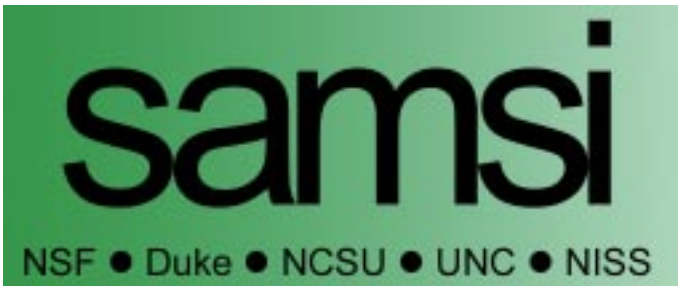


# A Note on Epidemic Models with Infective Immigrants and Vaccination

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# A NOTE ON EPIDEMIC MODELS WITH INFECTIVE IMMIGRANTS AND VACCINATION

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Abstract. The roles of immigration and vaccination on disease dynamics are explored in a simple setting that considers the possibility of conferred immunity. We focus on SIR and SIS models with a vaccinated class. Conditions for the existence of multiple endemic steady states and a fold bifurcation are discussed.

1. **Introduction.** It is well known that immigrants play a critical role in disease dynamics, and yet there is hardly any theoretical work [1], [2], [3]. Epidemics "ignited" or "enhanced" by the immigration of infectious cases include HIV [4], severe acute respiratory syndrome(SARS) [5], [6], avian influenza and measles [5]. The effect of vaccination on simple epidemic models with immigration is the subject of this note, which is motivated by the research in [1], as expanded in [2]. Extensions of the work in [1] and [2] to an SIRS model with vaccination have recently been published [3].

Some diseases, such as gonorrhea, do not confer immunity and can be modeled using a Susceptible-Infective-Susceptible (SIS) framework. The SIS model is the core of any disease transmission model and serves as a basic template to be expanded for special cases. Thus a full understanding of a simple SIS model is essential regardless of how well any particular disease can be forced into its framework. The use of control measures, such as the use of condoms (for example, in the case of gonorrhea), can be interpreted as a partially effective "vaccine." Hence, a vaccinated class is added to the classic SIS model in a population where the impact of infective immigrants is considered. "Vaccine" efficacy is a function of the disease. Our models (SVIS and SVIR) consider various levels of efficacy and waning effect (e.g., influenza vaccines have a 70% to 90% efficacy rate among healthy young adults but only 30% to 40% among the elderly [3]).

Models that include a constant flow of infective immigrants cannot eliminate a disease through vaccination or "standard" control measures. These standard policies are based on the *basic reproduction number*, which cannot be applied to models with a constant flow of infective individuals [7], [8]. Here, the value of a critical ratio, computed as the fraction of infective immigrants approaches zero, is used as a "limiting" threshold for the study of disease invasion and control. In addition, the result of partially effective vaccines is evaluated within appropriate submodels [9]. The possibilities of subthreshold coexistence and bistability are

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investigated. This paper is organized as follows: in Section 2, the SVIS model with infective immigrants is modified to include a vaccinated class [1], [10], [11] and their impact on disease dynamics is explored; in Section 3, the SVIR model with infective immigrants is modified to include a vaccinated class. However, this note only outlines my earlier results in [2] and more similar results were obtained recently in the context of the flu [3]; Section 4 focuses on discussion of the work in this note.

## 2. SVIS model with infective immigrants.

A population with a constant flow of infective immigrants within the simple SIS framework proposed by Kermack and McKendrick in [12] is considered in a model that incorporates the impact of a vaccination program. Diseases that fit within the above crude model include influenza [13], if "vaccination" is interpreted as a partially-effective control measure [9] or as the result of "natural" partially effective immunity (cross immunity) [13]. The SVIS framework can also be used in the context of sexually transmitted diseases, such as gonorrhea, as long as the vaccinated class is defined as the class of individuals who use prophylactics (condoms) with some regularity or who receive treatment that provides some (possibly quite short) temporary protection against reinfection [9].

