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DYNAMICS OF AN INFECTIOUS DISEASES WITH MEDIA/PSYCHOLOGY INDUCED NON-SMOOTH INCIDENCE

Yanni Xiao and Tingting Zhao

Department of Applied Mathematics
Xi'an Jiaotong University, Xi'an, 710049, China

Sanyi Tang

College of Mathematics and Information Science
Shaanxi Normal University, Xi'an, 710062, China.

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Abstract. This paper proposes and analyzes a mathematical model on an infectious disease system with a piecewise smooth incidence rate concerning media/psychological effect. The proposed models extend the classic models with media coverage by including a piecewise smooth incidence rate to represent that the reduction factor because of media coverage depends on both the number of cases and the rate of changes in case number. On the basis of properties of Lambert W function the implicitly defined model has been converted into a piecewise smooth system with explicit definition, and the global dynamic behavior is theoretically examined. The disease-free is globally asymptotically stable when a certain threshold is less than unity, while the endemic equilibrium is globally asymptotically stable for otherwise. The media/psychological impact although does not affect the epidemic threshold, delays the epidemic peak and results in a lower size of outbreak (or equilibrium level of infected individuals).

1. **Introduction.** During the outbreak of influenza A (H1N1) in 2009, media coverage plays an important role in helping both the government authority make interventions to contain the disease and people response to the disease [14]. Individuals' reactions range from avoiding social contact with infected individuals to wearing protective masks, or vaccination. Human behavior change consequently leads to reduction in number of real susceptible individuals or effective contact rates. The mass media have been used as a way of delivering preventive health messages due to their potential influence on people's behavior [7]. To curb the spread of infectious diseases it is then crucial to examine the role of media coverage on disease outbreaks.

Although, the precise functioning of media coverage of epidemics is not well understood, a number of mathematical models has been formulated to describe

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the impact of media coverage on the transmission dynamics of infectious diseases [19, 11, 10, 24, 25]. To model the reduction in contacts due to behavioral change, as awareness of the presence of the disease, functions of the number of infectives are formulated to response to the reported information of cases [28]. The negative exponential function βe^{-mI} or $\beta e^{-\alpha_1 E-\alpha_2 I-\alpha_3 H}$, where H denotes hospitalized individuals, has been embedded into the transmission term by Cui et al. [10] and Liu et al. [19] to examine the impact of media on the spreading of the disease. While Cui et al. [11], Tchuenche et al. [24] and Sun et al. [23] incorporated non-linear function of the number of infectives, $c(I) = c_1 - c_2 f(I)$, in their transmission term to investigate the effects of media coverage on the transmission dynamics. Liu et al.[19] obtained the potential for multiple outbreaks and sustained oscillations of emerging infectious diseases, and Cui et al. [10] concluded that multiple positive equilibria are possible when the media effect is sufficiently strong.

However, these changes of individual avoidance and contact patterns, as awareness of the presence of the disease, are response to not only the reported information of cases but also the changing rate in case number. During the early stage of outbreak of an emergent infectious disease like A/H1N1 influenza and SARS [18], the reported high growth rate of cases puts great pressure on people and cause people to stay home or wear face masks when going outside. While as disease further spreads, the massive cases number exhibits great influence on people's behaviors. So, during the whole outbreak people are aware of presence of disease either in the case number or in their changing rate or both reported by mass media.

The purpose of this paper is to formulate a particular function to describe the reduction factor induced either by large number of cases (I), or by significant changes in the number of cases (dI/dt). Awareness through media and education plays a tremendous role in changing behaviors or contact patterns, and hence in limiting the spread of infectious disease. Whether this media function induced by both cases and changes in cases affect the size of an epidemic outbreak or the peak time, or bring interesting findings remains unknown, and falls within the scope of this study. We then examine the system with incident function dependent on both cases number and their changing rate. Firstly we convert this implicitly defined system into a explicitly defined system, which is actually a piecewise smooth system [4, 6, 29], by using the property of the Lambert W function [9]. The piecewise smooth system is then theoretically and technically analyzed. Finally the epidemiological conclusions on effect of media coverage are addressed.

2. **The SIR model.** We consider the dynamics of susceptibles S, infecteds I and recovereds R. Let Λ be the (constant) recruitment rate and μ be the natural death rate of the population. Assume that β is basic the transmission coefficient, γ is the recovery rate from infection, and α is disease-related death rate. Such media/psychological impact, as awareness of the presence of the disease, is simply described by an exponential decreasing factor, resulting the transmission coefficient as $\beta e^{-M(I,dI/dt)}$, where

$$M(I, dI/dt) = \max\left\{0, p_1 I(t) + p_2 \frac{dI(t)}{dt}\right\}, \tag{1}$$

where p_1, p_2 are non-negative parameters to measure the effect of media/psychological impact of media reported cases and changing rate. Then we have the model

$$\begin{cases} \dfrac{\mathrm{d}S}{\mathrm{d}t} &= \Lambda - e^{-M(I,dI/dt)}\beta IS - \mu S, \\ \dfrac{\mathrm{d}I}{\mathrm{d}t} &= e^{-M(I,dI/dt)}\beta IS - \gamma I - \mu I - \alpha I, \\ \dfrac{\mathrm{d}R}{\mathrm{d}t} &= \gamma I - \mu R. \end{cases} \tag{2}$$

The choice of M function ensure that it is non-negative and is simply denoted by $M(t)$ afterwards. For simplification we denote $m = \gamma + \mu + \alpha$. It is not difficult to get the system (2) is well defined, that is, any solution initiating from R^3_+ will stay in it and of course is eventually bounded [3]. Note that this is an implicitly defined system, which bring lots of difficulties in theoretical analysis. Can we convert this implicitly defined system into a explicit system? In the following we shall address this issue.

