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# Global asymptotic stability of a compartmental model for a pandemic



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#### KEYWORDS

Influenza; Basic reproduction number; Equilibrium; Lyapunov function; Global asymptotic stability **Abstract** With influenza as a prototype, we propose a compartmental model for a pandemic by taking into account of recruitment. The model has a threshold dynamics. Precisely, when the basic reproduction number  $\mathcal{R}_0 \leq 1$ , the disease free equilibrium is globally asymptotically stable; when  $\mathcal{R}_0 > 1$ , the disease free equilibrium is unstable and there is a unique endemic equilibrium which globally attracts all solutions except the trivial one (the disease free equilibrium). These results are established by applying the LaSalle's invariance principle.

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#### 1. Introduction

Influenza is one of the most common contagious respiratory illnesses caused by viruses related to negative-sense RNA orthomyxovirade family [1]. The virus can spread from person to person through air by coughs, sneezes or from infected surfaces, and by the direct contact of infected persons. It is also able to shift from species to species and to change its form rapidly. This highly spreadable disease causes about three to five million cases of acute respiratory infections and 250,000–500,000 deaths every year worldwide [2,3]. Even in the developed countries such as USA, Europe, and Canada,

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the morbidity and the mortality are very high. As an example, in USA more than 200,000 people are hospitalized from flu complication that results in an average 23,600 (approximately) annual deaths [4].

Anyone infected by flu may have symptoms of fever, sore throat, muscle pains, headache, coughing and fatigue. Individuals incubate the virus for nearly 1–3 days before becoming infectious. The infectious period is generally 3–6 days, and the duration of the disease is typically 2–7 days [5].

Epidemic models are important to study the transmission dynamics of infectious diseases and their future risks to human population, and to seek the optimum prevention and control strategies. They provide us with useful information, such as disease transmission, spread of disease agent, epidemiological trends, and preparedness for the disease outbreak.

Arino et al. [6] argued that "as a general policy in preparing for an outbreak of a disease whose parameters are not yet known, it would be better to use a general compartmental model involving relatively few parameters and not depending critically on the particular as yet unknown setting." As a

1110-256X © 2014 Production and hosting by Elsevier B.V. on behalf of Egyptian Mathematical Society. http://dx.doi.org/10.1016/j.joems.2014.04.001 result, they proposed a compartmental SLIAR epidemic model with influenza being a prototype. This model was built on the assumption that a significant fraction of the infected individuals never develop symptoms (called asymptomatic cases). The people with asymptomatic infection are able to transmit the disease although they do not have any sign of the disease. Therefore, infectious population is divided into two compartments according to whether or not they develop the symptoms after being infected. They calculated the basic reproduction number and obtained the final size relation. In their study, they neglected the important factor of recruitment.

The purpose of this paper is to study the effect of recruitment. It turns out that the dynamics is quite different from that in [6]. The remaining of this paper is organized as follows. First we formulate the model in Section 2. Then, in Sections 3 and 4, we study the stability of the disease free equilibrium and the endemic equilibrium, respectively. The paper concludes with a brief discussion.

#### 2. Model formulation

The total population N(t) is divided into five classes: susceptible (S(t)), latent (L(t)), symptomatically infective (I(t)), asymptomatically infective (A(t)), and recovered (R(t)). It is assumed that there is an incubation period between infection and development of disease before an infected person is being infectious. Thus after being infected the susceptible individuals first move to latent class, then to infectious class (either I(t) or A(t)), and finally progress to recovered class.

To build a concrete model, we make the following assumptions.

- There is a constant recruitment rate  $\Lambda$  into the susceptible class and the natural death rate is  $\mu$ .
- The transmission coefficient of the symptomatic infective is β, whereas the infectiousness due to asymptomatic individuals is reduced by a factor δ.
- The rate of having infectiousness is *k* while the probability being symptomatic infective is *p*.
- The recovered rates for symptomatic and asymptomatic classes are  $r_1$  and  $r_2$ , respectively, and the death rates due to symptomatic and asymptomatic infection are  $d_1$  and  $d_2$ , respectively.

Based on the above assumptions, we can sketch the transmission diagram in Fig. 1. These assumptions lead to the model

$$\frac{dS}{dt} = \Lambda - \lambda_s(t)S - \mu S,$$

$$\frac{dL}{dt} = \lambda_s(t)S - kL - \mu L,$$

$$\frac{dI}{dt} = kpL - r_1 I - (\mu + d_1)I,$$

$$\frac{dA}{dt} = k(1 - p)L - r_2 A - (\mu + d_2)A,$$

$$\frac{dR}{dt} = r_1 I + r_2 A - \mu R,$$
(2.1)

where  $\lambda_s(t) = \beta(I + \delta A)$ . Since the fifth equation in (2.1) is decoupled from the other four equations, we only focus on the first four equations of (2.1) in the sequel, namely,



Fig. 1 The transmission diagram for an *SLIAR* model of influenza.

$$\frac{dS}{dt} = \Lambda - \lambda_s(t)S - \mu S,$$

$$\frac{dL}{dt} = \lambda_s(t)S - kL - \mu L,$$

$$\frac{dI}{dt} = kpL - r_1 I - (\mu + d_1)I,$$

$$\frac{dA}{dt} = k(1 - p)L - r_2 A - (\mu + d_2)A.$$
(2.2)

It is not difficult to show that the feasible region of (2.2)

$$\Gamma = \left\{ (S, L, I, A) \in \mathbb{R}^4_+ : S + L + I + A \leqslant \frac{A}{\mu} \right\}$$

is a positively invariant and attracting set that attracts all solutions of (2.2) with nonnegative initial conditions. For the long term behavior of (2.2), we only consider solutions in  $\Gamma$ . In the following two sections, we study the stability of the disease free equilibrium and the endemic equilibrium.

#### 3. The global asymptotic stability of the disease free equilibrium

It is easy to see that (2.2) has a unique disease free equilibrium  $E^0 = (S_0, 0, 0, 0)$ , where  $S_0 = \Lambda/\mu$ . We first study the local stability of  $E^0$  by linearization.

Let

$$\mathcal{R}_0 = \frac{\beta S_0 k p}{(k+\mu)(r_1+\mu+d_1)} + \frac{\beta \delta S_0 k(1-p)}{(k+\mu)(r_2+\mu+d_2)}$$

Note that  $\mathcal{R}_0$  is called the *basic reproduction number* and it can be calculated by the next generation matrix method [7].

**Theorem 3.1.** The disease free equilibrium  $E^0$  of (2.2) is locally exponentially stable if  $\mathcal{R}_0 < 1$  and is unstable if  $\mathcal{R}_0 > 1$ .

**Proof.** The Jacobian matrix of (2.2) at  $E^0$  is

$$J(E^0) = \begin{bmatrix} -\mu & 0 & -\beta S_0 & -\beta \delta S_0 \\ 0 & -(k+\mu) & \beta S_0 & \beta \delta S_0 \\ 0 & kp & -(r_1+\mu+d_1) & 0 \\ 0 & k(1-p) & 0 & -(r_2+\mu+d_2) \end{bmatrix}.$$

Denote

$$A_{22} = \begin{bmatrix} -(k+\mu) & \beta S_0 & \beta \delta S_0 \\ kp & -(r_1+\mu+d_1) & 0 \\ k(1-p) & 0 & -(r_2+\mu+d_2) \end{bmatrix}.$$

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