Pergamon

Mathl. Comput. Modelling Vol. 23, No. 4, pp. 67–91, 1996

0895-7177/96 $15.00 + 0.00

S0895-7177(96)00004-0

Sex Ratio Features of Two-Group SIR Model for Asymmetric Transmission of Heterosexual Disease

C. KOIDE
Department of Mathematics, Faculty of Science
Hiroshima University, Kagamiyama 1-3-1
Higashi-Hiroshima, Hiroshima, 739 Japan

H. SENO*
Information and Computer Sciences
Nara Women's University, Kita-uoya-nishi-machi, Nara, 630 Japan
seno@ics.nara-wu.ac.jp

(Received and accepted April 1995)

Abstract—We consider the SIR model with two sexual groups to discuss the effect of the asymmetricity in the transmission dynamics of STDs. It is shown that the asymmetric transmission between two groups results in the asymmetric structure of infective populations.

Keywords—Epidemics, SIR model, Sex ratio.

1. INTRODUCTION

In the modern communities, many diseases once rampant have virtually disappeared, except perhaps for some sporadic cases. However, the others like influenza, poliomyelitis, infective hepatitis, etc., not only continue to defy prevention but still lack the specifically distinct cures. All methods of study are therefore welcome for them, whether clinical, biological, ecological or mathematical.

Today, the AIDS/HIV infection is one of the most serious contemporary social problems. Many mathematical modelling analyses are challenging the problem, and some could be expected to play an important role for understanding its outbreaking prevalence. It appears that there exist many difficult points for demographic and mathematical researchers on the dynamics of AIDS/HIV transmission. One of them is that, for example, people with HIV have a wide and variable range of incubation periods, 4–15 years [1,2], and there has not yet been found any successful preventive treatment or cure. In addition, its dynamics must reflect the social structure involving the infection: the number of drug users, the frequency of homosexual contacts, the degree of sexual activities, the number of spouses, the frequency of medical accidents, etc. Moreover, there are few confidentially sufficient statistical data in any country besides Japan, since AIDS/HIV infection appears very serious just in recent years, and since many infectives tend to hide their infection.

The authors greatly thank M. Kakehashi and Y. Takeuchi for their helpful comments to complete this paper.
*Author to whom all correspondence should be addressed.

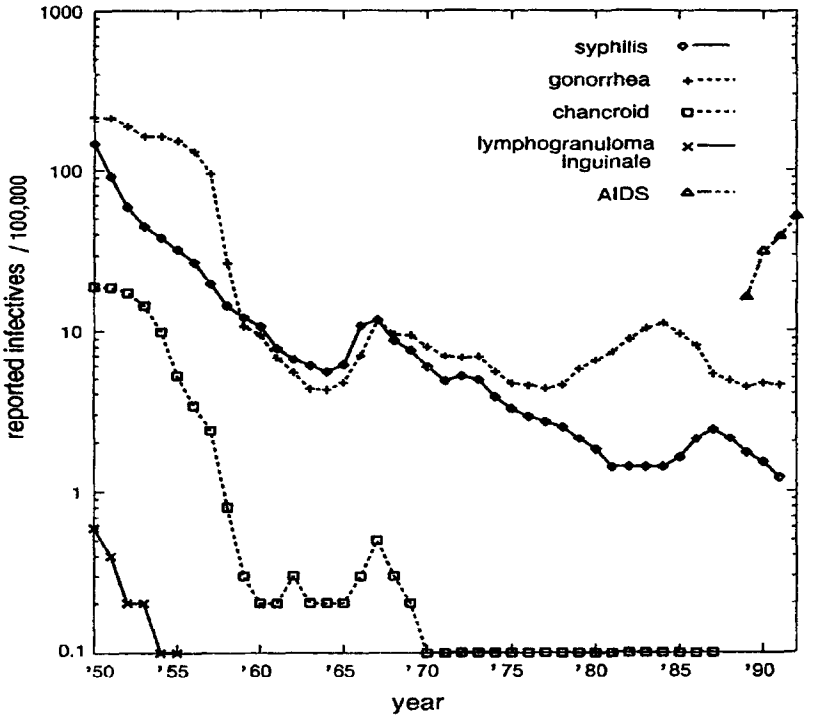


Figure 1. Reported variation of infectives for STDs in Japan [4]. Those numbers of reported patients per 100,000 are plotted.

On the other hand, in regards to other *sexually transmitted diseases* (STDs), many records remain through the past. In Japan, the first time that syphilis was reported was in 1512 [3]. After World War II, various institutions were established, and the law obliges infectives to report infection with STDs. So statistical data has been accumulated since 1949 (Figure 1) [4]. The number of infectives could reflect some developments of social background. For a striking example, the law to prohibit prostitution was established in 1958. Although some STDs were very serious in the past, almost all of them are now cured without mortality and vertical transmission.

In mathematical biology, the study on the epidemic models is one of the most successful fields because we have been able to apply various statistical data, especially with regard to human epidemics. Indeed, the history of mathematical models for infectious diseases is quite long, traced back to at least the 1920's. In that period, the work by Kermack and MacKendrick [5] appeared. In the Kermack-MacKendrick model, total population is presumed to be constant in size and to be divided into three classes. There are infectives, I who can pass on the disease to susceptibles, S who have not yet contracted the disease. The remaining class consists of members R who have been infected and have become unable to transmit the disease for some reasons, for instance, because of isolation from the rest of population. In the Kermack-MacKendrick system, one of the most basic SIR models is

$$\begin{aligned}\frac{dS(t)}{dt} &= -\lambda SI,\\ \frac{dI(t)}{dt} &= \lambda SI - \gamma I,\\ \frac{dR(t)}{dt} &= \gamma I.\end{aligned}$$

To consider the nature of epidemic dynamics by SIR model, there must be initially some infectious and some susceptible population for infection, so $I(0) > 0$ and $S(0) > 0$. The basic idea of the Kermack-MacKendrick model is that the susceptibles become infected at a rate proportional to the number of contacts between individuals of S and I, assuming that the contact depends only

on the population size of each class, which can be regarded, for example, as the uniform mixing of the whole population. The rate at which individuals become unable to transmit the disease is assumed proportional to the population size of infectives. It could be regarded as to represent some kind of average of the process in which particular individuals take different lengths of time to reach the state in which they neither contract nor pass on the infection.

Recently, Beretta and Capasso [6,7] mathematically studied some multigroup SIR models. Epidemic process among heterogeneous populations can be considerably contributed to asymmetric relationships among them. Such asymmetric relationships corresponds to, for example, some social structures or some genetic factors. In this paper, we consider two sexual groups: one corresponds to male group and another to female. We analyze an SIR model with two sexual groups to consider some qualitative natures of STDs, and also try to discuss some real cases of nonmortal STDs. In our modelling analysis, the sex ratio appears to be one of complications in the study of such two sexual models. Further, it is interesting that slight differences in the dynamic structure of modelling lead to different results.

2. SIR MODEL FOR ASYMMETRIC TRANSMISSION OF HETEROSEXUAL DISEASE

2.1. Assumptions and Modelling

We make the following assumptions on the epidemic dynamics for our modelling:

(i) There are two sexual groups, whose total populations are temporally variable due to the recruitment of newborns and death. Group 1 and group 2 at time t have, respectively, male population $N_1(t)$ and female one $N_2(t)$.

(ii) Each of populations N_1 and N_2 is divided into three classes:
   (a) Susceptible class $S_i(t)$: subpopulation capable of contracting disease and becoming infective.
   (b) Infective class $I_i(t)$: subpopulation capable of transmitting disease to susceptible subpopulation.
   (c) Removed class $R_i(t)$: subpopulation which has contracted disease, and died or recovered, permanently immune or been isolated, so as to be unable to contract and transmit the disease.

(iii) Newborn population is supplied by female population. The sex ratio of newborn is generally assumed constant as follows:

$$\frac{\{\text{male newborn}\}}{\{\text{female newborn}\}} = \frac{k}{1-k} \qquad 0 < k < 1.$$

(iv) All newborns are susceptible without any vertical transmission. Birth rate depends in general on the class of the mother, denoted by B_S, B_I and B_R, respectively, for S, I and R classes.

(v) The death process is exponential decay with the rate $\mu_i > 0$ $(i = 1, 2)$.

(vi) A susceptible individual of S_i becomes infected at a rate proportional to the frequency of contacts with infectives of both groups, which are given by the multiplication $S_i I_j$ $(i, j = 1, 2)$. Infective individuals of group j $(j = 1, 2)$ transmit the disease to susceptible individuals of group i $(i = 1, 2)$ with the transmission rate $\lambda_{ij} \geq 0$.

(vii) For group i, the transition of individuals from the class I_i to the class R_i is exponential with the rate $\gamma_i > 0$ $(i = 1, 2)$.

(viii) All parameters μ_i, λ_{ij} and γ_i $(i, j = 1, 2)$ are time-independent constants.

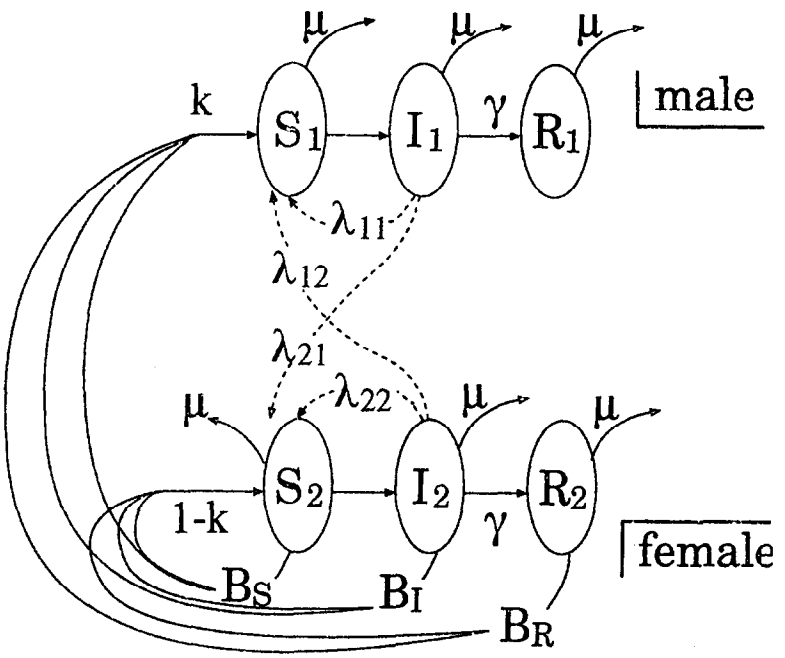


Figure 2. Scheme of population dynamics for SIR model with two sexual groups.

2.2. Basic Model System

With the above assumptions, we consider the following system (Figure 2):

$$
\begin{cases}
\dfrac{dS_1(t)}{dt} = k\left(B_S S_2(t) + B_I I_2(t) + B_R R_2(t)\right) - \left(\lambda_{11} I_1(t) + \lambda_{12} I_2(t)\right) S_1(t) - \mu S_1(t) \\
\dfrac{dI_1(t)}{dt} = \left(\lambda_{11} I_1(t) + \lambda_{12} I_2(t)\right) S_1(t) - (\mu + \gamma) I_1(t) \\
\dfrac{dR_1(t)}{dt} = \gamma I_1(t) - \mu R_1(t) \\
\dfrac{dS_2(t)}{dt} = (1-k)\left(B_S S_2(t) + B_I I_2(t) + B_R R_2(t)\right) - \left(\lambda_{21} I_1(t) + \lambda_{22} I_2(t)\right) S_2(t) - \mu S_2(t) \\
\dfrac{dI_2(t)}{dt} = \left(\lambda_{21} I_1(t) + \lambda_{22} I_2(t)\right) S_2(t) - (\mu + \gamma) I_2(t) \\
\dfrac{dR_2(t)}{dt} = \gamma I_2(t) - \mu R_2(t).
\end{cases}
\tag{1}
$$

The total population $N_i(t)$ $(i = 1, 2)$ is defined by $N_i(t) = S_i(t) + I_i(t) + R_i(t)$.