Let $M_1(t) = p_1 I(t) + p_2 dI(t)/dt$. When $M_1(t) > 0$, then $M(t) = M_1(t)$. It follows from the second equation of (2) that we have

$$p_2\left(\frac{\mathrm{d}I}{\mathrm{d}t} + mI\right)\exp\left[p_2\left(\frac{\mathrm{d}I}{\mathrm{d}t} + mI\right)\right] = p_2\beta SI\exp\left[-p_1 I + p_2 mI\right],$$

and then by employing the definition of Lambert W function (see Appendix A and [9]) we have

$$\frac{\mathrm{d}I}{\mathrm{d}t} = \frac{1}{p_2}W\left[p_2\beta SI\exp\left(-p_1 I + p_2 mI\right)\right] - mI. \tag{3}$$

Thus $M(t)$ reads

$$\begin{aligned} M(t) &= M_1(t) = p_1 I + p_2\tfrac{dI}{dt} \\ &= W\left[p_2\beta SI\exp\left(-p_1 I + p_2 mI\right)\right] - (-p_1 I + p_2 mI). \end{aligned} \tag{4}$$

Now we examine the condition under which $M_1(t) > 0$. To this end, we consider $M_1(t) = 0$, which is

$$W\left[p_2\beta SI\exp\left(-p_1 I + p_2 mI\right)\right] - (-p_1 I + p_2 mI) = 0. \tag{5}$$

Following from properties of Lambert W function (5) gives

$$(-p_1 I + p_2 mI)\exp(-p_1 I + p_2 mI) = p_2\beta SI\exp\left(-p_1 I + p_2 mI\right),$$

which yields,

$$S = \frac{-p_1 + p_2 m}{p_2\beta} \doteq S_c. \tag{6}$$

Since $M_1(t)$ defined by (4) is strictly monotone function with respect to S, then we have $M_1(t) > 0$ is equivalent to $S > S_c$.

Then the system (2) without considering dynamics of the removed individuals becomes as follows

$$\begin{cases} \dfrac{\mathrm{d}S}{\mathrm{d}t} &= \Lambda - e^{-\epsilon M_1(t)}\beta IS - \mu S, \\ \dfrac{\mathrm{d}I}{\mathrm{d}t} &= e^{-\epsilon M_1(t)}\beta IS - \gamma I - \mu I - \alpha I, \end{cases} \tag{7}$$

with

$$\epsilon = \begin{cases} 0, & S - S_c \le 0, \\ 1, & S - S_c > 0, \end{cases} \tag{8}$$

where the function $M_1(t) = W\left[p_2\beta SI\exp\left(-p_1I + p_2mI\right)\right] - \left(-p_1I + p_2mI\right)$ and S_c is given in (6). Systems (7) with (8) say that there is a threshold for susceptibles under which no media/psychological impact is present, whilst above which media exhibits impact in reduction in transmission. Note that when a disease breaks out in a human population, not all people are susceptible, the susceptible class is actually the number of individuals who were exposed to the virus. Therefore, when the number of susceptibles is relatively large, possibly reaches a certain level, media/psychological impact would be effective to response to disease outbreak by affecting susceptible behavior changes such as giving up risky behavior or taking precautionary measures.

Let $H(Z) = S - S_c$ with vector $Z = (S, I)^T$, and

$$\begin{aligned} F_{G_1}(Z) &= \left(\Lambda - \beta IS - \mu S,\ \ \beta IS - \gamma I - \mu I - \alpha I\right)^T, \\ F_{G_2}(Z) &= \left(\Lambda - e^{-M_1(t)}\beta IS - \mu S,\ \ e^{-M_1(t)}\beta IS - \gamma I - \mu I - \alpha I\right)^T, \end{aligned}$$

then the model (7) with (8) can be rewritten as following non-smooth system [13]

$$\dot{Z}(t) = \begin{cases} F_{G_1}(Z),\ Z \in G_1, \\ F_{G_2}(Z),\ Z \in G_2, \end{cases} \tag{9}$$

where

$$G_1 = \{Z \in R_+^2 :\ \ H(Z) \le 0\}, \quad G_2 = \{Z \in R_+^2 :\ \ H(Z) > 0\}$$

and $R_+^2 = \{Z = (S, I) :\ S \ge 0, I \ge 0\}$. Note that R_+^2 is the invariant set of system (2). We assume that $-p_1 + p_2m > 0$ holds true, suggesting $S_c > 0$. Otherwise, we have $S_c < 0$, then the set G_1 becomes empty, and the non-smooth system (9) becomes the system $\dot{Z}(t) = F_{G_2}(Z)$, which is smooth one and will be examined in the following section.

We define the switching line Σ by

$$\Sigma = \{Z \in R^2 :\ \ H(Z) = 0\}.$$

From now on, we call non-smooth system (9) defined in region G_1 as system S_{G1}, and defined in region G_2 as system S_{G2}. It is interesting to note that the implicitly defined system (2) can be converted into a piece-wise smooth (PWS) system [5] by using properties of Lambert W function. Many dynamical systems arising in applications are non-smooth such as the occurrence of impacting motion in mechanical systems [8], switchings in electronic circuits [4], and hybrid dynamics in control systems [6]. There are many different approaches to the study of non-smooth dynamics such as complementarity systems [17], differential inclusions [12, 2], and Filippov systems [13]. The system (9), a particular form of Filippov system, can also be theoretically investigated by using general dynamical method but with great mathematical techniques (see details in [29]). The following definitions on all types of equilibria of non-smooth system (9)[5] are necessary throughout the rest paper.

Definition 2.1 A point Z^* is called a regular equilibrium of system (9) if $F_{S_{G1}}(Z^*) = 0,\ H(Z^*) \le 0,$ or $F_{S_{G2}}(Z^*) = 0,\ H(Z^*) > 0$. A point Z^* is called a virtual equilibrium of system (9) if $F_{S_{G1}}(Z^*) = 0,\ H(Z^*) > 0,$ or $F_{S_{G2}}(Z^*) = 0,\ H(Z^*) \le 0$.

3. Global dynamics of PWS system (9).

In this section we pay attention to the global dynamics of non-smooth system (9). To this end we initially examine the dynamics of system S_{G1} and S_{G2} separately.

3.1. **Dynamics of system S_{G1}.** Obviously, the dynamics of system S_{G1} is classic. There is a disease-free equilibrium for system S_{G1}, denoted by $E_0 = (\Lambda/\mu, 0)$. It is easy to get that if $R_0 = \beta\Lambda/(m\mu) < 1$, the equilibrium E_0 is locally stable for system S_{G1}. Note that

$$\frac{\Lambda}{\mu} - S_c = \frac{p_2 m\mu(R_0 - 1) + p_1\mu}{p_2\beta\mu},$$

then, if $R_0 > 1$ the disease-free equilibrium E_0 lies in the region G_2 and is unstable. While $R_0 < 1$, E_0 could lie either in the region G_1 if $p_2 m\mu(1 - R_0) \geq p_1\mu$, or in the region G_2 if $p_2 m\mu(1 - R_0) < p_1\mu$ (as shown in Fig.1).

