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STOCHASTIC MODELS FOR COMPETING SPECIES WITH A SHARED PATHOGEN

Linda J. S. Allen

Department of Mathematics and Statistics
Texas Tech University
Lubbock, Texas 79409-1042, USA

Vrushali A. Bokil

Department of Mathematics
Oregon State University
Corvallis, OR 97331-4605, USA

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Abstract. The presence of a pathogen among multiple competing species has important ecological implications. For example, a pathogen may change the competitive outcome, resulting in replacement of a native species by a non-native species. Alternately, if a pathogen becomes established, there may be a drastic reduction in species numbers. Stochastic variability in the birth, death and pathogen transmission processes plays an important role in determining the success of species or pathogen invasion. We investigate these phenomena while studying the dynamics of deterministic and stochastic models for n competing species with a shared pathogen. The deterministic model is a system of ordinary differential equations for n competing species in which a single shared pathogen is transmitted among the n species. There is no immunity from infection, individuals either die or recover and become immediately susceptible, an SIS disease model. Analytical results about pathogen persistence or extinction are summarized for the deterministic model for two and three species and new results about stability of the infection-free state and invasion by one species of a system of $n-1$ species are obtained. New stochastic models are derived in the form of continuous-time Markov chains and stochastic differential equations. Branching process theory is applied to the continuous-time Markov chain model to estimate probabilities for pathogen extinction or species invasion. Finally, numerical simulations are conducted to explore the effect of disease on two-species competition, to illustrate some of the analytical results and to highlight some of the differences in the stochastic and deterministic models.

1. **Introduction.** Interspecific interactions between multiple species and their relation to species coexistence has long been an important issue in ecology [28]. The interactions of hosts with their parasites or pathogens are also important to species

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coexistence [26, 28], and to the emergence of novel infectious diseases [13]. Mathematical models aid in understanding how these different interactions, such as competition, predation and mutualism, affect individual species or the entire system and how a pathogen affects these interactions.

The interaction between competition and disease has received a lot of attention in the context of non-spatial deterministic models of two competing hosts with a shared pathogen spread directly either by density-dependent (mass action) [39] or by frequency-dependent (standard) disease incidence [12, 24, 28, 37]. In particular, an important question in this context is: can the presence of disease reverse the outcome of competition, leading to invasion of native species by exotic, non-native species? Some examples of hosts and pathogens where this behavior has been observed include the red and grey squirrel populations and the pathogen *Parapoxvirus* in the United Kingdom [51], larval amphibians and a pathogenic water mold *Saprolegnia ferax* [33] and annual and perennial grass species in western California and the Barley/Cereal yellow dwarf suite of viruses [11, 44]. These example systems suggest the ability of pathogens and disease to change the outcome of direct competition, or to change the outcome of species interactions by parasite-mediated competition (apparent competition) [18, 22, 30, 46, 50].

The interaction between competition and disease in the stochastic setting has received much less attention. Models based on stochastic differential equations have been formulated for multiple hosts and pathogens but with no assumptions about interspecific interactions (e.g., [5, 41, 42]). There is also a collection of work that has examined competition and coexistence or host-pathogen coexistence in stochastic models such as interacting particle systems (spatial and non-spatial) [15, 16, 17, 35, 36, 45]). The models in [17, 35, 36] consider disease by including parameters such as the degree of specificity of the pathogens and the effect of the pathogens on the fecundity and mortality of their hosts. Competition for space is included in these models through an additional component: a spatial structure that takes the form of a connected graph. The models that we consider in this paper are non-spatial and include parameters which measure the strength of the competition between species.

In this investigation, we formulate new stochastic models based on continuous-time Markov chains (CTMCs) and stochastic differential equations (SDEs) for n competing species with a shared pathogen and consider important questions about species or pathogen invasion and species coexistence. The stochastic models are based on an underlying deterministic system of ordinary differential equations (ODEs) for n competing species with a single shared pathogen. There is no immunity from infection, individuals either die or recover. If they recover, they become immediately susceptible again, referred to as an SIS disease model. Both density-dependent and frequency-dependent incidence rates are considered as well as birth and death rates that have an explicit competitive regulation. Analytical results about pathogen persistence or extinction are summarized for the ODE model for two and three species. New results for the ODE model regarding stability of the infection-free state in terms of the basic reproduction number, and invasion by one species of a system of $n-1$ species are obtained. Branching process theory is applied to the CTMC model to estimate probabilities for successful pathogen or species invasion when the basic reproduction number is greater than unity. Numerical examples are presented, with sample paths from the CTMC and SDE models compared to solutions of the ODE model, to illustrate three distinct cases. In two cases, the probability of an outbreak is computed (successful pathogen invasion) when the

two-species competitive (infection-free) equilibrium becomes unstable after the introduction of disease. In case 1, the disease introduction results in extinction of one of the species and in case 2, a new two-species infected coexistence equilibrium is established with reduced population densities. In case 3, a single stable host-pathogen system is invaded and replaced by a new host-pathogen system.

2. **ODE model.** We consider a general Susceptible (S)-Infected (I) model of n competing species where all species share a single pathogen that is directly transmitted among the n species. The system of ODEs consists of $2n$ equations, n equations for S_i, the population density of healthy and susceptible individuals and n equations for I_i, the population density of infected and infectious individuals, $i = 1, 2, \ldots, n$:

$$\frac{dS_i}{dt} = b_i N_i \left(1 - \sum_{j=1}^{n} a_{ij} \frac{N_j}{\theta_{ij}}\right) - d_i S_i \left(1 + \sum_{j=1}^{n} (1 - a_{ij}) \frac{N_j}{\psi_{ij}}\right) - S_i \sum_{j=1}^{n} \alpha_{ij}(N_j) \frac{I_j}{N_j} + \gamma_i I_i, \tag{1}$$

$$\frac{dI_i}{dt} = S_i \sum_{j=1}^{n} \alpha_{ij}(N_j) \frac{I_j}{N_j} - \gamma_i I_i - d_i I_i \left(1 + \sum_{j=1}^{n} (1 - a_{ij}) \frac{N_j}{\psi_{ij}}\right) - \delta_i I_i, \tag{2}$$

$$\frac{dN_i}{dt} = r_i N_i \left(1 - \sum_{j=1}^{n} \frac{N_j}{K_{ij}}\right) - \delta_i I_i, \tag{3}$$

where $N_i = S_i + I_i$ is the total population density of species i. Initial conditions are non-negative, $S_i(0) \geq 0$, $I_i(0) \geq 0$, $i = 1, \ldots, n$ but not all zero. All parameters are strictly positive with the exception that $0 \leq a_{ij} \leq 1$, $\delta_i \geq 0$ and $\gamma_i \geq 0$. The competitive interactions are of Lotka-Volterra form.

In model (1)-(3), it is assumed that increased densities through intraspecific crowding and interspecific competition have a negative impact on reproduction and survival. In particular, the birth rate can be reduced,

$$b_i N_i \left(1 - \sum_{j=1}^{n} a_{ij} \frac{N_j}{\theta_{ij}}\right), \tag{4}$$

whereas the death rate can be increased,

$$d_i N_i \left(1 + \sum_{j=1}^{n} (1 - a_{ij}) \frac{N_j}{\psi_{ij}}\right), \tag{5}$$

when population densities $N_j, j = 1, \ldots, n$, increase. The parameters $0 \leq a_{ij} \leq 1$ subdivide the density-dependent effects into those affecting births (a_{ij}) and those affecting deaths ($1 - a_{ij}$). The intrinsic per capita growth rate for each species i is defined as $r_i = b_i - d_i > 0$. The parameters $\theta_{ij} = (b_i K_{ij})/r_i$, $\psi_{ij} = (d_i K_{ij})/r_i$ are susceptibilities to crowding, with K_{ii} the carrying capacity of species i (in the absence of competition and pathogen effects) and $K_{ij} = K_{ii}/c_{ij}, i \neq j$, where c_{ij} are the competition coefficients. The quantity $1/K_{ij}$ can be defined as the inhibition strength of species j on species i [49].

The coefficients $\alpha_{ii}(N_i)$ are pathogen transmission rates within species i and $\alpha_{ij}(N_j)$, $i \neq j$ are the transmission rates between different species, i and j. These transmission rates are either constant, $\alpha_{ij}(N_j) = \beta_{ij}$, which results in incidence rates that are frequency-dependent

$$\beta_{ij} S_i \frac{I_j}{N_j},$$

or proportional to the population density, $\alpha_{ij}(N_j) = \beta_{ij} N_j$, resulting in density-dependent incidence rates,

$$\beta_{ij} S_i I_j,$$

[39]. We assume there is no vertical transmission and that the disease does not impact reproduction, although the disease may shorten the life span of those infected, via the parameter δ_i = disease-related mortality rate. The disease does not confer immunity, so that individuals may recover and become infected again, where γ_i is the rate of recovery of infected individuals. For each species i, the model is of SIS type.

It is straightforward to show that solutions are non-negative and bounded. Boundedness follows from the non-negativity and from the fact that

$$\frac{dN_i}{dt} \leq r_i N_i \left(1 - \frac{N_i}{K_{ii}}\right).$$

The birth rate (4) must also be non-negative which requires

$$\left(1 - \sum_{j=1}^{n} a_{ij} \frac{N_j}{\theta_{ij}}\right) \geq 0. \tag{6}$$

We assume that parameters a_{ij}/θ_{ij} are chosen sufficiently small and the initial conditions restricted so that solutions $(N_1, \ldots, N_n)$ lie in a bounded region $\Omega \subset \mathbb{R}^n_+$, where (6) holds.

The inclusion of density-dependent effects in both births and deaths, equations (4) and (5), in the competition model (1)-(3) has been studied in special cases for $n = 2$ or 3 species [10, 12, 20, 24, 37, 49, 51, 53]. In the next section, we summarize some of the known results for model (1)-(3) in these two cases. Then we present some new results for the n-species case.