### 2.1. Model formulation.

A constant flow of $A$ new members arrives into the population in unit time with the fraction $p$ of $A$ arriving infected ($0 \leq p \leq 1$). The susceptible class is vaccinated (educated) at the per capita rate $\phi$ and standard incidence is used. Infection can invade the susceptible or vaccinated classes (depending on vaccine efficacy). The vaccine (education program) is assumed to reduce the likelihood of infection by a factor of $\sigma$ ($0 \leq \sigma \leq 1$). The case $\sigma = 0$ corresponds to the case where the vaccine is completely effective, while $\sigma = 1$ models the situation where the vaccine is totally ineffective. It is further assumed that the vaccine provides temporary immunity, that is, wears off at the per capita rate $\theta$. The case $\theta = \infty$ corresponds to the case where there is absolutely no vaccine-induced immunity, while the case $\theta = 0$ corresponds to the case when immunity is life-long.

The population is divided into three epidemiological classes: susceptibles ($S$), infectives ($I$) and vaccinated ($V$). The above assumptions and the flow diagram (Figure 1) lead to the following epidemic model:

$$\begin{aligned} S' &= \Lambda + (1-p)A - \frac{\beta SI}{N} - (\mu+\phi)S + \gamma I + \theta V, \\ V' &= \phi S - \frac{\sigma\beta VI}{N} - (\mu+\theta)V, \\ I' &= pA + \frac{\beta SI}{N} + \frac{\sigma\beta VI}{N} - (\mu+\gamma)I. \end{aligned} \tag{1}$$

Model (1) is well posed: solutions remain nonnegative for nonnegative initial conditions. The population is replenished in two ways: from births and immigration. Here, it is assumed that newborns enter the susceptible class at the constant rate $\Lambda$, while immigrants enter at the constant rate $A$. Obviously, other scenarios are possible. These assumptions imply that the population under consideration is essentially constant. The per capita natural death rate is $\mu > 0$ in each class. There is no disease-induced mortality; that is, the number of individuals killed by the disease per unit of time is assumed to be negligible. Consequently, without loss of generality [14], it is assumed that the total population is asymptotically constant;

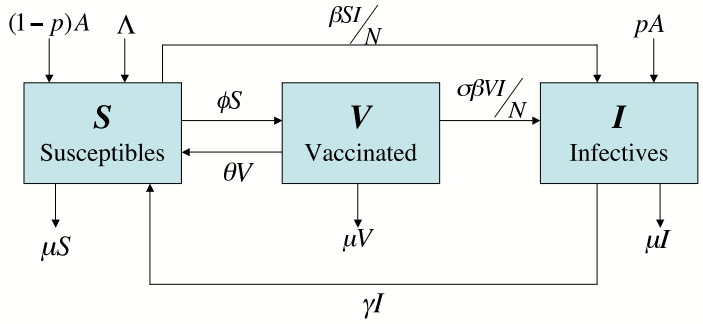


FIGURE 1. The SVIS model

that is, $N = K := \frac{A+\Lambda}{\mu}$. The disease's incidence rate is $\beta \cdot \frac{S}{N} \cdot I = \frac{\beta SI}{K}$, where $\beta$ is the transmission rate. A constant fraction $\gamma > 0$ of infectives recovers in unit time. System (1) can be reduced to a two-dimensional system by replacing $S$ by $K - I - V$ [14]. The use of the dimensionless variables ($i = I/K$, $v = V/K$ and $a = A/K$) leads to further simplifications. System (1) is replaced by

$$\begin{aligned} v' =& \phi[1 - i] - \sigma\beta vi - (\mu + \theta + \phi)v, \\ i' =& pa + \beta i[1 - i - (1 - \sigma)v] - (\mu + \gamma)i. \end{aligned} \tag{2}$$