The interior equilibrium for the system S_{G1}, denoted by $E_1^* = (S_1^*, I_1^*)$ with

$$S_1^* = \frac{m}{\beta},\ I_1^* = \frac{\mu}{\beta}\left(\frac{\Lambda}{m}\frac{\beta}{\mu} - 1\right) = \frac{\mu}{\beta}(R_0 - 1),$$

exists if $R_0 > 1$. Moreover, it is asymptotically stable if it is feasible for the system S_{G1}. Note that $S_1^* > S_c$ holds true, which implies that the equilibrium E_1^* lies in the region G_2 and hence it is a virtual equilibrium. It implies that for $R_0 > 1$, any trajectory starting from the region G_1 follows the system S_{G1} initially, and then enters into the region G_2 due to its stable endemic state E_1^* is virtual. Since trajectories initiating from G_2 follows the system S_{G2}, then the asymptotically stable equilibrium E_1^* can not be reached. Hence there is no closed orbit which is totally in the region G_1 since no interior equilibrium is in it.

3.2. **Dynamics of system S_{G2}.** Note that although system S_{G2} is smooth and explicitly defined system, it is difficult to investigate dynamics of system S_{G2} since the model equations involve the Lambert W function. The system S_{G2} reads

$$\begin{cases} \dfrac{\mathrm{d}S}{\mathrm{d}t} &= \Lambda - e^{-M_1(t)}\beta IS - \mu S, \\ \dfrac{\mathrm{d}I}{\mathrm{d}t} &= e^{-M_1(t)}\beta IS - \gamma I - \mu I - \alpha I, \end{cases} \qquad S > S_c \tag{10}$$

with function $M_1(t)$ given in (4). The disease-free equilibrium for system (10) or S_{G2} exists, which is $(\Lambda/\mu, 0)$, and is coincided with its counterpart for system S_{G1}. We also denote it by E_0. It is easy to get that if $R_0 < 1$, the equilibrium E_0 is locally stable for system S_{G2}.

Existence of endemic equilibrium To show the existence of the endemic equilibrium, we shall change the subsystem (10) into a simple form. For this purpose, in the function $M_1(t)$ we denote

$$f_1(S, I) \triangleq p_2\beta SI\exp(-p_1 I + p_2 mI), \quad g_1(I) \triangleq -p_1 I + p_2 mI,$$

then $M_1(t) = W(f_1(S, I)) - g_1(I)$. According to the properties of Lambert W function, we have

$$\begin{aligned} \exp(-M_1(t)) &= \exp(-W(f_1(S, I)) + g_1(I)) \\ &= \frac{W(f_1(S,I))}{f_1(S,I)}\exp(g_1(I)) = \frac{W(f_1(S,I))}{p_2\beta SI}. \end{aligned} \tag{11}$$

Substituting above equation into the model (10), then subsystem (10) becomes

$$\begin{cases} \dfrac{\mathrm{d}S}{\mathrm{d}t} &= \Lambda - \frac{W(f_1(S,I))}{p_2} - \mu S, \\ \dfrac{\mathrm{d}I}{\mathrm{d}t} &= \frac{W(f_1(S,I))}{p_2} - mI, \end{cases} \qquad S > S_c. \tag{12}$$

Let the endemic equilibrium be denoted by $E_2^* = (S^*, I^*)$. Then $dI/dt = 0$ gives $W(f_1(S,I)) = p_2 mI$. By using property of the Lambert W function we have

$$S^* = \frac{m}{\beta}\exp(p_1 I^*). \tag{13}$$

Substituting it into $dS/dt = 0$ and applying $W(f_1(S,I)) = p_2 mI$ yield

$$\frac{m\mu}{\beta}\exp(p_1 I^*) + mI^* = \Lambda. \tag{14}$$

Rearranging above equation, one yields

$$p_1\left(\frac{\Lambda}{m} - I^*\right)\exp\left[p_1\left(\frac{\Lambda}{m} - I^*\right)\right] = \frac{p_1\mu}{\beta}\exp\left(\frac{p_1\Lambda}{m}\right). \tag{15}$$

According to the definition of Lambert W function and solving above equation with respect to I^* we have

$$I^* = \frac{\Lambda}{m} - \frac{1}{p_1}W\left[\frac{p_1\mu}{\beta}\exp\left(\frac{p_1\Lambda}{m}\right)\right]. \tag{16}$$

It is feasible (i.e. $I^* > 0$) provided

$$\frac{p_1\Lambda}{m} > W\left[\frac{p_1\mu}{\beta}\exp\left(\frac{p_1\Lambda}{m}\right)\right]. \tag{17}$$

which is equivalent to

$$R_0 = \frac{\Lambda\beta}{\mu m} > 1. \tag{18}$$

Substituting (16) into (13) and using the properties of Lambert W function we have

$$\begin{aligned} S^* &= \frac{m}{\beta}\exp(p_1 I^*) = \frac{m}{\beta}\exp\left(\frac{p_1\Lambda}{m}\right)\exp\left[-W\left(\frac{p_1\mu}{\beta}\exp\left(\frac{p_1\Lambda}{m}\right)\right)\right] \\ &= \frac{m}{\beta}\exp\left(\frac{p_1\Lambda}{m}\right)\frac{W\left(\frac{p_1\mu}{\beta}\exp\left(\frac{p_1\Lambda}{m}\right)\right)}{\frac{p_1\mu}{\beta}\exp\left(\frac{p_1\Lambda}{m}\right)} \\ &= \frac{m}{p_1\mu}W\left(\frac{p_1\mu}{\beta}\exp\left(\frac{p_1\Lambda}{m}\right)\right). \end{aligned} \tag{19}$$

Therefore, the endemic state $E_2^* = (S^*, I^*)$ with S^* and I^* defined in (19) and (16) exists if $R_0 > 1$. Note that the susceptible component of both the interior equilibria E_2^* is no less than S_c (that is, $S^* > S_c$), which indicates the E_2^* lies in the region G_2, and hence it is a regular equilibrium.