We consider the population which has a temporally variable size. The sex ratio of newborns is in general assumed to be $k : 1 - k$ $(0 < k < 1)$, and the birth rate is commonly constant B. For convenience, male birth rate is denoted by B_1, and female one is by B_2:

$$B_1 \equiv kB, \qquad B_2 \equiv (1-k)B.$$

We consider only the case without the homosexual transmission, when $\lambda_{11} = \lambda_{22} = 0$. For instance, in regards the general STDs in Japan, there is insignificant infection owed to it, compared with heterosexual transmission. To consider the dependence of results on the model structure, we comparably analyze the following three systems depending on which female classes could contribute to the recruitment of newborns. Actually, it is known that once a female contracts certain kinds of STDs, for example, the papilloma virus, it is highly probable she becomes sterile.

MODEL I. With birth from every female class:

$$\begin{cases} \dfrac{dS_1}{dt} = B_1(S_2 + I_2 + R_2) - \lambda_{12} I_2 S_1 - \mu S_1 \\ \dfrac{dI_1}{dt} = \lambda_{12} I_2 S_1 - (\mu + \gamma) I_1 \\ \dfrac{dR_1}{dt} = \gamma I_1 - \mu R_1 \end{cases}$$

$$\begin{cases} \dfrac{dS_2}{dt} = B_2(S_2 + I_2 + R_2) - \lambda_{21} I_1 S_2 - \mu S_2 \\ \dfrac{dI_2}{dt} = \lambda_{21} I_1 S_2 - (\mu + \gamma) I_2 \\ \dfrac{dR_2}{dt} = \gamma I_2 - \mu R_2. \end{cases} \tag{2}$$

MODEL II. With birth from S and I classes:

$$\begin{cases} \dfrac{dS_1}{dt} = B_1(S_2 + I_2) - \lambda_{12} I_2 S_1 - \mu S_1 \\ \dfrac{dI_1}{dt} = \lambda_{12} I_2 S_1 - (\mu + \gamma) I_1 \\ \dfrac{dR_1}{dt} = \gamma I_1 - \mu R_1 \end{cases}$$

$$\begin{cases} \dfrac{dS_2}{dt} = B_2(S_2 + I_2) - \lambda_{21} I_1 S_2 - \mu S_2 \\ \dfrac{dI_2}{dt} = \lambda_{21} I_1 S_2 - (\mu + \gamma) I_2 \\ \dfrac{dR_2}{dt} = \gamma I_2 - \mu R_2. \end{cases} \tag{3}$$

MODEL III. With birth only from S class:

$$\begin{cases} \dfrac{dS_1}{dt} = B_1 S_2 - \lambda_{12} I_2 S_1 - \mu S_1 \\ \dfrac{dI_1}{dt} = \lambda_{12} I_2 S_1 - (\mu + \gamma) I_1 \\ \dfrac{dR_1}{dt} = \gamma I_1 - \mu R_1 \end{cases}$$

$$\begin{cases} \dfrac{dS_2}{dt} = B_2 S_2 - \lambda_{21} I_1 S_2 - \mu S_2 \\ \dfrac{dI_2}{dt} = \lambda_{21} I_1 S_2 - (\mu + \gamma) I_2 \\ \dfrac{dR_2}{dt} = \gamma I_2 - \mu R_2. \end{cases} \tag{4}$$

3. ANALYSIS

In our mathematical analysis of those models, we focus on the behaviour of the infective population at the early stage, and at the stationary state, to consider how the sex ratio of infectives is related to epidemic parameters, especially to the asymmetric transmission rates.

3.1. Invasion of Heterosexual Disease

Could the disease successfully invade and persist after a few infectives come into the group which formerly had no infectives? In this section, we consider this question by examining some phases of the development of an infective population.

3.1.1. Early spread of disease

At the early stage of invasion of disease, *the early spread of disease* is defined as follows.

DEFINITION 1 (EARLY SPREAD OF DISEASE). *Early spread of disease in group i is defined for the case when, with sufficiently small initial infectives within the group, the infective population increases just after its initial invasion.*

From the definition, the early spread of disease within group i for the SIR model can be mathematically defined to occur, iff the following condition is satisfied:

$$\frac{dI_i}{dt}(0) > 0, \qquad (i = 1, 2). \tag{5}$$

Let us consider the condition for the early spread of disease, defined in the above for the SIR model. We assume that $I_1(0)$ and $I_2(0)$ are sufficiently small. From (5), we get another equivalent inequality $\widehat{R_i} > 1$, where $\widehat{R_i}$ is defined as a nondimensional value, given commonly among three models as follows:

$$\widehat{R_1} = S_1(0)\frac{\lambda_{12}}{\mu + \gamma} \qquad \text{for group 1,} \tag{6}$$

$$\widehat{R_2} = S_2(0)\frac{\lambda_{21}}{\mu + \gamma} \qquad \text{for group 2.} \tag{7}$$

Iff $\widehat{R_i} > 1$, $\frac{dI_i}{dt}$ is positive at $t = 0$, and early spread of the disease occurs. $\widehat{R_i}$ is called *the basic reproductive rate* of epidemic disease within group i [8]. $\widehat{R_i}$ corresponds to the expected value of susceptibles that the unit infective population can transmit during its expected infective period until transition to the removed class. If the expected number is more than the unity, then the disease will spread, since the infectives are expected to gain new recruits.

From (6) and (7), the larger the summation is of the natural death rate μ and the recovery rate γ, the harder it is for early spread to occur, because $\widehat{R_i}$ becomes smaller. It is interesting that even if we can increase the recovery rate γ, $\widehat{R_i}$ may not become smaller when the natural death rate decreases. This can be often observed in medically advanced countries. When the initial susceptible population $S_i(0)$ or the transmission rate λ_{ij} is large, $\widehat{R_i}$ is large and early spread easily occurs. Since $\widehat{R_1}$ is in general not equal to $\widehat{R_2}$ except when $S_1(0)\lambda_{12} = S_2(0)\lambda_{21}$, it is likely that one of two groups takes the early spread and another does not (for instance, see Figure 3).

On the other hand, again from (5), the threshold $\widehat{S_i}$ for the initial susceptible population $S_i(0)$ in terms of the early spread within group i can be also obtained

$$\widehat{S_i} = \frac{\mu + \gamma}{\lambda_{ij}}. \tag{8}$$

If $S_i(0) > \widehat{S_i}$, we have the early spread within group i. Since $S_i(0) \approx N_i(0)$, (8) can be regarded as the threshold for the total population of group i, in order to cause the initial spread of disease within group i. Further, in the same way, the threshold $\widehat{\lambda_i}$ for the transmission rate λ_{ij} can also be defined

$$\widehat{\lambda_i} = \frac{\mu + \gamma}{S_i(0)}.$$

When $\lambda_{ij} > \widehat{\lambda_i}$, early spread occurs within group i.

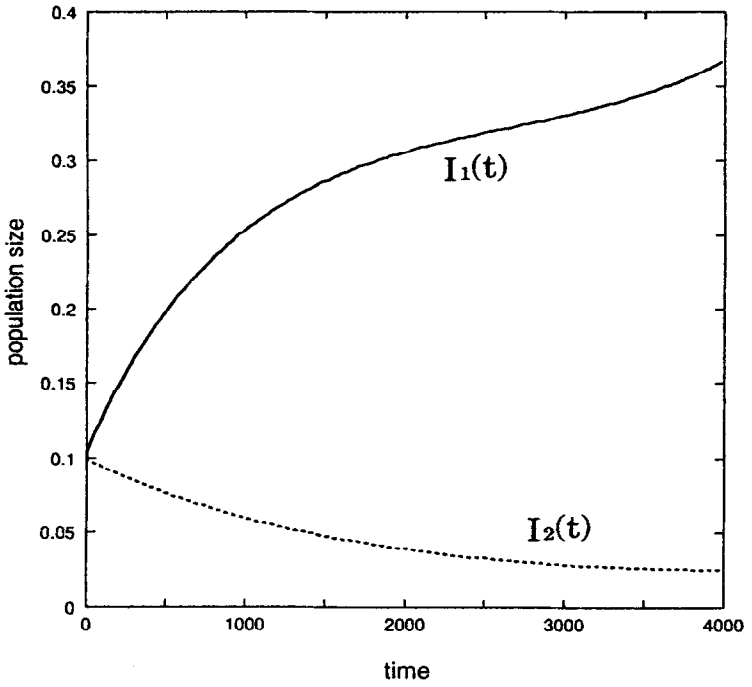


Figure 3. A behaviour of I_i $(i = 1, 2)$. $\mu = 0.02$, $\gamma = 0.0333$, $B = 0.106$, $k = 0.50$, $\lambda_{12} = 0.30$, $\lambda_{21} = 0.90 \times 10^{-3}$, $S_1(0) = S_2(0) = 1.0$, $I_1(0) = I_2(0) = 0.1$.

Along the same lines, if $I_1(0) \ll 1$, $I_2(0) \ll 1$ and $I_1(0)/I_2(0) \approx q$ (q: constant), we can estimate the basic reproductive rate for the total population

$$\widehat{R}_0 = \frac{\lambda_{12} S_1(0) + \lambda_{21} S_2(0)}{(1+q)(\mu+\gamma)}. \tag{9}$$

Iff $\widehat{R}_0 > 1$, the total infective population increases at the initial stage of invasion.

3.1.2. Persistence of disease

We consider if the disease can persist after the invasion. We define *the persistence of disease* as follows.

DEFINITION 2 (PERSISTENCE OF DISEASE). *Persistence of disease is defined for the case when infectives can exist at any time once the invasion is successful.*

There are mathematically three distinct cases of the persistence with positive infective population: nontrivial positive equilibrium, periodic solution, and chaotic behaviour. When infectives disappear, we can distinguish two types: one is the case when the total population becomes extinct, another is when the susceptibles come to occupy the whole group, that is, when the invasion is not completely successful.

We can analyze the local stability of the equilibrium states without any infectives. If all of them are unstable, the infectious disease can necessarily persist after the invasion. If the disease free equilibrium is locally stable, it can be regarded as to imply that infectious disease with initially very small infective population is eventually excluded. Another standing point of the analysis is the existence of the nontrivial equilibrium state with infectives. With what condition of parameters could it exist? We can argue the condition which is necessary for the persistence of disease with an equilibrium state.

MODEL I. From (2), we obtain the following closed linear system of ordinary differential equations for $N_1(t)$ and $N_2(t)$:

$$\begin{aligned} \frac{dN_1}{dt} &= B_1 N_2 - \mu N_1, \\ \frac{dN_2}{dt} &= (B_2 - \mu) N_2. \end{aligned} \tag{10}$$

Solving this system, we can get

$$N_1(t) = \left\{ N_1(0) - \frac{k}{1-k} N_2(0) \right\} e^{-\mu t} + \frac{k}{1-k} N_2(0) e^{(B_2 - \mu)t}, \tag{11}$$

$$N_2(t) = N_2(0) e^{(B_2 - \mu)t}. \tag{12}$$

Therefore, iff $B_2 < \mu$, $(S_1^*, S_2^*, I_1^*, I_2^*, R_1^*, R_2^*) = (0,0,0,0,0,0)$ is the stable equilibrium for any initial condition.

