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FINAL SIZE OF AN EPIDEMIC FOR A TWO-GROUP SIR MODEL*

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Abstract. In this paper we consider a two-group SIR epidemic model. We study the finale size of the epidemic for each subpopulation. The qualitative behavior of the infected classes at the earlier stage of the epidemic is described with respect to the basic reproduction number. Numerical simulations are also preformed to illustrate our results.

Key words. epidemic models, final size, two group, crisscross transmission

AMS subject classifications. 92D25, 92D30

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1. Introduction.

In this article we study a two-group epidemic model. In order to focus on the dynamical properties of an infectious disease itself, here we neglect the demography, namely, the birth and death processes, and the immigration/emigration process. The classical SIR model takes the following form (Anderson and May [1])

$$\left\{\begin{array}{l} \dfrac{dS(t)}{dt} = -\beta S(t)I(t), \\ \dfrac{dI(t)}{dt} = \beta S(t)I(t) - \eta I(t), \\ \dfrac{dR(t)}{dt} = \eta I(t) \end{array}\right. \tag{1}$$

with the initial distributions

$$S(0) = S_0 \in \mathbb{R}_+, I(0) = I_0 \in \mathbb{R}_+, \text{ and } R(0) = R_0 \in \mathbb{R}_+,$$

where $S(t)$ is the number of susceptible individuals, $I(t)$ is the number of infectious individuals (i.e., individuals who are infected and capable of transmitting the disease), $R(t)$ is the number of recovered individuals at time t, respectively. The parameter $\beta > 0$ is called the infection rate (i.e., the contact rate times the probability of infection; see Thieme [40]), and $\eta > 0$ is the recovery rate (i.e., the rate at which infectious individuals recover).

Epidemic models have a long history and starts with the pioneering work of Bernoulli [7] in 1760 in which he aimed at evaluating the effectiveness of inoculation against smallpox. The susceptible-infectious-recovered (SIR) model as we know today takes its origin in the fundamental works on "a priori pathometry" by Ross [38] and Ross and Hudson [37, 36] in 1916–1917 in which a system of ordinary differential

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equations was used to describe the transmission of infectious diseases between susceptible and infected individuals. In 1927–1933, Kermack and McKendrick [22, 23, 24] extended Ross's ideas and model, proposed the cross quadratic term βIS linking the sizes of the susceptible (S) and infectious (I) populations from a probabilistic analysis of the microscopic interactions between infectious agents and/or vectors and hosts in the dynamics of contacts, and established the threshold theorem. Since then epidemic models have been extensively developed in several directions; we refer to the monographs of Bailey [5], Bartlett [6], Muench [31], Anderson and May [1], Busenberg and Cooke [10], Capasso [11], Murray [33], Daley and Gani [13], Mode and Sleeman [30], Brauer and Castillo-Chavez [9], Diekmann and Heesterbeek [15], Thieme [40], and Keeling and Rohani [25] on these topics.

The main tool to understand the dynamical properties of (1) is the following conservation formula

$$\frac{d}{dt}\left[S(t)+I(t)-\frac{\eta}{\beta}\ln(S(t))\right]=0. \tag{2}$$

By exploiting the above conservation formula, Hethcote [19, 20] obtained the following classical result.

THEOREM 1. *Let $(S(t),I(t))$ be a solution of* (1). *If $R_0:=\beta S_0/\eta\le 1$, then $I(t)$ decreases to zero as $t\to+\infty$. If $R_0:=\beta S_0/\eta>1$, then $I(t)$ first increases up to a maximum value $I_{\max}=S_0+I_0-\frac{\eta}{\beta}\ln(S_0)-\frac{\eta}{\beta}+\frac{\eta}{\beta}\ln(\frac{\eta}{\beta})$ and then decreases to zero as $t\to+\infty$. The susceptible $S(t)$ is a decreasing function and the limiting value $S(+\infty)$ is the unique root in $(0,\frac{\eta}{\beta})$ of the equation*

$$S(+\infty)-\frac{\eta}{\beta}\ln(S(+\infty))=S_0+I_0-\frac{\eta}{\beta}\ln(S_0)$$

or, equivalently,

$$\ln\left(\frac{S(+\infty)}{S_0}\right)=R_0\left(\frac{S(+\infty)}{S_0}-1\right)-\frac{R_0}{S_0}I_0. \tag{3}$$

In this article, we focus on a two-group SIR epidemic model. Our motivation is coming from vector borne diseases as well as two-group populations with asymmetric transmission probability or susceptibility. Probably the first example is coming from malaria as well as other disease transmitted mosquitoes [29]. Another example of a population with two subgroups are the male and the female in the context of HIV, since there is the probability of transmission is not the same from male to female than from female to male [26]. Another example of asymmetric probability of transmission is the hospital-acquired infection where the probability of transmission from the health care worker and the patients are not symmetric [14, 28]. The probability of transmission can also be strongly influenced by the coinfection [32, 35]. An example of coinfection is provide by HIV and tuberculosis as well as other diseases, since the susceptibility to tuberculosis of people infected by HIV is much higher than other people [35]. Differences in the susceptibility between individuals can also come from educational campaigns which may influence the susceptibility of individuals [21].

Many examples of application of a two-group (or multigroup) model can be observed practically.

In this article, we will focus on the theoretical aspects of the system of equations for the two group SIR model. We remark that our results for the final size of the two group SIR model are similar to the results given in [34]. Our method of proof, however, is very different, much simpler, and more intuitive for applications. The system considered here is the following:

$$\begin{cases} \dfrac{dS(t)}{dt} = -\mathrm{diag}\,(S(t))\,BI(t), \\ \dfrac{dI(t)}{dt} = \mathrm{diag}\,(S(t))\,BI(t) - EI(t), \\ \dfrac{dR(t)}{dt} = EI(t) \end{cases} \tag{4}$$

with the initial distributions

$$S(0) = S_0 \in \mathbb{R}^2_+, I(0) = I_0 \in \mathbb{R}^2_+, \text{ and } R(0) = R_0 \in \mathbb{R}^2_+,$$

where $S(t)$ are the susceptible, $I(t)$ are the infectious, and $R(t)$ are the recovered individuals and are decomposed according to the populations 1 and 2:

$$S(t) = \begin{pmatrix} S_1(t) \\ S_2(t) \end{pmatrix},\ I(t) = \begin{pmatrix} I_1(t) \\ I_2(t) \end{pmatrix},\ R(t) = \begin{pmatrix} R_1(t) \\ R_2(t) \end{pmatrix},\ t > 0.$$