Local stability of the endemic equilibrium E_2^* In the following we shall check the Jacobian matrix at the endemic equilibrium E_2^* for the subsystem (10). To this end we still consider the system (12) and let $b = -p_1 + p_2 m$, and

$$P_2(S,I) = \Lambda - \frac{W(f_1(S,I))}{p_2} - \mu S,\ Q_2(S,I) = \frac{W(f_1(S,I))}{p_2} - mI,$$

then the Jacobian matrix can be calculated as follows

$$J = \begin{pmatrix} \frac{\partial P_2}{\partial S} & \frac{\partial P_2}{\partial I} \\ \frac{\partial Q_2}{\partial S} & \frac{\partial Q_2}{\partial I} \end{pmatrix} = \begin{pmatrix} -\frac{1}{p_2}F_1 - \mu & -\frac{1}{p_2}F_2 \\ \frac{1}{p_2}F_1 & \frac{1}{p_2}F_2 - m \end{pmatrix}, \tag{20}$$

where F_1 and F_2 are defined by

$$\begin{aligned} F_1 := \frac{\partial W(f_1(S,I))}{\partial S} &= \frac{W(f_1(S,I))}{f_1(S,I)(1+W(f_1(S,I)))}\frac{\partial f_1}{\partial S}, \\ F_2 := \frac{\partial W(f_1(S,I))}{\partial I} &= \frac{W(f_1(S,I))}{f_1(S,I)(1+W(f_1(S,I)))}\frac{\partial f_1}{\partial I}, \end{aligned}$$

with $\partial f_1/\partial S = p_2\beta I\exp(bI)$, $\partial f_1/\partial I = p_2\beta S\exp(bI)(1+bI)$. By simple calculation at equilibrium $E^* = (S^*, I^*)$ we have

$$\begin{aligned} F_1 &= \frac{p_2 mI^*}{f_1(S^*,I^*)(1+p_2mI^*)}p_2\beta I^*\exp(bI^*) = \frac{p_2mI^*}{S^*(1+p_2mI^*)}, \\ F_2 &= \frac{p_2mI^*}{f_1(S^*,I^*)(1+p_2mI^*)}p_2\beta S^*\exp(bI^*)(1+bI^*) = \frac{p_2m(1+bI^*)}{1+p_2mI^*}. \end{aligned} \tag{21}$$

Therefore, the characteristic equation at E_2^* with respect to λ is given as follows

$$\lambda^2 + \left[\frac{F_1}{p_2} - \frac{F_2}{p_2} + \mu + m\right]\lambda + \frac{m}{p_2}F_1 - \frac{\mu}{p_2}F_2 + m\mu = 0$$

with F_1 and F_2 defined in (21). It is easy to determine that this characteristic equation has two negative roots, which means that the interior equilibrium E_2^* is locally asymptotically stable in the region G_2.

The nonexistence of closed orbits We shall exclude existence of closed orbits of system (10) by constructing the suitable Dulac function. Here, we choose the Dulac function $B_2(S,I) = e^{M(S,I)}/SI$ and get

$$\begin{aligned} \frac{\partial(B_2P_2)}{\partial S} + \frac{\partial(B_2Q_2)}{\partial I} &= \frac{\Lambda e^{M_1(S,I)}}{S^2I}\left(\frac{W(f_1)}{1+W(f_1)} - 1\right) - \frac{e^{M_1(S,I)}W(f_1)}{1+W(f_1)}\cdot\frac{\mu}{SI} \\ &\quad - \frac{me^{M_1(S,I)}}{S}\left(\frac{W(f_1)}{1+W(f_1)}\cdot\frac{1+bI}{I} - b\right). \end{aligned} \tag{22}$$

Note that $S > S_c = b/(p_2\beta)$ is equivalent to $f_1 > bIe^{bI}$, which implies $W(f_1) > bI$, then we have

$$\frac{W(f_1)}{1+W(f_1)}\cdot\frac{1+bI}{I} - b > 0.$$

Then, we get $\partial(B_2P_2)/\partial S + \partial(B_2Q_2)/\partial I < 0$, which implies that system (10) does not have closed orbits totally in the region G_2. Therefore, the regular equilibrium E_2^* is globally asymptotically stable in the region G_2.

3.3. **Dynamics of system (9).** When $R_0 < 1$ we know that the disease-free equilibria for system S_{G1} or S_{G2} coincide with each other (i.e. $(\Lambda/\mu, 0)$) and are locally asymptotically stable in the region G_1 or G_2 respectively. Moreover, the disease-free equilibrium could either be in the region G_1 or in G_2. Without loss of generality, assume it lies in the region G_1, then any trajectory starting from G_2 hits the line $S = S_c$ initially and enters into the region G_1; while trajectories initiating from G_1 will finally approach the disease-free equilibrium (as shown in Fig.1).

For $R_0 > 1$, the endemic state E_1^* (or E_2^*) is locally asymptotically stable for system S_{G1} (or S_{G2}) in the region G_1 (or G_2). Moreover, we have shown that system S_{G1} or S_{G2} does not have closed orbits in the region G_1 or G_2, respectively. In the following, we shall show there is no closed orbit of system (9) across the

switching line Σ. Generally speaking, the approach of Dulac functions applies to smooth vector fields. Then whether it can still be applicable to the piecewise smooth system (7) with (8) where the vector field is not smooth at the switching line Σ? Making use of the distribution theory, we extend the classic theory of Bendixson-Dulac in the two-dimensional case to Filippov systems where the vector field is not smooth at the switching line, and a Lemma is given in Appendix B in order to exclude the existence of closed orbits.