2.1. **Summary of results for $n = 2, 3$.** Bowers and Turner [12] studied model (1)-(3) with density-independent death rates, $a_{ij} = 1$, and density-dependent incidence. Their emphasis was on understanding how forces of competition and infection combine to determine the long-term equilibrium structure of the system. Their conclusion was that these forces do not add linearly, but there is an additional positive factor which is a combination of the two forces that indicates resistance to invasion. They also distinguish different ways in which infected coexistence is possible. In particular, two hosts that would not coexist stably in the absence of disease, do so through pathogen-mediated host coexistence when there is strong intraspecific infection and/or weak interspecific infection. Manore [37] studied this case in more detail using persistence theory, and showed that the persistence of the system of two species is determined by a few key ecologically relevant parameters including the basic reproduction numbers, parameters that measure the relative magnitudes of interspecific and intraspecific competition, and parameters related to invasion criteria.

Saenz and Hethcote [49] studied two-species models with frequency-dependent incidence for two cases (1) $a_{ij} = 1$, density-independent death rates, and (2) $a_{ij} = 0$, density-independent birth rates but with a reduction in the birth rate of infectives. In case (2), the classic endemic model behavior is seen; a modified reproduction number determines the asymptotic behavior, either the disease dies out if the basic reproduction number $\mathcal{R}_0 < 1$ or approaches a globally attractive endemic equilibrium if $\mathcal{R}_0 > 1$. The four competitive outcomes (species 1 wins; species 2 wins; stable coexistence; winner depends on initial conditions) are possible. However, there is also an additional outcome in which the disease may drive one or both host populations to extinction due to disease-related deaths and/or disease-reduced reproduction. This latter outcome does not occur with density-dependent incidence rates [12, 24, 37]. Case (1) was studied in more detail by Manore [37], who proved existence of a unique endemic equilibrium and showed for the case $\delta_i = 0, i = 1, 2$ that the endemic equilibrium is globally asymptotically stable if $\mathcal{R}_0 > 1$, and the disease-free equilibrium is globally asymptotically stable if $\mathcal{R}_0 < 1$.

Han and Pugliese [24] analyzed model (1)-(3) with density-dependent incidence in the case $0 \leq a_{ii} \leq 1, a_{ij} = 0, i \neq j$. Their model has explicit competition in the death rate and self-regulation in the birth rate. Applying persistence theory the authors came to three conclusions: (1) a species i that becomes extinct in the absence of disease, may weakly persist in the presence of disease; that is, there exists $\epsilon > 0$ such that

$$\limsup_{t\to\infty} N_i(t) > \epsilon,$$

(2) a species that would coexist with its' competitor without infection, may be driven to extinction with infection, and (3) an infection that would die out may strongly persist in both species if cross-species transmission is present,

$$\liminf_{t\to\infty} \min\{I_1(t), I_2(t)\} > \epsilon.$$

Hopf bifurcation theory and numerical investigations revealed complicated model behavior. In particular, periodic solutions may exist, dependent on the form of the disease incidence rate. Periodic solutions were also shown by Greenman and Hudson [20] and van den Driessche and Zeeman [53] for some special cases with two hosts and density-dependent incidence rates. With frequency-dependent incidence rates in the models of Saenz and Hethcote [49], no periodic solutions were observed.

Finally, Bokil and Leung [10] considered a simplified three-species model with density-dependent incidence and density-independent deaths, $a_{ij} = 1$. The species have the same birth and death rates, except that interspecific competition is asymmetric and the intraspecific and interspecific disease transmission rates are the same (as in the red, grey squirrel system considered in [51]). There are no disease-related deaths or recovery. For this simplified model, equilibrium analysis is tractable, and it is shown that the presence of disease does not reverse the competitive outcome. A similar result was shown in [37] for a two-species model with either density-dependent or frequency-dependent incidence.

Other types of models with competition and a shared pathogen for two species have been considered, including SIRS models, models with disease affecting only one species and models when there is no explicit competition among the species (see e.g., [6, 9, 23, 29, 40, 42, 49, 53, 54]). We do not consider these types of models. In our models, disease impacts all species and there is explicit competition between species.

2.2. **Analytical results,** $n \geq 2$**.** We consider two cases: pathogen invasion or species invasion. In the first case, we derive conditions for a pathogen to invade a persistent healthy competitive system. In the second case, we derive conditions for a new species to invade a system with a pathogen already established.

In the first case, formulas are derived for the basic reproduction number via the next generation matrix approach [52]. We assume there exists a stable positive equilibrium, $\bar{N}_j$ =positive constant, $j = 1, \ldots, n$ for the n-species competition model. That is, $\bar{N}_j$ is a solution to

$$\sum_{j=1}^{n} \frac{\bar{N}_j}{K_{ij}} = 1; \ i = 1, 2, \ldots, n. \tag{7}$$

For two competing species, the positive stable equilibrium is well-known for the Lotka-Volterra system. For the general case of three or more competing species, conditions for existence and stability of equilibria are much more complex (e.g., [38, 56]). We will assume that a unique stable equilibrium exists in the interior of $\mathbb{R}^n_+$. It is straightforward to show for the disease-free equilibrium (DFE) $\bar{N}_j$, $j = 1, \ldots, n$, that the next generation matrix has the following form [52]:

$$K = \begin{bmatrix} \dfrac{\hat{\alpha}_{11}}{\mathcal{D}_1} & \dfrac{\hat{\alpha}_{12}}{\mathcal{D}_2} & \cdots & \dfrac{\hat{\alpha}_{1n}}{\mathcal{D}_n} \\ \vdots & \vdots & \cdots & \vdots \\ \dfrac{\hat{\alpha}_{n1}}{\mathcal{D}_1} & \dfrac{\hat{\alpha}_{n2}}{\mathcal{D}_2} & \cdots & \dfrac{\hat{\alpha}_{nn}}{\mathcal{D}_n} \end{bmatrix}, \tag{8}$$

where $\hat{\alpha}_{ij} = \bar{N}_i \alpha_{ij}(\bar{N}_j)/\bar{N}_j$, $i, j = 1, \ldots, n$ and

$$\mathcal{D}_i = \gamma_i + \delta_i + d_i \left(1 + \sum_{j=1}^{n} (1 - a_{ij}) \frac{\bar{N}_j}{\psi_{ij}} \right), \ i = 1, \ldots, n. \tag{9}$$

Similar expressions for matrix K have been obtained by others for n-species models [14, 37, 42]. The basic reproduction number $\mathcal{R}_0$ is the spectral radius of K, $\mathcal{R}_0 = \rho(K)$. If $\mathcal{R}_0 < 1$, then the pathogen cannot invade; the DFE is locally asymptotically stable.

Suppose there are $n-1$ species that persist in the presence of a shared pathogen, that is, the following stable equilibrium exists, $(\bar{S}_1, \bar{I}_1, \ldots, \bar{S}_{n-1}, \bar{I}_{n-1})$, with $\bar{N}_j = \bar{S}_j + \bar{I}_j, 1 \leq j \leq n-1$. We consider whether it is possible for the nth species (S_n, I_n) to invade this system for the case of density-dependent incidence, $\alpha_{ij}(N_j) = \beta_{ij} N_j$. Calculating the Jacobian matrix of system (1)-(3), evaluated at the equilibrium $(\bar{S}_1, \bar{I}_1, \ldots, \bar{S}_{n-1}, \bar{I}_{n-1}, 0, 0)$, leads to a matrix with the following form:

$$J = \begin{bmatrix} J_{11} & J_{12} \\ \mathbf{0} & J_{22} \end{bmatrix}.$$

where the $2(n-1) \times 2(n-1)$ matrix J_{11} is stable, the $2 \times 2(n-1)$ matrix $\mathbf{0}$ is a matrix of zeros, and the 2×2 matrix J_{22} is equal to

$$\begin{bmatrix} r_n \left(1 - \displaystyle\sum_{j=1}^{n-1} \frac{\bar{N}_j}{K_{nj}} \right) - \sum_{j=1}^{n-1} \beta_{nj} \bar{I}_j & b_n \left(1 - \displaystyle\sum_{j=1}^{n-1} \frac{a_{nj} \bar{N}_j}{\theta_{nj}} \right) + \gamma_n \\ \displaystyle\sum_{j=1}^{n-1} \beta_{nj} \bar{I}_j & -d_n \left(1 + \displaystyle\sum_{j=1}^{n-1} \frac{(1 - a_{nj}) \bar{N}_j}{\psi_{nj}} \right) - \delta_n - \gamma_n \end{bmatrix}. \tag{10}$$

TABLE 1. Five types of events and the infinitesimal probabilities $\mathcal{P}_j\Delta t + o(\Delta t)$ for species $i = 1, 2, \ldots, n$.

Event j	Change $\Delta\mathbf{X}$	Type	Probability $\mathcal{P}_j\Delta t$
$5i-4$	$\mathbf{e}_{2i-1}$	Birth	$b_iN_i\left(1-\sum_{\ell=1}^{n}a_{i\ell}\frac{N_\ell}{\theta_{i\ell}}\right)\Delta t$ $= B_i\Delta t$
$5i-3$	$-\mathbf{e}_{2i-1}$	Susceptible Death	$d_iS_i\left(1+\sum_{\ell=1}^{n}(1-a_{i\ell})\frac{N_\ell}{\psi_{i\ell}}\right)\Delta t$ $= D_i\Delta t$
$5i-2$	$-\mathbf{e}_{2i}$	Infected Death	$d_iI_i\left(1+\sum_{\ell=1}^{n}(1-a_{i\ell})\frac{N_\ell}{\psi_{i\ell}}\right)\Delta t$ $+\delta_iI_i\Delta t = \mu_i\Delta t$
$5i-1$	$-\mathbf{e}_{2i-1}+\mathbf{e}_{2i}$	Infection	$S_i\sum_{\ell=1}^{n}\alpha_{i\ell}(N_\ell)\frac{I_\ell}{N_\ell}\Delta t = T_i\Delta t$
$5i$	$\mathbf{e}_{2i-1}-\mathbf{e}_{2i}$	Recovery	$\gamma_iI_i\Delta t$

Species n is able to invade if either the Trace$(J_{22}) > 0$ or $\det(J_{22}) < 0$.