The recovery of individuals (or quarantine of the infectious) is described by the matrix

$$E = \begin{pmatrix} \eta_1 & 0 \\ 0 & \eta_2 \end{pmatrix}$$

while the transmission of pathogen is described by the matrix

$$B = \begin{pmatrix} \beta_{11} & \beta_{12} \\ \beta_{21} & \beta_{22} \end{pmatrix}.$$

The diagram flux of system (4) is described in Figure 1. System (4) can be rewritten as the following system:

$$\begin{cases} \dfrac{dS_1(t)}{dt} = -S_1(t)(\beta_{11}I_1(t) + \beta_{12}I_2(t)), \\ \dfrac{dS_2(t)}{dt} = -S_2(t)(\beta_{21}I_1(t) + \beta_{22}I_2(t)), \\ \dfrac{dI_1(t)}{dt} = S_1(t)(\beta_{11}I_1(t) + \beta_{12}I_2(t)) - \eta_1 I_1(t), \\ \dfrac{dI_2(t)}{dt} = S_2(t)(\beta_{21}I_1(t) + \beta_{22}I_2(t)) - \eta_2 I_2(t), \\ \dfrac{dR_1(t)}{dt} = \eta_1 I_1(t), \\ \dfrac{dR_2(t)}{dt} = \eta_2 I_2(t). \end{cases} \tag{5}$$

We make the following assumption on the parameters.

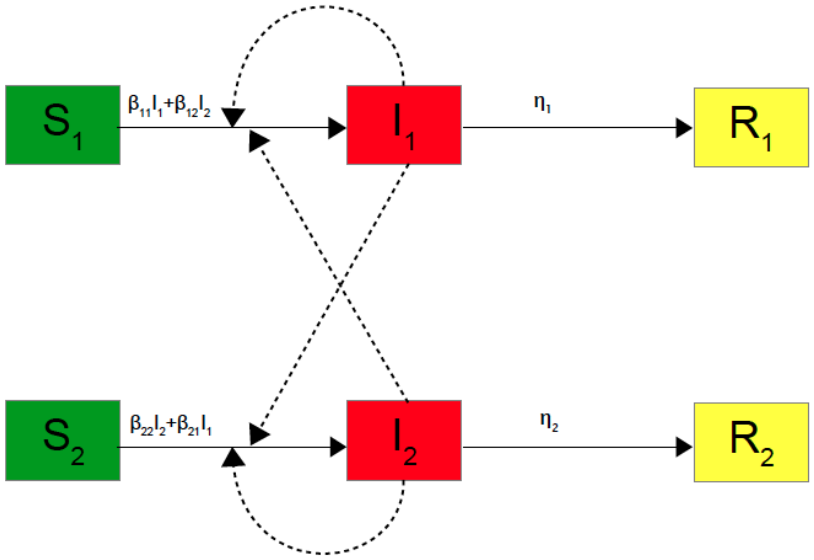

FIG. 1. *The figure represents a transfer diagram of the individual fluxes of system* (4). *In this diagram each solid arrow represents a flux of individuals, while the dashed arrows represent the influence of either infectious of subpopulation* 1 *or infectious of subpopulation* 2.

Assumption 2. We assume that
(i) B is a nonnegative irreducible matrix;
(ii) $\eta_1 > 0$ and $\eta_2 > 0$.

Remark 3. One may observe that B irreducible is equivalent to assuming that

$$\beta_{12} > 0 \text{ and } \beta_{21} > 0.$$

When we assume in addition that the transmission of pathogen occurs by crisscross transmission only (i.e., $\beta_{11} = \beta_{22} = 0$); this of course implies that B is invertible.

One may observe that such a system, SIR, has an infinite number of equilibria. Namely, every three nonnegative vectors

$$\overline{S} \geq 0, \overline{I} = 0, \text{ and } \overline{R} \geq 0$$

is an equilibrium of the system.

Moreover system (4) preserves the total number of individuals in each subpopulation. Namely, for each $t \geq 0$

$$S(t) + I(t) + R(t) = \begin{pmatrix} N_1 \\ N_2 \end{pmatrix}, \tag{6}$$

where $N_1 > 0$ (respectively, $N_2 > 0$) is the number of individuals in subpopulation 1 (respectively, subpopulation 2).

It is trivial to verify that $t \to S(t)$ is nonincreasing and $t \to R(t)$ is nondecreasing (since the solutions are nonnegative). Therefore by using the equality (6) we deduce that the limits

$$\lim_{t\to\infty} S(t) = S^{+\infty}, \lim_{t\to\infty} I(t) = I^{+\infty}, \text{ and } \lim_{t\to\infty} R(t) = R^{+\infty}$$

exist. Moreover the final distribution of infectious $I^{+\infty}$ is 0. The final distribution of susceptible individuals $S^{+\infty}$ is the number of individuals who escape the epidemic. The final distribution of recovered individuals $R^{+\infty}$ is the total number of individuals who have been infected during the epidemic.

We can also rewrite the model (4) by using the fraction of individuals instead of the number of individuals. Consider

$$D := \operatorname{diag}\begin{pmatrix} N_1 \\ N_2 \end{pmatrix},$$

then the fraction of individuals are given by

$$s(t) := D^{-1}S(t), i(t) := D^{-1}I(t), \text{ and } r(t) := D^{-1}R(t)$$

and the model (4) rewrites as

$$\begin{cases} \dfrac{ds(t)}{dt} = -\operatorname{diag}(s(t))\, BDi(t), \\ \dfrac{di(t)}{dt} = \operatorname{diag}(s(t))\, BDi(t) - Ei(t), \\ \dfrac{dr(t)}{dt} = Ei(t). \end{cases} \tag{7}$$

The goal of this article is to extend Theorem 1 to a two-group epidemic model. Actually Theorem 1 can be decomposed into two parts: (1) the computation of the final size of the epidemic; (2) the qualitative behavior of the infected class. As we will see it is possible to extend the first part of Theorem 1 concerning the final size of the epidemic. But we will not be able to describe the qualitative behavior of the infected classes in the two-group case. We should mention the work of Andreasen [2], Arino et al. [3, 4], Ma and Earn [27], and Brauer [8] for some works going into the same direction. To our best knowledge, the computation of the final size of the epidemic for system (1) has not been obtained in the literature. In section 4 we will see an example of numerical simulation showing that the behavior of the infected classes can be more complex for a two-group model than for a single-group model (see Figure 4).

This article is organized as follow. In section 2 we first compute the final size of the epidemic. In the second part of section 2 we describe the behavior of the infectious classes at time $t = 0$ depending on the reproduction number. Section 3 is devoted to numerical simulations. We will conclude this article by considering an application to superspreader in the context of SARS in section 4.