When $B_2 = \mu$, since the female population is assumed constant N_2, we can obtain the explicit solution for N_1 as follows:

$$N_1(t) = \left\{ N_1(0) - \frac{k}{1-k} N_2 \right\} e^{-\mu t} + \frac{k}{1-k} N_2. \tag{13}$$

So, it can be immediately seen that, as $t \to \infty$, the male population N_1 asymptotically approaches to

$$N_1^* = \frac{k}{1-k} N_2.$$

Thus, in the stationary state, the sex ratio of the total population is the same with that of newborn. The local stability of the disease free trivial equilibrium $(S_1^*, S_2^*, I_1^*, I_2^*) = ((k/(1-k))N_2, N_2, 0, 0)$ is determined by the eigenvalues of the following Jacobian matrix:

$$J\left(\frac{k}{1-k} N_2, N_2, 0, 0 \right) = \begin{pmatrix} -\mu & B_1 & 0 & -\lambda_{12} \dfrac{k}{1-k} N_2 \\ 0 & 0 & -\lambda_{21} N_2 & \mu \\ 0 & 0 & -(\mu + \gamma) & \lambda_{12} \dfrac{k}{1-k} N_2 \\ 0 & 0 & \lambda_{21} N_2 & -(\mu + \gamma) \end{pmatrix}. \tag{14}$$

From the eigenvalue analysis, the trivial equilibrium is unstable, iff

$$(\mu + \gamma)^2 - \lambda_{12} \lambda_{21} \frac{k}{1-k} N_2^2 < 0. \tag{15}$$

If the nontrivial equilibrium exists, from (2), it turns out to satisfy the following:

$$S_1^* = \frac{(\lambda_{21} I_1^* + \mu)(\mu + \gamma)^2}{\mu \lambda_{12} \lambda_{21} N_2}, \tag{16}$$

$$S_2^* = \frac{(\mu + \gamma)^2}{\lambda_{12} \lambda_{21} S_1^*}, \tag{17}$$

$$I_1^* = \frac{\mu \{ k \lambda_{12} \lambda_{21} N_2^2 - (1-k)(\mu + \gamma)^2 \}}{(1-k)(\mu + \gamma) \lambda_{21} (\mu + \gamma + \lambda_{12} N_2)}, \tag{18}$$

$$I_2^* = \frac{(\mu + \gamma) I_1^*}{\lambda_{12} S_1^*}, \tag{19}$$

$$R_1^* = \frac{\gamma}{\mu} I_1^*, \tag{20}$$

$$R_2^* = \frac{\gamma}{\mu} I_2^*. \tag{21}$$

Table 1. Feasible asymptotic states for Model I with $\mu < B_2$ and for Model II with $\mu + \gamma < B_2$. The symbol * indicates an unknown finite value.

	S_1	S_2	I_1	I_2	R_1	R_2
(a)	∞	∞	∞	∞	∞	∞
(b)	*	∞	∞	∞	∞	∞
(c)	∞	*	∞	∞	∞	∞
(d)	*	*	∞	∞	∞	∞

The necessary and sufficient condition for the existence of the above nontrivial equilibrium, that is, for $0 < S_i^* < N_i^*$ $(i = 1, 2)$, is equivalent to (15).

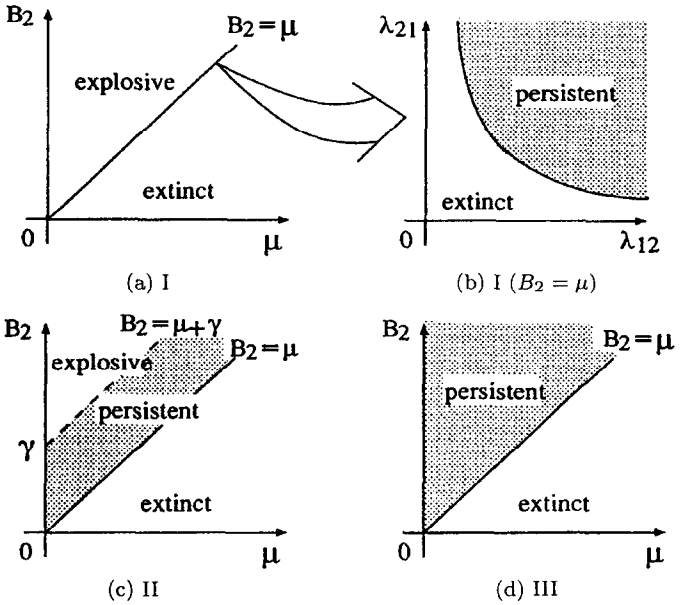
In the case when

$$(\mu + \gamma)^2 - \lambda_{12}\lambda_{21}\frac{k}{1-k}N_2^2 > 0, \tag{22}$$

since Jacobian matrix (14) has a zero-eigenvalue, the stability of the nontrivial equilibrium is not trivial from the eigenvalue analysis for the linearized system around the equilibrium. However, we know that the total male population asymptotically approaches a constant, and hence we can argue the nature of the ω-limit set for this model, by analyzing the corresponding system with $B_2 = \mu$ and constant N_1. In such condition, it can be shown that the trivial equilibrium is locally stable if (22) is satisfied from the eigenvalue analysis.

When $\mu < B_2$, $N_i(t) \to \infty$ $(i = 1, 2)$; i.e., population is explosive. In this case, the feasible asymptotic states of S, I and R classes are given in Table 1 (for detail way of the derivation, see Appendix A). Consequently, it is proved that the infective population is explosive (see Figures 4a,b).

MODEL II. For this model, $N_1(t)$ and $N_2(t)$ themselves cannot be analytically solved, although it can be proved that $N_1(t)/N_2(t)$ tends asymptotically to $k/(1-k)$ as $t \to \infty$ (see Appendix B).



(a) I (b) I ($B_2 = \mu$)

(c) II (d) III

Figure 4. Persistence of disease. For detailed explanation, see text.

When $B_2 < \mu$, it can be easily shown that the trivial equilibrium

$$(S_1^*, S_2^*, I_1^*, I_2^*, R_1^*, R_2^*) = (0, 0, 0, 0, 0, 0)$$

is locally stable from the eigenvalue analysis for the corresponding Jacobian matrix.

When $B_2 = \mu$, the unique equilibrium point in the domain $\{\mathbf{R}_+^6 - \mathcal{D}\}$ is the following trivial one:

$$(S_1^*, S_2^*, I_1^*, I_2^*, R_1^*, R_2^*) = (0, 0, 0, 0, 0, 0),$$

where we define

$$\mathcal{D} = \{(S_1, S_2, I_1, I_2, R_1, R_2) \mid S_1 \geq 0,\ S_2 > 0,\ I_1 = I_2 = R_1 = R_2 = 0\},$$
$$\mathbf{R}_+^6 = \{(S_1, S_2, I_1, I_2, R_1, R_2) \mid S_1 \geq 0,\ S_2 \geq 0,\ I_1 \geq 0,\ I_2 \geq 0,\ R_1 \geq 0,\ R_2 \geq 0\}.$$

It can be proved that the trivial equilibrium is globally stable in $\mathbf{R}_+^6 - \mathcal{D}$, and that the trajectory with the initial state in $\mathcal{D}$ asymptotically approaches to

$$(S_1^*, S_2^*, I_1^*, I_2^*, R_1^*, R_2^*) = \left(\frac{B_1}{\mu} S_2(0), S_2(0), 0, 0, 0, 0\right),$$

and this equilibrium state exists uniquely determined by the initial state (for the proof of these results, see Appendix C). This is a disease free case for $t \geq 0$.

On the other hand, if the nontrivial equilibrium exists, it must satisfy the following:

$$S_1^* = \frac{(\mu + \gamma) I_1^*}{\lambda_{12} I_2^*}, \tag{23}$$

$$S_2^* = \frac{\mu + \gamma - B_2}{B_2 - \mu} I_2^*, \tag{24}$$

$$I_1^* = \frac{(\mu + \gamma)(B_2 - \mu)}{\lambda_{21}(\mu + \gamma - B_2)}, \tag{25}$$

$$I_2^* = \frac{1}{2B_1\gamma(\mu + \gamma - B_2)\lambda_{12}\lambda_{21}} \left\{ (B_2 - \mu)^2(\mu + \gamma)^2\lambda_{12} + \sqrt{(B_2 - \mu)^4(\mu + \gamma)^4\lambda_{12}^2 + 4B_1\gamma(\mu + \gamma - B_2)^2\lambda_{12}\lambda_{21}^2(B_2 - \mu)\mu(\mu + \gamma)} \right\}, \tag{26}$$

$$R_1^* = \frac{\gamma}{\mu} I_1^*, \tag{27}$$

$$R_2^* = \frac{\gamma}{\mu} I_2^*. \tag{28}$$

Iff the right-hand sides of (23)–(28) are positive, this nontrivial equilibrium exists. From equations (23)–(28), we can easily prove that the necessary and sufficient condition for the existence of the above nontrivial positive equilibrium is $\mu < B_2 < \mu + \gamma$.

As for the case when $\mu + \gamma < B_2$, it can be proved that $S_2 + I_2$ diverges as $t \to \infty$ (see Appendix D). In this case, the feasible asymptotic states of S, I and R classes are shown in Table 1, qualitatively the same as for Model I. For Model II, we can prove that $S_1(t) \to B_1/\lambda_{12}$ as $t \to \infty$ in the state (d) of Table 1 (see Appendix D). Anyway, the obtained result is that the infective population is explosive. Some numerical calculations implied that the state (d) of Table 1 would be as asymptotically attainable equilibrium state.

MODEL III. Also for this model, with the argument analogous to that for the previous model, we can prove that

$$\frac{N_1(t)}{N_2(t)} \to \frac{k}{1 - k} \qquad t \to \infty.$$

When $B_2 < \mu$, it is easily proved that the trivial equilibrium

$$(S_1^*, S_2^*, I_1^*, I_2^*, R_1^*, R_2^*) = (0, 0, 0, 0, 0, 0)$$

is locally stable.

When $B_2 = \mu$, the same as for Model II, the unique equilibrium point in $\mathbf{R}_+^6 - \mathcal{D}$ is the following trivial one:

$$(S_1^*, S_2^*, I_1^*, I_2^*, R_1^*, R_2^*) = (0, 0, 0, 0, 0, 0).$$

This trivial equilibrium is globally stable in $\mathbf{R}^6_+ - \mathcal{D}$. With the initial state in $\mathcal{D}$, the same argument as for Model II can be applied, and the same result of the disease free asymptotic state can be drawn.

Iff the condition $\mu < B_2$ is satisfied, the nontrivial equilibrium exists. This results from such requirements that the following (29)–(34) must all be positive:

$$S_1^* = \frac{(\mu+\gamma)(B_2-\mu)}{\lambda_{12}\lambda_{21}I_2^*}, \tag{29}$$

$$S_2^* = \frac{\mu+\gamma}{B_2-\mu}I_2^*, \tag{30}$$

$$I_1^* = \frac{B_2-\mu}{\lambda_{21}}, \tag{31}$$

$$I_2^* = \frac{1}{2B_1\lambda_{12}\lambda_{21}}\left\{(B_2-\mu)^2\lambda_{12} + \sqrt{(B_2-\mu)^4\lambda_{12}^2 + 4B_1\lambda_{12}\lambda_{21}\mu(B_2-\mu)^2}\right\}, \tag{32}$$

$$R_1^* = \frac{\gamma}{\mu}I_1^*, \tag{33}$$

$$R_2^* = \frac{\gamma}{\mu}I_2^*. \tag{34}$$

Moreover, along the same line of argument as for Model I and II, we can prove that the state $(S_1(t), S_2(t), I_1(t), I_2(t), R_1(t), R_2(t)) = (N_1(t), N_2(t), 0, 0, 0, 0)$ cannot be the asymptotically attainable one as $t \to \infty$.

Results from the above analyses about the local stability of trivial equilibria and the existence of nontrivial ones for each model are displayed in Figure 4. For the region of extinction in Figure 4, the total population dies out, which is determined only by the relation between the birth and the death rates. That is, for that region in Figure 4, since the death rate exceeds the birth rate, the total population goes extinct.

