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Global stability of disease-free equilibria in a two-group SI model with feedback control*

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Abstract. In this letter, a two-group SI epidemic model is tackled with an eye to population mobility. Using the method of Lyapunov functions, global stability of the disease-free equilibria with respect to one group as well as both groups is investigated. We find that the disease outbreak can be effectively controlled through adjusting the feedback control variables. Examples are worked out to illustrate the theoretical results.

Keywords: global stability, epidemiological model, feedback control, population mobility.

1 Introduction

As is known, many infectious diseases, such as influenza, HIV/AIDS and SARS, are often highly contagious, inflicting pain on millions of people and involving billions of dollars in health care cost every year. One of the foremost tasks in biomathematics is to study the transmission of infectious diseases quantitatively—hence control the diseases effectively—by establishing mathematical models [5]. Generally, individuals in a population belong to one of the two states: susceptible and infective; the fraction of the population contained in each state is denoted by S and I, respectively. Most of the existing works on SI (and other types of) epidemic models focus on homogeneous population, that is, all individuals respond homogeneously regarding the disease; see e.g., [2,5,9,11,13,19] and references therein.

In this letter, we introduce a general two-group SI epidemic model, which characterizes individual movement between the two groups, signified by A and B. A transfer

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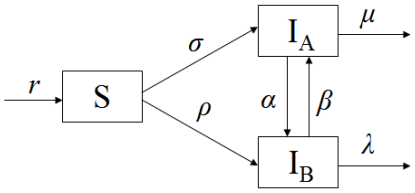


Fig. 1. Flow diagram of the two-group SI model.

diagram for this model is shown in Fig. 1. The model mathematically can be stated as (see Remark 1)

$$\begin{aligned} S'(t) &= S(t)\big(r - \sigma I_A(t) - \rho I_B(t) - aS(t)\big), \\ I'_A(t) &= I_A(t)\big(\sigma S + \beta - \mu - \alpha I_B(t)\big), \\ I'_B(t) &= I_B(t)\big(\rho S + \alpha - \lambda - \beta I_A(t)\big), \end{aligned} \tag{1}$$

where the parameters r, σ, ρ, a, α, β, μ, and λ are all positive constants; r is the recruitment rate of susceptible individuals; σ and ρ represent the infection rates from infected individuals to susceptible individuals in groups A and B, respectively; μ and λ are the death rates of the infectives in groups A and B, respectively; α and β describe "turnover" rates between the two groups. Here, the classification into groups A and B may find its applications not only in obvious physical settings—for example, individuals living in two geographic regions [12] migrate with rates α and β—but also in more remarkably psychological settings—for example, individuals gain and lose disease awareness [3] with rates α and β. It is recently reported that [4, 14] whether possessing awareness or not has pronounced impact on the spread of infectious diseases, which goes some way to highlighting the importance of developing multi-group epidemic models.

Remark 1. The last two equations of (1) more naturally take the following form:

$$I'_A(t) = I_A(t)\big(\sigma S + \beta I_B(t) - \mu - \alpha I_B(t)\big), \tag{2}$$

$$I'_B(t) = I_B(t)\big(\rho S + \alpha I_A(t) - \lambda - \beta I_A(t)\big). \tag{3}$$

We come up with system (1) particularly taking into consideration a couple of things. First, when groups A and B are classified according to whether disease awareness is present, the rates of change in infectives rely on the multiplication of I_A and I_B [8]. This explains why the term $I_A I_B$ appears instead of something like $I_A + I_B$. Second, in view of recent study in swarm dynamics [15] which reveals that an individual in the real world approximately interacts with only a fixed number of neighbors (as opposed to the fully mixed assumption), we simplify $\beta I_A I_B$ (in (2)) and $\alpha I_A I_B$ (in (3)) as βI_A and αI_B in the second and third equations of (1), respectively.

Compartmental epidemic models in the literature such as above are mainly delineated by ordinary differential equations. One of the fundamental questions is to determine the

asymptotical stability of the disease-free equilibrium, which corresponds to the vanishing of a disease in a region. Lyapunov functions are often constructed to obtain the desired global asymptotical stability for the equilibria of the models. Recently, Chen and Sun [1] first deal with the stability of a homogeneous SI epidemic model by introducing feedback control variables—which capture the unpredictable disturbances and uncertain environments in realistic situations—although such control techniques have already been used in ecosystems [7,20]. Some optimal control strategies have also been studied in SIS models [16,17], which have implications in cyber security.