2. Main results.

2.1. Final size of an epidemic. By using the S-equation of (4) we have for each $t \geq 0$

$$\frac{d\ln S(t)}{dt} = -BI(t);$$

therefore

$$\ln(S(t)) - \ln(S(0)) = \int_0^t \frac{d\ln S(s)}{ds} ds = -B \int_0^t I(s) ds \tag{8}$$

and by summing the S-equation and the I-equation we obtain

$$\frac{d(S+I)(t)}{dt} = -EI(t).$$

Hence for each $t \geq 0$

$$(S+I)(t) - (S+I)(0) = \int_0^t \frac{d(S+I)(s)}{ds} = -E \int_0^t I(s) ds \tag{9}$$

and by combining (8)–(9) we obtain

$$\ln(S(t)) - \ln(S(0)) = BE^{-1} \left[(S+I)(t) - (S+I)(0) \right].$$

Therefore the equivalent of formula (2) is the following:

$$\frac{d}{dt} \left[BE^{-1}(S+I)(t) - \ln(S(t)) \right] = 0 \ \forall t \geq 0. \tag{10}$$

By integrating (10) between 0 and $+\infty$ we obtain

$$BE^{-1}(S+I)(+\infty) - \ln(S(+\infty)) = BE^{-1}(S+I)(0) - \ln(S(0))$$

and since $I(+\infty) = 0$ we obtain

$$BE^{-1}S(+\infty) - \ln(S(+\infty)) = BE^{-1}(S+I)(0) - \ln(S(0)).$$

Hence we deduce that $S(+\infty)$ satisfies the following fixed point problem

$$S(+\infty) = \operatorname{diag}(S(0)) \exp\left(BE^{-1} \left[S(+\infty) - V \right] \right), \tag{11}$$

where

$$V := (S+I)(0).$$

The fixed point problem (11) reads to find $0 \leq S(+\infty) \leq S(0)$ satisfying

$$\begin{cases} S_1(+\infty) = S_1(0) \exp\left(\frac{\beta_{11}}{\eta_1} \left[S_1(+\infty) - V_1 \right] + \frac{\beta_{12}}{\eta_2} \left[S_2(+\infty) - V_2 \right] \right), \\ S_2(+\infty) = S_2(0) \exp\left(\frac{\beta_{21}}{\eta_1} \left[S_1(+\infty) - V_1 \right] + \frac{\beta_{22}}{\eta_2} \left[S_2(+\infty) - V_2 \right] \right). \end{cases} \tag{12}$$

In the following we will use the following notations,

$$\begin{aligned} &X \leq Y \Leftrightarrow X_j \leq Y_j \text{ for all } j = 1, 2, \\ &X < Y \Leftrightarrow X \leq Y \text{ and } X_j < Y_j \text{ for some } j = 1, 2, \\ &X \ll Y \Leftrightarrow X_j < Y_j \text{ for all } j = 1, 2. \end{aligned}$$

Consider $T : \mathbb{R}^2 \to \mathbb{R}^2$ is the map defined by the second member of system (12). Namely,

$$T \begin{pmatrix} x_1 \\ x_2 \end{pmatrix} = \begin{pmatrix} T_1(x_1, x_2) \\ T_2(x_1, x_2) \end{pmatrix}$$

with

$$T_1(x_1,x_2) := S_1(0)\exp\left(\frac{\beta_{11}}{\eta_1}[x_1-V_1]+\frac{\beta_{12}}{\eta_2}[x_2-V_2]\right)$$

and

$$T_2(x_1,x_2) := S_2(0)\exp\left(\frac{\beta_{21}}{\eta_1}[x_1-V_1]+\frac{\beta_{22}}{\eta_2}[x_2-V_2]\right).$$

Then it is clear that T is monotone increasing. This means that

$$X \le Y \Rightarrow T(X) \le T(Y) \tag{13}$$

and by using the fact that $\beta_{21} > 0$ and $\beta_{12} > 0$ we obtain

$$X \ll Y \Rightarrow T(X) \ll T(Y). \tag{14}$$

Moreover it is not difficult to see that

$$0 \ll T(0) < T(S(0)) < S(0).$$

Therefore by using induction arguments we deduce that for each $n \ge 1$

$$0 \ll T(0)\cdots \ll T^n(0) \ll T^{n+1}(0) \le T^{n+1}(S(0)) < \cdots < T^n(S(0)) < S(0)$$

so that by taking the limit when n goes to $+\infty$ we obtain

$$0 \ll \lim_{n\to+\infty} T^n(0) =: S^- \le S^+ := \lim_{n\to+\infty} T^n(S(0)) < S(0).$$

Then by continuity of T we have

$$T(S^-) = S^- \text{ and } T(S^+) = S^+.$$

By using the above arguments we obtain the following lemma.

LEMMA 4. *All the fixed points of T in $[0,S(0)]$ are contained in the smaller interval $[S^-,S^+]$.*

The irreducibly of B gives the following property.

LEMMA 5. *If $S^- < S^+$ then $S^- \ll S^+$.*

Proof. Assume, for example, that $S_1^- < S_1^+$. Then since $\beta_{21} > 0$ we have

$$S_2^- = T_2(S_1^-,S_2^-) \le T_2(S_1^-,S_2^+) < T_2(S_1^+,S_2^+) = S_2^+,$$

hence,

$$S_1^- < S_1^+ \Rightarrow S_2^- < S_2^+.$$

Similarly $\beta_{12} > 0$ gives $S_2^- < S_2^+ \Rightarrow S_1^- < S_1^+$. ∎

LEMMA 6. *For each $\lambda > 1$ and $X \gg 0$ we have the following inequality:*

$$T\left(\lambda X + S^-\right) - T\left(S^-\right) \gg \lambda\left[T\left(X + S^-\right) - T\left(S^-\right)\right].$$

Proof. We have

$$T\left(\lambda X + S^-\right) - T\left(S^-\right) = \int_0^1 DT\left(l\lambda X + S^-\right)(\lambda X)\,dl = \lambda\int_0^1 DT\left(l\lambda X + S^-\right)X dl$$

and the differential of T is given by the following formula:

$$DT(X) = \begin{pmatrix} \frac{\beta_{11}}{\eta_1} T_1(x_1, x_2) & \frac{\beta_{12}}{\eta_2} T_1(x_1, x_2) \\ \frac{\beta_{21}}{\eta_1} T_2(x_1, x_2) & \frac{\beta_{22}}{\eta_2} T_2(x_1, x_2) \end{pmatrix}. \tag{15}$$

Since $\lambda > 1$ and $X \gg 0$ we deduce that

$$DT\left(l\lambda X + S^-\right) X \gg DT\left(lX + S^-\right) X \ \forall l \in [0,1].$$

It follows that

$$\begin{aligned} T\left(\lambda X + S^-\right) - T\left(S^-\right) &\gg \lambda \int_0^1 DT\left(lX + S^-\right) X dl \\ &= \lambda \left[T\left(X + S^-\right) - T\left(S^-\right)\right]. \end{aligned}$$