2.2. **Equilibria of the SVIS model.** To understand the role of infected immigrants on the dynamics of System (2), conditions for the existence of positive equilibria with and without infective immigrants are presented here. The case when $p = 0$ was extensively explored in [11], and the disease-free equilibrium in this case is $E_0 = (i^*, v^*) = (0, \frac{\phi}{\mu+\theta+\phi})$. Also, $E_0$ is locally asymptotically stable as long as the reproduction number,

$$\Re(\phi) = \frac{\beta(\mu + \theta + \sigma\phi)}{(\gamma + \mu)(\mu + \theta + \phi)} := \Re_0 \left[1 - \frac{(1 - \sigma)\phi}{\mu + \theta + \phi}\right], \tag{3}$$

is less than one, where $\Re_0 \equiv \frac{\beta}{\gamma+\mu} = \Re(0)$ [11]. The introduction of vaccination implies that $\Re(\phi) \leq \Re_0$ and, consequently, if $\Re_0 < 1$, then $\Re(\phi) < 1$ when $\phi > 0$. From equation (3) one can deduce that

$$\sigma\Re_0 = \frac{\sigma\beta}{\gamma + \mu} \leq \Re(\phi) \leq \Re_0,$$

since $\phi \geq 0$ and $0 \leq \sigma \leq 1$. Thus if $\frac{\sigma\beta}{\gamma+\mu} > 1$ (i.e., $\sigma > \sigma_c \equiv \frac{\gamma+\mu}{\beta}$), then $\Re(\phi) > 1$, and therefore no amount of vaccination can bring $\Re(\phi)$ below 1 [2]. Here $\sigma_c$ defines the critical value for the vaccine-related reduction rate of infection. Furthermore, setting $\Re(\phi) = 1$ and solving for $\phi$ give the threshold vaccination rate, $\phi_c$:

$$\phi_c = \frac{(\Re_0 - 1)(\mu + \theta)}{1 - \sigma\Re_0},$$

where $\gamma + \mu < \beta < \frac{\gamma+\mu}{\sigma}$.

To better understand the role of vaccination, we consider the special case where permanently effective vaccination ($\theta = 0$) begins immediately ($\phi \to \infty$) and infective immigrants exist ($p > 0$). The dynamics in System (2) in this case ($p > 0$) reduce to the study of a single equation, $i' = -\sigma\beta i^2 + (\sigma\beta - \mu - \gamma)i + pa$, which supports two equilibria, $i_1^* \geq 0$ and $i_2^* \leq 0$. The biologically relevant endemic equilibrium is given by

$$i_1^* = \frac{\Re_0^* - 1 + \sqrt{(\Re_0^* - 1)^2 + \frac{4pa}{\gamma+\mu}\Re_0^*}}{2\Re_0^*}, \tag{4}$$

where $\Re_0^* = \frac{\sigma\beta}{\gamma+\mu}$ and the level curves of $i_1^*$ are shown in Figure 2. The formula for $\Re_0^*$ is typical; that is, it is given by the product of the transmission rate, the vaccine efficacy and the mean infective period. We also observe that $\Re_0^* = \sigma\Re(0) = \lim_{\phi\to\infty}\Re(\phi)$ [11]. As $p$ gets close to zero, the limit of $i_1^*$ is

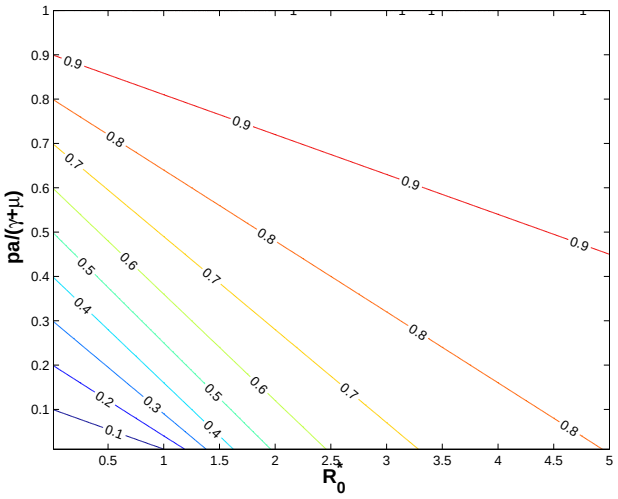


FIGURE 2. The level curves for the fraction of infected population ($i_1^*$) using equation (4).

