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COMPETING SPECIES MODELS WITH AN INFECTIOUS DISEASE

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Abstract. The frequency-dependent (standard) form of the incidence is used for the transmission dynamics of an infectious disease in a competing species model. In the global analysis of the SIS model with the birth rate independent of the population size, a modified reproduction number $\mathbf{R}_1$ determines the asymptotic behavior, so that the disease dies out if $\mathbf{R}_1 \leq 1$ and approaches a globally attractive endemic equilibrium if $\mathbf{R}_1 > 1$. Because the disease-reduced reproduction and disease-related death rates are often different in two competing species, a shared disease can change the outcome of the competition. Models of SIR and SIRS type are also considered. A key result in all of these models with the frequency-dependent incidence is that the disease must either die out in both species or remain endemic in both species.

1. **Introduction.** Population sizes of species are affected not only by ecological interactions such as competition, predation, and parasitism, but also by the effects of infectious diseases [13, 18, 23]. Infectious diseases are said to be of SIS type if animals have no immunity after an infection, so that susceptibles move to the infective class when infected and then back to the susceptible class after recovery. If there is temporary immunity in a recovered class after an infection, then the disease is of SIRS type. An SIR model is a special case of an SIRS model in which the immunity is permanent, so that recovered animals never lose their immunity. Here we consider SIS and SIRS modifications with disease-related deaths and disease-reduced reproduction of the usual competing species model. For these models we show how the infectious disease can affect the outcome of the interspecies competition.

The infection rate per unit time of susceptible animals through their contacts with infectious animals is called the incidence of the disease. Let $X(t)$ be the number of susceptibles at time t, $Y(t)$ be the number of infectives, and $N(t)$ be the host population size. Let β be the average number of adequate contacts (i.e., contacts sufficient for transmission) of a susceptible animal per unit time. Since Y/N is the proportion of animals that are infectious, $\beta Y/N$ is the average number of adequate contacts with infectious animals per unit time of one susceptible. The number of new cases per unit time $(\beta Y/N)X$ is the average number of adequate contacts with infectious animals per unit time of all susceptibles. Hethcote [14, 15, 16, 17] calls

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this form, $\beta XY/N$, the standard incidence. Because it depends on the frequency of infections Y/N, Begon et al. [3] called the form $\beta XY/N$ frequency-dependent incidence.

The mass action law ηXY, with η as a mass action coefficient, has also been used for the incidence in infectious disease models [2]. In this case the parameter η has no epidemiological interpretation, but comparing it with frequency-dependent formulation shows that $\beta = \eta N$, so that this form implicitly assumes that the contact rate β is a linearly increasing function of population size. However, this linear dependence is not supported by data, since the transmission between two animals is determined primarily by the local interactions among animals, which are usually independent of or only weakly dependent on the population size. Numerous studies have also found that frequency-dependent (standard) incidence is more realistic than the mass action incidence [3, 8, 29]. Hethcote, Wang, and Li [19] gave careful derivations of the frequency-dependent and mass action incidence for population sizes and densities.

For two species let the contact rate β_{ij} be the average number of adequate contacts of species i susceptible animals with species j animals per unit time (cf. Hethcote [16, 17]). The fraction of animals in species j with size N_j that are infectious is Y_j/N_j, so that the average number of adequate contacts of one species i susceptible animal with species j infectious animals per unit time is $\beta_{ij}Y_j/N_j$. Since the number of species i susceptibles is X_i, the average number of adequate contacts of species i susceptibles with species j infectious animals per unit time is $(\beta_{ij}Y_j/N_j)X_i$; so, this is the number of new cases in species i due to contacts with species j. Thus the frequency-dependent (standard) incidence, which is the total number of new cases in species i due to contacts with their own species i and the other species j, is given by $[\beta_{ii}Y_i/N_i + \beta_{ij}Y_j/N_j]X_i$. Hethcote, Wang, and Li [19] showed that the frequency-dependent incidence in terms of species densities has this same form. The mass-action incidence for two species is $[\eta_{ii}Y_i + \eta_{ij}Y_j]X_i$, where η_{ij} are mass-action coefficients for the interactions between species i and j animals.

Models for two species which share a disease but do not compete have been studied previously. Holt and Pickering [21], Begon et al. [5], and Greenman and Hudson [12] found that two host SIS models with the mass action incidence can have complicated behaviors such as one, two, or three infected coexistence equilibria and one or more attractive periodic solutions. Hethcote, Wang, and Li [19] found that similar models with frequency-dependent incidence have the classic endemic model behavior, in which the disease dies out below the threshold and approaches an endemic equilibrium above the threshold.

In their study of a competing species model with mass action incidence in which one species was affected by a pathogen, Anderson and May [1] described how the disease could influence the competition. A comprehensive survey of two host models with a pathogen was given by Begon and Bowers [4]. In a thorough study of an SIS competing species model with mass action incidence, density-independent death rates, and disease-related deaths, Bowers and Turner [7] developed invadability criteria to show how the forces of competition and infection combine. In a paper on the cowpox virus in coexisting populations of bank voles and wood mice, Begon et al. [6] found that frequency-dependent incidence was clearly superior. Venturino [30] formulated competing species models with a disease in one species and both

types of incidence; he found periodic solutions numerically in one model with mass action incidence.

Tompkins, White, and Boots [28] used a variation of the model of Bowers and Turner [7] with density-independent death rates and mass action incidence to study the effects of a parapoxvirus in competing squirrel species in the United Kingdom. Their model was SIR type for invading grey squirrels and SI type with disease-related deaths for native red squirrels. Using parameters estimated from data, they found that the invading grey squirrels eventually win the competition, and the disease speeds up the process. They also used computer simulations to examine the speed of the spatial spread of the grey squirrels into red squirrel territory.

The model considered here differs from previous models for a disease in two competing species, because it uses the frequency-dependent incidence and the disease can affect both species. The behaviors of an SIS model with logistic growth and a competing species model are given in sections 2 and 3. After a general competing species model with an SIS disease is formulated in section 4, we analyze this model with birth and death rates independent of size in sections 5 and 6, respectively. Results on an SIRS model with logistic growth are presented in section 7 and a competing species model with an SIRS disease is formulated in section 8. This model with size-independent birth rates is analyzed in section 9 and the results in this paper are discussed in section 10.