3.2. Sex Ratio of Infective Population

We consider the sex ratios of infective population at the equilibrium states.

3.2.1. Sex ratio at equilibrium state

For the nontrivial equilibrium, the stationary infective population for each model has been obtained by the previous analysis. In this section, we consider the sex ratio of the infective population at the equilibrium state. We must note that the following analysis should be under the restriction of parameters in order for the existence of the nontrivial equilibrium.

MODEL I.

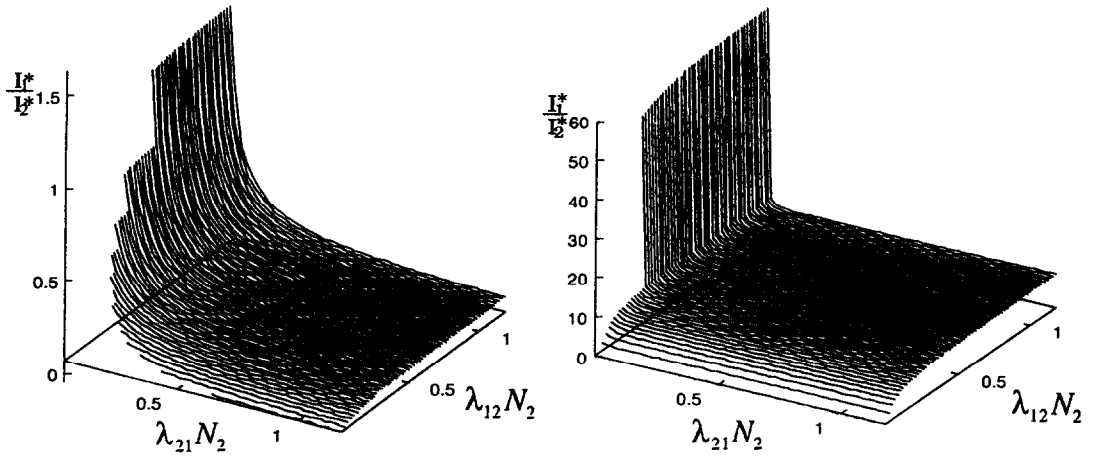
$$\frac{I_1^*}{I_2^*} = \frac{\{(1-k)(\mu+\gamma) + k\lambda_{21}N_2\}\lambda_{12}}{(1-k)(\lambda_{12}N_2 + \mu + \gamma)\lambda_{21}}. \tag{35}$$

I_1^*/I_2^* is a monotonically increasing function of k $(0 < k < 1)$ (Figure 5).

MODEL II.

$$\frac{I_1^*}{I_2^*} = \frac{2(\mu+\gamma)(B_2-\mu)B_1}{(B_2-\mu)^2(\mu+\gamma)^2 + \sqrt{(B_2-\mu)^4(\mu+\gamma)^4 + 4B_1\gamma(\mu+\gamma-B_2)^2\frac{\lambda_{21}^2}{\lambda_{12}}(B_2-\mu)\mu(\mu+\gamma)}}. \tag{36}$$

As $\lambda_{12} \to \infty$, I_1^*/I_2^* approaches the constant $B_1\gamma/\{(B_2-\mu)(\mu+\gamma)\}$ independent of λ_{21} (see Figure 6a and Table 2). When λ_{21} is sufficiently small, I_1^*/I_2^* is asymptotically $B_1\gamma/\{(B_2-\mu)(\mu+\gamma)\}$. We consider that for such sufficiently small λ_{21}, the transmission of disease from male to female occurs very slowly. Then, I_2^* could be quasi-stationary determined by only female



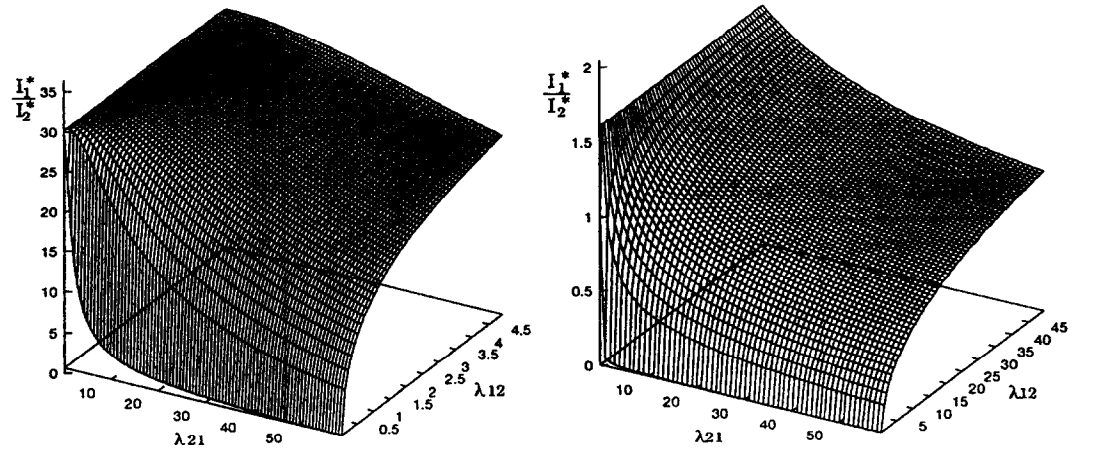
(a) $k = 0.10$ (b) $k = 0.90$

Figure 5. Sex ratio of infective population at the equilibrium state for Model I. $\mu = 0.02$, $\gamma = 0.0333$.

population dynamics, so that λ_{12} and λ_{21} do not contribute to the sex ratio of infective population in such case.

MODEL III.

$$\frac{I_1^*}{I_2^*} = \frac{2B_1}{B_2 - \mu + \sqrt{(B_2 - \mu)^2 + 4B_1(\lambda_{21}/\lambda_{12})\mu}}. \tag{37}$$



(a) (b)

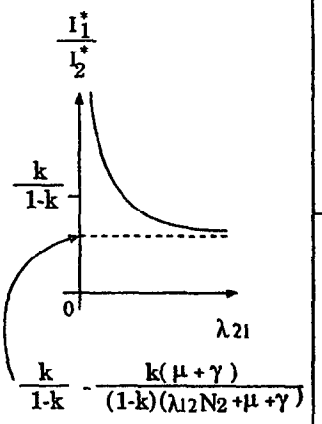
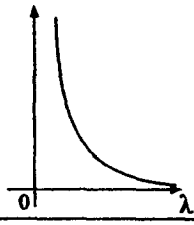
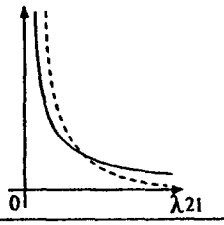
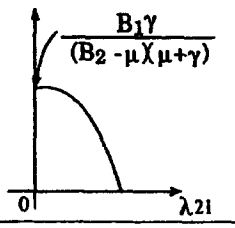
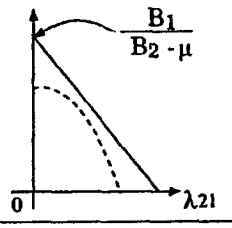
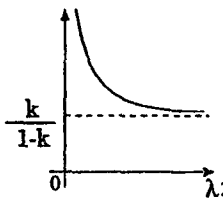
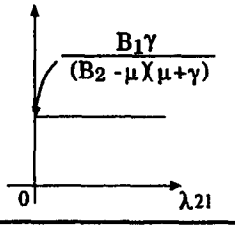
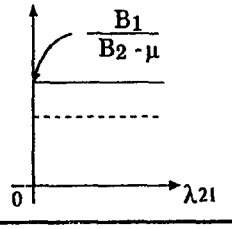
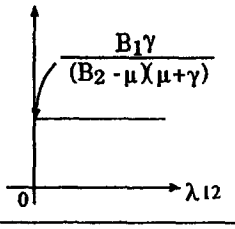
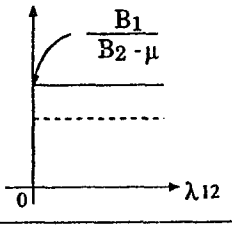
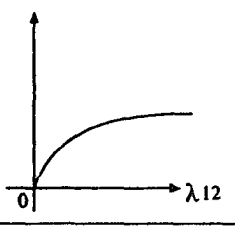
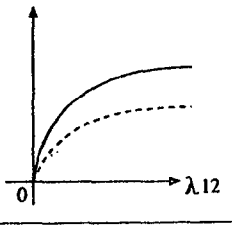
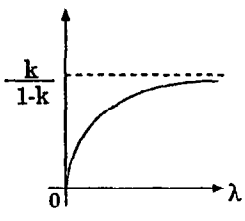
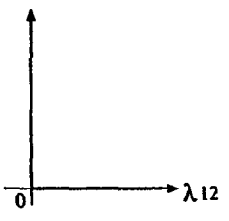
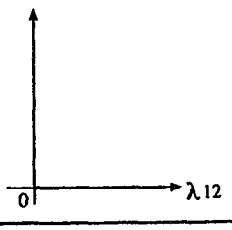
Figure 6. Sex ratio of infective population at the equilibrium state: (a) for Model II; (b) for Model III. $\mu = 0.02$, $\gamma = 0.0333$, $B = 0.106$, $k = 0.50$.

As well as for Model II, if $\lambda_{12} \to \infty$ or $\lambda_{21} \to 0$, I_1^*/I_2^* is asymptotically constant, $B_1/(B_2 - \mu)$, independent of both transmission rates, which is larger than that for Model II (Figure 6b and Table 2). The infective sex ratio for this model behaves qualitatively the same as that for Model II except for its λ_{21}-dependence. It would be due to the structural difference such that the mother class is only S_2 in Model III while $S_2 + I_2$ in Model II. When we compared the result for Model III with that for Model II, the sex ratio for Model III tended to take the bigger value than that for Model II, except for when λ_{12} is sufficiently small (see Table 2).

3.2.2. Sex ratio at explosive state

For Model I, we could not find any mathematical result about the sex ratio at the explosive state.

Table 2. Sex ratio of infective population at the nontrivial equilibrium for each model. The parenthesized numbers in the second and the third rows indicate the corresponding conditions and formulas in the text. Dashed lines in III show the results for II.

	I	II	III
	$B_2 = \mu$ (22)	$\mu < B_2 < \mu+\gamma$	$\mu < B_2$
I_1^*/I_2^*	(35)	(36)	(37)
sufficiently small λ_{12}			
sufficiently large λ_{12}			
$\lambda_{12} \to \infty$			
sufficiently small λ_{21}			
sufficiently large λ_{21}			
$\lambda_{21} \to \infty$			

MODEL II. In Table 1, the states (c) and (d) lead to a sex ratio of infective population which asymptotically tends to a constant value (see Appendix E), which is

$$\frac{I_1(t)}{I_2(t)} \to \frac{B_2}{\lambda_{21} S_2^*}, \qquad t \to \infty.$$

For Model III, we consider the sex ratio of the infective population, assuming that both $I_1(t)$ and $I_2(t)$ diverge exponentially as $t \to \infty$.

MODEL III. As described in Appendix E, we can prove that

$$\frac{I_1(t)}{I_2(t)} \to 0, \qquad t \to \infty.$$

3.2.3. Sex ratio at extinctive state

For our models, it can be proved that $I_1(t)/I_2(t)$ converges to a finite constant (see Appendix F). However, we could not distinctly determine the converged constant (for example, see Figure 7).