$$\lim_{p\to 0} i_1^* = \begin{cases} 0 & \text{if } \Re_0^* < 1 \\ 1 - \frac{1}{\Re_0^*} & \text{if } \Re_0^* > 1. \end{cases}$$

For general $\theta$ and $\phi$, the equilibria supported by model (2) can be found from the study of the roots of the cubic polynomial:

$$\mathcal{F}(i,p) = Ei^3 + Bi^2 + Ci + D := i\mathcal{G}(i) - p\sigma ai - pa(\mu+\theta+\phi)/\beta = 0, \tag{5}$$

where

$$\mathcal{G}(i) = Ei^2 + Bi + C_0$$

and

$$\begin{aligned} E =&\beta\sigma, \\ B =&(\mu+\theta+\sigma\phi)+\sigma(\mu+\gamma)-\sigma\beta, \\ C =&(\mu+\gamma)(\mu+\theta+\phi)/\beta-(\mu+\theta+\sigma\phi)-p\sigma a, \\ C_0 =&(\mu+\gamma)(\mu+\theta+\phi)/\beta-(\mu+\theta+\sigma\phi), \\ D =&-pa(\mu+\theta+\phi)/\beta. \end{aligned}$$

Notice that when $p=0$, $\mathcal{F}(i,p)=0$ is reduced to $i\mathcal{G}(i)=0$ and $\mathcal{F}(i,p)<i\mathcal{G}(i)$ for $p>0$. Also, $C_0=(\mu+\theta+\sigma\phi)(\frac{1}{\Re(\phi)}-1)$, and $C_0<0$ is equivalent to $\phi<\phi_c$. There are three cases to be considered (depending on the signs of $B$ and $C_0$) to study the number of positive roots of $\mathcal{F}(i,p)=0$. A similar approach is followed in the study of the $SVIR$ model [2], [3].

*Case 1*: $C_0<0$. In this case, by Descarte's Rule of Signs, $\mathcal{F}(i,p)=0$ has at most one positive root regardless of the sign of $B$, since $E>0$, $C<0$ and $D<0$. $\mathcal{G}(i)=0$ has a unique positive root, $i^*$. It follows that $\mathcal{F}(i^*,p)<0$, since $\mathcal{F}(i,p)<i\mathcal{G}(i)$ for $p>0$. Furthermore, $\mathcal{F}(i,p)\to\infty$ as $i\to\infty$ and $\mathcal{F}(1,p)>0$ when $\Re_0<\frac{\beta}{ap}$, which proves that $\mathcal{F}(i,p)$ has a positive root smaller than one for $p<\frac{\beta}{a\Re_0}$.

*Case 2*: By Descarte's Rule of Signs, $C_0>0$ and $B>0$. $\mathcal{F}(i,p)$ has a positive root smaller than one regardless of the sign of $C$ if $p<\frac{\beta}{a\Re_0}$.

*Case 3*: $C_0>0$ and $B<0$. If $B^2-4EC_0>0$, then $i\mathcal{G}(i)=\mathcal{F}(i,0)=0$ has two positive and one zero roots. Since $\mathcal{F}(i,p)$ is a decreasing function of $p$ for $i>0$, there exists a positive $p^*$ such that $\mathcal{F}(i,p)$ has three positive roots if $0<p<p^*$; two positive roots (with a smaller positive root of multiplicity 2) if $p=p^*$; and a unique positive root if $p>p^*$ where $p^*<p_0$ and

$$p_0=\frac{(\mu+\gamma)(\mu+\theta+\phi)/\beta-(\mu+\theta+\sigma\phi)}{\sigma a}.$$