2. **The SIS model with logistic growth.** In the SIS model with size-dependent regulation of one species with disease-reduced reproduction and disease-related deaths, the number of susceptibles is $X(t)$ and the number of infectives is $Y(t)$ in host population with size $N(t)$, so that $N(t) = X(t) + Y(t)$. This SIS model with logistic growth and frequency-dependent incidence $\beta XY/N$ is

$$\begin{aligned} dN/dt &= \left[a - \frac{\chi r N}{K}\right][X + (1-f)Y] - \left[b + \frac{(1-\chi)rN}{K}\right]N - \varepsilon_0 Y \qquad (2.1)\\ &= r(1-N/K)N - (\varepsilon_0 + af)Y + f\chi rYN/K \\ dX/dt &= \left[a - \frac{\chi r N}{K}\right][X + (1-f)Y] - \left[b + \frac{(1-\chi)rN}{K}\right]X - \beta XY/N + \gamma Y, \\ dY/dt &= \beta XY/N - \left[\gamma + b + \varepsilon_0 + \frac{(1-\chi)rN}{K}\right]Y, \end{aligned}$$

where a is the intrinsic per capita birth rate for susceptibles and f is the reduction in birth rate due to the disease, so that $a(1-f)$ is the intrinsic per capita birth rate for infectives. Here b is the intrinsic natural per capita death rate, $r = a - b$ is the positive intrinsic per capita net growth rate, χ is the convex combination constant with $0 \le \chi \le 1$, K is the environmental carrying capacity, γ is the per capita recovery rate, and ε_0 is the per capita disease-related death rate. This SIS model with frequency-dependent (standard) incidence, disease-related deaths, and vertical transmission, but without disease-reduced reproduction, was studied by Gao and Hethcote [11].

With no disease, the differential equation for N is the logistic differential equation for restricted growth given by

$$\begin{aligned} dN/dt &= [a - \chi rN/K]N - [b + (1-\chi)rN/K]N \\ &= r(1 - N/K)N, \end{aligned}$$

where the first term is the birth rate, the second term is the death rate, and χ is a convex combination parameter in the interval $[0, 1]$. Solutions with $N(0) > 0$ approach the carrying capacity K. For $0 < \chi < 1$, the per capita birth rate $a - \chi rN/K$ decreases and the per capita death rate $b + (1 - \chi)rN/K$ increases as the population size N increases. These size-dependent birth and death rates are consistent with the effects of limited resources in a population. The birth rate is independent of the population size when $\chi = 0$, so that all of the size dependence is in the per capita death rate, and the death rate is independent of size when $\chi = 1$. We consider the positively invariant subset of the first quadrant in XY space with $N < aK/\chi r$, so that the birth rate is always positive. Since $N' < 0$ for $N > K$, all solution paths in the subset above approach, enter, or stay in the subset with $N \leq K$. Solution paths with $N(0) > K$ which do not enter the region $N \leq K$ in finite time have their omega limit sets on the $N = K$ plane. Thus it suffices to analyze solution paths in the subset of the first quadrant of XY space with $N = X + Y \leq K$.

The system (2.1) can be reduced to the following system of two equations by using $I = Y/N$ and $X/N = 1 - I$:

$$\begin{aligned} dN/dt &= [r(1 - N/K) - (af + \varepsilon_0)I + f\chi rIN/K]N, \\ dI/dt &= \beta I(1 - I) - [\gamma + a + \varepsilon_0 - (af + \varepsilon_0)I - \chi r(1 - fI)N/K]I. \end{aligned} \tag{2.2}$$

This model is well posed in the positively invariant region $D = \{(N, I) : 0 \leq N \leq K, 0 \leq I \leq 1\}$. The behavior is governed by the three threshold quantities:

$$\begin{aligned} \mathbf{R}_2 &= \beta/(\gamma + a + \varepsilon_0 - \chi r), \\ \mathbf{R}_1 &= \beta/(\gamma + a + \varepsilon_0), \\ \phi &= \frac{r}{(af + \varepsilon_0)[1 - (\gamma + b + \varepsilon_0)/\beta]}, \end{aligned} \tag{2.3}$$

where $\mathbf{R}_2 \geq \mathbf{R}_1$. The asymptotic behaviors summarized in the table below were proved in Hethcote, Wang, and Li [19].

Table 1. Asymptotic behaviors for the SIS logistic model

Cases	Asymptotic behavior for large time
case 1: $1 \geq \mathbf{R}_2 \geq \mathbf{R}_1$	$N(0) > 0 \Rightarrow (N, I) \to E_2 = (K, 0)$
case 2: $\mathbf{R}_2 > 1 \geq \mathbf{R}_1$	$N(0) > 0, I(0) > 0 \Rightarrow (N, I) \to E_4 = (N_4, I_4)$
case 3: $\mathbf{R}_1 > 1$ & $\phi < 1$	$I(0) > 0 \Rightarrow (N, I) \to E_3 = (0, I_3)$
case 4: $\mathbf{R}_1 > 1$ & $\phi > 1$	$N(0) > 0, I(0) > 0 \Rightarrow (N, I) \to E_4 = (N_4, I_4)$

The system always has the equilibrium points $E_1 = (0, 0)$ and $E_2 = (K, 0)$. If $\mathbf{R}_1 > 1$, then the equilibrium point $E_3 = (0, I_3)$ is on the boundary of D with

$$I_3 = \frac{\beta - (\gamma + a + \varepsilon_0)}{\beta - (af + \varepsilon_0)}. \tag{2.4}$$

If $\mathbf{R}_2 > 1$ and $\phi > 1$, there is an equilibrium point $E_4 = (N_4, I_4)$ in the interior of D. The intuitive explanation of case 2 in Table 1 is that the disease just barely remains endemic since $\mathbf{R}_2 > 1 \geq \mathbf{R}_1$, but the natural per capita growth rate r dominates the per capita disease-induced death rate given by $af + \varepsilon_0$ because $\phi > 1$, so that the population size goes to a steady state N_4 that is less than the carrying capacity K.

In case 3 the disease remains endemic since $\mathbf{R}_2 > 1$ and the per capita disease-induced death rate given by $af + \varepsilon_0$ overpowers the per capita natural growth rate

r since $\phi < 1$, so that the population is driven to extinction by the endemicity of the disease. In case 4 the disease remains endemic since $\mathbf{R}_2 > 1$, and the per capita disease-induced death rate given by $af + \varepsilon_0$ is dominated by the natural per capita growth rate r since $\phi > 1$, so that the population size goes to a steady state N_4 that is less than the carrying capacity K.

3. **The competing species model.** The usual model [10] for two species competing for a limited resource such as food or habitat is

$$\begin{aligned} dN_1/dt &= r_1[1 - (N_1 + \alpha_{12}N_2)/K_1]N_1, \\ dN_2/dt &= r_2[1 - (N_2 + \alpha_{21}N_1)/K_2]N_2, \end{aligned} \tag{3.1}$$

where N_i is the number of individuals in species i, r_i is the intrinsic per capita growth rate of species i, and K_i is the environmental carrying capacity for species i. The parameter α_{ij} gives the per capita inhibiting effect of species j on the population growth rate of species i, as compared to the effect of species i on its own population growth rate. One can interpret $1/K_i$ as the inhibition of species i on its own growth and α_{ij}/K_i as the inhibition of species j on the growth of species i.