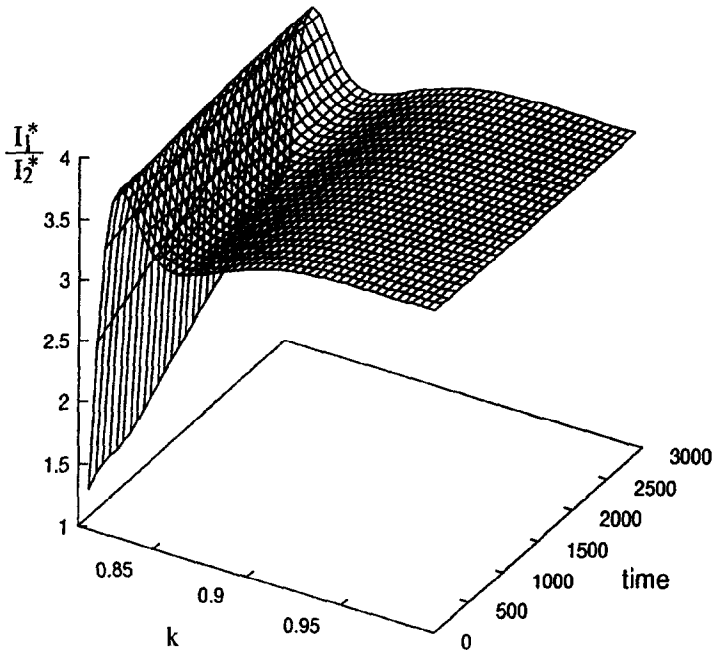


Figure 7. Sex ratio of the infective population at the extinctive state for Model II. Numerically obtained for k with which the extinctive state is attainable. $\mu = 0.02$, $\gamma = 0.0333$, $B = 0.106$, $\lambda_{12} = 0.90$, $\lambda_{21} = 0.50$, $S_1(0) = S_2(0) = 1.1$, $I_1(0) = I_2(0) = 0.1$.

3.3. Stationary Incidence of Disease

In this section, we consider the *incidence* of disease that is the ratio of the new infective recruitment.

DEFINITION 3 (INCIDENCE OF DISEASE). *Incidence of disease is the ratio of population transferred from the susceptible class to the infective in the total population.*

In ordinary statistical use, the incidence is reported as the ratio of recruited infective population to a certain total population. For our model, to somehow give corresponding meaning, we consider the incidence of disease at the nontrivial equilibrium state. We define the male and the female incidences of disease, respectively, as follows:

$$\mathrm{Inc}_1^* = \frac{\lambda_{12} S_1^* I_2^*}{N_1^*}, \tag{38}$$

$$\mathrm{Inc}_2^* = \frac{\lambda_{21} S_2^* I_1^*}{N_2^*}, \tag{39}$$

where Inc_1^* is the male incidence and Inc_2^* is the female one. Further, we introduce the ratio $\mathrm{Inc}_1^*/\mathrm{Inc}_2^*$ denoted by INC*:

$$\mathrm{INC}^* = \frac{\lambda_{12} S_1^* I_2^* N_2^*}{\lambda_{21} S_2^* I_1^* N_1^*}. \tag{40}$$

For our models, INC* can be computed as follows:

$$\mathrm{INC}^* = \frac{(1-k) I_1^*}{k I_2^*}. \tag{41}$$

It turns out that INC* behaves qualitatively the same as the sex ratio of infective population I_1^*/I_2^* does for transmission ratios λ_{12} and λ_{21}. However, for the sex ratio k of newborn, INC* has natures different from those of I_1^*/I_2^*.

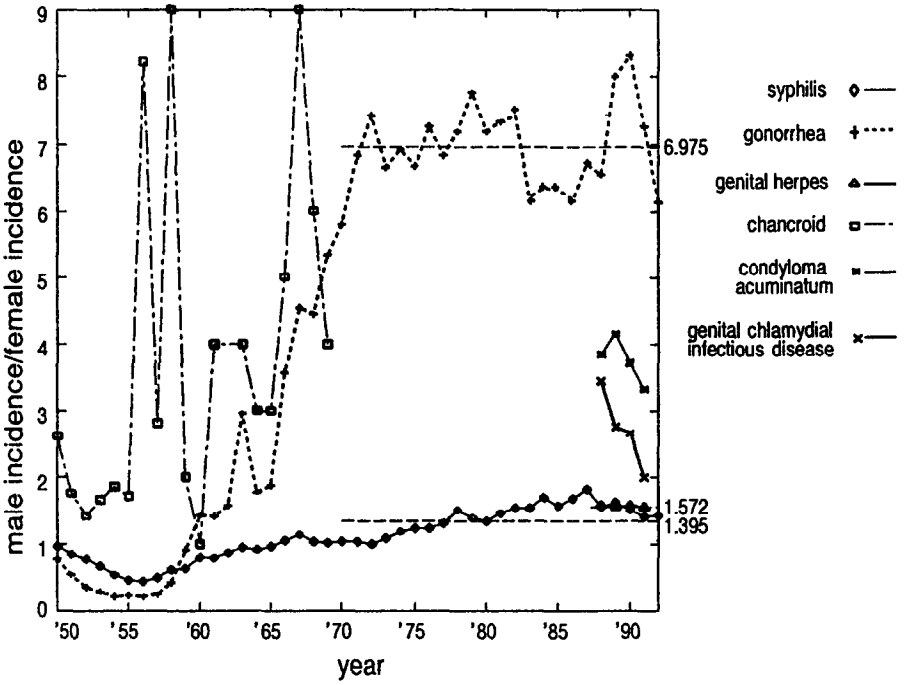


Figure 8. Sex ratio of incidences of STDs in Japan [4].

3.4. A Qualitative Application

Reported numbers of STD infectives in postwar Japan are shown in Figure 1 [4]. Those numbers can be regarded as to correspond to the incidence of STDs. However these numbers are changing due to the radical changes of social background. Compared with the social upheaval just after World War II, the temporal variation of them to some drastic changes of those incidences of STDs appears smaller after 1970s. In this chapter, along with our results in the previous section, we consider the incidences of some STDs from the statistical data.

We focus on the ratio of incidences of STDs between male and female population. The temporal variation obtained from some statistical data in Japan [4] is given in Figure 8.

In our models, $k = 0.50$, $\mathrm{INC}^* = I_1^*/I_2^*$ from (41). Especially in some cases of strongly biased transmission (see Table 2), using $B = 0.106$, $\mu = 0.020$, $\gamma = 0.033$ by May and Anderson [1], we get

$$\frac{B_1 \gamma}{(B_2 - \mu)(\mu + \gamma)} = 1.0044, \tag{42}$$

$$\frac{B_1}{B_2 - \mu} = 1.6060, \tag{43}$$

where (42) is for Model II, and (43) is for Model III.

The statistical data from 1970 in Figure 8 gives the average values for gonorrhea, syphilis and genital herpes: 6.975, 1.395, and 1.572, respectively. In regards genital herpes, the average seems relatively near the value of (43). Also for genital chlamydial, which is a disease known to make the infected female sterile, it appears roughly near the value of (43). Hence, the genital chlamydial infectious disease might be corresponding to Model III, in which I_2 and R_2 are sterile.

INC* for Model I is given by

$$\mathrm{INC}^* = \frac{\left\{(1-k)(\mu+\gamma)+k\widetilde{\lambda}_{21}\right\}\widetilde{\lambda}_{12}}{k\left(\widetilde{\lambda}_{12}+\mu+\gamma\right)\widetilde{\lambda}_{21}}, \tag{44}$$

where

$$\widetilde{\lambda}_{12} = \lambda_{12}N_2; \qquad \widetilde{\lambda}_{21} = \lambda_{21}N_2.$$

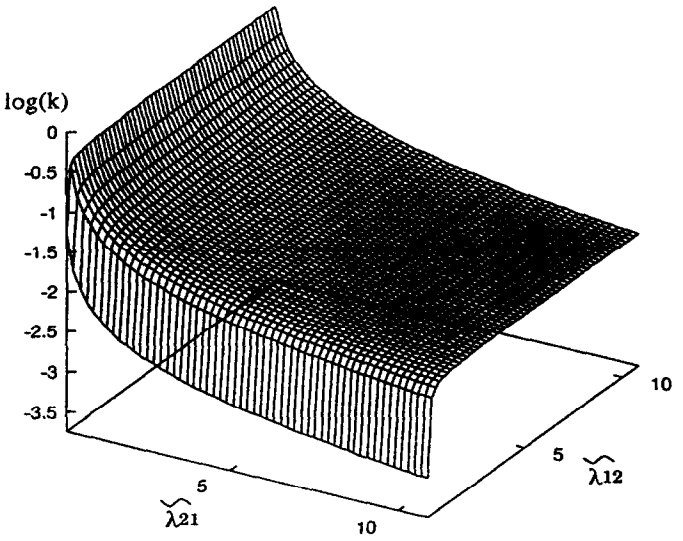
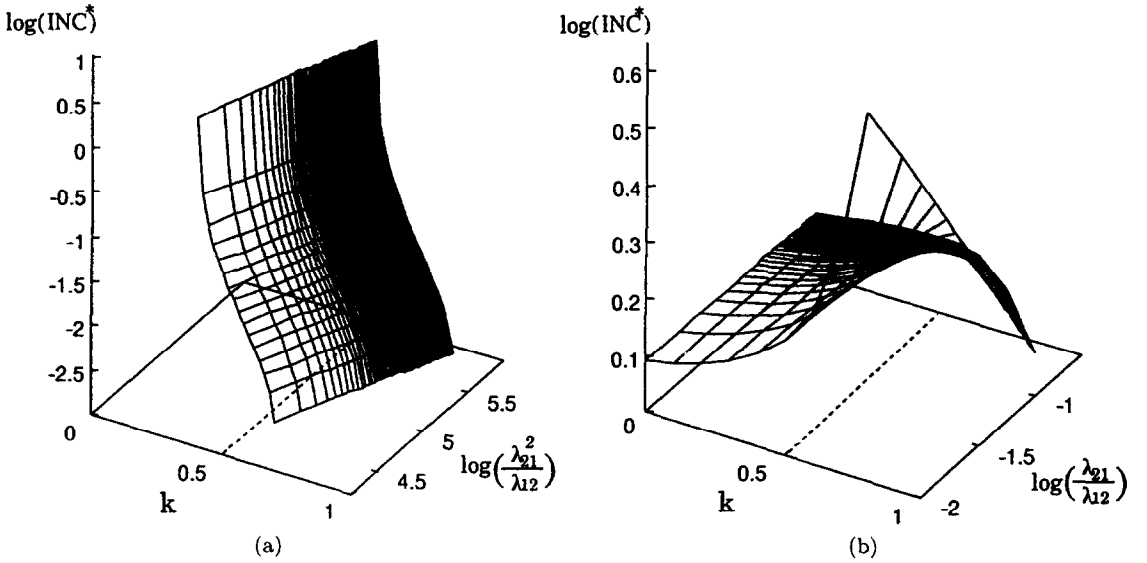


Figure 9. Relation among k, $\widetilde{\lambda}_{12}$ and $\widetilde{\lambda}_{21}$ with INC* = 1.572 for genital herpes. Drawn from Model I. $\mu = 0.02$, $\gamma = 0.0333$.

With $\mu = 0.020$, $\gamma = 0.033$ by May and Anderson [1], parameters k, $\widetilde{\lambda}_{12}$ and $\widetilde{\lambda}_{21}$ determine INC*. From (41), for a fixed INC* = 1.572, in case of genital herpes, the other parameters k, $\widetilde{\lambda}_{12}$ and $\widetilde{\lambda}_{21}$ have the relation shown in Figure 9. In the cases of gonorrhea and syphilis, a similar relation can be drawn.