Let $x = S - S_c,\ y = I$, then model (7) with (8) becomes

$$\begin{cases} \frac{dx(t)}{dt} = \Lambda - e^{-H(x)M_1^*(t)}\beta(x(t)+S_c)y(t) - \mu(x(t)+S_c) := P(x,y), \\ \frac{dy(t)}{dt} = e^{-H(x)M_1^*(t)}\beta(x(t)+S_c)y(t) - my(t) := Q(x,y), \end{cases} \tag{23}$$

where $M_1^*(t) = W[p_2\beta(x+S_c)y\exp(-p_1y+p_2my)] - (-p_1y+p_2my)$ and the Heaviside function H satisfies

$$H(x) = \begin{cases} 0, & x \le 0, \\ 1, & x > 0. \end{cases} \tag{24}$$

The general derivative of $H(x)$ with respect to x is the Dirac function given as follows

$$\delta(x) = \begin{cases} 0, & x \neq 0, \\ +\infty, & x = 0. \end{cases} \tag{25}$$

Take a Dulac function $B = e^{H(x)M_1^*(t)}/((x(t)+S_c)y(t))$, and from (23) we have

$$\begin{aligned} \mathscr{F} &:= \frac{\partial(BP)}{\partial x} + \frac{\partial(BQ)}{\partial y} \\ &= \frac{\Lambda e^{H(x)M_1^*}}{y(x(t)+S_c)^2}\left(\delta(x)M_1^*(x+S_c) + \frac{H(x)W(f_1^*)}{1+W(f_1^*)} - 1\right) \\ &\quad - \frac{\mu e^{H(x)M_1^*}}{y}\left(\delta(x)M_1^* + \frac{H(x)W(f_1^*)}{(x+S_c)(1+W(f_1^*))}\right) \\ &\quad - \frac{mH(x)e^{H(x)M_1^*}}{x+S_c}\left(\frac{W(f_1^*)}{1+W(f_1^*)}\frac{1+by}{y} - b\right), \end{aligned}$$

where $f_1^* = p_2\beta(x+S_c)y\exp(-p_1y+p_2my)$. Making use of the definition of functions $H(x)$ and $\delta(x)$ and noting that $M_1^* = 0$ for $x = 0$, we get the $\mathscr{F}$ is negative for any x and y (≥ 0) due to

$$\mathscr{F} = \begin{cases} -\frac{\Lambda}{y(x(t)+S_c)^2} < 0, & x < 0, \\ \frac{\Lambda e^{M_1^*}}{y(x(t)+S_c)^2}(A-1) - \frac{\mu e^{M_1^*}}{y}\frac{A}{x+S_c} - \frac{me^{M_1^*}}{x+S_c}\left(\frac{A(1+by)}{y} - b\right) < 0, & x > 0, \\ -\frac{\Lambda}{yS_c^2} < 0, & x = 0, \end{cases} \tag{26}$$

where $A = W(f_1^*)/(1+W(f_1^*))$. Therefore, it follows from the Lemma in the Appendix B that the system (7) with (8) does not have a limit cycle.

In a word, any trajectory starting from G_1 hits the switching line Σ initially and enters into the region G_2 due to its asymptotically stable equilibrium E_1^* is virtual and lies in the region G_2. While trajectories initiating from the region G_2 will finally tend to the endemic equilibrium E_2^* since E_2^* is regular and is globally asymptotically stable in the region G_2 (as shown in Fig.2). We also showed that no closed orbit of system (7) with (8) crosses the switching line Σ, which gives the globally stability of the endemic state E_2^*. Hence we have the following conclusion.

Proposition 3.1 *For the PWS system (9), the disease-free equilibrium E_0 is globally asymptotically stable if $R_0 < 1$, while the positive equilibrium E_2^* is globally asymptotically stable if $R_0 > 1$.*

It is interesting to note that the media parameters p_1 and p_2 affect the disease evolution. To understand how these two media parameters influence the model dynamics, and particular the number of infected individuals we examine variation in the endemic equilibrium with parameters p_1 and p_2. It follows from (16) and (19) that the endemic equilibrium E_2^* is independent on parameter p_2 but on parameter p_1. Further, it is easy to get

$$\begin{aligned} \frac{\partial S^*}{\partial p_1} &= \frac{m}{\mu p_1^2}\frac{W\left(\frac{p_1\mu}{\beta}\exp\left(\frac{p_1\Lambda}{m}\right)\right)}{1+W\left(\frac{p_1\mu}{\beta}\exp\left(\frac{p_1\Lambda}{m}\right)\right)}\left[-W\left(\frac{p_1\mu}{\beta}\exp\left(\frac{p_1\Lambda}{m}\right)\right)+\frac{p_1\Lambda}{m}\right] \\ &> 0 \end{aligned} \tag{27}$$

due to $R_0 > 1$, and hence

$$\frac{\partial I^*}{\partial p_1} = -\frac{\mu}{m}\frac{\partial S^*}{\partial p_1} < 0. \tag{28}$$

This shows that the equilibrium value of I decreases with increasing parameter p_1. Note that p_1 represents the efficacy level of the prevention and control strategies implemented when facing disease prevalence due to media coverage. So, the higher value of parameter p_1 the less the eventual level of the infectives. Therefore, it is important to maximize the impact of communications campaigns that promote protective behavior during the outbreak of diseases [22, 27].

3.4. **Dynamics of system (9) without demography.** It is interesting to examine the media effect for the SIR model without population dynamics and investigate how media/psychology impact influences the size of outbreaks and peak time. Similarly, the system with media effect can also be converted into the following PWS system with $\bar{S}_c := (-p_1 + p_2\gamma)/(p_2\beta)$

$$\begin{cases} \dfrac{\mathrm{d}S}{\mathrm{d}t} &= -e^{-\epsilon M_2(t)}\beta IS, \\ \dfrac{\mathrm{d}I}{\mathrm{d}t} &= e^{-\epsilon M_2(t)}\beta IS - \gamma I, \end{cases} \tag{29}$$

where $M_2(t) = W\left[p_2\beta SI\exp\left(-p_1 I + p_2\gamma I\right)\right] - \left(-p_1 I + p_2\gamma I\right)$ and

$$\epsilon = \begin{cases} 0, & S - \bar{S}_c \le 0, \\ 1, & S - \bar{S}_c > 0. \end{cases} \tag{30}$$

To examine when the number of infected individuals reaches its maximum, let's set $dI/dt = 0$ for $S > \bar{S}_c$, that is,

$$e^{-M_2(t)}\beta IS - \gamma I = 0,$$

which indicates that

$$\exp(W(f_2(S,I))) = \frac{\beta S}{\gamma}\exp(g_2(I))$$

with $f_2(S,I) = p_2\beta SI\exp\left(-p_1 I + p_2\gamma I\right)$, $g_2(I) = -p_1 I + p_2\gamma I$. It follows from the relation of Lambert W function given by (A1) that we have

$$\frac{f_2(S,I)}{W(f_2(S,I))} = \frac{\beta S}{\gamma}\exp(g_2(I)), \tag{31}$$

i.e. $W(f_2(S,I)) = p_2\gamma I$. Solving the equation with respect to S yields

$$S = \frac{\gamma}{\beta}\exp(p_1 I) := \bar{S}_{th}. \tag{32}$$

This shows that any solution of system (29) with initial value satisfying

$$S_0 > \gamma \exp(p_1 I_0)/\beta$$

will reach its maximum value along the curve given by (32). It follows from the classic SIR model without media coverage [1] that the number of infectives reaches its maximum when susceptibles are at the threshold $S_{th} = \gamma/\beta$. Note that $\bar{S}_{th} > S_{th}$, which implies that with media coverage a relatively large threshold for susceptibles is obtained below which outbreak can not occur.