This competing species model always has three equilibrium points, $E_0 = (0, 0)$, $E_1 = (K_1, 0)$, and $E_2 = (0, K_2)$, on the boundary of the positively invariant first quadrant. These boundary equilibria correspond to both species being absent, or one species being absent while the other is at its carrying capacity. Solutions starting on a positive axis approach the carrying capacity equilibrium on that axis. A frequency-dependent phase plane analysis using nullclines yields four cases. In **case 1**, in which species 1 inhibits species 2 more that it inhibits itself ($\alpha_{21}/K_2 > 1/K_1$) and species 2 inhibits itself more than it inhibits species 1 ($1/K_2 > \alpha_{12}/K_1$), species 1 wins the competition and all paths with $N_1(0) > 0$ approach the equilibrium $E_1 = (K_1, 0)$. In **case 2**, in which species 1 inhibits itself more that it inhibits species 2 ($1/K_1 > \alpha_{21}/K_2$) and species 2 inhibits species 1 more than it inhibits itself ($\alpha_{12}/K_1 > 1/K_2$), species 2 wins the competition and all paths with $N_2(0) > 0$ approach the boundary equilibrium $E_2 = (0, K_2)$.

In **case 3**, in which each species inhibits the other more than it inhibits itself ($\alpha_{21}/K_2 > 1/K_1$ and $\alpha_{12}/K_1 > 1/K_2$), the nullclines intersect at an unstable saddle interior equilibrium $E_3 = (N_1^e, N_2^e)$. In this case there is a separatrix curve through the interior equilibrium and the origin with solutions starting below the separatrix going to the equilibrium $E_1 = (K_1, 0)$, and solutions starting above it going to the boundary equilibrium $E_2 = (0, K_2)$. Intuitively, whichever species is initially dominant is the winner of the competition.

In **case 4**, in which each species inhibits itself more than it inhibits the other species ($1/K_1 > \alpha_{21}/K_2$ and $1/K_2 > \alpha_{12}/K_1$), the interior equilibrium is attractive, and all solutions starting with $N_1(0) > 0$ and $N_2(0) > 0$ approach this interior equilibrium $E_3 = (N_1^e, N_2^e)$. In this case the two species coexist and approach a coexistence equilibrium. The interior equilibrium is found as the intersection of the straight line nullclines $N_1 + \alpha_{12}N_2 = K_1$ and $N_2 + \alpha_{21}N_1 = K_2$. Thus

$$\begin{aligned} N_1^e &= (K_1 - \alpha_{12}K_2)/(1 - \alpha_{12}\alpha_{21}), \\ N_2^e &= (K_2 - \alpha_{21}K_1)/(1 - \alpha_{12}\alpha_{21}), \end{aligned}$$

where the numerators and denominators are negative in case 3 and positive in case 4.

4. **Competing species with an SIS disease.** The SIS model for two competing host populations is

$$\begin{aligned}
dN_1/dt &= r_1(1-(N_1+\alpha_{12}N_2)/K_1)N_1-(\varepsilon_{10}+a_1f_1)Y_1 \\
&\quad + f_1\chi_1 r_1 Y_1(N_1+\alpha_{12}N_2)/K_1, \\
dX_1/dt &= (a_1-\frac{\chi_1 r_1(N_1+\alpha_{12}N_2)}{K_1})[X_1+(1-f_1)Y_1]+\gamma_1 Y_1 \\
&\quad -(\beta_{11}Y_1/N_1+\beta_{12}Y_2/N_2)X_1-(b_1+\frac{(1-\chi_1)r_1(N_1+\alpha_{12}N_2)}{K_1})X_1, \\
dY_1/dt &= (\beta_{11}Y_1/N_1+\beta_{12}Y_2/N_2)X_1 \\
&\quad -(\gamma_1+b_1+\frac{(1-\chi_1)r_1(N_1+\alpha_{12}N_2)}{K_1}+\varepsilon_{10})Y_1, \\
dN_2/dt &= r_2(1-(N_2+\alpha_{21}N_1)/K_2)N_2-(\varepsilon_{20}+a_2f_2)Y_2 \\
&\quad + f_2\chi_2 r_2 Y_2(N_2+\alpha_{21}N_1)/K_2, \\
dX_2/dt &= (a_2-\frac{\chi_2 r_2(N_2+\alpha_{21}N_1)}{K_2})[X_2+(1-f_2)Y_2]+\gamma_2 Y_2 \\
&\quad -(\beta_{21}Y_1/N_1+\beta_{22}Y_2/N_2)X_2-(b_2+\frac{(1-\chi_2)r_2(N_2+\alpha_{21}N_1)}{K_2})X_2, \\
dY_2/dt &= (\beta_{21}Y_1/N_1+\beta_{22}Y_2/N_2)X_2 \\
&\quad -(\gamma_2+b_2+\frac{(1-\chi_2)r_2(N_2+\alpha_{21}N_1)}{K_2}+\varepsilon_{20})Y_2,
\end{aligned} \tag{4.1}$$

where the variables and parameter values are analogous to those in the SIS and competing species models. We assume that the competing species do interact, so that all contact rates β_{ij} including β_{12} and β_{21} are positive.

The system (4.1) can be reduced to the following system of four equations for the population sizes and the infective fractions in them by using $X_i = N_i - Y_i$ and $I_i = Y_i/N_i$.

$$\begin{aligned}
dN_1/dt &= [r_1(1-(N_1+\alpha_{12}N_2)/K_1)-\varepsilon_1 I_1+f_1\chi_1 r_1 I_1(N_1+\alpha_{12}N_2)/K_1]N_1, \\
dI_1/dt &= (\beta_{11}I_1+\beta_{12}I_2)(1-I_1) \\
&\quad -[d_1-\varepsilon_1 I_1-\chi_1 r_1(1-f_1 I_1)(N_1+\alpha_{12}N_2)/K_1]I_1, \\
dN_2/dt &= [r_2(1-(N_2+\alpha_{21}N_1)/K_2)-\varepsilon_2 I_2+f_2\chi_2 r_2 I_2(N_2+\alpha_{21}N_1)/K_2]N_2, \\
dI_2/dt &= (\beta_{21}I_1+\beta_{22}I_2)(1-I_2) \\
&\quad -[d_2-\varepsilon_2 I_2-\chi_2 r_2(1-f_2 I_2)(N_2+\alpha_{21}N_1)/K_2]I_2,
\end{aligned} \tag{4.2}$$

where $r_i = a_i - b_i$, $\varepsilon_i = \varepsilon_{i0} + a_i f_i$, and $d_i = \gamma_i + a_i + \varepsilon_{i0}$. The system (4.2) is mathematically well posed in the four-dimensional region

$$D = \{(N_1, I_1, N_2, I_2) : 0 \le N_i \le K_i, 0 \le I_i \le 1\}. \tag{4.3}$$

For equilibrium points of the system (4.2), it is easy to see that if one of the equilibrium values I_i^e is zero, then the other I_j^e must also be zero. Thus, the disease must either die out in both species or remain endemic in both species.