□

THEOREM 7. *The map T has at most two equilibria. More precisely we have the following alternative, either*

(i) *$S^- = S^+$ and T has only one equilibrium in $[0, S(0)]$*
or
(ii) *$S^- \ll S^+$ and the only equilibria of T in $[0, S(0)]$ are S^- and S^+.*

Proof. Assume that $S^- \neq S^+$. Then $S^- < S^+$ which implies $S^- \ll S^+$. Assume that there exists $\overline{X} \in [S^-, S^+]$ a fixed point T such that

$$S^- \neq \overline{X} \text{ and } \overline{X} \neq S^+.$$

Then by using the same arguments as in Lemma 5 we deduce that

$$S^- \ll \overline{X} \ll S^+.$$

Define

$$\gamma := \sup\left\{\lambda \geq 1 : \lambda\left(\overline{X} - S^-\right) + S^- \leq S^+\right\}.$$

Since $\overline{X} \ll S^+$ this implies that

$$\gamma > 1.$$

We have

$$\gamma\left(\overline{X} - S^-\right) + S^- \leq S^+$$

and by applying T on both side of this last inequality we obtain

$$T\left(\gamma\left(\overline{X} - S^-\right) + S^-\right) \leq S^+.$$

By using Lemma 6 we have

$$T\left(\gamma\left(\overline{X} - S^-\right) + S^-\right) - T\left(S^-\right) \gg \gamma\left[T\left(\left(\overline{X} - S^-\right) + S^-\right) - T\left(S^-\right)\right] = \gamma\left[\overline{X} - S^-\right];$$

therefore,

$$S^+ \geq T\left(\gamma\left(\overline{X} - S^-\right) + S^-\right) \gg \gamma\left[\overline{X} - S^-\right] + S^-$$

which contradict the definition of γ. □

In the rest of this section we will focus on the case

$$S^- \ll S^+.$$

By using formula (15) we deduce that

$$DT\left(S^{\pm}\right) = \begin{pmatrix} \frac{\beta_{11}}{\eta_1} S_1^{\pm} & \frac{\beta_{12}}{\eta_2} S_1^{\pm} \\ \frac{\beta_{21}}{\eta_1} S_2^{\pm} & \frac{\beta_{22}}{\eta_2} S_2^{\pm} \end{pmatrix}. \tag{16}$$

LEMMA 8. *The spectral radius of the matrices $DT(S^-)$ and $DT(S^+)$ satisfy the following property:*

$$r\left(DT\left(S^-\right)\right) < 1 < r\left(DT\left(S^+\right)\right).$$

Proof. We observe that

$$S^+ - S^- = T\left(S^+\right) - T\left(S^-\right) = T\left(\left(S^+ - S^-\right) + S^-\right) - T\left(S^-\right)$$

$$= \int_0^1 DT\left(l\left(S^+ - S^-\right) + S^-\right)\left(S^+ - S^-\right) dl$$

and since $S^+ - S^- \gg 0$ we have

$$DT\left(S^+\right)\left(S^+ - S^-\right) \gg \int_0^1 DT\left(l\left(S^+ - S^-\right) + S^-\right)\left(S^+ - S^-\right) dl$$

$$\gg DT\left(S^-\right)\left(S^+ - S^-\right).$$

Therefore

$$DT\left(S^+\right)\left(S^+ - S^-\right) \gg \left(S^+ - S^-\right) \gg DT\left(S^-\right)\left(S^+ - S^-\right)$$

and since both matrices are nonnegative and irreducible the result follows by using the Perron–Frobenius theorem. ∎

THEOREM 9 (final size of the epidemic). *Let*

$$S(0) = S_0 \gg 0 \text{ and } I(0) = I_0 > 0.$$

Then the final size of an epidemic of model (4) *is given by*

$$\lim_{t\to+\infty} S(t) = S^-, \quad \lim_{t\to+\infty} I(t) = 0, \text{ and } \lim_{t\to+\infty} R(t) = \begin{pmatrix} N_1 \\ N_2 \end{pmatrix} - S^-.$$

Remark 10. Due to the above theorem and due to the approximation formula $S^- = \lim_{n\to+\infty} T^n(0)$, it is clear that we can compute numerically the final size of the epidemic.

Proof. If $S^- = S^+$ there is nothing to prove. Otherwise, let

$$S^- \ll S^+.$$

Assume that

$$\lim_{t\to+\infty} S(t) = S^+.$$

We can rewrite the I-equation of system (5) as

$$\frac{dI(t)}{dt} = \begin{bmatrix} S_1(t)\beta_{11} & S_1(t)\beta_{12} \\ S_2(t)\beta_{21} & S_2(t)\beta_{22} \end{bmatrix} I(t) - EI(t)$$

and since $t \to S(t)$ is decreasing we have

$$\frac{dI(t)}{dt} \geq \begin{bmatrix} S_1^+\beta_{11} & S_1^+\beta_{12} \\ S_2^+\beta_{21} & S_2^+\beta_{22} \end{bmatrix} I(t) - EI(t) = \left[\begin{pmatrix} \frac{\beta_{11}}{\eta_1}S_1^+ & \frac{\beta_{12}}{\eta_2}S_1^+ \\ \frac{\beta_{21}}{\eta_1}S_2^+ & \frac{\beta_{22}}{\eta_2}S_2^+ \end{pmatrix} - I \right] EI(t).$$

By using the theory of monotone dynamical systems, we deduce that

$$I(t) \geq Y(t) \ \forall t \geq 0, \tag{17}$$

where $Y(t)$ is the solution of the ordinary differential equation

$$\frac{dY(t)}{dt} = \begin{bmatrix} S_1^+\beta_{11} & S_1^+\beta_{12} \\ S_2^+\beta_{21} & S_2^+\beta_{22} \end{bmatrix} Y(t) - EY(t) \text{ for all } t \geq 0$$

and

$$Y(0) = I(0) > 0.$$

By using (16), we have

$$\begin{bmatrix} S_1^+\beta_{11} & S_1^+\beta_{12} \\ S_2^+\beta_{21} & S_2^+\beta_{22} \end{bmatrix} - E = \left[\begin{pmatrix} \frac{\beta_{11}}{\eta_1}S_1^+ & \frac{\beta_{12}}{\eta_2}S_1^+ \\ \frac{\beta_{21}}{\eta_1}S_2^+ & \frac{\beta_{22}}{\eta_2}S_2^+ \end{pmatrix} - I \right] E = \left[DT\left(S^+\right) - I \right] E.$$