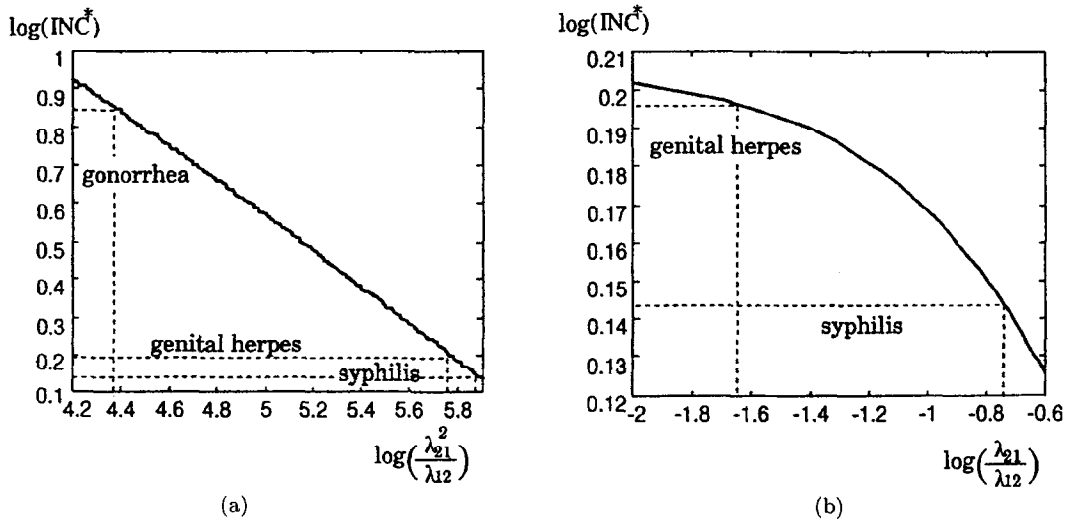
In regards to Models II and III with $B = 0.106$, $\mu = 0.020$, $\gamma = 0.033$ by May and Anderson [1], we can draw the relation among INC* and the other parameters as shown in Figure 10, where $\lambda_{21}^2/\lambda_{12}$ and $\lambda_{21}/\lambda_{12}$ are used as characteristic parameters for Models II and III, respectively.

If we suppose $k = 0.5$, the contribution of parameters $\lambda_{21}^2/\lambda_{12}$ and $\lambda_{21}/\lambda_{12}$ for INC* can be obtained as shown in Figure 11. From some statistical data in Japan [4], we can pick up the values of incidence ratio. Applying the data of incidence ratio to our INC* with $B = 0.106$, $\mu = 0.020$, $\gamma = 0.033$ by May and Anderson [1], the parameters $\lambda_{21}^2/\lambda_{12}$ and $\lambda_{21}/\lambda_{12}$ can be estimated. Indeed, applying the data for gonorrhea, genital herpes and syphilis, we obtain Table 3. For Model II, since $\lambda_{21}^2/\lambda_{12} \sim O(10^4 \sim 10^5)$ we can suggest that λ_{21} might be larger than λ_{12} with $O(10^2 \sim 10^3)$. As in Model II, referred to in Table 2, this corresponds to the case when λ_{12} is sufficiently small or λ_{21} is sufficiently large. For Model III, $\lambda_{21}/\lambda_{12}$ is negative in case of gonorrhea. This indicates that Model III could not be applied in this case. For the other STDs, λ_{21} appears smaller than λ_{12} by $O(10^{-2} \sim 10^{-1})$, as in Model II (Table 2), corresponding to the case when λ_{12} is sufficiently large or λ_{21} is sufficiently small. Therefore, it might be appropriate



(a) (b)

Figure 10. INC*: (a) for Model II; (b) for Model III. $B = 0.106$, $\mu = 0.02$, $\gamma = 0.0333$.



(a) (b)

Figure 11. INC* with $k = 0.5$: (a) for Model II; (b) for Model III. $B = 0.106$, $\mu = 0.02$, $\gamma = 0.0333$. Some values of the incidence ratio from the data for STDs in Japan [4] are indicated.

Table 3. Estimated $\lambda_{21}^2/\lambda_{12}$ and $\lambda_{21}/\lambda_{12}$ for the data of incidence ratios for some STDs in Japan [4].

Disease	Incidence ratio	$\frac{\lambda_{21}^2}{\lambda_{12}}$ for Model II	$\frac{\lambda_{21}}{\lambda_{12}}$ for Model III
gonorrhea	6.975	2.457×10^4	-1.821×10^{-1}
genital herpes	1.572	5.965×10^5	2.274×10^{-2}
syphilis	1.395	7.622×10^5	1.790×10^{-1}

that we consider the constant $B_1/(B_2 - \mu)$ by (43), especially for genital herpes. This drastic difference between the resulting estimations of $\lambda_{21}^2/\lambda_{12}$ and $\lambda_{21}/\lambda_{12}$ in Table 3 could be due to the structural difference between Model II and III, that is, the difference of reproductive and sterile classes.

With the estimated values of parameters $\lambda_{21}^2/\lambda_{12}$ and $\lambda_{21}/\lambda_{12}$ in Table 3, we consider the k-dependence of INC* (Figure 12). There are distinct differences between results for Models II and III. The k-dependence for Model II appears monotonically decreasing independently of STDs.

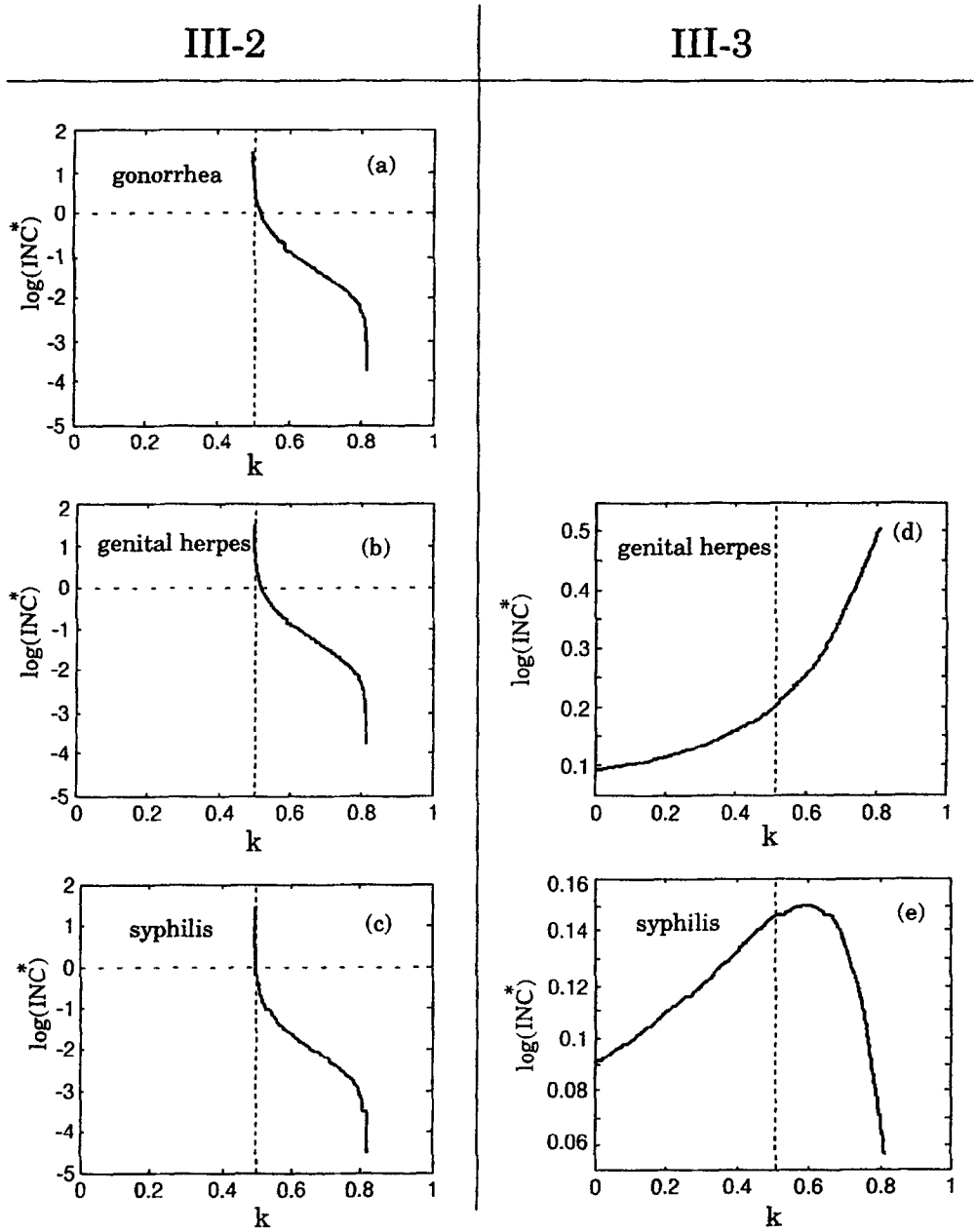


Figure 12. k-dependence of INC* with the estimated parameters $\lambda_{21}^2/\lambda_{12}$ and $\lambda_{21}/\lambda_{12}$ in Table 3. Some parts are not drawn due to the nonexistence of nontrivial positive equilibrium.

However, for Model III, it has a peak in case of the syphilis, while monotonically increasing for genital herpes. It should be noted that, since the estimated values in Table 3 are for $k = 0.5$, we should especially pay attention to the nature of INC* around $k = 0.5$ in Figure 12. This difference of results between Model II and Model III could again be considered due to the difference of model structure.

On the other hand, we can consider INC* with the assumption $\lambda_{12} = \lambda_{21}$. Even if the transmission is symmetric, the relation between INC* and k is not clear from the sexually asymmetric structure of the dynamical system. When $\lambda_{12} = \lambda_{21}$, the INC* applied to the data for genital herpes and syphilis leads to the result $k > 0.5$ (Figure 13). This result implies that in the considered community, a male newborn might be expected more than a female. Most of the previous mathematical considerations provided that $k = 0.5$. However, the sex ratio of newborn is indeed

not exactly 1 : 1 in Japan [9]. The averaged sex ratio calculated from the statistical data after 1970 for (male):(female) is 96.6 : 100. Since at the equilibrium state the sex ratio of total population must be equivalent to that of newborn, we now consider this sex ratio as that of newborn; then we have $k = 4.91 \times 10^{-1}$. For a while, let us consider the case of $k = 4.91 \times 10^{-1}$. Then, for Model II, this value of k turns out not to satisfy the condition for the existence of nontrivial equilibrium. In case of Model III, we can estimate $\lambda_{21}/\lambda_{12}$ only for syphilis: $\lambda_{21}/\lambda_{12} = 1.23 \times 10^{-1}$. With the data for the other STDs, it appears negative.

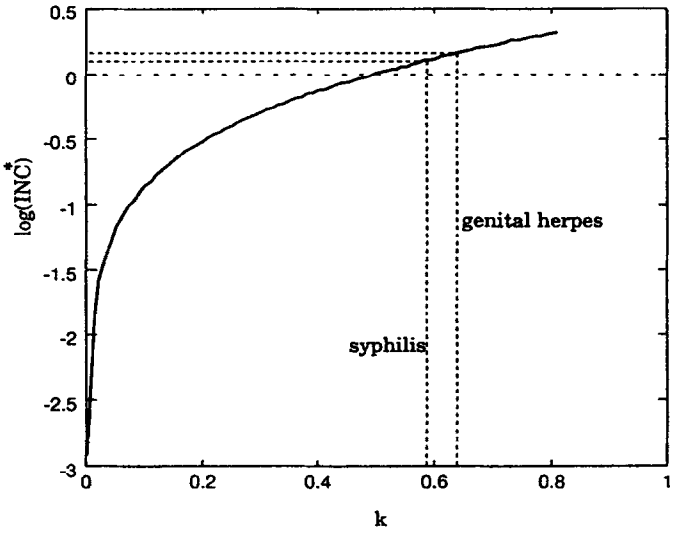


Figure 13. Relation between k and INC* for Model III with $\lambda_{12} = \lambda_{21}$. $\mu = 0.02$, $\gamma = 0.0333$.