5. **SIS model with size-independent birth rates.** One simplification of the model is to assume that the per capita birth rates are independent of size ($\chi_i = 0$),

so that the competing species model with an SIS infectious disease (4.2) becomes

$$\begin{aligned} dN_1/dt &= [r_1(1-(N_1+\alpha_{12}N_2)/K_1)-\varepsilon_1 I_1]N_1, \\ dI_1/dt &= (\beta_{11}I_1+\beta_{12}I_2)(1-I_1)-[d_1-\varepsilon_1 I_1]I_1, \\ dN_2/dt &= [r_2(1-(N_2+\alpha_{21}N_1)/K_2)-\varepsilon_2 I_2]N_2, \\ dI_2/dt &= (\beta_{21}I_1+\beta_{22}I_2)(1-I_2)-[d_2-\varepsilon_2 I_2]I_2. \end{aligned} \tag{5.1}$$

This system (5.1) uncouples, since the differential equations for the infective fractions I_i are independent of the N_i variables. This I_1I_2 system

$$\begin{aligned} dI_1/dt &= (\beta_{11}-d_1)I_1+\beta_{12}I_2(1-I_1)+(\varepsilon_1-\beta_{11})I_1^2, \\ dI_2/dt &= \beta_{21}I_1(1-I_2)+(\beta_{22}-d_2)I_2+(\varepsilon_2-\beta_{22})I_2^2. \end{aligned} \tag{5.2}$$

was studied in Hethcote, Wang, and Li [19]. The feasible region for (5.2) is the unit square $\hat{D}=\{(I_1,I_2): 0\le I_1\le 1, 0\le I_2\le 1\}$. Because the off-diagonal entries in the Jacobian matrix of the system (5.2) are positive, the system is cooperative in $\hat{D}$. Thus solutions must approach equilibria [20, 26]. The modified reproduction number $\mathbf{R}_1$ for this model is the spectral radius ρ (the maximum absolute value of an eigenvalue) of the next generation matrix [9, 16] given by

$$\mathbf{R}_1=\rho\begin{bmatrix} \beta_{11}/d_1 & \beta_{12}/d_2 \\ \beta_{21}/d_1 & \beta_{22}/d_2 \end{bmatrix}=\frac{1}{2}\left\{\frac{\beta_{11}}{d_1}+\frac{\beta_{22}}{d_2}+\sqrt{\left(\frac{\beta_{11}}{d_1}-\frac{\beta_{22}}{d_2}\right)^2+\frac{4\beta_{12}\beta_{21}}{d_1d_2}}\right\}. \tag{5.3}$$

From the trace and determinant of the Jacobian, we find that the equilibrium $\hat{E}_0=(0,0)$ is asymptotically stable if

$$\beta_{12}\beta_{21}<(d_1-\beta_{11})(d_2-\beta_{22}), \tag{5.4}$$

where the factors on the right side must be positive. This condition is equivalent to $\mathbf{R}_1<1$.

The nullclines of (5.2) are

$$\begin{aligned} I_2 &= \frac{I_1[d_1-\varepsilon_1 I_1-\beta_{11}(1-I_1)]}{\beta_{12}(1-I_1)}, \\ I_1 &= \frac{I_2[d_2-\varepsilon_2 I_2-\beta_{22}(1-I_2)]}{\beta_{21}(1-I_2)}. \end{aligned} \tag{5.5}$$

Because the second derivatives of the expressions above are positive, the first nullcline is concave and the second is convex in $\hat{D}$. These two nullclines pass through the origin of $\hat{D}$ and pass out the top and right side of $\hat{D}$, respectively. Geometrically we see that these nullclines do not intersect in $\hat{D}$, if at the origin the slope $(d_1-\beta_{11})/\beta_{12}$ of the first nullcline is greater than the slope $\beta_{21}/(d_2-\beta_{22})$ of the second nullcline, which is the same as condition (5.4). However, if the slope of the first nullcline at the origin is less than the slope of the second nullcline, then the nullclines do intersect inside $\hat{D}$.

Thus, if $\mathbf{R}_1\le 1$, then $\hat{E}_0=(0,0)$ is the only equilibrium in $\hat{D}$ and it is locally asymptotically stable. Because cooperativity implies monotonicity, all solution paths in $\hat{D}$ must approach $\hat{E}_0=(0,0)$. If $\mathbf{R}_1>1$, then the disease-free equilibrium $\hat{E}_0=(0,0)$ is unstable with a repulsive direction into $\hat{D}$, since the Perron Theorem implies that the spectral radius is a real positive eigenvalue and the corresponding eigenvector has positive entries [22]. Moreover, $\mathbf{R}_1>1$ implies

that there is a unique endemic equilibrium $\hat{E}_e = (I_1^e, I_2^e)$ in $\hat{D}$, which is globally attractive in $\hat{D} - \{(0,0)\}$ by the monotonicity result above.

For the system (5.1), the limiting differential equations when $\mathbf{R}_1 \leq 1$ are the competing species model (3.1). By the last result in the appendix, the asymptotic behavior of the system (5.1) is that the disease dies out and the behavior of the two species corresponds to one of the four cases given in section 3. These four cases with $\mathbf{R}_1 \leq 1$ are cases 1 to 4.

If $\mathbf{R}_1 > 1$, so that the disease remains endemic, then the limiting differential equations are a modified competing species system given by

$$dN_1/dt = [(r_1 - \varepsilon_1 I_1^e)(1 - (N_1 + \alpha_{12}N_2)/K_1^*)]N_1,$$
$$dN_2/dt = [(r_2 - \varepsilon_2 I_2^e)(1 - (N_2 + \alpha_{21}N_1)/K_2^*)]N_2,$$

where $K_1^* = (r_1 - \varepsilon_1 I_1^e)K_1/r_1$ and $K_2^* = (r_2 - \varepsilon_2 I_2^e)K_2/r_2$ are called modified carrying capacities. Using the last result on limiting systems in the appendix, we find that the asymptotic behavior of the system (5.1) is that $I_i(t) \to I_i^e$ as $t \to \infty$ and the behavior of the two population sizes when K_1^* and K_2^* are positive corresponds to one of the four cases given in section 3 with K_1 and K_2 replaced by the modified carrying capacities K_1^* and K_2^*. Since $K_1^* < K_1$ and $K_2^* < K_2$, the endemicity of the disease changes the parameter ranges for which the conditions of each of the first four cases are satisfied, so that the asymptotic behavior of the two species with an endemic disease may be given by a different case. Even if the case does not change, the attractive equilibria are different.

In **case 5**, in which species 1 inhibits species 2 more than it inhibits itself ($\alpha_{21}/K_2^* > 1/K_1^*$) and species 2 inhibits itself more than it inhibits species 1 ($1/K_2^* > \alpha_{12}/K_1^*$), species 1 wins the competition and all paths with $N_1(0) > 0$ approach the equilibrium $E_1^* = (K_1^*, I_1^e, 0, I_2^e)$. Because of the endemicity of the disease, the new equilibrium E_1^* has a value K_1^* that is lower than the original carrying capacity K_1. Thus the disease has decreased the equilibrium population size of species 1.

In **case 6**, in which species 1 inhibits itself more than it inhibits species 2 ($1/K_1^* > \alpha_{21}/K_2^*$) and species 2 inhibits species 1 more than it inhibits itself ($\alpha_{12}/K_1^* > 1/K_2^*$), species 2 wins the competition and all paths with $N_2(0) > 0$ approach the boundary equilibrium $E_2^* = (0, I_1^e, K_2^*, I_2^e)$, where $K_2^* < K_2$.