Moreover the matrix $DT\left(S^+\right)$ is nonnegative irreducible, so by the Perron–Frobenius theorem, we can find $W = (W_1, W_2)$ with

$$W \gg 0$$

and such that

$$W DT\left(S^+\right) = r\left(DT\left(S^+\right)\right) W.$$

We have

$$\frac{dWY(t)}{dt} = \lambda W E Y(t),$$

where $\lambda := \left[r\left(DT\left(S^+\right)\right) - 1 \right]$. By Lemma 8 we know that $\lambda > 0$, hence,

$$\frac{dWY(t)}{dt} \geq \min\left(\eta_1, \eta_2\right) \lambda W Y(t)$$

and since

$$WY(0) = WI(0) > 0$$

this implies that

$$\lim_{t\to+\infty} WY(t) = +\infty.$$

This gives a contradiction with (17) and the fact that $\lim_{t\to+\infty} I(t) = 0$. ∎

2.2. Basic reproduction number. We can also extend the result for the basic reproduction number of the general case. We define R_0 the basic reproduction number as the spectral radius of

$$L := \operatorname{diag}(S_0)\, BE^{-1}.$$

More precisely following the next generation method [16, 41] we have

$$L = \begin{pmatrix} \dfrac{S_{10}\beta_{11}}{\eta_1} & \dfrac{S_{10}\beta_{12}}{\eta_2} \\ \dfrac{S_{20}\beta_{21}}{\eta_1} & \dfrac{S_{20}\beta_{22}}{\eta_2} \end{pmatrix} \text{ and } R_0 = r(L). \tag{18}$$

Since L is nonnegative and irreducible, by using the Perron–Frobenius theorem we can find a left eigenvector $W = (W_1, W_2)$ and a right eigenvector $V = \binom{V_1}{V_2}$ such that

$$W \gg 0 \text{ and } V \gg 0$$

with

$$W\operatorname{diag}(S_0)\, BE^{-1} = R_0 W \text{ and } \operatorname{diag}(S_0)\, BE^{-1}V = R_0 V.$$

Recall that the I-equation in system (4) is given by

$$\frac{dI(t)}{dt} = \operatorname{diag}(S(t))\, BI(t) - EI(t) = [\operatorname{diag}(S(t))\, BE^{-1} - I]EI(t),\ t \geq 0.$$

Then the following lemmas hold true.

LEMMA 11. *Assume that $EI(0)$ is proportional to V, the eigenvector associated with the dominant eigenvalue (i.e., R_0) of the matrix* $\operatorname{diag}(S(0))BE^{-1}$. *Then at time $t = 0$*

$$\frac{dI(0)}{dt} = (R_0 - 1)EI(0).$$

Moreover if we assume that $R_0 > 1$ and $EI(0)$ is proportional to V, then both components $I_1(t)$ and $I_2(t)$ are increasing locally around $t = 0$. Similarly, if we assume that $R_0 < 1$ and $EI(0)$ is proportional to V then both components $I_1(t)$ and $I_2(t)$ are decreasing locally around $t = 0$.

Furthermore for any initial distribution $I(0)$ we have

$$\begin{aligned} &W\frac{dI(0)}{dt} = (R_0 - 1)WEI(0) \\ &\Leftrightarrow W_1\frac{dI_1(0)}{dt} + W_2\frac{dI_2(0)}{dt} = (R_0 - 1)\left(W_1\eta_1 I_1(0) + W_2\eta_2 I_2(0)\right). \end{aligned}$$

Remark 12. It is obvious to see that when $R_0 > 1$ we always have at least one component increasing locally around $t = 0$. Indeed when $R_0 > 1$ we may obtain very complex dynamics at the onset of the epidemic (See Figure 4).

Note that the explicit form of the I-equation in system (4) is given by

$$\begin{cases} \dfrac{dI_1(t)}{dt} = S_1(t)\left(\beta_{11}I_1(t) + \beta_{12}I_2(t)\right) - \eta_1 I_1(t), \\ \dfrac{dI_2(t)}{dt} = S_2(t)\left(\beta_{21}I_1(t) + \beta_{22}I_2(t)\right) - \eta_2 I_2(t), \end{cases}$$

which is equivalent to

$$\begin{cases} \dfrac{dI_1(t)}{dt} = \left[S_1(t)\beta_{12}\frac{I_2(t)}{I_1(t)} - (\eta_1 - \beta_{11}S_1(t))\right] I_1(t), \\ \dfrac{dI_2(t)}{dt} = \left[S_2(t)\beta_{21}\frac{I_1(t)}{I_2(t)} - (\eta_2 - \beta_{22}S_2(t))\right] I_2(t). \end{cases} \tag{19}$$

By using the above system we also deduce the following lemma.

LEMMA 13. *Let $S_1(0) > 0$ and $S_2(0) > 0$ be fixed. Assume that $R_0 > 1$. Then the following properties hold true:*

(i) *If $\eta_1 > \beta_{11}S_1(0)$ then by choosing $\frac{I_2(0)}{I_1(0)}$ small enough, the map $I_1(t)$ is decreasing and $I_2(t)$ is increasing locally around $t = 0$.*

(ii) *If $\eta_2 > \beta_{22}S_2(0)$ then by choosing $\frac{I_1(0)}{I_2(0)}$ small enough, the map $I_2(t)$ is decreasing and $I_1(t)$ is increasing locally around $t = 0$.*

2.3. Relationship between the final size and R_0. In this section we will give the relationship between the final size of the epidemic and R_0 defined in (18). More precisely we give a generalization of (3) for our two-group SI epidemic model. Recall that

$$\ln(S(t)) - \ln(S_0) = BE^{-1}\left(S(t) + I(t) - S_0 - I_0\right) \ \forall t \geq 0. \tag{20}$$

Then since $I(+\infty) = 0$ by letting t goes to $+\infty$ in (20) we obtain

$$\ln(S(+\infty)) - \ln(S_0) = BE^{-1}\left(S(+\infty) - S_0 - I_0\right). \tag{21}$$

Hence using the fact that $L = \mathrm{diag}(S_0)BE^{-1}$ we obtain

$$\mathrm{diag}(S_0)\left[\ln(S(+\infty)) - \ln(S_0))\right] = L\left(S(+\infty) - S_0 - I_0\right).$$

Finally recalling that L is an irreducible matrix and $R_0 = r(L)$ we can find a left eigenvector $W = (W_1, W_2) \gg 0$ such that $WL = R_0W$ providing that

$$W\mathrm{diag}(S_0)\left[\ln(S(+\infty)) - \ln(S_0)\right] = R_0W\left(S(+\infty) - S_0 - I_0\right). \tag{22}$$

Note that (22) generalized the relation between R_0 and the final size of the epidemic for the one dimensional SIR model. In fact for the one dimensional SIR model we trivially have $\mathrm{diag}(S_0) = S_0$ and since W becomes a positive real number we trivially obtain

$$\ln\left(\frac{S(+\infty)}{S_0}\right) = R_0\left(\frac{S(+\infty)}{S_0} - 1\right) - \frac{R_0}{S_0}I_0.$$

3. Numerical simulations. In this section we illustrate the theoretical results obtained in section 2 as well as the complex dynamic that can exhibit a two-group

SIR model at an earlier stage of the epidemic. Here we will restrict our attention to the crisscross model, namely, when $\widehat{\beta}_{11} = \widehat{\beta}_{22} = 0$.