4. CONCLUSION

In this paper, we discussed the effect of the asymmetricity in the transmission dynamics of STDs on the population structure. Some parameters, the transmission rate λ_{ij} and the newborn sex ratio k, appear to contribute fundamentally to the features of population structure with STDs. Indeed, for example, the transmission rate could reflect the average number of sexual partners, the sexual activity, the physical nature, and so on. In this paper, we construct our models to consider differences of population dynamics. Interestingly, it is shown that the results are significantly different depending on which classes can contribute to the recruitment of newborns.

As for the early stage of infection in our models, we can obtain the basic reproductive rates given by (6)–(8). May and Anderson [2] suggest for the total population with HIV/AIDS infection: $\widetilde{R}_0 \propto \sqrt{\lambda_{12}\lambda_{21}}$. Different from our traditional SIR modellings, in their model, it is assumed that susceptible individuals become infected proportionally to the ratio of infective population to the total number. They also neglect natural death rate in comparison with the transition rate from I class to R class. The basic reproductive rate by May and Anderson [2] is proportional to the geometric mean of transmission rates of both classes, while our result given by (27) is proportional to the arithmetic mean of them, weighted with the sex ratio of the initial invading infective population.

The sex ratio of the infective population long after the disease invasion reveals some differences among models. May and Anderson [2] give the sex ratio of infective population at the early stage: {HIV/AIDS among male}/{HIV/AIDS among female} $\simeq \sqrt{\lambda_{12}/\lambda_{21}}$. We consider the corresponding sex ratio at the equilibrium state (Section 3.2). Only the resulted sex ratio for Model III could be regarded to be relatively corresponding to that by May and Anderson [2]. Consequently, the sex ratio of infective population strongly depends on the dynamical structure of the transmission of disease.

Applying the incidence ratios from the statistical data for STDs in Japan [4], we consider the corresponding ratios for each of our models. As a result, we are able to suggest that the considered STDs have a much more asymmetric (biased) transmission rate, which were estimated not to have small order of difference.

As for mathematical analysis along our modelling, we still have many open-problems to consider general STDs. One of them is the effect of vertical transmission, in which the mother's disease transmits to the fetus. Another is the mortality due to disease.

We expect that a variety of mathematical studies, including ours, can contribute to the epidemiology in the other fields so as to understand present diseases. We expect that our work will somehow contribute to the way the dynamics of some STDs are understood, as one of such works in mathematical biology.

APPENDIX A
FEASIBLE ASYMPTOTIC STATES FOR MODEL I

We can easily find, from the differential equation for $R_i(t)$, that $I_i(t)$ and $R_i(t)$ have similar asymptotic behaviour as $t \to \infty$. So, iff $N_i(t) = S_i(t) + I_i(t) + R_i(t)$ diverges, $S_i(t) + I_i(t)$ also diverges.

At first, suppose that $I_2(t) \to 0$. Then, it is required that as $t \to \infty$, $\frac{dI_2(t)}{dt} \to 0$, so that $\lambda_{21} I_1 S_2 \to 0$ from equation for $I_2(t)$. Since $I_2(t)$ converges to zero, $S_2(t)$ must diverge and asymptotically behave as $\exp\{(B_2 - \mu)t\}$ from (12). Hence, it is additionally required that $I_1(t) \to 0$ with a smaller order than $\exp\{-(B_2 - \mu)t\}$. Therefore, if $I_2(t) \to 0$, $(S_1(t), S_2(t), I_1(t), I_2(t)) \to (N_1(t), N_2(t), 0, 0)$. However, it can be proved that this state cannot be attained as $t \to \infty$. (For proof, see below.) Similarly, provided that $I_1(t) \to 0$, $(S_1(t), S_2(t), I_1(t), I_2(t))$ is required to attain asymptotically to $(N_1(t), N_2(t), 0, 0)$, which is unattainable.

Next, suppose that $I_2(t)$ converges to a positive finite constant. From the divergence of $N_2(t)$, it is required that $S_2(t) \to \infty$. Since $\frac{dI_2(t)}{dt} \to 0$, $\lambda_{21} I_1 S_2 \to 0$, so that $I_1(t) \to 0$. Then, $\frac{dI_1(t)}{dt} \to 0$, and therefore $\lambda_{12} I_2 S_1 \to 0$. Since I_2 converges to a positive finite constant, it is required that $S_1 \to 0$. So, $S_1(t) + I_1(t) \to 0$, and this is contradictory to the condition that $N_1 \to \infty$. Along the same line of argument, it is proved that $I_1(t)$ positively diverges.

On the other hand, suppose that $S_1(t)$ converges to zero; then $\frac{dS_1}{dt} \to 0$ as $t \to \infty$. Now, consider $\frac{dS_1}{dt}$ as follows:

$$\frac{dS_1}{dt} = I_2 \left\{ B_1 \left(\frac{S_2}{I_2} + \frac{R_2}{I_2} + 1 \right) - \lambda_{12} S_1 - \mu \frac{S_1}{I_2} \right\}.$$

Since $I_2 \to \infty$ from the above argument, $\frac{dS_2}{dt} \approx I_2 B_1$ for sufficiently large t. This is contradictory to the convergence of S_1. In the same way, we can show that $S_2(t)$ cannot converge to zero. These arguments lastly give the result given as Table 1.

For unattainable state $(N_1(t), N_2(t), 0, 0)$, from (2), provided that $I_i(0) = R_i(0) = 0$, then $I_i(t) = R_i(t) = 0$ for any t, and system (2) is reduced to

$$\frac{dS_1(t)}{dt} = B_1 S_2(t) - \mu S_1(t),$$
$$\frac{dS_2(t)}{dt} = (B_2 - \mu) S_2(t).$$

Now, let us denote the solution for this system by $(\overline{S_1}(t), \overline{S_2}(t))$, which can be explicitly obtained as follows:

$$\overline{S_1}(t) = \left[S_1(0) - \frac{k}{1-k} S_2(0) \right] e^{-\mu t} + \frac{k}{1-k} S_2(0) e^{(B_2 - \mu)t},$$
$$\overline{S_2}(t) = S_2(0) e^{(B_2 - \mu)t},$$

when $\mu < B_2$, $\overline{S_1}(t)$, and $\overline{S_2}(t)$ explode as $t \to \infty$.

We consider the perturbation from the trajectory $(S_1(t), S_2(t), I_1(t), I_2(t)) = (\overline{S_1}(t), \overline{S_2}(t), 0, 0)$. The Jacobian matrix governing the asymptotic behaviour of $(S_1(t), S_2(t), I_1(t), I_2(t))$ around $(\overline{S_1}(t), \overline{S_2}(t), 0, 0)$ is the following:

$$J(\overline{S_1}(t), \overline{S_2}(t), 0, 0) = \begin{pmatrix} -\mu & B_1 & 0 & B_1 - \lambda_{12}\overline{S_1}(t) \\ 0 & B_2 - \mu & -\lambda_{21}\overline{S_2}(t) & B_2 \\ 0 & 0 & -(\mu+\gamma) & \lambda_{12}\overline{S_1}(t) \\ 0 & 0 & \lambda_{21}\overline{S_2}(t) & -(\mu+\gamma) \end{pmatrix}.$$

We obtain the following closed system for the perturbation (i_1, i_2) from $(I_1, I_2) = (0, 0)$:

$$\begin{pmatrix} \dfrac{di_1}{dt} \\ \dfrac{di_2}{dt} \end{pmatrix} = \begin{pmatrix} -(\mu+\gamma) & \lambda_{12}\overline{S_1}(t) \\ \lambda_{21}\overline{S_2}(t) & -(\mu+\gamma) \end{pmatrix} \begin{pmatrix} i_1 \\ i_2 \end{pmatrix}.$$

Under the condition $\mu < B_2$, the matrix has positive real eigenvalue for $t > \tilde{t}$, where

$$\tilde{t} = \frac{1}{2(B_2 - \mu)} \log \frac{(\mu+\gamma)^2(1-k)}{\lambda_{12}\lambda_{21}kS_2(0)^2} > 0. \tag{45}$$

Therefore, the trajectory $(\overline{S_1}(t), \overline{S_2}(t), 0, 0)$ is not attainable as one of equilibrium states for Model I with any infected initial state.

APPENDIX B
ASYMPTOTIC SEX RATIO IN MODEL II

From (3), we obtain the following:

$$\frac{dN_1}{dt} = B_1(S_2 + I_2) - \mu N_1,$$
$$\frac{dN_2}{dt} = B_2(S_2 + I_2) - \mu N_2.$$

From these, we derive

$$\frac{d}{dt}(B_2N_1 - B_1N_2) = -\mu(B_2N_1 - B_1N_2),$$

so that immediately

$$B_2N_1(t) - B_1N_2(t) = \{B_2N_1(0) - B_1N_2(0)\}\, e^{-\mu t}.$$

As $t \to \infty$, the right-hand side approaches to zero. Therefore,

$$\frac{N_1}{N_2} \to \frac{k}{1-k}, \qquad t \to \infty.$$

APPENDIX C
GLOBAL STABILITY OF THE TRIVIAL EQUILIBRIUM FOR MODEL II

We consider the female population $x = (S_2(t), I_2(t), R_2(t))$, and define the function

$$V(x) = S_2(t) + I_2(t) + R_2(t), \qquad x \in \mathbf{R}^{3*}_{+} \cup \{x^0\},$$

where

$$\mathbf{R}_+^{3*} = \{(S_2, I_2, R_2) \mid S_2 \geq 0,\ I_2 \geq 0,\ R_2 > 0\},$$
$$x^0 = (0,0,0).$$

Suppose that x^* is the unique equilibrium for the female system in $\mathbf{R}_+^{3*} \cup \{x^0\}$. Then, $V(x)$ satisfies

$$V(x) > 0, \qquad x \in \mathbf{R}_+^{3*},$$
$$V(x^0) = 0.$$

Since $\dot{V}(x) = -\mu R_2$,

$$\dot{V}(x) < 0, \qquad x \in \mathbf{R}_+^{3*},$$
$$\dot{V}(x^0) = 0.$$

Hence, $V(x)$ becomes the Lyapunov function, so that x^0 is GAS in $\mathbf{R}_+^{3*} \cup \{x^0\}$.

If $S_2(t) = I_2(t) = R_2(t) = 0$, then

$$\frac{dN_1}{dt} = -\mu N_1,$$

and N_1 approaches asymptotically to zero. Therefore, $(S_1, S_2, I_1, I_2, R_1, R_2) = (0,0,0,0,0,0)$ is GAS in $\mathcal{D}_1 \cup \{z^0\}$, where

$$\mathcal{D}_1 = \{(S_1, S_2, I_1, I_2, R_1, R_2) \mid S_1 \geq 0,\ S_2 \geq 0,\ I_1 \geq 0,\ I_2 \geq 0,\ R_1 \geq 0,\ R_2 > 0\},$$
$$z^0 = (0,0,0,0,0,0).$$

For convenience, we define the following subspace of six-dimensional space:

$$\mathcal{D}_2 = \{(S_1, S_2, I_1, I_2, R_1, R_2) \mid S_1 \geq 0,\ S_2 \geq 0,\ I_1 \geq 0,\ I_2 \geq 0,\ R_1 \geq 0,\ R_2 = 0\} - \{z^0\}.$$

The ω-limit set for trajectory with the initial state in $\mathcal{D}_2$ can be determined. Since $I_2(t) \equiv 0$ if $R_2(t) \equiv 0$, $I_1(t) \to 0$ and $R_1(t) \to 0$ as $t \to \infty$, as easily seen from the system (3). Hence, the ω-limit set is within the following subspace $\mathcal{D}_3$:

$$\mathcal{D}_3 = \{(S_1, S_2, I_1, I_2, R_1, R_2) \mid S_1 \geq 0, S_2 \geq 0, I_1 = I_2 = R_1 = R_2 = 0\}.$$

It is easily seen that $\mathcal{D}_3$ is the invariant set for the map given by the system (3). Since $\frac{dS_2}{dt} \equiv 0$ within $\mathcal{D}_3$, $S_2(t) \equiv S_2(0)$, and $S_1(t)$ can be explicitly obtained as follows:

$$S_1(t) = \left[S_1(0) - \frac{B_1}{\mu} S_2(0)\right] \frac{k}{1-k} S_2(0) e^{-\mu t} + \frac{B_1}{\mu} S_2(0),$$

and $S_1(t) \to (B_1/\mu) S_2(0)$ as $t \to \infty$. Hence, the considered ω-limit set consists of the point

$$(S_1, S_2, I_1, I_2, R_1, R_2) = \left(\frac{B_1}{\mu} S_2(0), S_2(0), 0, 0, 0, 0\right).$$

If $S_2(0) = 0$, this is equivalent with $\{z^0\}$. Therefore, any trajectory with the initial state in $\mathcal{D}_3$ leads to the ω-limit set included to $\mathcal{D}_3$. From these arguments, the trivial equilibrium is GAS in $\mathcal{D}_3 - \{z^0\}$, that is, $\mathcal{D}$ as defined in the main text.