Motivated by the work [1], the system (1) mediated by feedback control takes the following form:

$$\begin{aligned} S'(t) &= S(t)\big(r - \sigma I_A(t) - \rho I_B(t) - aS(t) - cu(t)\big),\\ I'_A(t) &= I_A(t)\big(\sigma S + \beta - \mu - \alpha I_B(t)\big),\\ I'_B(t) &= I_B(t)\big(\rho S + \alpha - \lambda - \beta I_A(t)\big),\\ u'(t) &= -eu(t) + dS(t), \end{aligned} \tag{4}$$

where $u(t)$ is feedback control variable and the parameters c, d and e are positive constants. The initial conditions are

$$S(0) > 0, \qquad I_A(0) > 0, \qquad I_B(0) > 0, \qquad u(0) > 0. \tag{5}$$

The vaccination term here is not a vaccinated population in the context of the controlled epidemic model. It is a feedback vaccination control driven by the susceptible population and with a decreasing transient term due to the initial condition of the vaccination. This is clearly seen by integrating through time the last equation of (4).

Remark 2. The solutions of the models (1) and (4) are always non-negative under the positive initial conditions (5) by the continuity of the state variables (populations and vaccination control) for all time. Indeed, starting from positive initial conditions, the variables continue to be positive always or instead until the corresponding time-derivative is zero. But in this last case, the corresponding time-derivative continues to be zero for all time so that the population remains at the same non-negative value reached at the first time instant at which its time-derivative was zero. Therefore, the model is well-posed and the Lyapunov function candidates used below are also well-posed.

The rest of the letter is organized as follows. In Section 2, we prove the global attractivity of the three concerned disease-free equilibria (with respect to A, B, and both) based on appropriate Lyapunov functions. In Section 3, we present numerical examples to illustrate the effectiveness of the results. Finally, a brief discussion is given in Section 4.

It is worthwhile to mention that there have been some publications considering stability of multi-group epidemic models, see e.g., [6, 10, 18], where nevertheless no feedback control variable is exerted.

2 Stability analysis

In what follows, we focus on the case of moderate turnover rates, i.e., $\beta < \mu$ and $\alpha < \lambda$. One easily checks that the solutions of (4) under initial conditions (5) stay positive for $t > 0$.

The points of equilibrium of the system are the solutions of (4):

$$S'(t) = I'_A(t) = I'_B(t) = u'(t) = 0.$$

Define two threshold values $f = (e\sigma r + (\beta - \mu)ae)/((\mu - \beta)cd)$ and $g = (e\rho r + (\alpha - \lambda)ae)/((\lambda - \alpha)cd)$. There always exists the disease-free equilibrium w.r.t. both groups A and B, which is given by $E^0(S^0, 0, 0, u^0)$ with $S^0 = re/(ae + cd)$ and $u = rd/(ae + cd)$. A simple calculation shows that, if $g > 1$, there exists a unique disease-free equilibrium w.r.t. group A, denoted $E^\wedge(S^\wedge, 0, I_B^\wedge, u^\wedge)$, where $S^\wedge = (\lambda - \alpha)/\rho$, $I_B^\wedge = (r\rho e + (\alpha - \lambda)(ae + cd))/(\rho^2 e)$, and $u^\wedge = d(\lambda - \alpha)/(e\rho)$. Likewise, if $f > 1$, there exists a unique disease-free equilibrium w.r.t. group B, denoted $E^\vee(S^\vee, I_A^\vee, 0, u^\vee)$, where $S^\vee = (\mu - \beta)/\sigma$, $I_A^\vee = (r\sigma e + (\beta - \mu)(ae + cd))/(\sigma^2 e)$, and $u^\vee = d(\mu - \beta)/(e\sigma)$. In the following, we investigate the global attractivity of these disease-free equilibria.

Theorem 1. *If $f < 1$ and $g < 1$, then the disease-free equilibrium $E^0(S^0, 0, 0, u^0)$ is globally asymptotically stable, i.e., the disease dies out w.r.t. both groups A and B.*

Proof. Define the Lyapunov candidate by

$$V(t) = \left(S - S^0 - S^0 \ln \frac{S}{S^0}\right) + I_A + I_B + \frac{c}{2d}\big(u - u^0\big)^2.$$

Along the trajectory of the solution of system (4), we have

$$\begin{aligned} V'(t) &= (S - S^0)\big(-a\big(S - S^0\big) - \sigma I_A - \rho I_B - c\big(u - u^0\big)\big) + I_A(\sigma S + \beta - \mu - \alpha I_B) \\ &\quad + I_B(\rho S + \alpha - \lambda - \beta I_A) + \frac{c}{d}(u - u^0)\big(-e\big(u - u^0\big) + d\big(S - S^0\big)\big) \\ &= -a\big(S - S^0\big)^2 + \left(\beta - \mu + \frac{\sigma r e}{ae + cd}\right) I_A + \left(\alpha - \lambda + \frac{\rho r e}{ae + cd}\right) I_B \\ &\quad - (\alpha + \beta) I_A I_B - \frac{ce}{d}\big(u - u^0\big)^2. \end{aligned}$$