In the absence of media coverage (i.e. $p_1 = p_2 = 0$) the system (29) with (30) becomes the classic SIR model from which we could easily obtain the final size and peak time for the classic SIR model [1]. In the presence of media coverage it is interesting to examine how two parameters (p_1 and p_2) introduced in the definition of media impact in (1) affect disease dynamics such as the final outbreak size and peak time. Unfortunately, we can not get the theoretical results due to quite complicated media signal function $M_2(t)$ in system (29). However, numerical studies on system (29) could suggest some interesting conclusions. We plot the phase plane S-I for both system (29) and the classic SIR model, showing the switching line $S = \bar{S}_c$ and the lines $S = \bar{S}_{th}$ or $S = S_{th}$ where disease peaks in Fig.3(A). The thick and the following thin curves denote the trajectories of system (29), and the dash-dot curves represent trajectories of the classic SIR model. Fig.3(B) shows that media coverage postpones the arrival of the infection peak. It also follows from the Fig.3(A) and (B) that media coverage lowers the outbreak size definitely, and hence decreases the severity of the outbreak. This is in agreement with the conclusion obtained in [25, 15].

We explore the numerical studies to show how sensitive would the peak time and final size be depending on the two parameters p_1 and p_2 in Fig.4. Fig.4(A) shows that peak time increases as parameter p_1 or p_2 increases, which further consolidates our conclusion that media coverage delays the peak of an epidemic. It follows from Fig.4(B) that media coverage results in a smaller final size and hence reduces the severity of an epidemic. It is interesting to note that for relatively small value of parameter p_1 final size declines with increasing p_2, whilst for relatively large p_1 final size increases with increasing p_2. It indicates that parameters p_1 and p_2 have a marked and complicated influence on the final size of an epidemic.

4. **Conclusion and discussion.** It has been observed that media coverage can affect the spread and control of infectious diseases [19]. General speaking, awareness through media and education and consequently changes in behavior play a tremendous role in limiting the spread of infectious disease. In this study, media coverage is described by a piecewise smooth function which is dependent both on the number of cases and the rate of change in cases. Then the resulting system is non-smooth and implicitly defined system, which bring the difficulties for theoretical analysis. By using Lambert W function we initially convert this non-smooth, implicitly defined system into a switching system with explicit definition. It is interesting to note that the switching systems (7) with (8) says the classic SIR system is present when the number of susceptibles is less than a threshold defined in (6), otherwise the nonlinear function of the number of cases is embedded in the incidence rate of the SIR model. It seems that only when the number of susceptibles exceeds a threshold the reduction factor induced by media coverage will be effective.

For this kind of systems with implicit definition, Tchuenche and Bauch [25] directly investigated the original SIHR system, where H denotes hospitalized individuals, and obtained the threshold and local dynamics of the model. Interestingly in this study we initially converted this implicitly defined system (2) into an explicitly defined system (7) with (8) by technically employing Lambert W function. Then we obtained the global dynamics by theoretically analyzing the converted switching systems (7) with (8). The disease-free equilibrium $(\Lambda/\mu, 0)$ is globally asymptotically stable if the basic reproduction $R_0 = \beta\Lambda/m\mu < 1$; while the endemic equilibrium E_2^* is globally asymptotically stable for $R_0 > 1$. The epidemic threshold R_0 is not changed due to media coverage, which is agreement with that obtained by Tchuenche and Bauch [25] and Funk et al. [15]. Comparing the infective components of the endemic states E_1^* for system S_{G1} and E_2^* for system S_{G2} gives that $I^* < I_1^*$. It indicates that the media effect alone cannot drive an endemic disease extinct, but it plays a significant role in reducing the number of infectives and its proportion to the total population. Note that when $R_0 > 1$ trajectories initiating from the region G_1 tend to its stable endemic state E_1^* which lies in the region G_2 and can not be reached. When trajectories cross the switching line Σ they have to follow the dynamics of system S_{G2} and approach the endemic state E_2^* (as shown in Fig.2). Therefore, media effect does not destabilize an endemic steady-state, but can significantly reduce the number of infectives and prevalence.

We note that media coverage does not eradicate the disease because the media signal function fades when the number of cases declines to small value and the changing rate in cases becomes negative, but it limits the spread of infectious disease and contributes in lowering the epidemic size (shown in Fig.3 and Fig.2). When considering demography media coverage does not affect the epidemic threshold in that sense the basic reproduction number R_0 is not changed. It is worthy noting that the peak of an epidemic is postponed due to media/psychology impact no matter whether we consider demography or not. This indicates that although awareness through media or education does not influence the epidemic threshold, it does change the timing of spread, delay the epidemic peak and result in a lower size of outbreak (or low equilibrium level of infectives). In fact, continually updating the media about the number of infected individuals or changing rate in cases will immediately pass on the information to the general population, which causes some susceptible individuals to choose to self-isolate such as using face masks, ceasing risky behavior, using barrier protections and etc. Prompt depletion of susceptibles due to self-isolation could slow down the spread of disease among the population, then postpone the epidemic peak, and result in a dramatic decrease in the severity of the outbreak, which agrees with the conclusion in [25, 15, 20].

Based on the classic SIR-type model Handel et al. [16] found that the best strategy for multiple infectious disease outbreaks was to apply intervention measures in such a way that the number of susceptibles reaches exactly the threshold level. Note that here our results indicate that the threshold level for susceptibles varies with the number of infectives due to media/psychological impact. Therefore, it is important to emphasize that the intervention measures should be adjusted instantaneously such that the number of susceptibles tends to the various threshold levels.