3.1. Final size of the epidemic. In Figures 2–3 we plot some phase plane representations of the solutions. These simulations illustrate Theorem 9 about the final size of the epidemic. In all these figures the parameters $\widehat{\beta}_{11} = \widehat{\beta}_{22} = 0$, $\widehat{\beta}_{12}$, $\widehat{\beta}_{21}$, η_1, and η_2 and the initial fractions of infectious are fixed while the initial values are varying with different constraints.

3.2. Behavior of the infectious classes. Figure 4 shows that the number of infected are not always either (1) decreasing or (2) increasing and then decreasing.

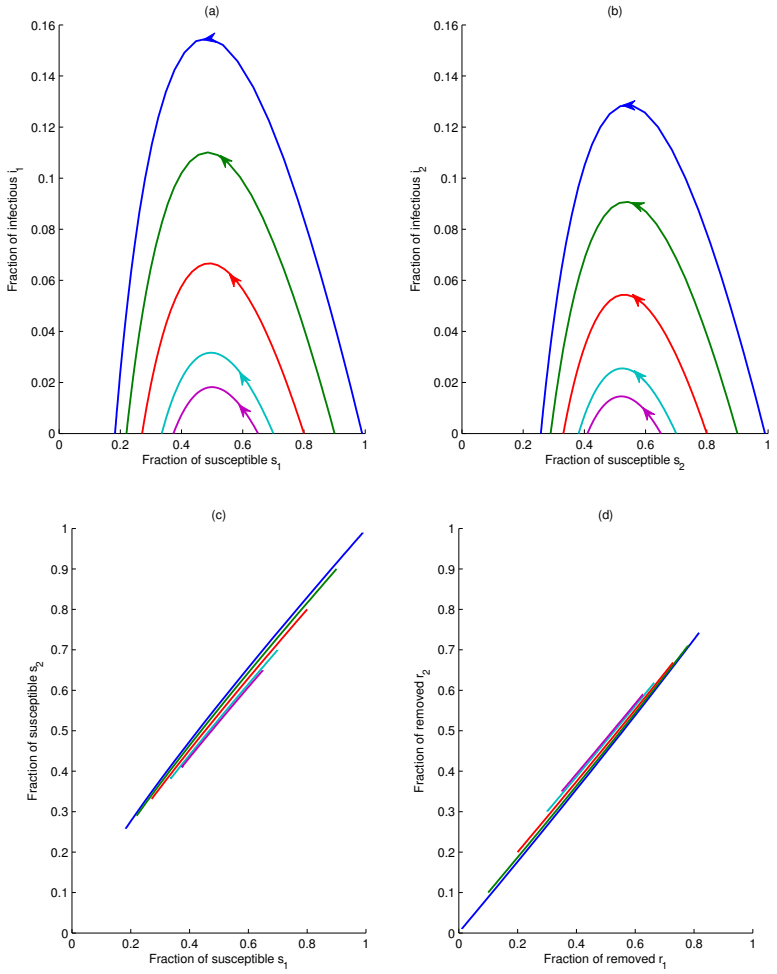

FIG. 2. (a) *(respectively,* (b)*) represents the evolution of the fraction of susceptible* s_1 *of subpopulation* 1 *(respectively,* s_2 *of subpopulation* 2*) with respect to the fraction of infectious* i_1 *of subpopulation* 1 *(respectively,* i_2 *of subpopulation* 2*).* (c) *(respectively,* (d)*) represents the evolution of the fraction of susceptible* s_2 *(respectively, removed* r_2*) of subpopulation* 2 *with respect to the fraction of susceptible* s_1 *(respectively, removed* r_1*) of subpopulation* 1*. We fix* $\widehat{\beta}_{11} = \widehat{\beta}_{22} = 0$*;* $\widehat{\beta}_{12} = 0.3$*;* $\widehat{\beta}_{21} = 0.2$*;* $\eta_1 = 0.12$*, and* $\eta_2 = 0.13$*. The fraction of infectious of each subpopulation is fixed with* $i_{10} = i_{20} = 10^{-5}$*. The fractions of susceptible takes different values with the constraint* $s_{10} = s_{20}$ *while the fraction of removed satisfies* $r_{10} = 1 - s_{10} - i_{10}$ *and* $r_{20} = 1 - s_{20} - i_{20}$*.*