To see this, suppose $p\geq p_0$. If $\mathcal{F}(i,p)=0$ has exactly one real root, then this must be positive, since $i_1i_2i_3=-D/E>0$. If the roots of $\mathcal{F}(i,p)=0$ are all real, then there is still a unique positive root, since

$$i_1i_2i_3=-D/E>0$$

and

$$i_1i_2+i_2i_3+i_1i_3=\frac{C}{E}=\frac{(\mu+\gamma)(\mu+\theta+\phi)/\beta-(\mu+\theta+\sigma\phi)-p\sigma a}{\beta\sigma}\leq 0$$

when $p\geq p_0$.

These results are summarized in Result 2.1.

RESULT 2.1. *The number of positive endemic equilibria of model (2) is summarized below:*

1. *If $\phi<\phi_c$, then model (2) has a unique positive endemic equilibrium when $p<\frac{\beta}{a\Re_0}$.*
2. *If $\phi>\phi_c$ and $B>0$, then model (2) has a unique positive endemic equilibrium when $p<\frac{\beta}{a\Re_0}$.*
3. *If $\phi>\phi_c$, $B<0$ and $B^2-4EC_0>0$, then there exists a positive $p^*$ such that $\mathcal{F}(i,p)$ has*
   (a) *three endemic equilibria if $0<p<p^*$;*
   (b) *two endemic equilibria (with the smaller of multiplicity 2) if $p=p^*$;*

(c) *a unique endemic equilibrium if* $p > p^*$.

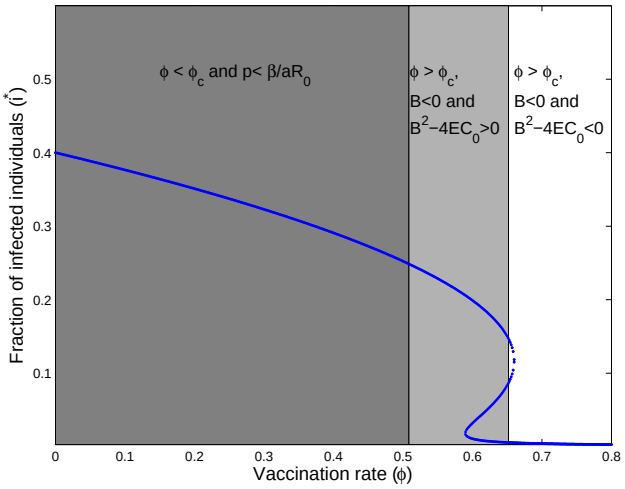


FIGURE 3. Bifurcation diagram of model (2). Parameters used are $\beta = 20$, $\sigma = 0.2$, $\mu = 0.01$, $\gamma = 12$, $\theta = 0.5$, $a = 0.025$ and $p = 0.2$.

To visualize the nature of the endemic steady states, we try to characterize the bifurcation curve using $\phi$ while keeping the parameters $\beta$, $\sigma$, $\mu$, $\gamma$, $\theta$, $a$ and $p$ fixed. Figure 3 shows the impact of vaccination on disease dynamics in the bifurcation diagrams (notice that $\phi_c$ is 0.5087 and that $B > 0$ when $\phi > 5.44$ in this case).

3. **The SVIR model with infective immigrants.** In this section the SVIR (Susceptible-Vaccinated-Infective-Recovered) system analogous to model (1) with recovery of infectives is presented. An analysis of this model is similar to the one of the SVIS model (Section 2) and was carried out as part of my master's thesis [2]. Consequently, only a brief summary of the results as well as additional notes on bifurcation associated with model (6) will be reported. This model (6) can be applied to diseases such as measles and whooping cough, which confer immunity after recovery [3], [15]. Also, analytical results on a similar model have been carried out in the context of influenza [3] under the assumption of acquired *temporary* immunity after natural infection. The assumptions made in Section 2 and the use of the parameters $\gamma$ as the recovery rate lead to the following SVIR model:

