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## Original articles

## Sliding Dynamic in a 3-dimensional epidemiological system

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## Abstract

This article analyzes the sliding behavior of a Filippov-type epidemiological system in three dimensions. Initially, a smooth model is mentioned, which involves the transmission of a disease with variables being the average number of susceptible, infected and recovered individuals. The model is analyzed at steady state and then, after locating a switching boundary in the number of infected people, and considering the application of a treatment to a certain number of infected people, a piecewise smooth model is proposed and analyzed in sliding mode. In the steady state, a local stability analysis of the equilibrium points is carried out based on the basic number of reproduction and then, in the sliding mode, the behavior of the number of infected individuals is analyzed from the commutation threshold, determining the existence of pseudo-equilibria, tangent folds, limit equilibria and singular points in the slide zone. The variation of the basic reproduction number of the system leads to a discontinuity-induced bifurcation in the sliding model. The results show that the application of a control is more efficient when the first infected are detected, compared to when there is already a significant number of infections.

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**Keywords:** Filippov systems; SIR model; Local bifurcation non-smooth

## 1. Introduction

In the field of applications of systems of ordinary differential equations, there are many phenomena that can be described by means of smooth models, especially epidemiological systems that describe the transmission of diseases, as in cases [16,17] where the global stability of SEIR (Susceptible, Exposed, Infected and Recovered) models is analyzed, or [9] who show a Susceptible, Infected and Recovered (SIR) model for the transmission of a disease, or the case of [19] who proposes two SIR models, one for the transmission of pneumonia and the other for the transmission of meningitis and, then a model that represents the transmission dynamics considering the presence of both diseases in the environment and the possible co-infection.

However, applications frequently require that one or more study variables change their dynamics depending on the region of the state space that is being considered [15], which gives rise to a non-smooth model. Biological systems are not exception to this type of modeling, although currently there have not been a large number of examples studied, since some theoretical aspects of non-smooth systems are still under development [11]. Within

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**Table 1**  
Variables and parameters for the construction of the model.

Variable/Parameter	Description
$S$	Average number of susceptible people at time $t$ ( $S(t)$ )
$I$	Average number of infected people at time $t$ ( $I(t)$ )
$R$	Average number of recovered people at time $t$ ( $R(t)$ )
$\Delta$	Average number of people per unit time who enter the susceptible population
$\mu$	Natural death rate in the human population.
$\alpha$	Rate of contagion
$\theta$	Recovery rate in humans

the Filippov-type biological systems there is the model of [12] who propose a linear non-smooth system that can be applied to an enzymatic or genetic network; the [21] model for understanding glucose–insulin behavior of people with type 1 and type 2 diabetes; the [20] model that describes the transmission of a Susceptible–Infected (SI) disease; the model [13] describes a fishing model with a control to avoid the extinction of the fish and model [18] which describes a prey predator model with a control to avoid extinction. It should be noted that most of the models studied are of two variables, since it is especially in three-dimensional systems where the theory is in a preliminary development, so the approach and analysis of a non-smooth epidemiological model in three dimensions can be of great use, not only to understand the transmission dynamics of a disease that requires a non-smooth three-dimensional model, but also in that the results can be useful to both Filippov’s theory and health entities.

## 2. Smooth model

Initially, for the non-smooth model approach, a smooth dynamic SIR-type system (Susceptible, Infected, Recovered) is considered [3, p. 415], which describes a disease that provides permanent immunity to people who have been infected and subsequently recovered from the disease. Assuming that there is a constant inflow to the population susceptible to contracting the disease and that the contagion occurs through direct contact with an infectious person, the smooth dynamic system is defined as:

$$\begin{aligned}\dot{S} &= \Delta - \mu S - \alpha SI \\ \dot{I} &= \alpha SI - (\mu + \theta)I \\ \dot{R} &= \theta I - \mu R,\end{aligned}\tag{1}$$

The system is defined in the set of biological interest:

$$\Omega = \{w = (S, I, R) : S \geq 0, I \geq 0, R \geq 0\}.$$

With initial conditions  $S(0) = S_0 > 0$ ,  $I(0) = I_0 \geq 0$  and  $R(0) = R_0 \geq 0$  and, for which  $\dot{S} = \frac{dS}{dt}$ ,  $\dot{I} = \frac{dI}{dt}$  and  $\dot{R} = \frac{dR}{dt}$ .

Table 1, describes the variables and parameters of the model.