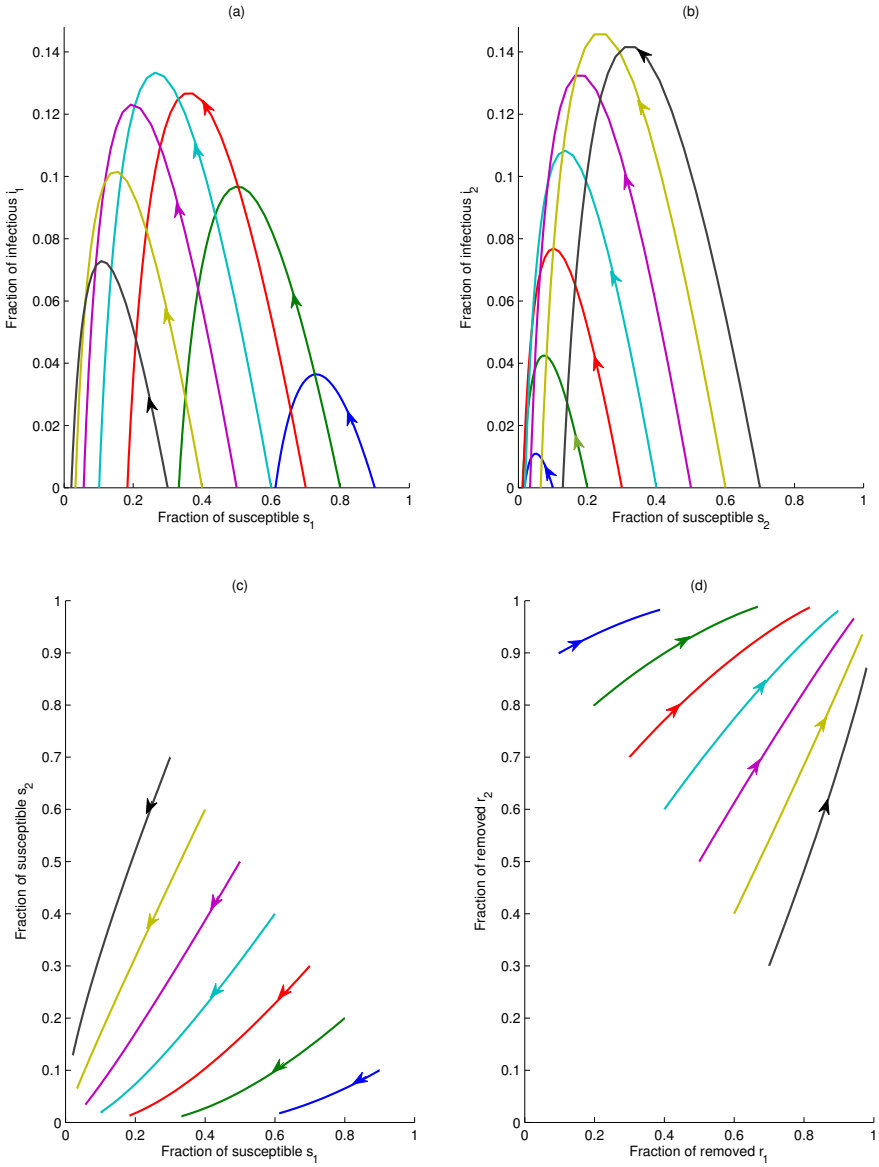

FIG. 3. (a) *(respectively,* (b)*) represents the evolution of the fraction of susceptible* s_1 *of subpopulation* 1 *(respectively,* s_2 *of subpopulation* 2*) with respect to the fraction of infectious* i_1 *of subpopulation* 1 *(respectively,* i_2 *of subpopulation* 2*).* (c) *(respectively,* (d)*) represents the evolution of the fraction of susceptible* s_2 *(respectively, removed* r_2*) of subpopulation* 2 *with respect to the fraction of susceptible* s_1 *(respectively, removed* r_1*) of subpopulation* 1*. We fix* $\widehat{\beta}_{11} = \widehat{\beta}_{22} = 0$*;* $\widehat{\beta}_{12} = 0.7$*;* $\widehat{\beta}_{21} = 0.91$*;* $\eta_1 = \eta_2 = 0.15$*. The fraction of infectious of each subpopulation is fixed with* $i_{10} = i_{20} = 10^{-5}$*. The fractions of susceptible takes different values with the constraint* $s_{10} + s_{20} = 1$ *while the fraction of removed satisfies* $r_{10} = 1 - s_{10} - i_{10}$ *and* $r_{20} = 1 - s_{20} - i_{20}$*.*

More precisely, the map $i_1(t)$ is first decreasing, then increasing to reach a peak and finally decreases to 0. This shows that the dynamic of the infectious classes is more complex in a two-group model than with a single group.

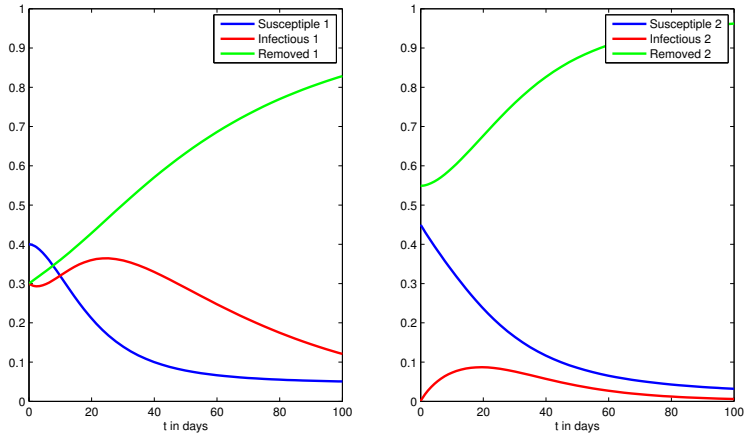

FIG. 4. *In this figure we plot the fraction of susceptible (blue line), the fraction of infectious (red line), and the fraction of removed (green line) for system* (7). *The subpopulation* 1 *is represented on the left side and the subpopulation* 2 *is represented on the right side. We fix* $\widehat{\beta}_{11} = \widehat{\beta}_{22} = 0$*;* $\widehat{\beta}_{12} = 0.5$*;* $\widehat{\beta}_{21} = 0.1$*;* $\eta_1 = 0.02$*;* $\eta_2 = 0.1$*;* $s_{10} = 0.4$*;* $i_{10} = 0.3$*;* $r_{01} = 0.3$*;* $s_{20} = 0.45$*;* $i_{20} = 0.001$*;* $r_{20} = 0.549$. *Here* $R_0 = 2.1213 > 1$. *The map* $i_2(t)$ *is decreasing, then increasing, and finally decreases to* 0. *The kind of behavior does exist for a single population model.*

4. The role of superspreaders in the 2003 SARS epidemic in Singapore. In this section we will subdivide the population into two classes; the superspreader individuals and the nonsuperspreader individuals. In the context of epidemiology the superspreader individuals are known as the 20/80 rule (i.e., 20% of the individuals within any given population are thought to contribute at least 80% to the transmission potential of a pathogen. Namely, the superspreaders have the capacity to infect more susceptible than other usual infectious individuals). We refer to Stein [39] for a nice survey on this topic. Here we focus on the role of superspreader in the context of the SARS outbreak in Singapore in 2003 according to the CDC [12]. We subdivide the population into two classes: the first class of individuals outside the hospital and the second class of individuals inside the hospital (patients and health care workers). We consider $S_1(t)$ (respectively, $I_1(t)$), the number of susceptible (respectively, infectious) outside the hospital at time t. We also consider $S_2(t)$ (respectively, $I_2(t)$), the number of susceptible (respectively, infectious) inside the hospital at time t. The number of new infected (per day) has been reported in [12]. The data used from this report go forward from March 25, 2003 to April 27, 2003. The superspreaders were patients, healthcare workers, and others in hospital and healthcare settings. They were responsible for approximately 75% of the approximately 200 total reported cases. In Figure 5 we plot the daily reported number of new infected inside and outside the hospital.

