Original articles

Global dynamics of a two-strain flu model with delay☆

Zhiting Xu [a,*], Liangcheng Qu [a], Yehui Huang [b]

[a] *School of Mathematical Sciences, South China Normal University, Guangzhou, 510631, PR China*
[b] *School of Computational Science, Zhongkai University of Agriculture and Engineering, Guangzhou, 510225, PR China*

Abstract

In this paper, we deal with the global dynamics of a two-strain flu model with delay. Using the method of Lyapunov functional, we show that if the basic reproduction number is less than one, then both strains die out; but when the number is larger than one, one or both of the strains become endemic. The main results are confirmed by some numerical simulations. The theoretical results obtained here provide some useful information on the impact of the vaccination rate of a single vaccine for one strain on the dynamics of the two strains.

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1. Introduction

Influenza, commonly called the flu, is an infectious disease caused by RNA viruses of the family Orthomyxoviridae, the influenza viruses. It spreads around the world in seasonal epidemics, resulting in about three to five million yearly cases of severe illness and about 250,000 to 500,000 yearly deaths, rising to millions in some pandemic years [16]. Since it causes public-health problems, there is an essential need for more information on understanding the transmission mechanism and control strategies [1,12]. Among various controlling infectious diseases strategies such as vaccination, isolation and the use of treatments, vaccination is considered to be one of the most effective methods. However, vaccines can cause the immune system to react as if the body were actually being infected, and general infection symptoms can appear [2,13,14]. On the other hand, when a virus mutates and resistant strains appear in a population, implementing a vaccine for one strain may affect the spread of other strains [4,16].

It is widely agreed that mathematical modeling is an effective tool for developing strategies to control possible outbreaks of diseases. To investigate such an effect of the vaccination of the current strain towards the newer strain, Rahman and Zou [13] recently developed a two-strain model and studied the effects of a single-strain vaccine on the dynamics of this two-strain model, which follows from the work of Castillo-Chavez et al. [3]. Here we shall briefly review the model proposed by Rahman and Zou [13]. We assume that a type of influenza virus, called strain 1, which is moderate in virulence, prevails in the population and a vaccine is available for this strain. A new strain, called

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strain 2, which is antigenically far related to the existing subtype and which has severe virulence effect, suddenly appears in the same host population. Substantial time is required to produce a safe and effective vaccine for the newer strain, and there is no pre-existing immunity in the population. To model the disease dynamics in such a scenario, we divide the total population N into five compartments: S-susceptible individuals; V-immunized individuals with the vaccination for strain 1; I_1-infected individuals with strain 1; I_2-infected individuals with strain 2; R-recovered individuals. That is, $N = S + V + I_1 + I_2 + R$. Here, for simplicity, as in [13], we assume that there is a constant recruitment into susceptible class through birth and/or immigration, and we assume that there is no double infection. Susceptible individuals are vaccinated with constant rate r for strain 1, and are infected by strains 1 and 2 with transmission coefficients β_1 and β_2, respectively. The vaccinated individuals (V) can also be infected by strain 2 at the rate of k. Once recovered from either strain 1 or 2, an individual remains in recovery class for good. The model introduces the following parameters: Λ-recruitment of individuals; μ-natural mortality rate; $r_i (i = 1, 2)$-recovery rate for infected individuals with stain 1, stain 2, $v_i i = 1, 2$ infection-induced death rate of stain 1, stain 2. All parameters are assumed positive. With the above assumptions, the disease dynamics is described by the following system of ordinary differential equations [13]

$$\begin{aligned}
\dot{S} &= \Lambda - (\beta_1 I_1 + \beta_2 I_2 + \lambda)S, \\
\dot{V} &= rS - (\mu + kI_2)V, \\
\dot{I}_1 &= \beta_1 I_1 S - \alpha_1 I_1, \\
\dot{I}_2 &= \beta_2 I_2 S + kI_2 V - \alpha_2 I_2, \\
\dot{R} &= r_1 I_1 + r_2 I_2 - \mu R,
\end{aligned} \tag{1.1}$$

where $\lambda = r + \mu$, $\alpha_1 = r_1 + v_1 + \mu$ and $\alpha_2 = r_2 + v_2 + \mu$. The readers are referred to [13] for the precise interpretation of the biological implication of (1.1).

Since the last equation of system (1.1) is independent of the other equations, Rahman and Zou [13] analyzed the global dynamics of the model (1.1) without the last equation for R in system (1.1). In particular, they showed that if the basic reproduction number is less than one, then both strains die out; but when the number is large than one, one or both of the strains become endemic depending on the parameter values.

It is well known that time delay should be and has been incorporated into many realistic models in applications [8]. Furthermore, as pointed in [13], it is worthwhile to consider the effect of time delay on vaccine-induced immunity. In fact, there always exists an intracellular phase of the viral life-cycle, defined as the time between infection of strain 1 (or 2) and production of new virus particles. Hence, the study of the effect of time delay on vaccine-induced immunity is an important research topic. In this paper, we present a mathematical model to describe the dynamics of a two-strain flu model with delay along the lines of [3,13], and to investigate the parameters to show how they affect on the infectious disease transmission.

This paper is organized as follows. In Section 2, we formulate a two-strain flu model with delay based on those in [3,13]. Also, the basic properties are discussed in the section. In Section 3, using the method of Lyapunov functional, we study the global stability of the model. Section 4 provides some numeric simulations to illustrate our main theoretical results. The paper ends with a brief remarks.

2. A two-strain flu model with delay and the basic properties

The model we present in this paper is a straightforward modification of (1.1) by incorporating two time delays. Here we assume that the flu virus production occurs after the flu virus entry by the positive constant delays τ_1, τ_2. The productions of I_1 (I_2) at time t is given by the number of newly infected susceptible and infected individuals at time $t - \tau_1$ ($t - \tau_2$), who are still alive at time t. Since we assume the natural mortality rate of the individuals is μ, the probability of surviving the time period from $t - \tau_i$ to t is $e^{-\mu\tau_i}$, $i = 1, 2$. Based on system (1.1), the dynamics of such a two-strain flu model with two delays is thus described by the following delay differential system

$$\begin{aligned}
\dot{S}(t) &= \Lambda - (\beta_1 I_1(t) + \beta_2 I_2(t) + \lambda)S(t), \\
\dot{V}(t) &= rS(t) - (\mu + kI_2(t))V(t), \\
\dot{I}_1(t) &= \beta_1 e^{-\mu\tau_1} I_1(t - \tau_1)S(t - \tau_1) - \alpha_1 I_1(t), \\
\dot{I}_2(t) &= \beta_2 e^{-\mu\tau_2} I_2(t - \tau_2)S(t - \tau_2) + k e^{-\mu\tau_2} I_2(t - \tau_2)V(t - \tau_2) - \alpha_2 I_2(t), \\
\dot{R}(t) &= r_1 I_1(t) + r_2 I_2(t) - \mu R(t).
\end{aligned} \tag{2.1}$$

Here, all of the parameters in system (2.1) are assumed positive.