Since $f < 1$ and $g < 1$, it follows that $V'(t) \leqslant 0$. Moreover, $V'(t) = 0$ if and only if $S = S^0$, $I_A = I_B = 0$ and $u = u^0$. Thus, we obtain $\lim_{t\to\infty} S(t) = S^0$, $\lim_{t\to\infty} I_A(t) = \lim_{t\to\infty} I_B(t) = 0$ and $\lim_{t\to\infty} u(t) = u^0$, which conclude the proof. □

Theorem 2. *If $f < 1$ and $g > 1$, then the disease-free equilibrium $E^\wedge(S^\wedge, 0, I_B^\wedge, u^\wedge)$ is globally asymptotically stable, i.e., the disease dies out w.r.t. group A.*

Proof. Define the Lyapunov function by

$$V(t) = \left(S - S^\wedge - S^\wedge \ln \frac{S}{S^\wedge}\right) + I_A + \left(I_B - I_B^\wedge - I_A^\wedge \ln \frac{I_B}{I_B^\wedge}\right) + \frac{c}{2d}(u - u^\wedge)^2.$$

Along the trajectory of the solution of system (4), we obtain

$$V'(t) = -a\big(S - S^{\wedge}\big)^2 - \beta\big(I_B - I_B^{\wedge}\big)^2 + \left(\beta - \mu + \frac{\sigma r e}{ae + cd}\right) I_A - \frac{ce}{d}\big(u - u^{\wedge}\big)^2.$$

Since $f < 1$, it follows that $V'(t) \leqslant 0$. Moreover, $V'(t) = 0$ if and only if $S = S^{\wedge}$, $I_A = 0$, $I_B = I_B^{\wedge}$ and $u = u^{\wedge}$. Consequently, we have $\lim_{t\to\infty} S(t) = S^{\wedge}$, $\lim_{t\to\infty} I_A(t) = 0$, $\lim_{t\to\infty} I_B(t) = I_B^{\wedge}$ and $\lim_{t\to\infty} u(t) = u^{\wedge}$, which conclude the proof. □

Theorem 3. *If $f > 1$ and $g < 1$, then the disease-free equilibrium $E^{\vee}(S^{\vee}, I_A^{\vee}, 0, u^{\vee})$ is globally asymptotically stable, i.e., the disease dies out w.r.t. group B.*

Proof. Consider the following Lyapunov function:

$$V(t) = \left(S - S^{\vee} - S^{\vee} \ln \frac{S}{S^{\vee}}\right) + \left(I_A - I_A^{\vee} - I_B^{\vee} \ln \frac{I_A}{I_A^{\vee}}\right) + I_B + \frac{c}{2d}\big(u - u^{\vee}\big)^2.$$

Along the trajectory of the solution of system (4), we obtain

$$V'(t) = -a\big(S - S^{\vee}\big)^2 - \alpha\big(I_A - I_A^{\vee}\big)^2 + \left(\alpha - \lambda + \frac{\rho r e}{ae + cd}\right) I_B - \frac{ce}{d}\big(u - u^{\vee}\big)^2.$$

The rest of the proof is in parallel with that of Theorem 2. □

Remark 3. At this stage, it might be tempting to conclude that, if $f > 1$ and $g > 1$, then the endemic equilibrium $E^*(S^*, I_A^*, I_B^*, u^*)$, where $S^* = (r\alpha\beta - \alpha\sigma(\alpha - \lambda) - \beta\rho(\beta - \mu))/((\alpha+\beta)\sigma\rho + \alpha\beta(a + cd/e))$, $I_A^* = (\sigma S^* + \beta - \mu)/\alpha$, $I_B^* = (\rho S^* + \alpha - \lambda)/\beta$ and $u^* = dS^*/e$, is globally asymptotically stable. This, however, is not true; by using the Lyapunov function $V(t) = (S - S^* - S^* \ln(S/S^*)) + (I_A - I_A^* - I_B^* \ln(I_A/I_A^*)) + (I_B - I_B^* - I_A^* \ln(I_B/I_B^*)) + c/(2d)(u - u^*)^2$, it is straightforward to check that E^* is locally asymptotically stable. Numerical computation in Fig. 2d further shows that the ultimate value of susceptible state can be either $S^{\wedge}$, or $S^{\vee}$, or S^* depending on the initial conditions.

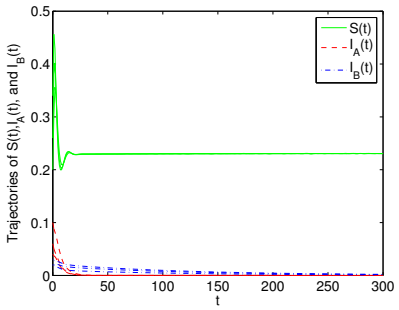
3 Numerical examples

Consider the system (4) with parameters $r = 1$, $\sigma = 0.6$, $\rho = 0.4$, $a = 1$, $\mu = 1.5$, $\lambda = 1$, $\alpha = 0.9$, and $\beta = 1.2$. We calculate that $f = e/(cd)$ and $g = 3e/(cd)$.

