B_1 I_1 + B_2 I_2 + B_{12} I_{12}) - \mu I_2, \]
\[ \frac{dI_{12}}{dt} = (b_2 I_1 + b_1 I_2)(B_1 I_1 + B_2 I_2 + B_{12} I_{12}) - \mu I_{12}. \]
Put into our previous notation we have

\[ y = (I_1, I_2, I_{12}), \quad z = S, \]

and the disease-free equilibrium is \( e = (0, 0, 0, \frac{\lambda}{\mu}) \).

There are two \( i \)-states at birth in this case, i.e., becoming first infected at part one and becoming first infected at part two. We label these two birth states with 1 and 2, respectively. The next generation matrix is hence a \( 2 \times 2 \) matrix, which, written for the case when an infected individual is introduced into a virgin environment, takes the form

\[ R = \frac{\lambda}{\mu^2} \begin{bmatrix} b_1 B_1 & b_1 B_2 \\ b_2 B_1 & b_2 B_2 \end{bmatrix}. \]

The basic reproduction ratio \( R_0 \) equals its dominant eigenvalue. Since \( R \) is a matrix of rank one, \( R_0 \) equals its trace,

\[ R_0 = \frac{\lambda}{\mu^2} (b_1 B_1 + b_2 B_2). \]

We shall again assume that the total population has reached an equilibrium and eliminate \( S \) by taking \( S = \frac{\lambda}{\mu} - I_1 - I_2 - I_{12} \).

To compute the direction of bifurcation we take

\[ G = \begin{bmatrix} b_1 B_1 S - b_2 Y - \mu & b_1 B_2 S & b_1 B_{12} S \\ b_2 B_1 S & b_2 B_2 S - b_1 Y - \mu & b_2 B_{12} S \\ b_2 Y & b_1 Y & -\mu \end{bmatrix}, \]

with \( Y = B_1 I_1 + B_2 I_2 + B_{12} I_{12} \) and \( S = \frac{\lambda}{\mu} - I_1 - I_2 - I_{12} \).

As was the case in the previous example, all bifurcation parameters yield the same direction of bifurcation from the disease-free steady state. However, we cannot determine the sign of \( M \) right away. We hence compute the left and the right zero eigenvectors of \( G_0 \) and obtain

\[ w = \begin{bmatrix} B_1, & B_2, & B_{12} \end{bmatrix}^T, \quad v = \begin{bmatrix} b_1, & b_2, & 0 \end{bmatrix}^T. \]

Then \( M = M_1 + M_2 + M_3 \), where

\[
\begin{align*}
M_1 &= B_1 \left( -2(b_2 + b_1) b_1^2 B_1 - 2(b_1 B_2 + b_2 B_2 + b_1 B_1) b_1 b_2 - 2b_1 b_2^2 B_2 \right), \\
M_2 &= B_2 \left( -2(b_2 + b_1) b_2^2 B_2 - 2(b_1 B_1 + b_2 B_1 + b_1 B_1) b_1 b_2 - 2b_2 b_1^2 B_1 \right), \\
M_3 &= B_{12} \left( 2b_1^2 b_2 B_1 + 2(b_1 B_1 + b_2 B_2) b_1 b_2 + 2b_1 b_2^2 B_2 \right).
\end{align*}
\]

In contrast with the previous example, bifurcation from a disease-free steady state may not always be supercritical. However, if we don’t expect any “amplification” of individual’s infectiousness by multiple infected parts, that is, if we assume that

\[ B_{12} \leq B_1 + B_2, \]

we expect a supercritical bifurcation, and indeed a simple computation (which we leave to the reader) shows that in such a case \( M < 0 \) and, as in the previous example, we are able to eradicate the disease by suppressing \( R_0 \) below 1.

**Appendix A. Some general considerations concerning physiologically structured population models.** In this section we review some basic definitions and results that we have used in the main part of the paper.
A.1. **Environmental condition.** We begin with the notion of *environmental condition* [14], [15], [16]. The defining property of the environmental condition (we shall denote it by $I$) is that individuals are independent of one another when $I$ is prescribed as a function of time.

The notion of the environmental condition can perhaps most easily be clarified by way of examples. We write the following ratio dependent predator-prey model and refer the reader to section 6 and [14], [15], [16] for more examples.

*Example 6.* Let us consider the following predator-prey model, the so-called Michaelis–Menten-type model:

\[
\begin{align*}
\dot{x} &= rx \left( 1 - \frac{x}{K} \right) - \frac{cry}{my + x}, \\
\dot{y} &= y \left( \frac{fx}{my + x} - D \right),
\end{align*}
\]

where $x(t)$ and $y(t)$ denote, respectively, the prey and predator densities at time $t$. In the absence of the predators the prey grows with constant intrinsic growth rate $r$ and constant carrying capacity $K$. The constants $D$, $c$, $m$, and $f$ stand for the predators’ per capita death rate, capturing rate, half saturation rate, and conversion rate, respectively.

In this case both the predator and prey densities influence the rates with which these two populations interact. Hence, by setting $I = (I_1, I_2) = (x, y)$, we can rewrite the equations so that all interactions are expressed in terms of the environmental variable $I$,

\[
\begin{align*}
\dot{x} &= r \left( 1 - \frac{I_1}{K} \right) - \frac{cI_2}{mI_2 + I_1} x, \\
\dot{y} &= \left( \frac{fI_1}{mI_2 + I_1} - D \right) y.
\end{align*}
\]

Indeed, one sees that when $I$ is prescribed as a function of time the individuals act independently of one another; i.e., equations are linear.

A.2. **The next generation operator and the basic reproduction ratio $R_0$.** A population model is described by a collection of rules for reproduction, maturation, and survival of individuals in a given community. The traditional way to study a population model is to separate reproduction from all other processes. One of the modeler’s first tasks is then to find the set of all conceivable $i$-states at birth and to construct the next generation operator [13], [15].