Model (1)-(3) serves as the deterministic skeleton in formulating analogous stochastic models that account for the variability in births, deaths, transmission and recovery. We derive a CTMC model, where time is continuous but the random variables for the states are discrete and an SDE model, where time is continuous and the random variables are also continuous.

3. **Continuous-Time Markov chain model.** We consider the random vector $\mathbf{X}(t) = (S_1(t), I_1(t), S_2(t), I_2(t), \ldots, S_n(t), I_n(t))^{\text{tr}}$, which takes values in a set $\mathcal{E} \subset \mathbb{N}_0^{2n}$, with $\mathbb{N}_0$ the set of non-negative integers. The superscript notation "tr" means transpose. We assume that in a small time interval each of the states $S_i(t)$ or $I_i(t)$ can change by -1, 0 or 1. There are a total of $2n$ states and $5n$ changes or events (n for births, n for deaths of susceptible individuals, n for deaths of infected individuals, n for infection and n for recovery). Define the infinitesimal transition probability for the jth event as

$$p_{\mathbf{x},\mathbf{y}}(\Delta t) = \text{Prob}(\mathbf{X}(t+\Delta t) = \mathbf{y} | \mathbf{X}(t) = \mathbf{x}) = \mathcal{P}_j\Delta t + o(\Delta t).$$

Order the $5n$ events according to species, with each set of five events for the n species representing birth, death of a susceptible individual, death of an infected individual, infection and recovery (Table 1). Suppose that the system is in the state $\mathbf{X}(t) = \mathbf{x}$ at time t, $\mathbf{x} \in \mathcal{E}$. Define $\mathbf{e}_i$ to be the ith unit column vector in the space $\mathbb{N}_0^{2n}$. Let

$$\Delta\mathbf{X} = \mathbf{X}(t+\Delta t) - \mathbf{X}(t) = (\Delta S_1, \Delta I_1, \Delta S_2, \Delta I_2, \ldots, \Delta S_n, \Delta I_n)^{\text{tr}}, \qquad (11)$$

be the change in the states in a small time interval Δt. The changes in $\Delta\mathbf{X}$ and their associated probabilities are listed in Table 1 (see e.g., [4, 32]). Because of the Markov assumption, the interevent time is exponentially distributed with a parameter that is equal to $\sum_{j=1}^{5n}\mathcal{P}_j$.

Techniques from branching processes are used to define probability generating functions (pgfs) for the offspring of this continuous-time process. Below, we present heuristic arguments to compute the probability of an outbreak and the probability of species invasion. The "offspring" can be new infections or new species trying to invade. We consider two cases similar to the two cases in the analytical results for

the ODE model discussed in Section 2.2. In the first case, we assume there is a competitive system of healthy individuals at a stable equilibrium and a pathogen is introduced resulting in new infections. In the second case, we assume there is an equilibrium consisting of $n-1$ species with a shared pathogen and a new species is introduced.

Continuous-time Galton-Watson branching process theory applied to the one-species SIS CTMC process provides a good approximation to the probability of an outbreak when the initial number of infectives is small and the population density is large. The approximation for the probability of an outbreak if $\mathcal{R}_0 = \hat{\alpha}_{11}/\mathcal{D}_1 > 1$ is

$$1 - (1/\mathcal{R}_0)^{i_1},$$

where $I_1(0) = i_1$ [7, 55]. The reason the branching process approximation works well for $\mathcal{R}_0 > 1$ is that sample paths of the branching process either hit zero with probability $(1/\mathcal{R}_0)^{i_1}$ or increase exponentially with probability $1-(1/\mathcal{R}_0)^{i_1}$. In the limit of the SIS CTMC model there is almost sure extinction of the entire population. However, prior to absorption at zero, the population reaches a quasistationary distribution, a distribution conditioned on nonextinction, whose mean is close to the endemic equilibrium of the deterministic model. For example, for practical purposes, an outbreak level O_L and time t_{end} can be defined for the stochastic model, $1 \ll O_L \leq \bar{I}$ ($\bar{I}$ the endemic level of infection), so that we can say "an outbreak has occurred" for a given sample path if there exists a time $t \leq t_{\text{end}}$ such that $I(t) > O_L$. If $\mathcal{R}_0 < 1$, then all sample paths of the branching process eventually hit zero with probability one. These results provide information about the stochastic process that cannot be obtained from the deterministic model. That is, if a small number of infected individuals are introduced, then the outbreak may not occur, even if $\mathcal{R}_0 > 1$. We apply this theory to the more complex CTMC model based on Table 1 to make predictions about the probability of an outbreak or the probability of species invasion.

3.1. **Probability of an outbreak.** Assume there is a stable equilibrium for the competitive system $S_j = \bar{N}_j$, given by (7). All variables $S_j = \bar{N}_j$ are approximately constant and we consider whether a pathogen can invade. We apply continuous-time, multitype Galton-Watson branching process theory to approximate the continuous-time process of the random variables $I_1, \ldots, I_n$ when the number of infected individuals is small and the population densities $\bar{N}_j$ are large [25, 31, 32, 43, 48]. It is important to note that this is only an approximation to ultimate pathogen extinction since an assumption in the Galton-Watson theory is that the offspring random variables are independent, a reasonable assumption for a small initial number of infected individuals. In addition, the number of infectives in the deterministic model must grow to a sufficiently large density near the unstable DFE to ensure that the exponential growth of the branching process is a good approximation. Given $I_1(0) = i_1, \ldots, I_n(0) = i_n$, the Galton-Watson branching process has a limiting probability of extinction [25, 31, 32, 43, 48],

$$P_0 = \lim_{t\to\infty} \text{Prob}\{(I_1(t), \ldots, I_n(t)) = \mathbf{0}\}.$$

The value $1 - P_0$ is a good approximation for the probability of an outbreak, assuming that the number of infected individuals introduced is small and the number of infected individuals in the deterministic model reach a sufficiently large density. On a practical level, we say "an outbreak has occurred" for a given sample path in

the stochastic model if for some $t \le t_{\text{end}}$, t_{end} large, and predefined outbreak level $O_L \gg 1$, $\sum_j I_j(t) > O_L$. The term "Galton-Watson" is more frequently applied to the discrete-time process since the original application considered by Galton and Watson was for extinction of family names in discrete generations (sometimes referred to as Bienaymé-Galton-Watson process). The extinction theory for discrete random variables in continuous time also applies to this process [25, 32, 43, 48].

Let $p_i(s_1, s_2, \dots, s_n)$ denote the probability an infected individual of type i has s_1 offspring of type 1, s_2 offspring of type 2, etc. The term offspring is used in a general sense in that offspring of an infected individual of type i (species i) are either susceptible individuals of the same type i or another type $j \neq i$ that become infected from type i. Let $\{Y_{ij}\}_{j=1}^n$ denote the collection of random variables for the offspring of type j from type i, where we assume that the offspring random variables are independent with the same pgf for each individual of type i. Then

$$p_i(s_1, s_2, \dots, s_n) = \text{Prob}\{Y_{i1} = s_1, \dots, Y_{in} = s_n\}.$$

We construct offspring pgfs for each infected state I_i given that $I_i(0) = 1$ and $I_j(0) = 0$ for $j \neq i$, defined as

$$f_i(u_1, u_2, \dots, u_n) = \sum_{s_n=0}^{\infty} \cdots \sum_{s_1=0}^{\infty} p_i(s_1, s_2 \dots, s_n) u_1^{s_1} u_2^{s_2} \cdots u_n^{s_n},$$

$i = 1, \dots, n$, where $\mathbf{u} = (u_1, \dots, u_n) \in [0,1]^n$. In addition, we assume that each f_i is not simple, where the term simple means f_i is linear in $u_1, u_1, \dots, u_n$, with no constant term [48]. The expectation matrix for the number of offspring of type j produced by an i type individual is given by a matrix M_1 of size $n \times n$ whose (i,j) element is

$$\left.\frac{\partial f_i}{\partial u_j}\right|_{\mathbf{u}^*=\mathbf{1}},$$

where $\mathbf{u}^* = \mathbf{1} = (1, 1, \dots, 1)$. It follows from the theory of multitype Galton-Watson branching processes that if the f_i are not simple, M_1 is irreducible and $\rho(M_1) < 1$, then the probability of extinction is one, $P_0 = 1$ [25, 32, 43, 48]. In this case, the pathogen does not become established and the process is known as subcritical. Alternately, if $\rho(M_1) > 1$, then there exists a unique fixed point $\mathbf{q} = (q_1, q_2, \dots, q_n) \in (0,1)^n$ of $\mathbf{f}$ $[f_i(\mathbf{q}) = q_i,\ i = 1, \dots, n]$ such that

$$P_0 = q_1^{i_1} q_2^{i_2} \cdots q_n^{i_n}, \tag{12}$$

with $I_k(0) = i_k, k = 1, 2, \dots, n$ [25, 32, 43, 48]. The irreducibility of M_1 ensures that $0 < q_i < 1$ when $\rho(M_1) > 1$. In this latter case, the process is referred to as supercritical. Although the value of P_0 is the limiting value, this limit is often approached rapidly in the subcritical or supercritcal cases.

Now we write the offspring pgf for I_i. Let the susceptible population be constant, $S_i = \bar{N}_i$. Then the offspring pgf for a type i infected individual, given $I_i(0) = 1$ and $I_j(0) = 0$, $j \neq i$, is

$$f_i(u_1, u_2, \dots, u_n) = \frac{\hat{\alpha}_{ii} u_i^2 + \mathcal{D}_i + \sum_{j \neq i}^{n} \hat{\alpha}_{ji} u_i u_j}{\mathcal{D}_i + \sum_{j=1}^{n} \hat{\alpha}_{ji}}, \tag{13}$$

where $\mathcal{D}_i$ is defined in (9). The first term in (13) represents new infections of type i, $\hat{\alpha}_{ii}u_i^2/(\mathcal{D}_i + \sum \hat{\alpha}_{ji})$. This term is multiplied by u_i^2 because the original type i individual remains infective and also causes one new infection of type i. The second term represents the probability the infective individual of type i dies without causing any new infections $\mathcal{D}_i/(\mathcal{D}_i + \sum \hat{\alpha}_{ji})$. The last term represents all interspecies infections, species i infects species j, $\sum_{j\neq i} \hat{\alpha}_{ji}u_iu_j/(\mathcal{D}_i + \sum \hat{\alpha}_{ji})$.