In **case 7**, in which each species inhibits the other more than it inhibits itself ($\alpha_{21}/K_2^* > 1/K_1^*$ and $\alpha_{12}/K_1^* > 1/K_2^*$), the nullclines intersect at an unstable saddle interior equilibrium $E_3^* = (N_1^*, I_1^e, N_2^*, I_2^e)$. In this case, there is a separatrix surface through the interior equilibrium and the origin, so that solutions starting on one side of the separatrix go to the equilibrium $E_1^* = (K_1^*, I_1^e, 0, I_2^e)$, and solutions starting on the other side go to the boundary equilibrium $E_2^* = (0, I_1^e, K_2^*, I_2^e)$. As in the competing species model in section 3, the coordinates of the interior equilibrium are

$$N_1^* = (K_1^* - \alpha_{12}K_2^*)/(1 - \alpha_{12}\alpha_{21}),$$
$$N_2^* = (K_2^* - \alpha_{21}K_1^*)/(1 - \alpha_{12}\alpha_{21}).$$

In **case 8**, in which each species inhibits itself more than it inhibits the other species ($1/K_1^* > \alpha_{21}/K_2^*$ and $1/K_2^* > \alpha_{12}/K_1^*$), the interior equilibrium is attractive, and all solutions starting with $N_1(0) > 0$ and $N_2(0) > 0$ approach this coexistence equilibrium $E_3^* = (N_1^*, I_1^e, N_2^*, I_2^e)$. In this case, the intersections of the two straight line nullclines with the axes decrease, so that the new coexistence

equilibrium $E_3^* = (N_1^*, I_1^e, N_2^*, I_2^e)$ has lower values for the population sizes than those of the original coexistence equilibrium $E_3 = (N_1^e, N_2^e)$. Thus the endemicity of the disease changes the parameter ranges leading to the case 4 and also reduces the species sizes at the attractive coexistence equilibrium.

Note that there are three additional cases when one or both of the modified carrying capacities K_1^* and K_2^* are nonpositive. In **case 9**, when $K_1^* > 0$ and $K_2^* \le 0$ so that $r_2 \le \varepsilon_2 I_2^e$, the intrinsic per capita net growth rate r_2 is dominated by the per capita disease-induced death rate $\varepsilon_2 I_2^e$. In this case, all paths with $N_1(0) > 0$ approach the boundary equilibrium $E_1^* = (K_1^*, I_1^e, 0, I_2^e)$, where $K_1^* < K_1$. Hence, the endemicity of the disease drives species 2 to extinction and reduces the carrying capacity of species 1 to K_1^*, so it does not drive species 1 to extinction.

In **case 10**, when $K_1^* \le 0$ and $K_2^* > 0$ so that $r_1 \le \varepsilon_1 I_1^e$, the intrinsic per capita net growth rate r_1 is dominated by the per capita disease-induced death rate $\varepsilon_1 I_1^e$. In this case all paths with $N_2(0) > 0$ approach the boundary equilibrium $E_2^* = (0, I_1^e, K_2^*, I_2^e)$, where $K_2^* < K_2$. Thus the endemicity of the disease drives species 1 to extinction and reduces the carrying capacity of species 2 to K_2^*, so it does not drive species 2 to extinction.

In **case 11**, when $K_1^* \le 0$ and $K_2^* \le 0$, both intrinsic per capita net growth rates are dominated by their per capita disease-induced death rates. In this case all paths with $N_1(0) \ge 0$ and $N_2(0) \ge 0$ approach the boundary equilibrium $E_0^* = (0, I_1^e, 0, I_2^e)$. Thus the endemicity of the disease drives both species to extinction.

6. **SIS model with size-independent death rates.** If the per capita death rates are independent of size ($\chi_i = 1$) and there is no reduction in the birth rate of infectives ($f_i = 0$), then the competing species model with an SIS infectious disease (4.2) becomes

$$\begin{aligned} dN_1/dt &= [r_1(1 - (N_1 + \alpha_{12}N_2)/K_1) - \varepsilon_1 I_1]N_1, \\ dI_1/dt &= (\beta_{11}I_1 + \beta_{12}I_2)(1 - I_1) - [d_1 - \varepsilon_1 I_1 - r_1(N_1 + \alpha_{12}N_2)/K_1]I_1, \\ dN_2/dt &= [r_2(1 - (N_2 + \alpha_{21}N_1)/K_2) - \varepsilon_2 I_2]N_2, \\ dI_2/dt &= (\beta_{21}I_1 + \beta_{22}I_2)(1 - I_2) - [d_2 - \varepsilon_2 I_2 - r_2(N_2 + \alpha_{21}N_1)/K_2]I_2. \end{aligned} \tag{6.1}$$

This model is similar to the model used in Bowers and Turner [7], except that they used mass action incidence instead of frequency-dependent incidence.

Recall from section 4 that the disease must either die out in both species or remain endemic in both species. When the disease dies out, the four equilibria are the trivial equilibrium $E_0 = (0, 0, 0, 0)$, equilibria $E_1 = (K_1, 0, 0, 0)$ and $E_2 = (0, 0, K_2, 0)$, in which one species wins, and the coexistence equilibrium $E_3 = (N_1^e, 0, N_2^e, 0)$. The equilibrium sizes K_1, K_2, N_1^e, and N_2^e are the same as in the competing species model in section 3. The parameter ranges for the cases in Table 2 are the same as those for the competing species model in section 3. Thus, in **case 1** species 1 inhibits species 2 more that it inhibits itself ($\alpha_{21}/K_2 > 1/K_1$) and species 2 inhibits itself more than it inhibits species 1 ($1/K_2 > \alpha_{12}/K_1$). In this case, in the competing species model in section 3, species 1 always wins. But in this model with an SIS disease, species 1 wins if condition 1 is satisfied, where condition 1 is $\beta_{11} - d_1 + r_1 + \beta_{22} - d_2 + r_2\alpha_{21}K_1/K_2 < 0$ and $(\beta_{11} - d_1 + r_1)(\beta_{22} - d_2 + r_2\alpha_{21}K_1/K_2) > \beta_{12}\beta_{21}$. If condition 1 is not satisfied, then equilibrium $E_1 = (K_1, 0, 0, 0)$ is unstable, so that the disease may remain endemic.

In **case 2**, species 1 inhibits itself more that it inhibits species 2 ($1/K_1 > \alpha_{21}/K_2$) and species 2 inhibits species 1 more than it inhibits itself ($\alpha_{12}/K_1 > 1/K_2$). Case 2

is the symmetric analogue of case 1, in which species 2 wins in the competing species model in section 3. This also occurs in the model with an SIS disease if condition 2 is satisfied, where condition 2 is $\beta_{11} - d_1 + r_1\alpha_{12}K_2/K_1 + \beta_{22} - d_2 + r_2 < 0$ and $(\beta_{11} - d_1 + r_1\alpha_{12}K_2/K_1)(\beta_{22} - d_2 + r_2) > \beta_{12}\beta_{21}$. If condition 2 is not satisfied, then equilibrium $E_2 = (0, 0, K_2, 0)$ is unstable, so that the disease may remain endemic.

