



A Filippov system describing media effects on the spread of infectious diseases



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ABSTRACT

A Filippov epidemic model with media coverage is proposed to describe the real characteristics of media/psychological impact in the spread of an infectious disease. We extend the existing models by incorporating a piecewise continuous transmission rate to describe that the media coverage exhibits its effect once the number of infected individuals exceeds a certain critical level. Mathematical and bifurcation analyses with regard to the local, global stability of equilibria and local sliding bifurcations are performed. Our main results show that the system stabilizes at either the equilibrium points of the two subsystems or the new endemic state induced by the on–off media effect, depending on the threshold levels. The finding suggests that a previously chosen level of the desired number of infected individuals can be reached when the threshold policy and other parameters are chosen properly.

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1. Introduction

When a type of contagious disease appears and starts to spread, people's response to the threat of disease is dependent on their perception of risk, which is affected by public and private information disseminated widely by the media. Massive news coverage and fast information flow can generate a profound psychological impact on the public. Media communications have played an important role in affecting the outcome of infectious disease outbreaks, such as the 2003 severe acute respiratory syndrome (SARS) and the recent 2009 H1N1 influenza epidemic [1–6]. In particular, the propagation of information and education by media have great influence not only on individual behavior but also on the implementation of public intervention and control measures. If susceptible individuals are alert enough to avoid unnecessary contact with infected individuals or all infected individuals stay in the region being treated and cancel their trips, new transmission will apparently decrease due to reduced contact rates [7,8]. The role of media coverage on the transmission of disease is thus crucial and should be given prominence in the study of disease dynamics.

There has been an increasing interest in models in which the impact of media coverage on disease spread in a given population is considered. Liu et al., in [1], described the impact of media coverage on the transmission coefficient by a decreasing factor, resulting in the transmission coefficient $\beta \exp(-a_1 E - a_2 I - a_3 H)$, where E , I , and H are the numbers of reported exposed, infectious, and hospitalized individuals, respectively. Cui et al., in [3], proposed a compartment model with incidence rate $\mu \exp(-mI)SI$ with $m > 0$ to reflect the impact of media coverage on the transmission. They have shown that Hopf bifurcation can occur when $m > 0$ is sufficiently small. Li et al., in [6], used $\beta_2 I / (m + I)$ to reflect the reduced amount of contact rate through media coverage and formulated an SIS (susceptible–infective–susceptible) epidemic model with incidence rate $(\beta_1 - \beta_2 I / (m + I))SI / N$. Moreover, other forms, such as $(\mu_1 - \mu_2 f(I))SI / (S + I)$, have been formulated to approximate the media-induced incidence rate (see details in [2,4,5]).

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A common assumption for the models in [1–6] is that the impact of media coverage on the transmission of the infectious disease occurs as soon as the disease emerges and remains during the whole process of the disease’s spreading. However, this is not really the case. Mostly, at the initial stages of an emerging infectious disease, both the general individuals and public mass media are unaware of the disease. Media reports, information processing, and individuals’ alerted responses to the information can only arise as the number of infected individuals reaches and exceeds a certain level. For example, the A/H1N1 virus was first reported in two US children in Mexico in March 2009, but health officials have stated that it apparently infected people as early as January 2009 in Mexico [9]. Hence, at the initial stage, the number of A/H1N1 cases and information about early precautionary measures against the disease were not available. With a diseases spreading further and information about infectious disease outbreaks being reported, media coverage about an epidemic disease can encourage the public to take precautionary measures against the disease such as wearing masks, avoiding public places, avoiding travel when sick, and frequent hand washing.

Therefore, it is necessary to refine the existing mathematical models with a media-induced effect to reflect this feature. Our main purpose is then to modify the known models in order to describe the impact of media coverage on disease transmission by introducing a piecewise continuous transmission term. We shall examine the effect of media coverage on disease transmission and to address whether the improved function representing a media-induced effect influences the global dynamics of disease transmission.

The rest of the paper is organized as follows. In Section 2, we propose a Filippov epidemic model to describe the media-induced effect on the transmission of infectious diseases. In Section 3, sliding mode dynamics and the existence of a pseudo-equilibrium are investigated. In Section 4, the bifurcation set and the sliding bifurcation of the proposed system are discussed. The global dynamics under variable threshold levels is examined in Section 5, and a discussion and biological conclusion are given in Section 6. Finally, for readers’ interests, we present a brief introduction to threshold policy in the Appendix.

2. Filippov epidemic model and preliminaries

When the number of infected individuals increases and reaches a certain level I_c , mass media often start to report information about the disease, including ways of transmission, number of infected individuals, and then the public try their best to avoid being infected. This consequently lowers the effective contact, resulting in a reduction in transmission rate which is usually represented by $\exp(-\alpha I)$. We consider a population that is divided into three types: susceptible, infective, and recovered. Let S , I , and R denote the proportions of susceptible, infective, and recovered individuals with respect to the total population of individuals, respectively. Then the model equations are

$$\begin{cases} \frac{dS(t)}{dt} = \mu - \beta \exp(-\alpha \epsilon I) SI - \mu S, \\ \frac{dI(t)}{dt} = \beta \exp(-\alpha \epsilon I) SI - (\mu + \gamma) I, \\ \frac{dR(t)}{dt} = \gamma I - \mu R, \end{cases} \tag{1}$$

with

$$\epsilon = \begin{cases} 0, & \sigma(S, I) < 0, \\ 1, & \sigma(S, I) > 0, \end{cases} \tag{2}$$

where $\sigma(S, I)$ is a given function which may depend on the number of susceptible and infected individuals, and ϵ is a discontinuous control function. But here we choose the special function $\sigma(S, I) = \sigma(I) = I - I_c$, which means that only the number of infected individuals is the index for mass media to exhibit its effect. We note that model (1) with $\epsilon = 0$ was introduced and examined in [10]. All other parameters are positive constants, where μ is the natural birth (death) rate, β denotes the basic transmission rate, and γ represents the removed/recovered rate. Model (1) with (2) reveals a dynamical system subject to a threshold policy (TP), which is referred to as an on–off control or as a special and simple case of variable structure control in the control literature. A more detailed introduction on the TP is presented in the Appendix.

Since the recovered class R does not influence the dynamics of the first and second equations of model (1), we only need to focus on the first two equations in the rest of this work. It is easy to show that

$$\{(S, I) \in R_+^2 | 0 < S(t) + I(t) \leq 1\} \doteq \Omega$$

is an attraction region of system (1). In fact, it follows from model (1) that $S(t) + I(t) + R(t) = 1$ and

$$\left. \frac{dS}{dt} \right|_{S=0} = \mu > 0, \quad \left. \frac{dI}{dt} \right|_{I=0} = 0, \quad \left. \frac{d(S+I)}{dt} \right|_{S+I=1} = \mu - \mu(S+I) - \gamma I < 0,$$

so all solutions will enter into the region Ω and remain in it ultimately. Hence, Ω is an attraction region. The (S, I) phase plane is split into three parts: $G^1 = \{(S, I) \in R_+^2 | \sigma(I) < 0\}$, $G^2 = \{(S, I) \in R_+^2 | \sigma(I) > 0\}$, and the discontinuous boundary

$$\Sigma = \{(S, I) \in R_+^2 | \sigma(I) = 0\}. \tag{3}$$

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