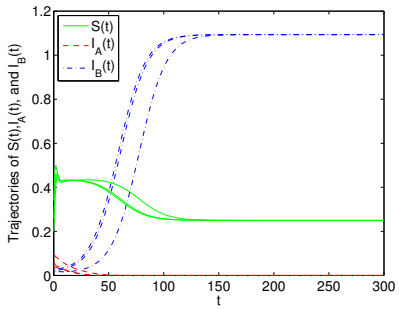
(i) Take $c = d = 1$ and $e = 0.3$. Then $f = 0.3 < 1$ and $g = 0.9 < 1$. We derive the disease-free equilibrium w.r.t. A and B as $E^0(0.2308, 0, 0, 0.7692)$. Figure 2a shows the dynamical behavior of the system, which is consistent with Theorem 1.

(ii) Take $c = d = 1$ and $e = 0.8$. Then $f = 0.8 < 1$ and $g = 2.4 > 1$. We derive the disease-free equilibrium w.r.t. A as $E^{\wedge}(0.25, 0, 1.0938, 0.3125)$. Figure 2b shows the dynamical behavior of the system, which agrees with Theorem 2.

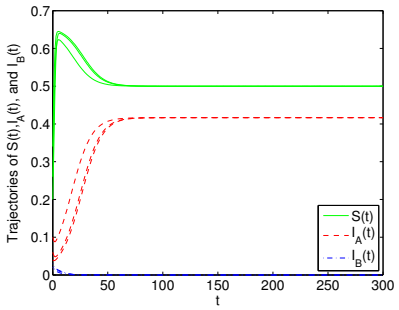
Reducing α to 0.7 yields $f = e/(cd)$ and $g = e/(3cd)$.



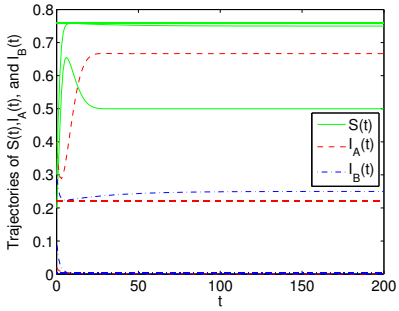
(a) The disease fades out in both A and B when $f < 1$ and $g < 1$.



(b) The disease fades out only in A when $f < 1$ and $g > 1$.



(c) The disease fades out only in B when $f > 1$ and $g < 1$.



(d) The disease may fade out or become endemic when $f > 1$ and $g > 1$.

Fig. 2. (a)–(c) Trajectories of system (4) under initial conditions $(0.34, 0.04, 0.02, 0.1)$, $(0.26, 0.1, 0.02, 0.1)$, and $(0.2, 0.06, 0.03, 0.2)$. (d) Trajectories of system (4) under initial conditions $(0.76, 0.222, 0.0026, 0.15)$, $(0.3, 0.02, 0.3, 0.1)$, and $(0.2, 0.4, 0.1, 0.2)$. System parameters are specified as in (i), (ii), (iii), and (iv), respectively.

(iii) Take $c = d = 1$ and $e = 2$. Then $f = 2 > 1$ and $g = 0.6667 < 1$. We derive the disease-free equilibrium w.r.t. B as $E^{\vee}(0.5, 0.4167, 0, 0.25)$. Figure 2c displays the dynamical behavior of the system, which again agrees with Theorem 3.

(iv) Take $c = d = 1$ and $e = 5$. Then $f = 5 > 1$ and $g = 1.6667 > 1$. We derive the endemic equilibrium and the two disease-free equilibria as $E^*(0.7582, 0.2213, 0.0028, 0.1516)$, $E^{\wedge}(0.75, 0, 0.25, 0.15)$, and $E^{\vee}(0.5, 0.6667, 0, 0.1)$, respectively. Figure 2d indicates that the ultimate state can be any of the three equilibria depending on the initial conditions.

4 Discussion

To summarize, we propose a generic two-group SI epidemic model with feedback control. By constructing Lyapunov functions, global stability of the disease-free equilibria

is tackled. The simulations above reveal that the outbreaks of disease in two groups can be finely tuned by choosing appropriate values of feedback control variables. Meanwhile, we find two critical threshold values f and g, which together determine the persistence or extinction of the disease.

In the present work, simple feedback control variable involving only susceptible state is considered. Although it proves to be very effective, design of more complex feedback is certainly interesting. On the other hand, we know that time-delay widely exists in realistic systems. Unmodelled delay effects in a feedback mechanism may destabilize an otherwise stable system. We leave the question of delayed feedback control for future study.

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