In **case 3** in Table 2, each species inhibits the other more than it inhibits itself ($\alpha_{21}/K_2 > 1/K_1$ and $\alpha_{12}/K_1 > 1/K_2$). In this case, in the competing species model in section 3, the boundary equilibria E_1 and E_2 are both stable and there is a separatrix through the unstable saddle interior equilibrium, so that solution paths on one side of the separatrix go to one boundary equilibrium and those on the other side go to the other boundary equilibrium. This also occurs in the model with an SIS disease if conditions 1 and 2 are both satisfied. If both are not satisfied, then the disease may remain endemic.

In **case 4**, each species inhibits itself more than it inhibits the other species ($1/K_1 > \alpha_{21}/K_2$ and $1/K_2 > \alpha_{12}/K_1$). In this case, both boundary equilibria E_1 and E_2 are always unstable. In the competing species model in section 3, there is a globally stable interior, coexistence equilibrium. The equilibrium $E_3 = (N_1^e, 0, N_2^e, 0)$ is locally asymptotically stable in the model with an SIS disease if condition 3 is satisfied, where condition 3 is $\beta_{11} - d_1 + r_1 + \beta_{22} - d_2 + r_2 < 0$ and $(\beta_{11} - d_1 + r_1)(\beta_{22} - d_2 + r_2) > \beta_{12}\beta_{21}$. If condition 3 is not satisfied, then the disease may remain endemic.

Table 2. Local stabilities of the disease-free equilibria

	case 1	case 2	case 3	case 4
E_0	unstable	unstable	unstable	unstable
E_1	stable if cond 1	unstable	stable if cond 1	unstable
E_2	unstable	stable if cond 2	stable if cond 2	unstable
E_3	not in region	not in region	unstable	stable if cond 3

It was shown in section 4 that the disease must either die out in both species or remain endemic in both species. Thus, when all of the equilibria without disease in Table 2 are unstable or not in the region, we expect that the disease remains endemic in both species. Equilibria with endemic disease in both species are given in Table 3, where $E_4 = (0, I_1^4, 0, I_2^4)$, $E_5 = (K_1^5, I_1^5, 0, I_2^5)$, $E_6 = (0, I_1^6, K_2^6, I_2^6)$, and $E_7 = (N_1^7, I_1^7, N_2^7, I_2^7)$. As in the model in section 5, the modified carrying capacities are $K_i^m = (r_i - \varepsilon_i I_i^m)K_i/r_i$ for $m = 5$, 6, or 7, and the interior equilibrium E_7 has coordinates given by

$$N_1^7 = (K_1^7 - \alpha_{12}K_2^7)/(1 - \alpha_{12}\alpha_{21}),$$
$$N_2^7 = (K_2^7 - \alpha_{21}K_1^7)/(1 - \alpha_{12}\alpha_{21}).$$

Conditions are given in Table 3 for the existence of the four equilibria; these are similar to cases 1 to 4 in section 5. Determining the local stability of these four endemic equilibria seems to be mathematically intractable, so that the complete global behavior is not known. However, the results in the tables give insight by providing conditions which determine when the disease dies out or remains endemic in both species. We expect that the behavior of this model with size-independent death rate would be similar to the behavior of the model with size-independent birth rate in section 5. For example, when the modified carrying capacities K_i^m are not positive, then we expect behavior similar to cases 9, 10, and 11 in section 5.

Table 3. Conditions for the existence of endemic equilibria

Equilibria	Conditions for existence
E_4	$\beta_{11} - d_1 > 0$ or $\beta_{22} - d_2 > 0$ or $(\beta_{11} - d_1)(\beta_{22} - d_2) < \beta_{12}\beta_{21}$
E_5	$K_1^5 > 0$ and condition 1 is not satisfied
E_6	$K_2^6 > 0$ and condition 2 is not satisfied
E_7	$N_1^7 > 0$, $N_2^7 > 0$, and condition 3 is not satisfied

7. **The SIRS model with logistic growth.** Consider the SIRS model for one population with size-dependent regulation, frequency-dependent incidence, and disease-related deaths, but without disease-reduced reproduction. The host population with size $N(t)$ has $X(t)$ susceptibles, $Y(t)$ infectives, and $Z(t)$ individuals who are removed with temporary immunity, so that $N(t) = X(t) + Y(t) + Z(t)$. This SIRS model with logistic growth is

$$\begin{aligned} dN/dt &= \left[a - \frac{\chi r N}{K}\right] N - \left[b + \frac{(1-\chi) r N}{K}\right] N - \varepsilon_0 Y \\ &= r(1 - N/K)N - \varepsilon_0 Y \\ dX/dt &= \left[a - \frac{\chi r N}{K}\right] N - \beta XY/N - \left[b + \frac{(1-\chi) r N}{K}\right] X + \delta Z, \\ dY/dt &= \beta XY/N - \left[\gamma + \varepsilon_0 + b + \frac{(1-\chi) r N}{K}\right] Y, \\ dZ/dt &= \gamma Y - \left[\delta + b + \frac{(1-\chi) r N}{K}\right] Z, \end{aligned} \tag{7.1}$$

where a is the intrinsic per capita birth rate for susceptibles, b is the intrinsic natural per capita death rate, $r = a - b$ is the positive intrinsic per capita net growth rate, χ is the convex combination constant with $0 \le \chi \le 1$, K is the environmental carrying capacity, ε_0 is the per capita disease-related death rate, γ is the per capita recovery rate, and δ is the per capita rate of loss of temporary infection-induced immunity. This SIRS model with different notation was studied by Gao and Hethcote [11]. As in the SIS model, it suffices to analyze solution paths in the subset of the first octant of XYZ space with $N = X + Y + Z \le K$.

The model (7.1) can be reduced to the following system of three equations for the host population size, the infective fraction I, and the removed fraction R by using $I = Y/N$ and $R = Z/N$, and $X/N = 1 - I - R$.

$$\begin{aligned} dN/dt &= [r(1 - N/K) - \varepsilon_0 I]N, \\ dI/dt &= \beta I(1 - I - R) - [\gamma + a + \varepsilon_0 - \varepsilon_0 I - \chi r N/K]I, \\ dR/dt &= \gamma I - [\delta + a - \varepsilon_0 I - \chi r N/K]R. \end{aligned} \tag{7.2}$$

This model is well posed in the positively invariant region

$$D = \{(N, I, R) : 0 \le N \le K, I \ge 0, R \ge 0, I + R \le 1\}.$$

This SIRS model has the same threshold quantities $\mathbf{R}_2 = \beta/(\gamma + a + \varepsilon_0 - \chi r)$ and $\mathbf{R}_1 = \beta/(\gamma + a + \varepsilon_0)$ as the SIS model (2.3), but the net growth threshold is now

$$\phi = \frac{r}{\varepsilon_0[1 - (\gamma + b + \varepsilon_0)/\beta]} \left(1 + \frac{\gamma}{\delta + b}\right).$$

The asymptotic behaviors are summarized in the table below. All results in the table were proved globally when the birth rate is size-independent ($\chi = 0$). For

$\chi > 0$, global stability was proved in case 1, and local asymptotic stabilities of the attractive equilibria were proved in the other cases.