When the set of all conceivable $i$-states at birth is finite, the next generation operator is a matrix which we shall denote by $R$ and which is defined as follows:

\[ R_{ij}(I) \text{ : } \text{the expected number of offspring with birth state } i \text{ born to one individual that was born with state } j, \text{ given a constant environmental condition } I. \]

The basic reproduction ratio, $R_0(I)$, is by definition [13], [15] the spectral radius of $R(I)$.

Now, by its very definition, $R(I)$ is a nonnegative matrix. When it is irreducible (see [1] and Appendix B for the definition), its spectral radius, i.e., $R_0(I)$, is a well-defined (dominant) eigenvalue, and the corresponding eigenvector can be chosen to be positive.
The literature where one can find special examples from population biology and
where the next generation matrices are constructed in the context of models is vast. We refer the reader to [13], [34] and the references therein and also to section 6 for some concrete examples.

Appendix B. On the notions of species, population, and a reproduc-
tively isolated subpopulation. In the main part of the paper we have used terms
such as population and reproducitively isolated subpopulation in a vague, intuitive way.

The aim of this section is to describe these notions in mathematical terms (here we are inspired by an unpublished note of Gyllenberg [23]).

First, the following definition.

Definitions. A square matrix $A$ is reducible if there exists a permutation matrix $P$ such that

$$P^{-1}AP = \begin{bmatrix} A_1 & 0 \\ B & A_2 \end{bmatrix}.$$ 

A matrix that is not reducible is irreducible.

Following [23], we shall call a matrix $A$ decomposable if the permutation matrix $P$ can be chosen so that $B = 0$. A matrix that is not decomposable is indecomposable.

Now, if an element of the next generation matrix, say $R_{ij}(I)$, is strictly positive for some environmental condition $I$, then, by definition, individuals with birth state $j$ can have offspring with birth state $i$ in this environment. Or, equivalently, the predecessors of individuals with birth state $i$ may, in the environment $I$, be individuals with birth state $j$.

If $R(I)$ is indecomposable for some environmental condition $I$, then all the $i$-states at birth are in this environment related by either ancestry or descent and hence belong to one species. On the other hand, if the next generation matrix is decomposable for some environmental condition $I$, then the set of $i$-states at birth partitions into (at least) two disjoint sets of birth states that are not reproductively connected in $I$.

We shall speak of reproductive isolation of two sets of $i$-states at birth (and of reproductive isolation of the corresponding subpopulations) when these two sets are reproductively isolated in any conceivable environment. Two sets of $i$-states at birth (and the corresponding subpopulations) that are not reproductively isolated are re-
productively connected.

A population is a collection of subpopulations that are reproductively connected and are at the same time the maximal connected collection in the sense that they are reproductively isolated from every subpopulation that is not included in the collection.

We now make these newly introduced terms more precise and make the following, almost mathematical definition (where “almost” refers to the unspecified “conceiv-
able” below).

Definitions. Consider a finite set $J$ of $i$-states at birth, and let $R(.)$ denote the corresponding next generation matrix. We say the following:

1. The set $J$ of $i$-states at birth (and the corresponding community) is reproducitively connected if there exists a conceivable environmental condition $I$ in which the corresponding next generation matrix $R(I)$ is indecomposable.

2. If the set $J$ of $i$-states at birth is not reproductively connected, it consists of (at least) two reproductively isolated subsets of $i$-states at birth. In other words, if $J$ is not reproductively connected, the matrix $R(I)$ is decomposable for every conceivable environmental condition $I$. (Note, however, that in principle, different environmental conditions may yield a different number of blocks in the next generation matrix.)
3. Let $J_1 \subseteq J$. We say that individuals with $i$-states at birth in $J_1$ form a population if

(i) $J_1$ is reproductively connected and

(ii) if $J_1 \subseteq J_2 \subseteq J$ and $J_2$ is reproductively connected, then $J_1 = J_2$.

Reproductive isolation is certainly a property that underlies the concept of species; i.e., two different species are reproductively isolated. Reproductive isolation alone, however, is not sufficient to deduce that we actually observe different species. Think of, for example, two groups of individuals that belong to the same species but live in areas that are not connected, say on two different continents.

Another display of this phenomenon would be the semelparous species, species that reproduce only once in their lives and die afterwards. Suppose that we observe a community whose individuals live for a fixed length of time, say $l$ years. We could characterize individuals by the year of their birth. Instead of doing so, we split the whole community into year classes according to the year of birth (modulo $l$); for example, if $l = 2$, we divide the community into two year classes, one consisting of individuals that were born in odd numbered years, and the other of individuals born in even numbered years. Different year classes are reproductively isolated subpopulations of the same species that interact (for example, compete for food, etc.), and we can study whether a missing year class is, after being introduced into the community, able to settle among the existing year classes. The reader can find one example in this spirit in section 6.

Consider now a community, consisting of several species perhaps, whose individuals are characterized by finitely many $i$-states. We introduce one new population and assume that the set of conceivable $i$-states of this population is also finite. We find the set of all possible $i$-states at birth and write the next generation matrix of the combined community, which for every conceivable environmental condition $I$ is of the form

$$R(I) = \begin{bmatrix} R(I)_{\text{new}} & 0 \\ 0 & R(I)_{\text{old}} \end{bmatrix}.$$ 

Since the resident community might consist of several populations, the matrix $R(I)_{\text{old}}$ may be decomposed further into indecomposable blocks. However, finding a way to deduce the number of reproductively isolated subpopulations from the next generation matrix and recognizing the set of $i$-states that constitute a population is not our aim here. We therefore refrain from these further decompositions.

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