It is easy to see that each f_i in (13) is not simple. There always exists at least one fixed point, $\mathbf{u}^* = \mathbf{1}$. The $n \times n$ expectation matrix is

$$M_1 = \begin{bmatrix} \dfrac{\hat{\alpha}_{11} + \sum_j \hat{\alpha}_{j1}}{\mathcal{D}_1 + \sum_j \hat{\alpha}_{j1}} & \dfrac{\hat{\alpha}_{21}}{\mathcal{D}_1 + \sum_j \hat{\alpha}_{j1}} & \cdots & \dfrac{\hat{\alpha}_{n1}}{\mathcal{D}_1 + \sum_j \hat{\alpha}_{j1}} \\ \vdots & \vdots & \cdots & \vdots \\ \dfrac{\hat{\alpha}_{1n}}{\mathcal{D}_n + \sum_j \hat{\alpha}_{jn}} & \dfrac{\hat{\alpha}_{2n}}{\mathcal{D}_n + \sum_j \hat{\alpha}_{jn}} & \cdots & \dfrac{\hat{\alpha}_{nn} + \sum_j \hat{\alpha}_{jn}}{\mathcal{D}_n + \sum_j \hat{\alpha}_{jn}} \end{bmatrix}. \tag{14}$$

Performing an element-wise comparison of M_1 with an $n \times n$ diagonal matrix $\mathbb{D}$ given as $\mathbb{D} = \text{diag}\left\{ \dfrac{\hat{\alpha}_{ii} + \sum_j \hat{\alpha}_{ji}}{\mathcal{D}_i + \sum_j \hat{\alpha}_{ji}} \right\}$, implies $\rho(\mathbb{D}) \leq \rho(M_1)$ [47]. Thus, a necessary condition for $\rho(M_1) \leq 1$ is that all diagonal elements be less than or equal to unity. Matrix M_1 is irreducible if $\hat{\alpha}_{ij} > 0$ for $i \neq j$.

For the special case of two species, it can be shown that $\mathcal{R}_0 > 1$ ($\mathcal{R}_0 \leq 1$) if and only if $\rho(M_1) > 1$ ($\rho(M_1) \leq 1$). (See the Appendix.) If $\mathcal{R}_0 > 1$ and interspecies transmission $\hat{\alpha}_{ij} > 0$, then there exists a unique fixed point of the pgfs $f_j(q_1, q_2) = q_j$, where $0 < q_j < 1$, $j = 1, 2$. The probability of an outbreak given $I_j(0) = i_j$, $j = 1, 2$, is approximately

$$1 - P_0 = 1 - q_1^{i_1} q_2^{i_2}. \tag{15}$$

Verification of the probability of an outbreak will be done through stochastic numerical simulations of the CTMC model in Section 5 and compared to the estimate $1 - P_0$ in the case of two species.

3.2. **Probability of species invasion.** We apply a similar technique in defining the pgfs as demonstrated in the previous section, except that we define the offspring pgfs for the nth species that is trying to invade a stable community consisting of $n-1$ species. Assume there is a stable equilibrium given by $(\bar{S}_1, \bar{I}_1, \ldots, \bar{S}_{n-1}, \bar{I}_{n-1})$ with density-dependent incidence so that the variables $S_j = \bar{S}_j$, $I_j = \bar{I}_j$, $j = 1, \ldots, n-1$ are approximately constant. We have $\bar{N}_j = \bar{S}_j + \bar{I}_j, 1 \leq j \leq n-1$. Next, we consider the invading species, the random variables S_n and I_n. Applying a similar technique as in the preceding section, we write offspring pgfs for S_n and I_n. Again we use the term offspring in a general sense. Assume $S_n(0) = 1$, $I_n(0) = 0$ and

$$1 - \sum_{j=1}^{n-1} a_{nj} \frac{\bar{N}_j}{\theta_{nj}} > 0. \tag{16}$$

The approximate pgf for the offspring of S_n is

$$g_1(u_1,u_2)=\frac{b_n\left(1-\sum_{j=1}^{n-1}a_{nj}\frac{\bar{N}_j}{\theta_{nj}}\right)u_1^2+d_n\left(1+\sum_{j=1}^{n-1}(1-a_{nj})\frac{\bar{N}_j}{\psi_{nj}}\right)+\sum_{j=1}^{n-1}\beta_{nj}\bar{I}_j u_2}{b_n\left(1-\sum_{j=1}^{n-1}a_{nj}\frac{\bar{N}_j}{\theta_{nj}}\right)+d_n\left(1+\sum_{j=1}^{n-1}(1-a_{nj})\frac{\bar{N}_j}{\psi_{nj}}\right)+\sum_{j=1}^{n-1}\beta_{nj}\bar{I}_j}$$
$$=\frac{A_1u_1^2+A_2+A_3u_2}{A_1+A_2+A_3}, \tag{17}$$

where the $A_i>0$, $i=1,2,3$ are the constant coefficients. The three terms $A_1u_1^2/(A_1+A_2+A_3)$, $A_2/(A_1+A_2+A_3)$ and $A_3u_2/(A_1+A_2+A_3)$ represent birth of a susceptible individual of species n, death of a susceptible individual of species n without becoming infective and a susceptible individual of species n becoming infective through contact with an infective of species $1,\ldots,n-1$. Since $S_n(0)$ is small, the intraspecific density-dependent effects are set to zero.

Assume $S_n(0)=0$, $I_n(0)=1$ and condition (16) holds. The approximate pgf for the offspring of I_n is

$$g_2(u_1,u_2)=\frac{b_n\left(1-\sum_{j=1}^{n-1}a_{nj}\frac{\bar{N}_j}{\theta_{nj}}\right)u_1u_2+d_n\left(1+\sum_{j=1}^{n-1}(1-a_{nj})\frac{\bar{N}_j}{\psi_{nj}}\right)+\delta_n+\gamma_nu_1}{b_n\left(1-\sum_{j=1}^{n-1}a_{nj}\frac{\bar{N}_j}{\theta_{nj}}\right)+d_n\left(1+\sum_{j=1}^{n-1}(1-a_{nj})\frac{\bar{N}_j}{\psi_{nj}}\right)+\delta_n+\gamma_n}$$
$$=\frac{A_1u_1u_2+A_2+\delta_n+\gamma_nu_1}{A_1+A_2+\delta_n+\gamma_n}. \tag{18}$$

The three terms $A_1u_1u_2/(A_1+A_2+\delta_n+\gamma_n)$, $(A_2+\delta_n)/(A_1+A_2+\delta_n+\gamma_n)$ and $\gamma_nu_1/(A_1+A_2+\delta_n+\gamma_n)$ represent birth of a susceptible individual from an infected individual of species n, death of an infected individual of species n without causing any new infections and recovery of an infected individual of species n.

The expectation matrix, calculated from the offspring pgfs, is given by

$$M_2=\begin{bmatrix}\frac{2A_1}{A_1+A_2+A_3} & \frac{A_3}{A_1+A_2+A_3}\\ \frac{A_1+\gamma_n}{A_1+A_2+\delta_n+\gamma_n} & \frac{A_1}{A_1+A_2+\delta_n+\gamma_n}\end{bmatrix}. \tag{19}$$

Since $A_i>0$, $i=1,2,3$, the offspring pgfs are not simple and matrix M_2 is irreducible. If $\rho(M_2)<1$, then species n cannot invade, that is, the probability of extinction for species n equals one. But if $\rho(M_2)>1$, then there is a unique fixed point, $g_i(q_1,q_2)=q_i$, $0<q_i<1$, $i=1,2$, such that the probability species n can invade is approximately $1-P_0$, where $P_0=q_1^{s_n}q_2^{i_n}$ with $S_n(0)=s_n$ and $I_n(0)=i_n$. In the numerical examples, we compute $\rho(M_2)$ and check the stability of the matrix J_{22} from the deterministic formulation, equation (10). In Section 5, we define a sufficiently large time t_{end} and species invasion level $S_{inv}\gg 1$ and say "a species invasion has occurred" for a given sample path if there exists $t\le t_{\text{end}}$ such that

$S_n(t)+I_n(t) > S_{inv}$. The probability of species invasion is calculated as the proportion of sample paths satisfying this criteria. Numerical simulations of the CTMC model will be compared with the estimate $1 - P_0$.

4. **Stochastic differential equation model.** To derive an SDE model corresponding to model (1)-(3), we calculate the expected change and the covariance matrix for the change $\Delta\mathbf{X}$, applying the method discussed in [1, 2, 4]. The SDE model corresponds closely to the CTMC model when the values of the random variables are sufficiently large [1, 2, 5, 41, 42]. We first compute the SDE model for the case of two interacting species and then do the computation for the general n-species case.