$$\begin{aligned} S' &= \Lambda + (1-p)A - \frac{\beta SI}{K} - (\mu+\phi)S + \theta V, \\ V' &= \phi S - \frac{\sigma\beta VI}{K} - (\mu+\theta)V, \\ I' &= pA + \frac{\beta SI}{K} + \frac{\sigma\beta VI}{K} - (\mu+\gamma)I, \\ R' &= \gamma I - \mu R, \end{aligned} \tag{6}$$

with $S(t)+V(t)+I(t)+R(t) = K$ where without loss of generality we take $K = \frac{\Lambda+A}{\mu}$. System (6) is rescaled by the introduction of the following variables: $v = V/K$,

$i = I/K$, $r = R/K$ and $a = A/K$:

$$\begin{aligned} v' &= \phi(1-i-r) - \sigma\beta v i - (\mu+\theta+\phi)v, \\ i' &= pa + \beta i[1-i-(1-\sigma)v - r] - (\mu+\gamma)i, \\ r' &= \gamma i - \mu r. \end{aligned} \tag{7}$$

The study of equilibria of system (7) reduces to the study of the roots of the cubic equation:

$$\mathcal{H}(i,p) = Ei^3 + Bi^2 + Ci + D := i\mathcal{J}(i) - pa\sigma\mu\beta i - pa\mu(\mu+\theta+\phi) = 0, \tag{8}$$

where

$$\mathcal{J}(i) = Ei^2 + Bi + C_0$$

and

$$\begin{aligned} E &= \beta^2\sigma(\mu+\gamma), \\ B &= \beta(\mu+\gamma)(\mu+\theta+\sigma\phi) + \sigma\beta\mu(\gamma+\mu) - \beta^2\sigma\mu, \\ C &= \mu(\mu+\gamma)(\mu+\theta+\phi) - \beta\mu(\mu+\theta+\sigma\phi) - pa\sigma\mu\beta, \\ C_0 &= \mu(\mu+\gamma)(\mu+\theta+\phi) - \beta\mu(\mu+\theta+\sigma\phi), \\ D &= -pa\mu(\mu+\theta+\phi). \end{aligned}$$

The reproduction number for vaccination "policy" ($\phi$), $\Re(\phi)$ (with $p = 0$) is given by [16]:

$$\Re(\phi) = \frac{\beta(\mu+\theta+\sigma\phi)}{(\gamma+\mu)(\mu+\theta+\phi)}, \tag{9}$$

and the basic reproduction number ($\phi = 0$) is [16],

$$\Re(0) \equiv \Re_0 = \frac{\beta}{\gamma+\mu}.$$

That is, $\Re(\phi) \leq \Re(0) = \Re_0$. Note that $\lim_{\phi\to\infty}\Re(\phi) \equiv \sigma\Re_0 \leq \Re(\phi)$ since $0 \leq \sigma \leq 1$. It can be shown that the infection-free equilibrium (when $p = 0$) is locally asymptotically stable if $\Re(\phi) < 1$ and unstable if $\Re(\phi) > 1$ [16].

The number of positive equilibria under the assumption of *temporary* immunity (acquired by recovery) was studied in [3] where the existence of the fold bifurcation was established. On the other hand, if the immunity acquired by recovery is *permanent* (see model (6)), then one can prove the nonexistence of a fold bifurcation (see Result 3.1). In other words, existence of multiple endemic equilibria depends critically on the assumption of the loss of immunity (see [17] for a "similar" scenario).

RESULT 3.1. *System (7) has at most one endemic equilibrium.*

*Proof.* One may see that when $p = 0$, $\mathcal{H}(i,p) = 0$ is reduced to $i\mathcal{J}(i) = 0$ and $\mathcal{H}(i,p) < \mathcal{J}(i)$ for $p > 0$. Depending on the signs of $B$ and $C$ in (8), there are three cases to be considered in the study of the number of positive roots of $\mathcal{H}(i,p) = 0$. *Case* 1 and 2 were covered in [3], so only a brief summary is provided here.