APPENDIX D
EXPLOSIVE STATE FOR MODEL II

For Model II, we can easily find that

$$\frac{d}{dt}(S_2(t)+I_2(t)) = (B_2-\mu)S_2(t) + \{B_2-(\mu+\gamma)\}I_2(t) > \{B_2-(\mu+\gamma)\}(S_2(t)+I_2(t)).$$

Thus, using the conventional comparison theorem,

$$S_2(t)+I_2(t) > (S_2(0)+I_2(0))\, e^{\{B_2-(\mu+\gamma)\}t},$$

for any t. Therefore, if $B_2-(\mu+\gamma)>0$, S_2+I_2 diverges as $t\to\infty$.

We denote by S_1^* the positive equilibrium value for $S_1(t)$. From the following transformation:

$$\frac{dS_1}{dt} = I_2\left\{B_1\left(\frac{S_2}{I_2}+1\right) - \lambda_{12}S_1 - \mu\frac{S_1}{I_2}\right\},$$

we can obtain $\frac{dS_1}{dt}\to I_2(B_1-\lambda_{12}S_1)$, if $I_2\to\infty$ and S_2 converges to a finite value. Therefore, for the state (d) in Table 1, it is required that $S_1^* = B_1/\lambda_{12}$.

We further consider the cases in Table 1. Provided that $I_1(t)$ and $I_2(t)$ behave as $\exp(\varphi t)$ and $\exp(\rho t)$ (φ, ρ: unknown constants), respectively, for sufficiently large t, we rewrite the differential equation for I_i as follows:

$$\frac{dI_1}{dt} = \left\{\lambda_{12}I_2S_1\frac{1}{I_1} - (\mu+\gamma)\right\}I_1,$$
$$\frac{dI_2}{dt} = \left\{\lambda_{21}I_1S_2\frac{1}{I_2} - (\mu+\gamma)\right\}I_2.$$

Then, for sufficiently large t, we obtain the following relations:

$$\varphi \approx \lambda_{12}I_2S_1\frac{1}{I_1} - (\mu+\gamma),$$
$$\rho \approx \lambda_{21}I_1S_2\frac{1}{I_2} - (\mu+\gamma).$$

Since φ and ρ are constant, it is required that $S_1(t)$ behaves as $\exp\{(\varphi-\rho)t\}$, and $S_2(t)$ as $\exp\{(\rho-\varphi)t\}$ for sufficiently large t.

In type (a) in Table 1, since $S_i(t)\to\infty$, $\varphi-\rho>0$. However, $S_1(t)\to\infty$ requires $\rho-\varphi>0$. This is contradictory. Similarly, we can lead to contradictions in the other types, except for type (d). In type (d), since both $S_1(t)$ and $S_2(t)$ converge, we get $\varphi=\rho$. From $\frac{dS_2}{dt}\to 0$, $\lambda_{21}I_1S_2 \approx (B_2-\mu)S_2 + B_2I_2$ for sufficiently large t. Therefore, for sufficiently large t,

$$\frac{dI_2}{dt} \approx (B_2-\mu)S_2 + \{B_2-(\mu+\gamma)I_2\}.$$

Then, we can obtain $\rho = B_2-(\mu+\gamma)$; i.e., both $I_1(t)$ and $I_2(t)$ exponentially behave as $\exp[\{B_2-(\mu+\gamma)\}t]$.

APPENDIX E
INFECTIVE SEX RATIO OF EXPLOSIVE POPULATION

We suppose that $I_1(t)$ and $I_2(t)$ diverge exponentially as same as in Appendix D for Model II. Then, we can obtain the result that $S_2(t)$ behaves as $\exp\{(\rho-\varphi)t\}$. Now, we suppose $I_2(t)=Ae^{\rho t}$, where A is unknown constant. Then, $R_2(t)$ becomes

$$R_2(t) = \left\{R_2(0) - \frac{\gamma A}{\rho+\mu}\right\}e^{-\mu t} + \frac{\gamma A}{\rho+\mu}e^{\rho t}$$
$$\approx \frac{\gamma A}{\rho+\mu}e^{\rho t} \qquad \text{for sufficiently large } t.$$

So, for sufficiently large t, we can denote $I_2(t)+R_2(t) \approx Ce^{\rho t}$. Moreover, we set $I_1(t)S_2(t) \approx De^{\rho t}$, because $\rho = \lambda_{21} I_1 S_2 \dfrac{1}{I_2} - (\mu+\gamma)$ is constant (Appendix D). Then, we can solve out $S_2(t)$

$$S_2(t) \approx \left\{ S_2(0) - \frac{B_2 C - \lambda_{21} D}{\rho - (B_2 - \mu)} \right\} e^{(B_2-\mu)t} + \frac{B_2 C - \lambda_{21} D}{\rho - (B_2 - \mu)} e^{\rho t}.$$

Hence, for sufficiently large t,

$$S_2 + I_2 + R_2 \approx E e^{(B_2-\mu)t} + F e^{\rho t},$$

where

$$E = S_2(0) - \frac{B_2 C - \lambda_{21} D}{\rho - (B_2 - \mu)},$$

$$F = \frac{B_2 C - \lambda_{21} D}{\rho - (B_2 - \mu)} + A + \frac{\gamma A}{\rho + \mu}.$$

From (3), we find that

$$\frac{d}{dt} N_2(t) = B_2 \left(S_2(t) + I_2(t) \right) - \mu N_2(t) \leq (B_2 - \mu) N_2(t).$$

So, with the conventional comparison theorem,

$$N_2(t) \leq N_2(0) e^{(B_2-\mu)t}$$

for any $t > 0$. Thus, with the obtained asymptotical behaviour of $N_2(t)$ in the above, we find that $\rho \leq B_2 - \mu$. Consequently, from the asymptotic behaviour of $S_2(t)$ for sufficiently large t, $\rho - \varphi = B_2 - \mu$. This brings such a result that $I_1(t) \to 0$ since $\varphi = \rho - (B_2 - \mu) \leq 0$. And similarly, $I_2(t) \to 0$. This argument proves that neither $I_1(t)$ nor $I_2(t)$ can diverge in an exponential manner.

When $S_2(t)$ converges to a finite value and $I_2(t) \to \infty$,

$$\frac{dS_2}{dt} = I_2 \left\{ B_2 \left(\frac{S_2}{I_2} + 1 \right) - \lambda_{21} S_2 \frac{I_1}{I_2} - \mu \frac{S_1}{I_2} \right\} \to I_2 \left(B_2 - \lambda_{21} S_2 \frac{I_1}{I_2} \right) \qquad \text{as } t \to \infty.$$

In order that $\frac{dS_2}{dt} \to 0$, it is required that $I_1^*/I_2^* = B_2/(\lambda_{21} S_2^*)$.

Along the same line of argument as for Model II, if $I_1(t)$ and $I_2(t)$ diverge in Model III, they do not behave in an exponential manner. It can be easily shown from (4) that, if $I_1(t) \to \infty$ as $t \to \infty$, then $I_2(t) \to \infty$, $S_1(t) \to 0$, $S_2(t) \to 0$, and $S_1(t)I_2(t) \to \infty$, $I_1(t)S_2(t) \to \infty$. $R_i(t)$ asymptotically behaves as $\gamma I_i(t)/\mu$ $(i = 1, 2)$. Hence, if the infective population in Model III is explosive, the population $N_i(t) \to (\mu+\gamma) I_i(t)/\mu$. Since $N_1(t)/N_2(t) \to k/(1-k)$ as $t \to \infty$ in Model III, this argument indicates that $I_1(t)/I_2(t) \to k/(1-k)$ for the explosive infective population.

APPENDIX F
SEX RATIO OF EXTINCTIVE POPULATION IN MODEL III

In this appendix, we describe the sex ratios of extinctive population only in the case of Model III. For Models I and II, we can apply the same line of argument.

At first, we can easily get the following:

$$\frac{d}{dt} \left(\frac{S_1}{S_2} \right) = B_1 - (\lambda_{12} I_2 + B_2 - \lambda_{21} I_1) \frac{S_1}{S_2}.$$

Since the total population goes extinct, $I_i(t)$ can be regarded as sufficiently small for sufficient large t. If $S_1/S_2 \to 0$, $\left(\frac{d}{dt}\right)(S_1/S_2) \to B_1 > 0$, and this is contradictory. Therefore, we prove that S_1/S_2 converges to $B_1/B_2 = k/(1-k)$.

Using this result, we next consider the following:

$$\frac{d}{dt} \left(\frac{I_1}{I_2} \right) = S_1 \left\{ \lambda_{12} - \lambda_{21} \frac{S_1}{S_2} \left(\frac{I_1}{I_2} \right)^2 \right\}.$$

Provided that I_1/I_2 converges to a positive finite value, the above equation is always satisfied without any additional condition, and we cannot determine the value. Moreover, in the similar line of argument as above, it can be shown that I_1/I_2 cannot converge to zero or diverge.

REFERENCES

1. R.M. May and R.M. Anderson, Transmission dynamics of HIV infection, *Nature* **326**, 137–142 (1987).
2. R.M. May and R.M. Anderson, The transmission dynamics of Human Immunodefficiency Virus (HIV), In *Applied Mathematical Ecology*, (Edited by L.J. Gross, T.G. Hallam and S.A. Levin), Springer-Verlag, Berlin, (1987).
3. S. Yamamoto, *From Syphilis To AIDS: Modern Japanese History of Prostitution and STDs (Baidoku kara AIDS he—baisyun to seibyou no nihon kindaishi)*, (in Japanese), Asakura-syoten, Tokyo, (1994).
4. The Japanese Ministry of Public Welfare, Editors, *Statistical Reports on Epidemics (Densenbyou toukei)*, (in Japanese), Tokyo, (1994).
5. W.O. Kermack and A.G. Mckendrick, Contributions to the mathematical theory of epidemics (Part I), *Proc. R. Soc.* **A115**, 700–721 (1927).
6. E. Beretta and V. Capasso, On the general structure of epidemic system: Global asymptotic stability, *Computers Math. Applic.* **12A** (6), 677–694 (1986).
7. E. Beretta and V. Capasso, Global stability results for a multigroup SIR epidemic model, In *Mathematical Ecology*, (Edited by L.J. Gross, T.G. Hallam, and S.A. Levin), World Scientific, Singapore, (1988).
8. N. Shigesada, *Mathematical Ecology on Invasion and Diffusion (Shinnyuu to denpan no suuri seitaigaku)*, (in Japanese), Tokyo University Press, Tokyo, (1992).
9. Yano Koutarou Memorial Association, Editors, *NIPPON (Kokusei-sha) a Charted Survey of Japan*, Tokyo, (1993).