4.1. **SDE for two interacting species.** Using the changes and probabilities from Table 1, the expected value to order Δt is

$$\begin{aligned}\mathbb{E}(\Delta\mathbf{X}) \approx \Delta t \{&\mathbf{e}_1(B_1 - D_1 - T_1 + \gamma_1 I_1) + \mathbf{e}_2(-\mu_1 + T_1 - \gamma_1 I_1) \\ &+\mathbf{e}_3(B_2 - D_2 - T_2 + \gamma_2 I_2) + \mathbf{e}_4(-\mu_2 + T_2 - \gamma_2 I_2)\},\end{aligned}$$

which is a 4×1 vector, that is, Δt times the right-hand side of the ODE system (1)-(2) for the variables $\mathbf{X} = (S_1, I_1, S_2, I_2)^{\text{tr}}$. The covariance matrix for the change $\Delta\mathbf{X}$ can be approximated to first order as

$$\mathbb{E}(\Delta\mathbf{X}(\Delta\mathbf{X})^{\text{tr}}) \approx \Delta t \begin{bmatrix} \mathcal{A}_1 & \mathbf{0} \\ \mathbf{0} & \mathcal{A}_2 \end{bmatrix},$$

where $\mathbf{0}$ is a 2×2 matrix of zeros, and the 2×2 block matrices $\mathcal{A}_i, i = 1, 2$ are given as

$$\mathcal{A}_i = \begin{bmatrix} B_i + D_i + \gamma_i I_i + T_i & -(\gamma_i I_i + T_i) \\ -(\gamma_i I_i + T_i) & \mu_i + \gamma_i I_i + T_i \end{bmatrix}. \tag{20}$$

Define $\mathbf{f}(t, \mathbf{X}) = \mathbb{E}(\Delta\mathbf{X})/\Delta t$. Since, Δt will be chosen to be small, and since $\mathbb{E}(\Delta\mathbf{X}(\Delta\mathbf{X})^{\text{tr}}) = o(\Delta t^2)$, we will set the covariance matrix

$$\Sigma(t, \mathbf{X}) = \mathbb{E}(\Delta\mathbf{X}(\Delta\mathbf{X})^{\text{tr}})/\Delta t.$$

Because of the difficulty of computing square roots of $2n \times 2n$ matrices for $n > 2$, instead of computing the 4×4 matrix $\Sigma^{1/2}$ in the associated SDE

$$d\mathbf{X} = \mathbf{f}dt + \Sigma^{1/2} d\mathbf{W}(t),$$

where $\mathbf{W}$ is a vector containing 4 independent Wiener processes, we will use an equivalent approach in which we construct a 4×8 matrix G such that $\Sigma = G(G)^{\text{tr}}$ [1, 2, 4]. In this case the corresponding SDE is

$$d\mathbf{X} = \mathbf{f}dt + Gd\mathbf{W}^*(t),$$

in which $\mathbf{W}^*$ is a vector containing 8 independent Wiener processes. The 4×8 matrix G is equal to

$$\begin{bmatrix} \sqrt{B_1 + D_1} & 0 & -\sqrt{T_1} & 0 & \sqrt{\gamma_1 I_1} & 0 & 0 & 0 \\ 0 & 0 & \sqrt{T_1} & 0 & -\sqrt{\gamma_1 I_1} & 0 & -\sqrt{\mu_1} & 0 \\ 0 & \sqrt{B_1 + D_1} & 0 & -\sqrt{T_2} & 0 & \sqrt{\gamma_2 I_2} & 0 & 0 \\ 0 & 0 & 0 & \sqrt{T_2} & 0 & -\sqrt{\gamma_2 I_2} & 0 & -\sqrt{\mu_2} \end{bmatrix}.$$

4.2. **SDE for n interacting species.** In this section, we extend the computation of the SDE to n interacting species. From Table 1, the expected value is approximately

$$\mathbb{E}(\Delta\mathbf{X}) \approx \sum_{i=1}^{n} \left\{ \mathbf{e}_{2i-1} \left(B_i - D_i - T_i + \gamma_i I_i \right) + \mathbf{e}_{2i} \left(-\mu_i + T_i - \gamma_i I_i \right) \right\} \Delta t.$$

The covariance matrix for the change $\Delta\mathbf{X}$ to first order is given by

$$\mathbb{E}(\Delta\mathbf{X}(\Delta\mathbf{X})^{\mathrm{tr}}) \approx \Delta t \left\{ \sum_{i=1}^{n} (B_i + D_i + \gamma_i I_i + T_i) \mathbf{e}_{2i-1} (\mathbf{e}_{2i-1})^{\mathrm{tr}} \right.$$
$$\left. + (\mu_i + \gamma_i I_i + T_i) \mathbf{e}_{2i} (\mathbf{e}_{2i})^{\mathrm{tr}} - (\gamma_i I_i + T_i) \left(\mathbf{e}_{2i} (\mathbf{e}_{2i-1})^{\mathrm{tr}} + \mathbf{e}_{2i-1} (\mathbf{e}_{2i})^{\mathrm{tr}} \right) \right\}.$$

The covariance matrix is Δt times a $n \times n$ block diagonal matrix in which the ith diagonal block representing events related to species i is $\mathcal{A}_i$ as defined in (20). Using the definition of $\mathcal{A}_i$, the covariance matrix can be compactly written as

$$\mathbb{E}(\Delta\mathbf{X}(\Delta\mathbf{X})^{\mathrm{tr}}) \approx \Delta t \sum_{j=1}^{n} \mathcal{I}_j \mathcal{A}_j \mathcal{I}_j^{\mathrm{tr}},$$

where the $n \times 2$ matrix $\mathcal{I}_j = (\mathbf{e}_{2i-1}, \mathbf{e}_{2i})$.

Following a similar process as in the case of two interacting species, we do not compute the $2n \times 2n$ square root matrix $\Sigma^{1/2}$ in the associated SDE

$$d\mathbf{X} = \mathbf{f} dt + \Sigma^{1/2} d\mathbf{W}(t),$$

where $\mathbf{W}$ is a vector containing $2n$ independent Wiener processes. Instead we apply an equivalent approach in which we construct a $2n \times 4n$ matrix G such that $\Sigma = G(G)^{\mathrm{tr}}$, and the corresponding SDE is

$$d\mathbf{X} = \mathbf{f} dt + G d\mathbf{W}^*(t)$$

(see e.g., [1, 2, 4]). In the SDE, $\mathbf{W}^*$ is a vector of $4n$ independent Wiener processes. The matrix G can be defined in terms of its columns, where the kth column of G, G_k, $k = 1, 2, \ldots, 4n$, is given as

$$G_k = \begin{cases} \sqrt{(B_k + D_k)} \mathbf{e}_{2k-1}, & k = 1, 2, \ldots, n \\ -\sqrt{T_{k-n}} \mathbf{e}_{2(k-n)-1} + \sqrt{T_{k-n}} \mathbf{e}_{2(k-n)}, & k = n+1, \ldots, 2n \\ \sqrt{\gamma_{k-2n} I_{k-2n}} \mathbf{e}_{2(k-2n)-1} - \sqrt{\gamma_{k-2n} I_{k-2n}} \mathbf{e}_{2(k-2n)}, & k = 2n+1, \ldots, 3n \\ -\sqrt{\mu_{k-3n}} \mathbf{e}_{2(k-3n)}, & k = 3n+1, \ldots, 4n. \end{cases}$$

5. **Numerical simulations.** Three numerical examples of the ODE, SDE and CTMC systems are presented for the case of $n = 2$ species. The SDE models are simulated using the Euler-Maruyama method [27, 34], and the CTMC models are simulated using the Stochastic Simulation Algorithm by Gillespie [19, 27]. The three cases illustrate pathogen and species invasion, where the presence of a pathogen causes a change in a stable two-species competitive equilibrium or a second species invades a stable one-species host-pathogen system. In cases 1 and 2, the probability of an outbreak computed by applying the theory of branching processes in Section 3.1 is verified against simulations from the CTMC model. In the third case, the probability of a species invasion computed by applying the theory of branching processes in Section 3.2 is verified against simulations from the CTMC model. Parameter values for the three cases are presented in Table 2 and

the existence and local stability results for the ODE model are summarized in Table 3. The values of the basic reproduction numbers are computed for each species (S_i, I_i), $\mathcal{R}_{0i}$, and for the system (S_1, I_1, S_2, I_2), $\mathcal{R}_0$, and are summarized in Table 4 for the three cases. In all three cases, the trivial equilibrium for the ODE model $(S_1, I_1, S_2, I_2) = (0, 0, 0, 0)$ is unstable.

TABLE 2. Parameter Values for Cases 1, 2 and 3

Parameter	Interpretation	Case 1	Case 2	Case 3
Density Dependence				
a_{ii}, $i = 1, 2$	Self regulation	0.5	1.0	1.0
a_{ij}, $i \neq j$	Cross-species regulation	0.5	1.0	1.0
Species 1				
b_1	Intrinsic birth rate	0.6	0.6	1.0
d_1	Natural mortality	0.4	0.2	0.4
K_{11}	Carrying capacity	8000	8000	6000
c_{12}	Competition coefficient	1.2	0.6	0.61
β_{11}	Intraspecies virus transmission rate	0.005	1	0.007
β_{12}	Interspecies virus transmission rate	0.001	1	0.007
δ_1	Disease related mortality	0.05	0.2	5
γ_1	Recovery rate	4.0	0	0
Species 2				
b_2	Intrinsic birth rate	0.5	0.8	1.2
d_2	Natural mortality	0.3	0.4	0.4
K_{22}	Carrying capacity	6000	8000	8000
c_{21}	Competition coefficient	0.3	0.5	1.65
β_{22}	Intraspecies virus transmission rate	0.001	1	0.007
β_{21}	Interspecies virus transmission rate	0.001	1	0.007
δ_2	Disease related mortality	0.5	0.2	0
γ_2	Recovery rate	0	0	13

TABLE 3. Equilibria in the form (S_1, I_1, S_2, I_2) and their local stability for the ODE model (1)-(3) with parameters given in Table 2, U=unstable, S=stable.

Case 1		Case 2		Case 3	
Equilibria	S/U	Equilibria	S/U	Equilibria	S/U
$(8000, 0, 0, 0)$	U	$(8000, 0, 0, 0)$	U	$(6000, 0, 0, 0)$	U
$(0, 0, 6000, 0)$	U	$(0, 0, 8000, 0)$	U	$(0, 0, 8000, 0)$	U
$(906, 5382, 0, 0)$	S	$(2240, 3360, 0, 0)$	U	$(771, 88, 0, 0)$	U
$(0, 0, 820, 385)$	U	$(0, 0, 3840, 2560)$	U	$(0, 0, 1914, 6086)$	S
$(1250, 0, 5625, 0)$	U	$(4571, 0, 5714, 0)$	U		
		$(527, 1982, 1108, 2778)$	S		

TABLE 4. The basic reproduction number $\mathcal{R}_0$ and species reproduction numbers $\mathcal{R}_{01}$, and $\mathcal{R}_{02}$ for the three cases.