In order to investigate this epidemic we will reconsider the two-group model

$$\begin{aligned} S_1'(t) &= -S_1(t)(\beta_{11}I_1(t) + \beta_{12}I_2(t)),\\ S_2'(t) &= -S_2(t)(\beta_{21}I_1(t) + \beta_{22}I_2(t)),\\ I_1'(t) &= S_1(t)(\beta_{11}I_1(t) + \beta_{12}I_2(t)) - \eta_1 I_1(t),\\ I_2'(t) &= S_2(t)(\beta_{21}I_1(t) + \beta_{22}I_2(t)) - \eta_2 I_2(t), \end{aligned} \tag{23}$$

where $\beta_{11} = 0.00008$ is the infection rate of susceptibles outside the hospital due to infectious cases outside the hospital, $\beta_{12} = 0.00006$ is the infection rate of susceptibles

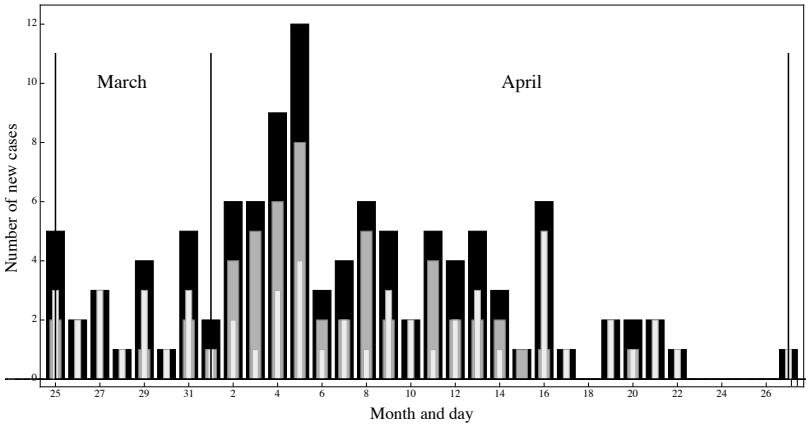

FIG. 5. *Case data from March* 25, 2003 *to April* 27, 2003 [12]. *Light gray bars: new* I_1 *cases (outside the hospital); dark gray bars: new* I_2 *cases (inside the hospital); black bars: total new cases.*

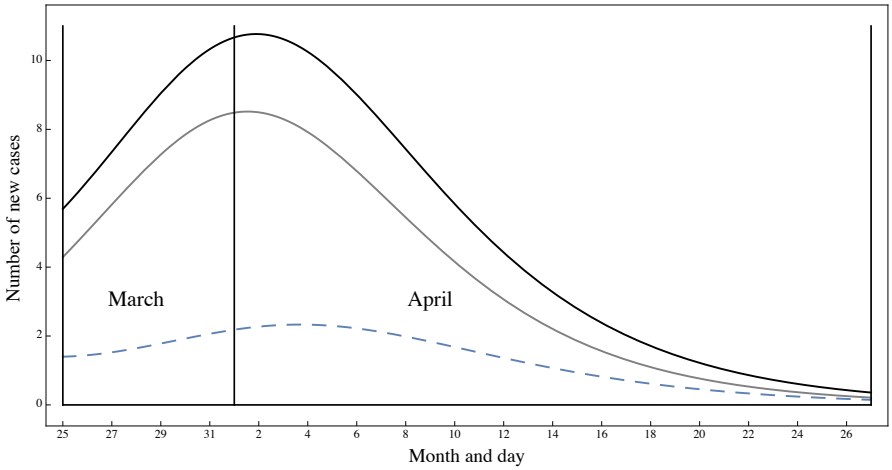

FIG. 6. *New cases from March* 25, 2003 *to April* 27, 2003. *Gray dashed graph: new* I_1 *cases (outside the hospital); gray solid graph: new* I_2 *cases (inside the hospital); black graph: total new cases. The simulation aligns with the data in the CDC report.*

outside the hospital due to infectious cases inside the hospital, $\beta_{21} = 0.00006$ is the infection rate of susceptibles intside the hospital due to infectious cases outside the hospital, $\beta_{22} = 0.0028$ is the infection rate of susceptibles intside the hospital due to infectious cases inside the hospital, $\eta_1 = 0.4$ is the removal rate of infectious cases outside the hospital (average infectious period = 2.5 days) and $\eta_2 = 0.66667$ is the removal rate of infectious cases inside the hospital (average infectious period = 1.5 days). These parameters were chosen to provide a reasonable fit to the data.

The initial distribution of population used in the simulation is the following:

$$S_1(0) = 2,000, \quad S_2(0) = 300, \quad I_1(0) = 5, \text{ and } I_2(0) = 5.$$

In Figures 6 and 7 we present a simulation of the model for the number of new infected and the cumulative number of case, respectively.

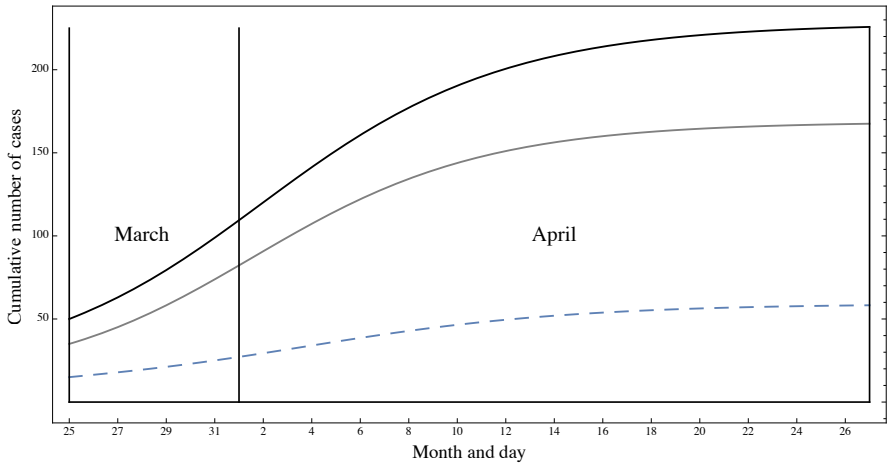

FIG. 7. *Cumulative cases from March* 25*,* 2003 *to April* 27*,* 2003*. Gray dashed graph: cumulative I_1 cases (outside the hospital); gray solid graph: cumulative I_2 cases (inside the hospital); black graph: total cumulative cases. The simulation aligns with the data in the CDC report.*

The two-group model of this SARS epidemic assists understanding of the reasons that the epidemic extinguished very rapidly in Singapore. The superspreaders were responsible for most of the cases which occurred in hospitals among patients and healthcare workers. Outside the hospital settings cases occurred, some caused by hospital cases, but many fewer than in the hospital settings. By the end of March, 2003, the medical community in Singapore understood the serious risk of SARS infection, and adopted stringent measures to control the epidemic in the hospitals. With these measures, which reduced greatly the number of susceptible individuals in hospitals, the number of hospital cases rapidly declined, and the epidemic rapidly extinguished. The two-group model reveals these features of the 2003 SARS epidemic in Singapore.

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