Table 4. Asymptotic behaviors for the SIRS logistic model

Cases	Asymptotic behavior for large time
case 1: $1 \geq \mathbf{R}_2 \geq \mathbf{R}_1$	$N(0) > 0 \Rightarrow (N, I, R) \to E_2 = (K, 0, 0)$
case 2: $\mathbf{R}_2 > 1 \geq \mathbf{R}_1$	$N, I > 0$ *at* $0 \Rightarrow (N, I, R) \to E_4 = (N_4, I_4, R_4)$
case 3: $\mathbf{R}_1 > 1$ & $\phi < 1$	$I(0) > 0 \Rightarrow (N, I, R) \to E_3 = (0, I_3, R_3)$
case 4: $\mathbf{R}_1 > 1$ & $\phi > 1$	$N, I > 0$ *at* $0 \Rightarrow (N, I, R) \to E_4 = (N_4, I_4, R_4)$

8. **Competing species with an SIRS disease.** The SIRS model for two competing species with size-dependent regulation, frequency-dependent incidence, and disease-related deaths, but without disease-reduced reproduction is

$$
\begin{aligned}
dN_1/dt &= r_1(1-(N_1+\alpha_{12}N_2)/K_1)N_1 - \varepsilon_{10}Y_1, \qquad (8.1)\\
dX_1/dt &= (a_1 - \frac{\chi_1 r_1(N_1+\alpha_{12}N_2)}{K_1})N_1 - (\beta_{11}Y_1/N_1 + \beta_{12}Y_2/N_2)X_1\\
&\quad - (b_1 + \frac{(1-\chi_1)r_1(N_1+\alpha_{12}N_2)}{K_1})X_1 + \delta_1 Y_1,\\
dY_1/dt &= (\beta_{11}Y_1/N_1 + \beta_{12}Y_2/N_2)X_1\\
&\quad - (\gamma_1 + b_1 + \frac{(1-\chi_1)r_1(N_1+\alpha_{12}N_2)}{K_1} + \varepsilon_{10})Y_1,\\
dZ_1/dt &= \gamma_1 Y_1 - \left[\delta_1 + b_1 + \frac{(1-\chi_1)r_1(N_1+\alpha_{12}N_2)}{K_1}\right] Z_1,\\
dN_2/dt &= r_2(1-(N_2+\alpha_{21}N_1)/K_2)N_2 - \varepsilon_{20}Y_2,\\
dX_2/dt &= (a_2 - \frac{\chi_2 r_2(N_2+\alpha_{21}N_1)}{K_2})N_2 - (\beta_{21}Y_1/N_1 + \beta_{22}Y_2/N_2)X_2\\
&\quad - (b_2 + \frac{(1-\chi_2)r_2(N_2+\alpha_{21}N_1)}{K_2})X_2 + \delta_2 Y_2,\\
dY_2/dt &= (\beta_{21}Y_1/N_1 + \beta_{22}Y_2/N_2)X_2\\
&\quad - (\gamma_2 + b_2 + \frac{(1-\chi_2)r_2(N_2+\alpha_{21}N_1)}{K_2} + \varepsilon_{20})Y_2,\\
dZ_2/dt &= \gamma_2 Y_2 - \left[\delta_2 + b_2 + \frac{(1-\chi_2)r_2(N_2+\alpha_{21}N_1)}{K_2}\right] Z_2,
\end{aligned}
$$

where the variables and parameter values are similar to those in the one-population model. When δ_1 and δ_2 are zero, the immunity is permanent, so that the model is of SIR type.

The system (8.1) can be reduced to the following system of six equations for the host population sizes, the infective fractions, and the removed fractions by using

$I_i = Y_i/N_i$ and $R_i = Z_i/N_i$, and $X_i/N_i = 1 - I_i - R_i$:

$$\begin{aligned} dN_1/dt &= [r_1(1-(N_1+\alpha_{12}N_2)/K_1) - \varepsilon_{10}I_1]N_1, \\ dI_1/dt &= (\beta_{11}I_1+\beta_{12}I_2)(1-I_1-R_1) \\ &\quad - [d_1 - \varepsilon_{10}I_1 - \chi_1 r_1(N_1+\alpha_{12}N_2)/K_1]I_1, \\ dR_1/dt &= \gamma_1 I_1 - [\delta_1 + a_1 - \varepsilon_{10}I_1 - \chi_1 r_1(N_1+\alpha_{12}N_2)/K_1]\,R_1, \\ dN_2/dt &= [r_2(1-(N_2+\alpha_{21}N_1)/K_2) - \varepsilon_{20}I_2]N_2, \\ dI_2/dt &= (\beta_{21}I_1+\beta_{22}I_2)(1-I_2-R_2) \\ &\quad - [d_2 - \varepsilon_{20}I_2 - \chi_2 r_2(N_2+\alpha_{21}N_1)/K_2]I_2, \\ dR_2/dt &= \gamma_2 I_2 - [\delta_2 + a_2 - \varepsilon_{20}I_2 - \chi_2 r_2(N_2+\alpha_{21}N_1)/K_2]\,R_2, \end{aligned} \tag{8.2}$$

where $r_i = a_i - b_i$, and $d_i = \gamma_i + a_i + \varepsilon_{i0}$. The system (8.2) is analyzed in the six-dimensional region

$$D = \{(N_1, I_1, R_1, N_2, I_2, R_2) : 0 \le N_i \le K_i, I_i \ge 0, R_i \ge 0, I_i + R_i \le 1\}. \tag{8.3}$$

This model with frequency-dependent incidence is similar to the model of Tompkins, White, and Boots [28], but they used mass action incidence and size-independent death rates.

9. **SIRS model with size-independent birth rates.** If the per capita birth rates are independent of the population sizes ($\chi_i = 0$), then the competing species model with an SIRS infectious disease (8.2) becomes

$$\begin{aligned} dN_1/dt &= [r_1(1-(N_1+\alpha_{12}N_2)/K_1) - \varepsilon_{10}I_1]N_1, \\ dI_1/dt &= (\beta_{11}I_1+\beta_{12}I_2)(1-I_1-R_1) - [d_1 - \varepsilon_{10}I_1]I_1, \\ dR_1/dt &= \gamma_1 I_1 - [\delta_1 + a_1 - \varepsilon_{10}I_1]\,R_1, \\ dN_2/dt &= [r_2(1-(N_2+\alpha_{21}N_1)/K_2) - \varepsilon_{20}I_2]N_2, \\ dI_2/dt &= (\beta_{21}I_1+\beta_{22}I_2)(1-I_2-R_2) - [d_2 - \varepsilon_{20}I_2]I_2, \\ dR_2/dt &= \gamma_2 I_2 - [\delta_2 + a_2 - \varepsilon_{20}I_2]\,R_2. \end{aligned} \tag{9.1}$$

In this case the equations for I_1, R_1, I_2, and R_2 are independent of N_1 and N_2, so that this four-dimensional subsystem uncouples from the original six-dimensional system (9.1). The feasible region for the subsystem is $\hat{D} = \{(I_1, R_1, I_2, R_2) : I_i \ge 0, R_i \ge 0, I_i + R_i \le 1\}$. For equilibrium points of the subsystem, it is easy to see that if one of the equilibrium values I_i^e is zero, then $R_i^e = 0$, $I_j^e = 0$, and $R_j^e = 0$. Thus, the disease must either die out in both species or remain endemic in both species.