This study is just a preliminary exploration of media/psychological impact on the spread of an infectious disease, and could be improved in many ways. In particular, there inevitably exists a difference between the time when data is collected and the time when audiences get to know it, so it may be reasonable to consider time

delay in the dynamic system. We have assumed in our model that media coverage is always beneficial in mitigating the spread of disease. However, it might also have negative consequences (e.g. messages inciting fears over vaccine safety). So more elaborate forms of the 'media coverage effect function' would be proposed. Further, the data on media coverage is hard to gather and in most cases when available are limited, which greatly affects the prior information on two parameters p_1 and p_2. Hence, we leave fitting our model to the real data as our future study.

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E-mail address: yxiao@mail.xjtu.edu.cn
E-mail address: zhaotingting1116@163.com
E-mail address: sytang@snnu.edu.cn

Appendix A Lambert W function

We initially introduce some properties of Lambert W function.

Definition. The Lambert W function is defined to be a multivalued inverse of the function $z \mapsto ze^z$ satisfying

$$\text{LambertW}(z)\exp(\text{LambertW}(z)) = z. \tag{A1}$$

It follows from above definition that we have

$$\text{LambertW}'(z) = \frac{\text{LambertW}(z)}{z(1+\text{LambertW}(z))}. \tag{A2}$$

First of all, the function $zexp(z)$ has the positive derivative $(z+1)exp(z)$ if $z > -1$. Define the inverse function of $zexp(z)$ restricted on the interval$[-1,\infty)$ to be $\text{LambertW}(0,z) = \text{LambertW}(z)$. For simplicity we use $W(z)$ to represent $\text{LambertW}(z)$ in the main text. Similarly, we define the inverse function of $zexp(z)$ restricted on the interval $(-\infty,-1]$ to be $\text{LambertW}(-1,z)$. For more details of the concepts and properties of the LambertW function, see Corless et al. [9].

Appendix B Nonexistence of closed orbits of the Filippov system

Consider the general Filippov system as follows:

$$\dot{X} = f(X), \tag{A3}$$

where $X = (x,y)'$ and $f = (P,Q)'$. We restrict the properties of the above system under consideration by the following four major assumptions.

(I) Ω is an open region in R^2, which is divided into a finite number of open subregion Ω_i satisfying $\bigcup\overline{\Omega}_i = \overline{\Omega}$.

(II) If $i \neq j$ and $\overline{\Omega}_i$ and $\overline{\Omega}_j$ are not disjoint, then $\overline{\Omega}_i \bigcap \overline{\Omega}_j = \Sigma_{i,j}$, where $\Sigma_{i,j}$ are piecewise smooth.

(III) f is Lipschitz in each Ω_i and discontinuous along $\Sigma_{i,j}$.

(IV) The vector field is directed from one side to the other at $\Sigma_{i,j}$, i.e. only transversal sliding mode exists.

Lemma. *(Extension of the Bendixson-Dulac criterion) Let the conditions (I)-(IV) be satisfied and f be bounded in the simply connected region Ω and C^1 in Ω_i for the Filippov system (A3). If there is a function B which is continuous in $intR^2_+$ and C^1 in Ω_i such that*

$$\frac{\partial(BP)}{\partial x}+\frac{\partial(BQ)}{\partial y}\leq 0(\geq 0),\ (x,y)\in\Omega,$$

and is not identically zero in Ω, which is caculated in distribution sense, then (A3) has no closed orbit in Ω.

Proof. On the basis of the method proposed by Melin [21] we give the simple proof of this lemma. Denote $f(x,y)=f_i(x,y)$, $f_i(x,y)=(p_i(x,y),q_i(x,y))$ and $B(x,y)=B_i(x,y)$, where $(x,y)\in\Omega_i$. Let χ_{Ω_i} be the characteristic function of Ω_i. Then $f=\sum_i f_i\cdot\chi_{\Omega_i}=\sum_i(p_i\cdot\chi_{\Omega_i},q_i\cdot\chi_{\Omega_i})$ and $Bf=\sum_i B_if_i\cdot\chi_{\Omega_i}=\sum_i(B_ip_i\cdot\chi_{\Omega_i},B_iq_i\cdot\chi_{\Omega_i})$. So we have

$$F:=\frac{\partial(BP)}{\partial x}+\frac{\partial(BQ)}{\partial y}=\sum_i\left(\partial_x(B_ip_i\cdot\chi_{\Omega_i})+\partial_y(B_iq_i\cdot\chi_{\Omega_i})\right),\tag{A4}$$

where F is defined in the sense of distribution theory and involves Dirac function. Now, suppose there is a closed, continuous and piecewise smooth orbit Γ in Ω. Let $T=(\dot{x},\dot{y})$ be the tangent vector of Γ, $N=(-\dot{y},\dot{x})$ be the normal vector of Γ, and $D=int\Gamma$. Next, we consider the line integral $\int_\Gamma\langle Bf,N\rangle ds$ in Ω_i, and assume t is in some interval Δ_i. Since

$$\int_\Gamma\langle B_if_i\cdot\chi_{\Omega_i},N\rangle ds=\int_{\Delta_i}(-B_ip_i\cdot\dot{y}+B_iq_i\cdot\dot{x})dt=\int_{\Delta_i}(-B_ip_i\cdot q_i+B_iq_i\cdot p_i)dt=0,$$

we can get that

$$\int_\Gamma\langle Bf,N\rangle ds=\int_\Gamma\left\langle\sum_i B_if_i\cdot\chi_{\Omega_i},N\right\rangle ds=\sum_i\int_\Gamma\langle B_if_i\cdot\chi_{\Omega_i},N\rangle\,ds=0.$$

However, according to the Green formula it can be seen that

$$\int_\Gamma\langle Bf,N\rangle ds=-\iint_D F dxdy,$$

which yields $\iint_D F dxdy=0$. This contradicts to the assumption of F, then we exclude the existence of closed orbits. This completes the proof. □