Case	$\mathcal{R}_0$	$\mathcal{R}_{01}$	$\mathcal{R}_{02}$
1	6.58	8.79	6.67
2	4.17	2.50	1.67
3	-	7.78	4.18

5.1. **Case 1: Probability of an outbreak: Density-dependent incidence.** For case 1, consider the ODE model (1)-(3) with density-dependent incidence and birth and death rates density-dependent. There is a stable coexistence equilibrium $(S_1, S_2) = (1250, 5625)$ in the pure competition model without disease. However, the corresponding equilibrium $(S_1, I_1, S_2, I_2) = (1250, 0, 5625, 0)$ is unstable in the two-species model (Table 3). The only stable equilibrium in model (1)-(3) is the single-species one host-pathogen equilibrium $(S_1, I_1, S_2, I_2) = (906, 5382, 0, 0)$. The presence of the pathogen results in replacement of the two-species equilibrium by a single species. Saenz and Hethcote considered model (1)-(3) in [49]; however, they provided analysis only for the cases of density-independent births or deaths. Thus, to our knowledge, this case has not been analyzed in the literature.

For the CTMC model, we apply branching process theory to compute the probability of pathogen invasion (outbreak). At the stable coexistence equilibrium, the offspring pgf $\mathbf{f}$ defined in equation (13), and the expectation matrix M_1 defined in (14), are easily computed using the parameter values in Table 2. The spectral radius of M_1, $\rho(M_1) \approx 1.7 > 1$, which implies there exists a unique fixed point $(q_1, q_2) \in (0,1) \times (0,1)$ of $\mathbf{f}$. The fixed point is computed numerically to be $q_1 = 0.336$, $q_2 = 0.137$. The probability of an outbreak $(1 - P_0)$, defined in (15) is then verified by computing the proportion of 1000 sample paths of the CTMC model in which there is an outbreak. That is, the CTMC model is simulated 1000 times over a given time period, $[0, t_{\text{end}}]$ ($t_{\text{end}} = 3$) with the initial conditions $S_1(0) = 1250, S_2(0) = 5625$ and a small number of infected individuals. In each run of the model, the condition $I_1(t_{\text{end}}) > O_L = 100$ is checked. For example, in the ODE model $I_1(t_{\text{end}} = 3) \approx 751$ when $I_1(0) = 0$ and $I_2(0) = 1$. The probability of an outbreak is computed as the fraction of simulations (out of 1000) in which the number of infected individuals of species 1 was above O_L at t_{end}. Longer time intervals gave similar results. The simulation results presented in Table 5 show good agreement with the predicted value of the probability of an outbreak.

Figure 1 illustrates three sample paths of the SDE model with the solution of the ODE model when $S_1(0) = 1250, S_2(0) = 5625, I_1(0) = 0, I_2(0) = 1$, i.e., we start at the DFE and introduce an infected individual of species two. All three sample paths of the SDE model follow the ODE solution (this was the case for all the simulations we performed for the SDE model). This behavior differs from the sample paths of the CTMC model in Figure 2. The sample paths of the simulated CTMC model were chosen to illustrate some cases of pathogen extinction ($P_0 = 0.131$). The sample paths from the CTMC and SDE models that follow the ODE solution illustrate the variability due to births, deaths, transmission and recovery.

5.2. **Case 2: Probability of an outbreak: Frequency-dependent incidence.** For the second case, consider the ODE model (1)-(3) with frequency-dependent incidence. The parameter values are given in Table 2. The natural mortality rates

TABLE 5. Case 1: Probability of an outbreak $(1 - P_0)$ computed from the theory of branching processes, and based on 1000 sample paths of the CTMC model for various initial values of I_1 and I_2.

$I_1(0)$	$I_2(0)$	$1 - P_0$	CTMC
1	0	0.658	0.657
0	1	0.869	0.870
1	1	0.969	0.952
2	0	0.892	0.892
0	2	0.979	0.980

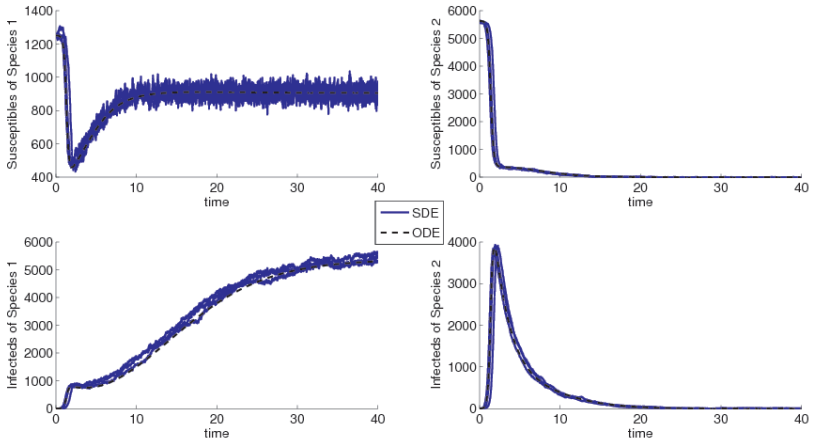

FIGURE 1. Comparison of ODE and SDE (3 sample paths) solutions for Case 1. We start the simulation at the DFE and add one infected individual of species 2, $S_1(0) = 1250, S_2(0) = 5625, I_1(0) = 0, I_2(0) = 1$.

(d_1, d_2) are density-independent, all the competitive effects are incorporated in the birth terms, and there is disease-related mortality, $\delta_i = 0.2$, $i = 1, 2$. There is a stable coexistence equilibrium $(S_1, S_2) = (4571, 5714)$ in the pure competition model with no disease. However, the corresponding equilibrium $(S_1, I_1, S_2, I_2) = (4571, 0, 5714, 0)$ is unstable in the two-species model, while the infected coexistence equilibrium $(S_1, I_1, S_2, I_2) = (527, 1981, 1108, 2778)$ is locally stable. In the ODE model, the presence of the pathogen results in a new stable infected coexistence equilibrium with reduced population densities.

The ODE model with frequency-dependent transmission and $a_{ij} = 1$, $\delta_i = 0 = \gamma_i$ was analyzed by Manore [37] which differs from case 2 (in case 2, $\delta_i = 0.2$, Table 2). Manore showed for $\delta_i = 0$ and $\mathcal{R}_0 > 1$ that the infected coexistence equilibrium is globally asymptotically stable. However, we expect that the infected coexistence equilibrium remains globally stable in this case too.

For the CTMC model, the offspring pgfs $\mathbf{f}$, defined in (13), and the expectation matrix M_1, defined in (14), are computed using the parameter values in Table 2.

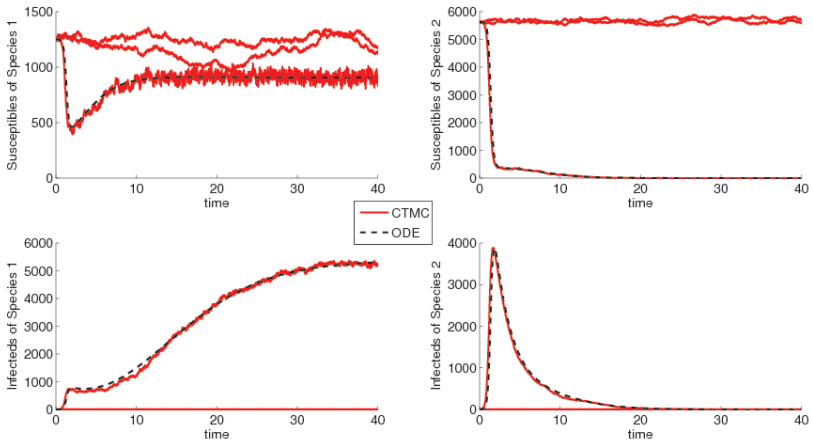

FIGURE 2. Comparison of ODE and CTMC (3 sample paths) solutions for Case 1. We start the simulation at the DFE and add one infected individual of species 2, $S_1(0) = 1250, S_2(0) = 5625, I_1(0) = 0, I_2(0) = 1$ ($P_0 = 0.131$).

The spectral radius of M_1 is $\rho(M_1) \approx 1.59 > 1$ so that a unique fixed point exists for $\mathbf{f}$ in $(0,1) \times (0,1)$. The fixed point is computed numerically to be $q_1 = 0.193$, $q_2 = 0.310$. The probability of an outbreak $(1 - P_0)$, defined in (15), is tested by computing the proportion of sample paths out of 1000 in which an outbreak occurs, see Table 6. One thousand sample paths were simulated until $t_{\text{end}} = 7$ with initial conditions $S_1(0) = 4571, S_2(0) = 5714$ and a small number of infective individuals. In each run of the model, the condition $\sum_j I_j(t_{\text{end}}) > O_L = 100$ was checked. For example, in the ODE model, $I_1(t_{\text{end}} = 7) \approx 2714$ and $I_2(t_{\text{end}} = 7) \approx 3105$ when $I_1(0) = 0$ and $I_2(0) = 1$. Longer time intervals gave similar results. The simulation results indicate that in both cases 1 and 2, Galton-Watson theory is a good approximation to the probability of outbreak or pathogen invasion.

TABLE 6. Case 2: Probability of an outbreak $(1 - P_0)$ computed using the theory of branching processes, and based on 1000 sample paths of the CTMC model for various initial values of I_1 and I_2.

$I_1(0)$	$I_2(0)$	$1 - P_0$	CTMC
1	0	0.807	0.793
0	1	0.690	0.681
1	1	0.940	0.938
2	0	0.963	0.967
0	2	0.904	0.899

Figures 3 and 4 illustrate three sample paths of the SDE model and CTMC model, respectively. In all cases simulated, the SDE model followed the ODE solution, but in the simulations for the CTMC model, sample paths were chosen to illustrate some cases of pathogen extinction ($P_0 = 0.31$).