The Jacobian at the equilibrium $\hat{E}_0 = (0,0,0,0)$ has two negative eigenvalues, $-(\delta_1 + a_1)$, $-(\delta_2 + a_2)$, and two eigenvalues that are roots of the quadratic equation

$$\lambda^2 - (\beta_{11} - d_1 + \beta_{22} - d_2)\lambda + (\beta_{11} - d_1)(\beta_{22} - d_2) - \beta_{12}\beta_{21} = 0.$$

This quadratic equation is the same as the characteristic equation for the two-dimensional SIS model in section 5, so that it has the same modified reproduction number $\mathbf{R}_1$ given by (5.3). Thus the equilibrium $\hat{E}_0 = (0,0,0,0)$ is locally asymptotically stable if $\mathbf{R}_1 \le 1$. For solutions starting near $\hat{E}_0$, the limiting differential equations for N_1 and N_2 are the competing species model (3.1). Hence the last result in the appendix implies that for $\mathbf{R}_1 \le 1$, the local asymptotic behavior of the system (9.1) is that the disease dies out and the behavior of the two species corresponds to one of the four competing species cases given in section 3.

In looking for interior equilibria for this model, we have $R_i = \gamma_i I_i/(\delta_i + a_i - \varepsilon_{i0} I_i)$, so that I_1 and I_2 must satisfy

$$I_2 = \frac{I_1\,(d_1 - \varepsilon_{10} I_1)}{\beta_{12}\,[1 - I_1 - \gamma_1 I_1/(\delta_1 + a_1 - \varepsilon_{10} I_1)]} - \frac{\beta_{11} I_1}{\beta_{12}}, \tag{9.2}$$

$$I_1 = \frac{I_2\,(d_2 - \varepsilon_{20} I_2)}{\beta_{21}\,[1 - I_2 - \gamma_2 I_2/(\delta_2 + a_2 - \varepsilon_{20} I_2)]} - \frac{\beta_{22} I_2}{\beta_{21}}.$$

Unfortunately, these curves do not have the nice monotonic behavior that was obtained for the SIS model (5.1) with size-independent birth rates, so that there can be more than one interior equilibrium point. Analysis of the global behavior of this model seems to be mathematically intractable.

10. **Discussion.** In our analyses of competing species models with an infectious disease of SIS, SIR, or SIRS type, in which the transmission is governed by the frequency-dependent incidence, we obtain the classic endemic model behavior. Specifically, the disease either dies out in both species or remains endemic in both species. For the SIS model in section 5 with birth rates that are independent of species population sizes, a modified reproduction number $\mathbf{R}_1$ determines the asymptotic behavior, so that the disease dies out if $\mathbf{R}_1 \leq 1$ and approaches a globally attractive endemic equilibrium if $\mathbf{R}_1 > 1$. When the disease dies out, the four usual competing species outcomes (species 1 wins; species 2 wins; stable coexistence; winner depends on initial conditions) are possible. These four outcomes with different equilibrium values are also possible when the disease remains endemic, but there is another possible outcome, in which the disease-related deaths and disease-reduced reproductions for the endemic disease drive one or both populations to extinction. Inequalities involving the parameters are given that determine which outcome occurs in the competition when the disease remains endemic.

The inequalities for the seven endemic cases in section 5 are not the same as the inequalities for the four disease-free cases. Thus, in our models a shared disease not only can change the equilibrium values, but also can change the outcome of the competition. For example, a disease could change the outcome from the winner being dependent on the initial conditions to one species winning. Or a disease that is more harmful to a superior competitor could allow the coexistence or dominance of an otherwise inferior competitor. Bowers and Turner [7] cite the following two instances in which a disease has a greater effect on one of the competing species, so that the winning species is switched. Park [24] found that the flour beetle *Tribolium castaneum* usually wins a competition in a culture with the flour beetle *Tribolium confusum*. But the sporozoan parasite, *Adelina triboli*, has a greater negative impact on *Tribolium castaneum*, so if the shared culture contains the parasite, then *Tribolium confusum* usually wins. Sibma et al. [25] also found winner reversal in competing oat and barley plants when a root-feeding nematode, *Heterodera amenae*, was introduced. In the case of introduced grey squirrels invading the territory of native red squirrels in the United Kingdom, the modeling of Tompkins, White, and Boots [28] suggests that the parapoxvirus, which has a harmful effect on red squirrels, has sped the replacement of the red squirrels by grey squirrels.

One important conclusion of this paper is that the form of the disease incidence strongly affects the asymptotic behavior of a competing species model. In the competing species model of Bowers and Turner [7] formulated using the mass action incidence, the disease could remain endemic in one species but die out in the other

species, even though the contact rates between the two species were positive. This behavior does not occur in our SIS competing species models formulated with the frequency-dependent (standard) incidence, since the disease either dies out in both species or remains endemic in both species. Because the contact rates β_{12} and β_{21} between the species are positive, it intuitively seems reasonable that infectives in one species would infect some susceptibles in the other species, so that endemicity of the disease in one species would imply endemicity in the other species. The form of the incidence used can also determine whether periodic solutions occur in a model. Periodic solutions were found numerically by Venturino [30] in an SIS competing species model formulated with the mass action incidence for the disease transmission. In contrast, our SIS competing species model with the frequency-dependent incidence in section 5 never has periodic solutions.

Appendix. Stability results using limit systems. Consider the following systems:

$$\dot{x} = f(t, x), \tag{A1}$$

$$\dot{y} = g(y), \tag{A2}$$

where f and g are continuous and locally Lipschitz in x in R^n, and solutions exist for all positive time. Equation (A1) is called asymptotically autonomous with limit system (A2) if $f(t, x) \to g(x)$ as $t \to \infty$ uniformly for x in R^n. Thieme [27] considered the situation in which e is a locally asymptotically stable equilibrium of (A2) and ω is the ω-limit set of a forward-bounded solution $x(t)$ of (A1). If ω contains a point y_0 such that the solution of (A2) with $y(0) = y_0$ converges to e as $t \to \infty$, then $\omega = \{e\}$, that is, $x(t) \to e$ as $t \to \infty$. Thus, it follows that if solutions of the system (A1) are bounded and the equilibrium e of the limit system (A2) is globally asymptotically stable, then any solution $x(t)$ of the system (A1) satisfies $x(t) \to e$ as $t \to \infty$.

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