### 2.1. Analysis of local stability

The local stability analysis of the smooth system (1) is summarized by the following theorems:

**Theorem 2.1 (Points of Equilibrium).** *The system (1) has two points of equilibrium given by:*

$$P_1 = \left(\frac{\Delta}{\mu}, 0, 0\right) \quad \text{and} \quad P_2 = \left(\frac{\mu + \theta}{\alpha}, \frac{\Delta}{\mu + \theta} - \frac{\mu}{\alpha}, \frac{\Delta\theta}{\mu(\mu + \theta)} - \frac{\theta}{\alpha}\right).$$

**Proof.** Setting the right hand side of the equations of system (1) equal to zero we get:

$$\Delta - \mu S - \alpha SI = 0\tag{2}$$

$$\alpha SI - (\mu + \theta)I = 0\tag{3}$$



$$\theta I - \mu R = 0. \quad (4)$$

Factorizing  $I$  from Eq. (3), we have that

$$I(\alpha S - (\mu + \theta)) = 0,$$

from where:

- If  $I = 0$ , Eq. (2) becomes,  $S = \frac{\Delta}{\mu}$  and, Eq. (4),  $R = 0$ . Thus, the first point of equilibrium is given by  $P_1 = \left(\frac{\Delta}{\mu}, 0, 0\right)$ .
- If  $I \neq 0$ , then  $S = \frac{\mu + \theta}{\alpha}$  and, from Eq. (2),  $I = \frac{\Delta}{\mu + \theta} - \frac{\mu}{\alpha}$ . Then, substituting  $I$  in Eq. (4), we get that  $R = \frac{\Delta\theta}{\mu(\mu + \theta)} - \frac{\theta}{\alpha}$ . Thus, the second point of equilibrium is given by

$$P_2 = \left(\frac{\mu + \theta}{\alpha}, \frac{\Delta}{\mu + \theta} - \frac{\mu}{\alpha}, \frac{\Delta\theta}{\mu(\mu + \theta)} - \frac{\theta}{\alpha}\right) \quad \square$$

**Theorem 2.2** (Stability Analysis of the Points of Equilibrium). Defining

$$\mathcal{R}_0 = \frac{\alpha\Delta}{\mu(\mu + \theta)},$$

we have that:

- For  $\mathcal{R}_0 < 1$ , the point of equilibrium  $P_1$  is asymptotically stable at the local level and,  $P_2$  makes no biological sense and is unstable.
- If  $\mathcal{R}_0 > 1$ , the point of equilibrium  $P_1$  is unstable and the point of equilibrium  $P_2$  is asymptotically stable at the local level.

**Proof.** The Jacobian matrix associated with the linearization of the system (1) about  $P_1$  is given by:

$$J(P_1) = \begin{pmatrix} -\mu & -\frac{\alpha\Delta}{\mu} & 0 \\ 0 & \frac{\alpha\Delta}{\mu} - (\mu + \theta) & 0 \\ 0 & \theta & -\mu \end{pmatrix}.$$

Thus, it has the characteristic equation:

$$(\lambda + \mu)^2 \left(\lambda - \frac{\alpha\Delta}{\mu} + \mu + \theta\right).$$

When  $\mathcal{R}_0 = \frac{\alpha\Delta}{\mu(\mu + \theta)}$ , the eigenvalues of the matrix  $J(P_1)$  are  $\lambda_1 = -\mu$  and  $\lambda_2 = (\mu + \theta)(\mathcal{R}_0 - 1)$ . From where, if  $\mathcal{R}_0 < 1$ ,  $P_1$  is locally asymptotically stable and, if  $\mathcal{R}_0 > 1$ ,  $P_1$  is unstable.

Given the definition of  $\mathcal{R}_0$ , the point of equilibrium  $P_2$  can be rewritten as  $P_2 = \left(\frac{\mu + \theta}{\alpha}, \frac{\mu}{\alpha}(\mathcal{R}_0 - 1), \frac{\theta}{\alpha}(\mathcal{R}_0 - 1)\right)$  and, so  $P_2$  does not make biological sense when  $\mathcal{R}_0 < 1$ .

Furthermore, the Jacobian matrix associated with the linearization of the system (1) about  $P_2$  is given by:

$$J(P_2) = \begin{pmatrix} -\mu\mathcal{R}_0 & -(\mu + \theta) & 0 \\ \mu(\mathcal{R}_0 - 1) & 0 & 0 \\ 0 & \theta & -\mu \end{pmatrix}.$$

Now, the characteristic equation is given by:

$$(\lambda + \mu)[\lambda^2 + \mu\mathcal{R}_0\lambda + \mu(\mu + \theta)(\mathcal{R}_0 - 1)] = 0.$$

According the Routh–Hurwitz criterion, all the roots of this equation will have a negative real part when  $\mathcal{R}_0 > 1$  and so,  $P_2$  will be asymptotically stable at the local level. However, when  $\mathcal{R}_0 < 1$ , there exists at least one root with a positive real part and thus  $P_2$  is unstable.  $\square$

### 3. Non-smooth model

To define the non-smooth system, suppose that  $\mathcal{R}_0 > 1$  and, when the health services detect a certain number  $\zeta$  of infected people, they apply a treatment to  $k$  infected people per unit time, such that those people immediately