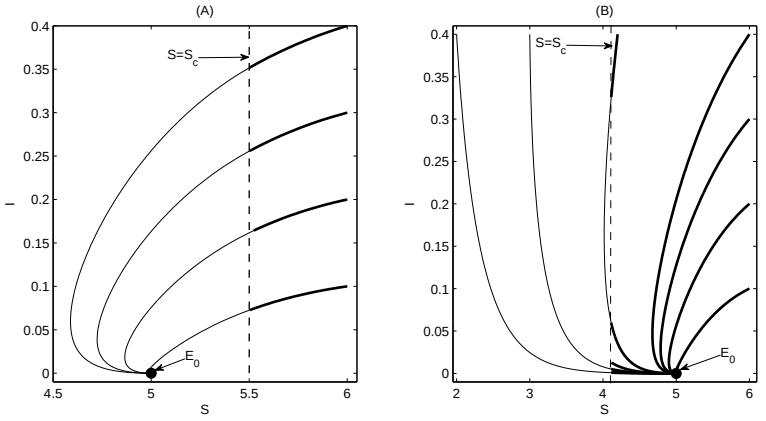

FIGURE 1. Phase plane S-I for SIR model, showing the global stability of the disease-free equilibrium. The thick and thin curves represent the general orbits in the phase plane for the system (7) with (8) in G_2 and in G_1. Parameters are $\beta = 0.3, \gamma = 1.5, \Lambda = 1, \mu = 0.2, \alpha = 0.2$ and (A) $p_1 = 0.2, p_2 = 0.8$; (B) $p_1 = 0.4, p_2 = 0.6$.

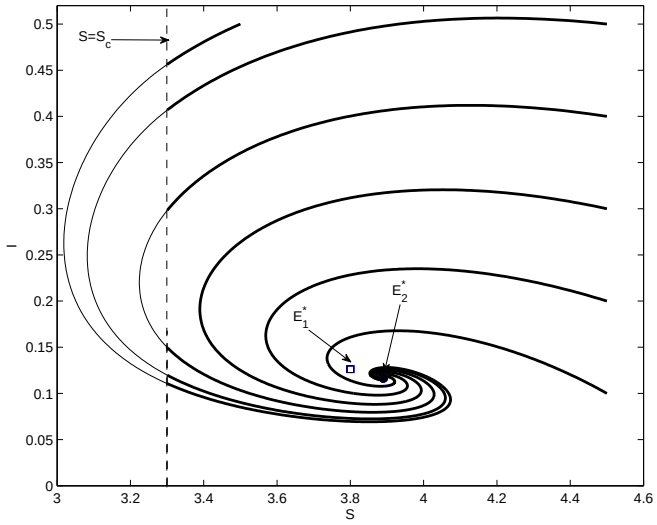

FIGURE 2. Phase plane S-I for SIR model, showing the global stability of the endemic equilibrium. The thick and thin curves represent the general orbits in the phase plane for the system (7) with (8) in G_2 and in G_1. Parameters are $\beta = 0.5, \gamma = 1.5, \Lambda = 1, \mu = 0.2, \alpha = 0.2, p_1 = 0.2, p_2 = 0.8$.

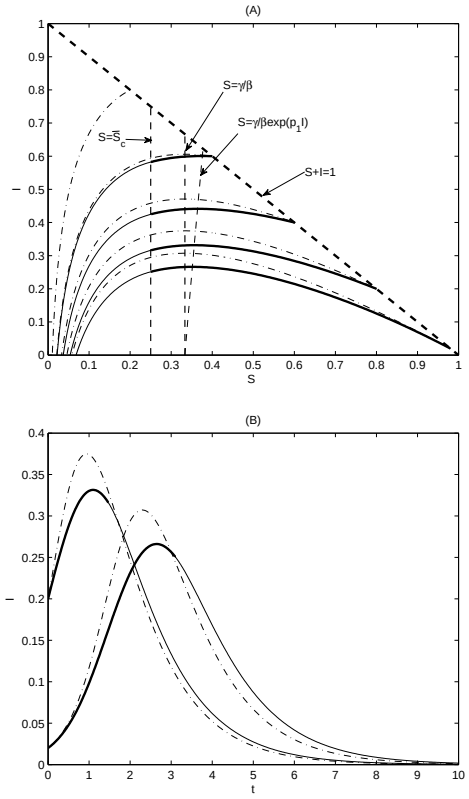

FIGURE 3. A comparison of the SIR model with media coverage (29) with (30) and the classic SIR model (i.e. $p_1 = p_2 = 0$ in system (29)). (A) Phase plane S-I for two SIR models, showing the switching line $S = \bar{S}_c$ and the lines $S = \gamma \exp(p_1 I)/\beta$ and $S = \gamma/\beta$ where disease peaks for two models. (B) Time series for the infected individuals $I(t)$ for two models. The thick and thin curves represent the orbits in the phase plane or solutions for the system (29) with (30), and the dash-dot curves are those for the classic SIR model. Parameters are $\beta = 3, \gamma = 1, p_2 = 0.8, p_1 = 0.2$.

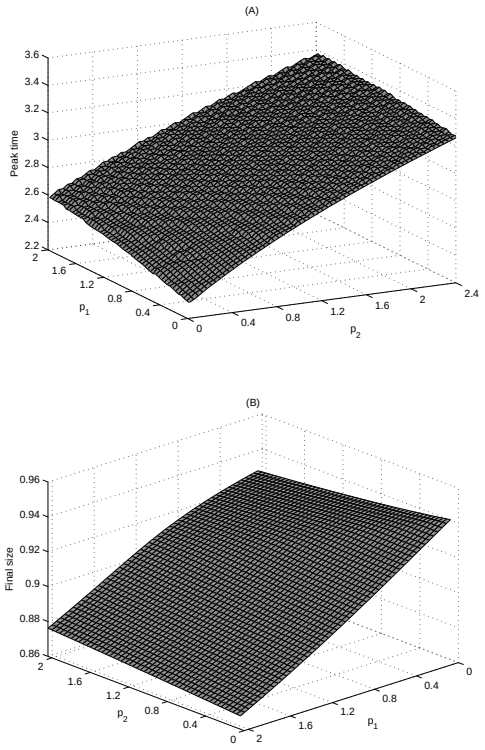

FIGURE 4. Dependence of peak time (A) and final epidemic size (B) on two parameters p_1 and p_2 in the definition of media impact (1). Parameters are $\beta = 3, \gamma = 1$ and initial data are $S(0) = 0.98, I(0) = 0.02$.