*Case* 1: $C_0 < 0$. $\mathcal{J}(i,p) = 0$ has a unique positive root for all $p > 0$.

*Case* 2: $C_0 > 0$ and $B > 0$. $\mathcal{J}(i,p) = 0$ has a unique positive root for all $p > 0$.

*Case* 3: $C_0 > 0$ and $B < 0$. This case is ruled out for the following reason. It can easily be shown that $C_0 > 0$ is equivalent to $\Re(\phi) < 1$, which implies that

$\sigma\Re_0 < 1$ since $\sigma\Re_0 \leq \Re(\phi)$. Under the assumption $\sigma\Re_0 < 1$ we have

$$\begin{aligned} B &= \beta(\mu+\gamma)(\mu+\theta+\sigma\phi) + \sigma\beta\mu(\mu+\gamma) - \beta^2\sigma\mu \\ &> +\beta(\mu+\gamma)(\mu+\theta+\sigma\phi) + \sigma\beta\mu(\mu+\gamma) - \beta\mu(\mu+\gamma) \ > 0, \end{aligned}$$

which rules out the case of $C_0 > 0$ and $B < 0$. □

Result 3.1 proves that multiple endemic equilibria in system (6) are not possible if the immigration rate of infective individuals is positive. On the other hand, if the immigration rate of infective individuals is negative (i.e., they are emigrants) then a fold bifurcation (although rare) is possible and some examples are shown in [2].

4. **Discussion.** The purpose of this paper is to take a close look at the endemic behavior of SIS and SIR models with infective immigrants and vaccination under standard incidence (bilinear incidence would produce effectively the same qualitative results in the absence of disease-induced mortality). Recent models including vaccination as a control measure show the existence of a fold bifurcation for some parameter values when the vaccine is not totally effective [3], [11], [16]. A basic reproduction ratio can be calculated only in the absence of a constant rate of infective immigrants. In the presence of a fold bifurcation, a basic reproduction number must be reduced below a certain threshold value (i.e., less than one) to eradicate diseases. The examples of a fold bifurcation include HIV/AIDS models in [17], [18], [19] and [20] and the bovine respiratory syncytial virus model in [21].

Under certain conditions, both SVIS and SVIR models *without immigrants* can support two endemic equilibria and a fold bifurcation when $\Re(\phi) < 1$ [11], [16]. A complete eradication of the disease is not possible in the presence of constant (incoming) flow of infectives (an open system). The SVIS model shows that the existence of multiple endemic equilibria (fold bifurcation) needs to be seriously examined to control an outbreak in the presence of infective immigrants. A range of vaccination rates that would preclude the existence of multiple endemic steady states is identified. Strikingly, our SVIR model does not support fold bifurcation unless there is an outflow of immigrants ($A < 0$). Nevertheless, if the immunity acquired by infection is temporary, the SVIR model can still support multiple endemic equilibria and therefore a fold bifurcation [3].

The role of immigration can have dramatic consequences on national public health programs, and the influx of illegal immigrants who may lack access to medical care creates additional challenges. For instance, more than 7,000 individuals in the U.S. were infected by leprosy (or Hansen's disease) between 2002 and 2005, when immigrants brought leprosy from India, Brazil, the Caribbean and Mexico [22]. Also the outbreak of dengue fever (in 1999) in Webb County, Texas, which borders Mexico, was due to infective immigrants or travelers [22]. Other examples of infectious diseases brought into the U.S. by immigrants include polio [22], [23] and tuberculosis [22], [24]. To assess the potential impact of infected immigrants and minimize the outbreaks of such infectious diseases, public health programs should find ways to monitor the flow of infectives into (or from) populations and to collaborate on global control policies. In addition, the possibility of providing access to health care for populations of illegal immigrants should be seriously assessed [25]. Immigrant populations often include a larger percentage of younger males who would tend to be subject to specific diseases that may be driven by age-dependent contact patterns. Hence, the study of demographic patterns is also critical.

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