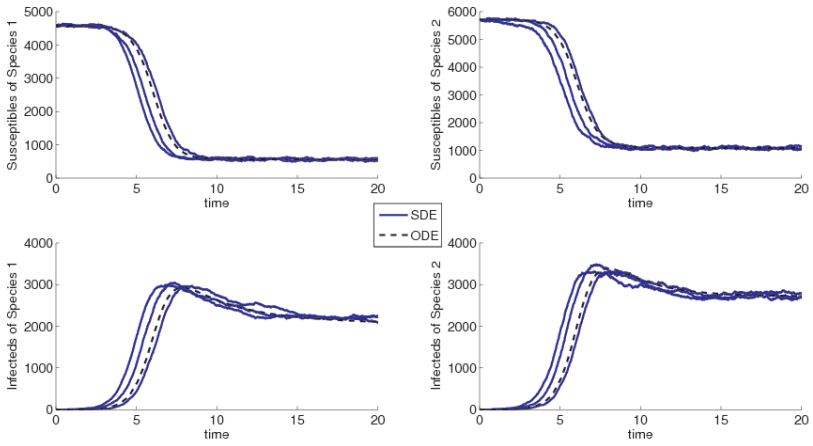

FIGURE 3. Comparison of ODE and SDE (3 sample paths) solutions for Case 2. We start the simulation at the DFE and add one infected individual of species 2, $S_1(0) = 4571, S_2(0) = 5714, I_1(0) = 0, I_2(0) = 1$.

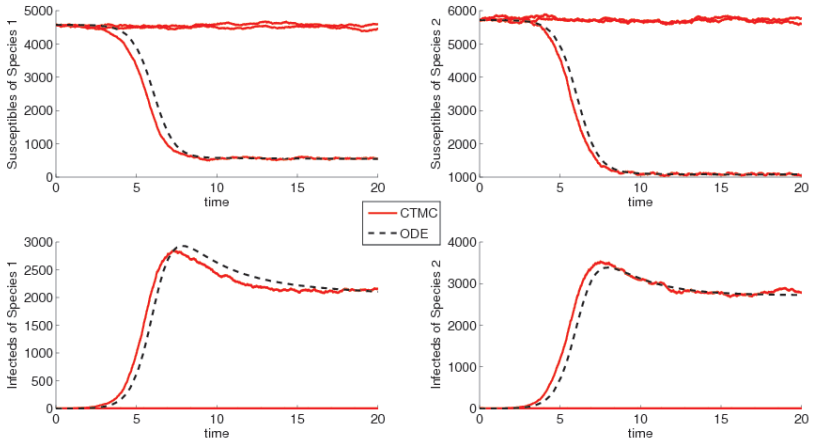

FIGURE 4. Comparison of ODE and CTMC (3 sample paths) solutions for Case 2. We start the simulation at the DFE and add one infected individual of species 2, $S_1(0) = 4571, S_2(0) = 5714, I_1(0) = 0, I_2(0) = 1$ ($P_0 = 0.31$).

5.3. **Case 3: Probability of invasion - density-dependent incidence.** In the third case, the ODE model (1)-(3) with density-dependent incidence illustrates species invasion. Parameter values are given in Table 2. The natural mortality rates (d_1, d_2) are density-independent, and all the competitive effects are incorporated in the birth terms. The pathogen causes disease-related mortality only in species 1, $\delta_1 = 5$ and $\delta_2 = 0$. There is no recovery for species 1 but the recovery rate for species

2 is very fast, $\gamma_1 = 0$ and $\gamma_2 = 13$. In the purely competitive system, species 1 wins the competition. However, the presence of disease changes the outcome. There is a stable host-pathogen equilibrium $(S_1, I_1) = (771, 88)$ for species 1. However, the corresponding equilibrium $(S_1, I_1, S_2, I_2) = (771, 88, 0, 0)$ is unstable in the two-species model as matrix J_{22}, defined in equation (10), is unstable. The equilibria and their local stability for the two-species model are summarized in Table 3.

Similar to this case, Manore [37] studied the ODE model (1)-(3) but with the assumption of no recovery ($\gamma_i = 0$). It was shown that if the parameters $\xi_1 = 1/K_{11} - c_{21}/K_{22}$, and $\xi_2 = 1/K_{22} - c_{12}/K_{11}$ have opposite signs $\xi_1\xi_2 < 0$, and if $\mathcal{R}_{01} > 1$, both species are present, and an infected individual of at least one species is present, then the disease persists uniformly strongly in at least one of the species. The parameters chosen for case 3 are such that the ξ_i's and $\mathcal{R}_{01}$ satisfy the inequalities above and we see stability of the infected one-species equilibrium for species 2.

In the CTMC model, the pgfs $\mathbf{g}$, defined in (17) and (18), and M_2, defined in (19), are computed using the parameters in Table 2. The spectral radius $\rho(M_2) \approx 1.26 > 1$ so that a unique fixed point of $\mathbf{g}$ exists in $(0,1) \times (0,1)$. The fixed point $\mathbf{q}$ is approximately $q_1 = 0.378$ and $q_2 = 0.378$. These extinction probabilities are used to compute the probability of invasion of species 1 by species 2, $(1 - P_0 = 1 - q_1^{S_2(0)} q_2^{I_2(0)})$, as described in Section 3.2, and verified with 1000 sample paths of the CTMC model in Table 7. One thousand simulations were run until $t_{\text{end}} = 15$ with the initial conditions $S_1(0) = 771, I_1(0) = 88$ and $S_2(0)$, $I_2(0)$ small. The probability of invasion was computed as the fraction of simulations in which $S_2(t_{\text{end}}) + I_2(t_{\text{end}}) > S_{inv} = 100$ at $t_{\text{end}} = 15$. For example, in the ODE model $S_2(t_{\text{end}} = 15) \approx 1946$ and $I_2(t_{\text{end}} = 15) \approx 4552$ when $S_2(0) = 1$ and $I_2(0) = 0$. Longer time intervals gave similar results.

Sample paths of the SDE model and CTMC model are compared with the ODE solution in Figures 5 and 6 for the initial conditions $S_1(0) = 771, S_2(0) = 1, I_1(0) = 88, I_2(0) = 0$, i.e., we start with the infected equilibrium of species one and add a susceptible individual of species two. In this case as in the two other cases, Galton-Watson theory is a good approximation to the probability of invasion.

TABLE 7. Case 3: Probability of invasion $(1 - P_0)$ of species 1 by species 2 computed using the theory of branching processes, and based on 1000 sample paths of the CTMC model for various initial values of S_2 and I_2.

$S_2(0)$	$I_2(0)$	$1 - P_0$	CTMC
1	0	0.622	0.620
0	1	0.622	0.600
1	1	0.875	0.861
2	0	0.875	0.862
0	2	0.875	0.863

6. **Discussion and conclusions.** In this investigation, a general deterministic model for n- species competition with a shared pathogen is introduced which serves as a skeleton on which to build new stochastic models, CTMC and SDE models. The competitive dynamics of the mixed community of species were studied,

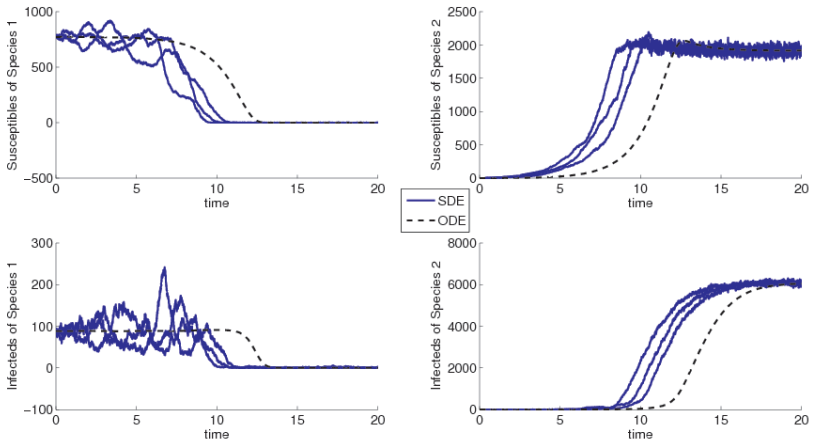

FIGURE 5. Comparison of ODE and SDE (3 sample paths) solutions for Case 3. We start the simulation at the infected one host equilibrium for species 1 and add one susceptible individual of species 2, $S_1(0) = 771, S_2(0) = 1, I_1(0) = 88, I_2(0) = 0$.

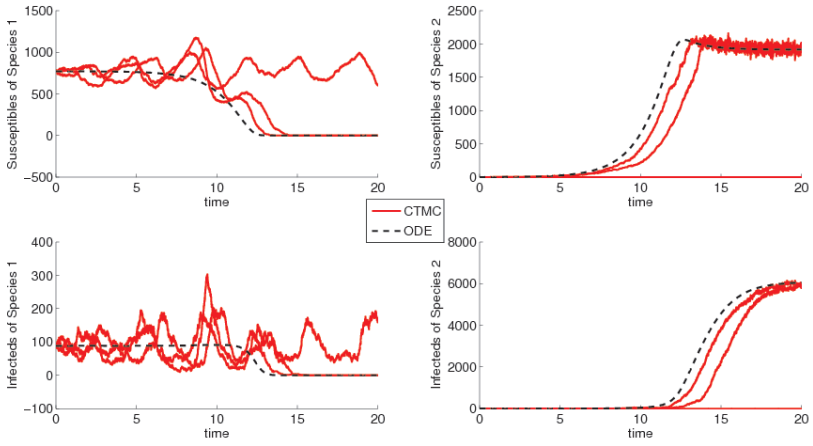

FIGURE 6. Comparison of ODE and CTMC (3 sample paths) solutions for Case 3. We start the simulation at the infected one host equilibrium for species 1 and add one susceptible individual of species 2, $S_1(0) = 771, S_2(0) = 1, I_1(0) = 88, I_2(0) = 0$ ($P_0 = 0.378$).

with a particular emphasis on the case $n = 2$. As documented in the literature [11, 33, 44, 51], the outcome of competition between two or more species can be changed in the presence of a shared pathogen. A native species may be invaded by an exotic, non-native species or a pathogen invasion may result in reduction of population densities because of disease-related mortalities. Whether the pathogen

or species invasion is successful depends on the initial density of invading pathogens or species. Thus, it is important to consider the stochastic variability due to births, deaths and transmission that impact the success of the invasion. We derive new CTMC and SDE models to account for this variability, using the assumptions in the deterministic model as the basis for our derivation.

New analytical results are presented for the ODE model in the case of $n \geq 2$ for pathogen or species invasion (Section 2.2). The basic reproduction number $\mathcal{R}_0 > 1$ indicates pathogen invasion and the instability of matrix J_{22} is an indication of species invasion for the ODE model, but when stochastic variability is included, $\mathcal{R}_0$ and J_{22} do not predict invasion success. Applying branching process theory to the CTMC model, we obtain estimates for probability of pathogen extinction P_0 or failure of a species invasion P_0 which depend on the initial number of pathogens or species invading and the fixed point of the offspring pgfs.

Application of branching process theory to the prediction of pathogen extinction is not new. For example, pathogen extinction was applied as early as 1955 by Whittle [55] to a simple SIR model, to a vector-host model in 1964 by Bartlett [8] and more recently by Griffiths and Greenhalgh to a respiratory disease in cattle [21]. However, application of branching process theory to multitype processes is not well-known in the epidemiological or ecological literature, nor is it well-known how to define the offspring pgfs. Our application of branching process theory to the n-species competitive system is new (Section 3). Careful attention must be paid to the underlying assumptions of the branching process theory. As with any linear approximation, these assumptions are only realistic near the equilibrium for small number of initial invaders in a large population. Whether the branching process approximation for probability of invasion is a good approximation must be verified for each model. After the invasion is successful, the SDE model provides an alternative to the CTMC model, especially useful for numerical simulation of large populations [1, 4, 27].

Our numerical examples highlight cases where the branching process predictions for pathogen or species invasion provide good estimates for the success of the invasion. Verification of the predicted estimate was checked via simulations of the CTMC model. A pathogen or species invader may not be successful on the first attempt but with repeated attempts or with a large number of invaders the probability of a successful invasion increases. These applications of branching processes to the n-species competition model with disease have a wider applicability to the emergence of new diseases and to species invasions in other settings.

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Appendix. We verify that $\rho(M_1) > 1$ iff $\rho(K) > 1$ for the case $n = 2$, where both M_1 and K are irreducible. Then it follows from continuous-time Galton-Watson

branching process theory that $\rho(K) > 1$ iff there exists a unique fixed point of the pgfs $f_i(q_1, q_2) = q_i$, $i = 1, 2$.

Theorem 6.1. *For the case $n = 2$, $\rho(M_1) > 1$ iff $\rho(K) > 1$, where matrices K and M_1 are defined in (8) and (14), respectively.*

Proof. The spectral radius of a 2×2 nonnegative matrix $B = [b_{ij}]$, $\rho(B) < 1$ iff the Jury conditions hold [3]:

$$\text{trace}(B) < 1 + \det(B) < 2. \tag{21}$$

Alternately, $\rho(B) > 1$ iff at least one of the inequalities in (21) is reversed, that is,

(i) $b_{ii} > 1$ for some $i = 1, 2$ or
(ii) $b_{ii} \leq 1$ for $i = 1, 2$ and $(1 - b_{11})(1 - b_{22}) < b_{12}b_{21}$.

For the 2×2 matrices $K = [k_{ij}]$ and $M_1 = [m_{ij}]$, defined in (8) and (14), respectively, the matrix elements are $k_{ii} = \hat{\alpha}_{ii}/\mathcal{D}_i$, $k_{ij} = \hat{\alpha}_{ij}/\mathcal{D}_i$, $m_{ii} = (\hat{\alpha}_{ii} + c_i)/(\mathcal{D}_i + c_i)$ and $m_{ij} = \hat{\alpha}_{ji}/(\mathcal{D}_i + c_i)$, with $c_i = \hat{\alpha}_{ii} + \hat{\alpha}_{ji}$, $i, j = 1, 2$, $i \neq j$. We show the conditions in (i) and (ii) are equivalent for b_{ij} replaced by k_{ij} or m_{ij}.

It is straightforward to show that $k_{ii} > 1$ (≤ 1) iff $m_{ii} > 1$ (≤ 1). The second condition in (ii) for matrix K is

$$(1 - k_{11})(1 - k_{22}) < k_{12}k_{21} \tag{22}$$

which is equivalent to

$$(\mathcal{D}_1 - \hat{\alpha}_{11})(\mathcal{D}_2 - \hat{\alpha}_{22}) < \hat{\alpha}_{12}\hat{\alpha}_{21}.$$

This latter inequality is equivalent to

$$(\mathcal{D}_1 + c_1 - [\hat{\alpha}_{11} + c_1])(\mathcal{D}_2 + c_2 - [\hat{\alpha}_{22} + c_2]) < \hat{\alpha}_{12}\hat{\alpha}_{21}$$

which, in turn, is equivalent to

$$(1 - m_{11})(1 - m_{22}) < m_{12}m_{21}.$$

Thus, $\rho(M_1) > 1$ iff $\rho(K) > 1$. □

It follows from the properties of the pgfs (13) (f_i are not simple), the properties of matrix M_1 (M_1 is nonnegative and irreducible) and the preceding theorem that a unique fixed point (q_1, q_2), $0 < q_i < 1$ exists to the pgfs (13) for $n = 2$ iff $\rho(K) > 1$ [25, 32, 43, 48].

Alternately, the preceding result, $\rho(K) > 1$ iff there exists a unique fixed point (q_1, q_2), $0 < q_i < 1$ of the pgfs (13) for $n = 2$, can be verified directly. Rewrite the equations for the pgfs $u_i = f_i(u_1, u_2)$ as the functions, $u_j = h_i(u_i)$, $i, j = 1, 2$, $i \neq j$. That is,

$$h_i(u_i) = \frac{-b_{1i}u_i^2 + u_i - b_{2i}}{b_{3i}u_i},$$

where

$$b_{1i} = \frac{\hat{\alpha}_{ii}}{\mathcal{D}_i + c_i}, \quad b_{2i} = \frac{\mathcal{D}_i}{\mathcal{D}_i + c_i} \quad \text{and} \quad b_{3i} = \frac{\hat{\alpha}_{ji}}{\mathcal{D}_i + c_i}, \ j \neq i.$$

Then the fixed point (q_1, q_2) is the unique point of intersection of these two functions in the region $(0, 1) \times (0, 1)$. Whether the curves h_i intersect in the open region $(0, 1) \times (0, 1)$ depends on the slope of these two curves at the point (1,1). It can be verified in a manner similar to the proof of Theorem 4.1 in reference [42] (page 24, where the fixed point at the origin is translated to the point (1,1)), that there exists a unique fixed point in the following four cases: (a) $k_{ii} > 1$, $i = 1, 2$, (b) $k_{11} > 1$ and $k_{22} < 1$, (c) $k_{11} < 1$ and $k_{22} > 1$, and (d) $k_{ii} \leq 1$, $i = 1, 2$ and inequality (22).

Cases (a)-(c) correspond to condition (i) and case (d) to condition (ii) in the proof of Theorem 6.1. See Figure 7.

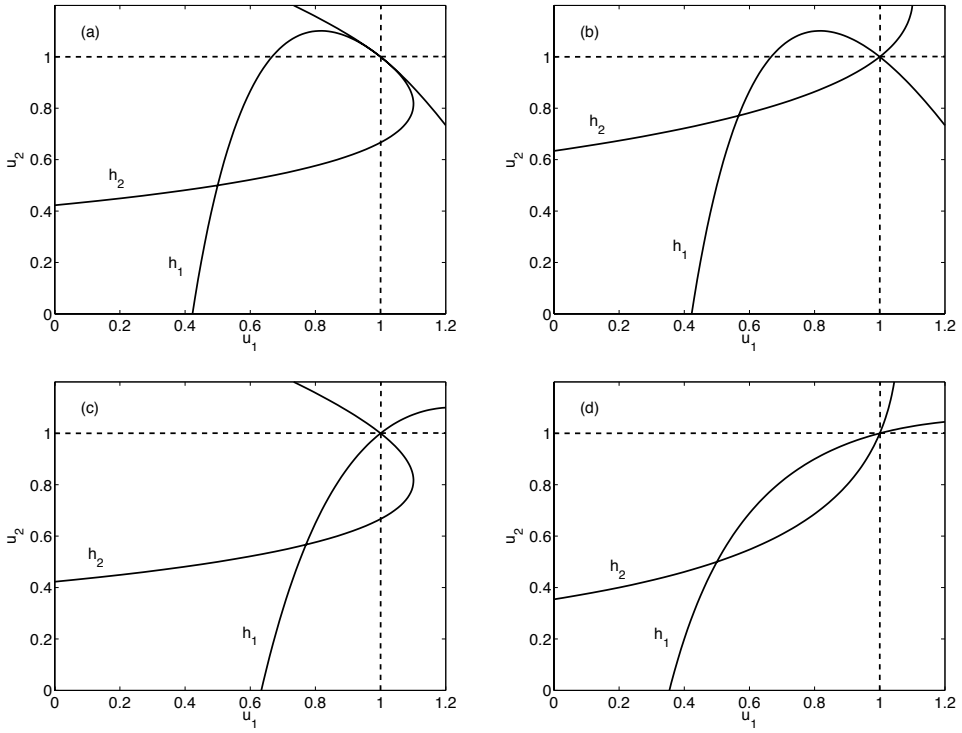

FIGURE 7. The functions $u_1 = h_2(u_2)$ and $u_2 = h_1(u_1)$ have a unique point of intersection in the region $(0,1) \times (0,1)$ in the four cases: (a) $k_{ii} > 1$, $i = 1, 2$; (b) $k_{11} > 1$ and $k_{22} < 1$; (c) $k_{11} < 1$ and $k_{22} > 1$; (d) $k_{ii} \leq 1$, $i = 1, 2$ and inequality (22).

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E-mail address: linda.j.allen@ttu.edu
E-mail address: bokilv@math.oregonstate.edu