Available online at http://scik.org
Commun. Math. Biol. Neurosci. 2021, 2021:80
https://doi.org/10.28919/cmbn/6569
ISSN: 2052-2541

A VIRUS DYNAMICS MODEL FOR INFORMATION DIFFUSION IN ONLINE SOCIAL NETWORKS

MOHAMED YASSER SAHNOUNE*, KHADIJA AKDIM, ADIL EZ-ZETOUNI, MEHDI ZAHID

Department of Mathematics, Faculty of Sciences and Technology, Cadi Ayyad University, B.P. 549, Marrakesh C.P. 40.000, Morocco

Abstract. Social media are increasingly influencing people's preferences and decisions. Modeling information diffusion on social media networks allows to understand the impact of viral information on individuals behaviors in economic, political and social fields. The aim of this paper is to propose a mathematical viral model to characterize the dynamic of information diffusion on social media platforms resulting from the spread of a viral information. To this end, the problem will be modelled by four differential equations that describe the interactions between "Uninfected Users", "Infected Users", "Inert Users" and "free viral information" based on the similarity between a virus dynamics and people interaction on social media networks. A saturated infection rate is incorporated into the model. First, the problem well-posedness is investigated in terms of existence, positivity and boundedness of solution. Moreover, the reproduction number R_0 associated to our problem is calculated using the next generation method. Next, the equilibrium points are calculated and their existence is proved. Therefore, the stability analysis and uniform persistence of the model are investigated according to R_0 threshold. Finally , some numerical simulations are carried out in order to illustrate the analytical results. It was revealed that our proposed model may be conductive to understand the viral information diffusion behavior on social media networks. The presented mathematical modeling approach is the first investigation of a virus dynamical model that is used to describe the viral information behavior.

Keywords: viral model; social media; viral information; stability analysis; uniform persistence.

2010 AMS Subject Classification: 91D30.

*Corresponding author

E-mail address: yassersahnoune@gmail.com

Received July 28, 2021

1. INTRODUCTION

In the last decade, the rapid development of smartphones and information technology has made social media one of the largest and powerful industries. With over 2.7 billion monthly active users, Facebook is currently one of the most important propagating tool for message spreading, rumors circulating, opinions sharing, campaigns and modern social movements mobilization. Recently, it was noticed that social media were widely used during the "Arab World Green Revolution" and Online Social Networks have been a valuable instrument in mobilizing protesters ([23, 25, 5]). More recently, several social movements have been perceived and widely distributed on social media platforms such as Morocco consumer boycott (2018 - 2019), the boycott of Algerian presidential election (2019) and the recent calls to boycott French goods (2020). Mathematical modeling of Diffusion in social media networks have been the interest of many scientific works. The aim is to well-understand the viral information spreading and to check the different means to control its progression. In the last few years, dynamical analysis of Information Diffusion in Social media Networks have played an undeniable role to understand the key parameters that impact the viral information spreading (see for instance [9, 6, 7, 14]). Epidemiological modeling is one of the most valuable tools used by many authors in order to describe the dynamics of viral information on social media (see for instance [12, 13, 27]). Indeed, at a large-scale, many mathematical models have been deployed a compartmental model in order to investigate the social interaction behavior in social media platforms ([21, 17, 10, 1]). The work of Daley et al.[4] was the first effort to present the similarity between epidemics and rumors using mathematical analysis . Lately, some other researchers studied rumor propagation modeling in various network topology (see for instance [18, 20]). Bailey et al. studied and showed how emerging data from social networks lead to better understanding the economic effects by taking Facebook as an illustrative example [2]. The results suggested that friendship networks give a process that can spread house price shocks across the economy and that at the country level. Liu and Zhang proposed a new dynamic information propagation model based on the susceptible-infected-recovered (SIR) epidemic model with fixed recovery time [16]. Rodrigues et al. analyzed the similarity between an epidemic and the viral marketing process. The authors proposed a SIR epidemic model in order to select the successful viral marketing strategy

[19]. Jin et al. showed that the epidemic SEIZ model was more specific in capturing the spread of information and rumors on Twitter [10]. Zhu et al. proposed a new epidemic model with two periods of infection in order to model human behavior in the social network [28]. On the other hand, mathematical modeling in Virology by differential systems can provide unique insights into the dynamics of host-pathogen interactions in *vivo*. Accordingly, it can be seen that there is a similarity between virus dynamics and the spread of viral information. Indeed, in this paper we propose a viral model in order to describe the viral information dynamics on social media networks by means of a virus dynamical model with saturated incidence and virus-to-cell infection. The organization of this work is as follows. In the next section we show the formulation of our proposed model. In section 3 we prove the positivity and boundedness of solutions. In section 4, the existence of equilibrium points and the reproduction number related to the model are carried out. Stability analysis and persistence of model are mainly discussed in Section 5. Section 6 is devoted to present some numerical simulations to support our analytical results. Finally, we provide some discussions and conclusion of our main results.

2. MODEL FORMULATION

In this section, we present a viral information dynamics model for social media networks with constant population environment. The mathematical model is constructed under the following hypothesis:

(1) We consider a homogeneous population in the sense that information spread on social media network has same effect on minds of all users.
(2) The free diffused viral information on social network $v(t)$ can have an "social mind effect" on social media users. In the virus dynamics viewpoint, it corresponds to the free virus.
(3) The population is divided to three compartments:
    - The "Uninfected Users" compartment $X(t)$ which represents the class of users that are yet to receive the viral information. In the virus dynamics viewpoint, it corresponds to the healthy cells.

- The "Infected Users" compartment $Y(t)$ which represents the individuals who came to know about the viral information. "Infected Users" can spread the viral information by "Liking", "Commenting" or "Sharing" online social media posts and messages. In the virus dynamics viewpoint, it corresponds to the infected cells.
- The "Inert Users" compartment $Z(t)$ which corresponds to the "Infected Users" that are losing interest by getting bored of the viral information.

(4) We assume that the total population is constant, which means that $X(t)+Y(t)+Z(t)=N>0$.

(5) All state variables and parameters of the proposed model are supposed positive.

The "uninfected users" are assumed to be produced at a constant rate μ and die at the same rate; birth and death can be viewed as events when people join or leave online social media platform; k, γ and c are the production rate of free virus by the "infected users", the transition rate from "infected" to "inert users" and the clearance rate of viral information, respectively. β is the rate at which a "uninfected user" comes in contact with a viral information; $1+av(t)$ is the crowding effect of viral posts (see [27]). α is the transition rate from "inert" to the class of "uninfected users" . Figure 1 shows the schematic diagram of the model.

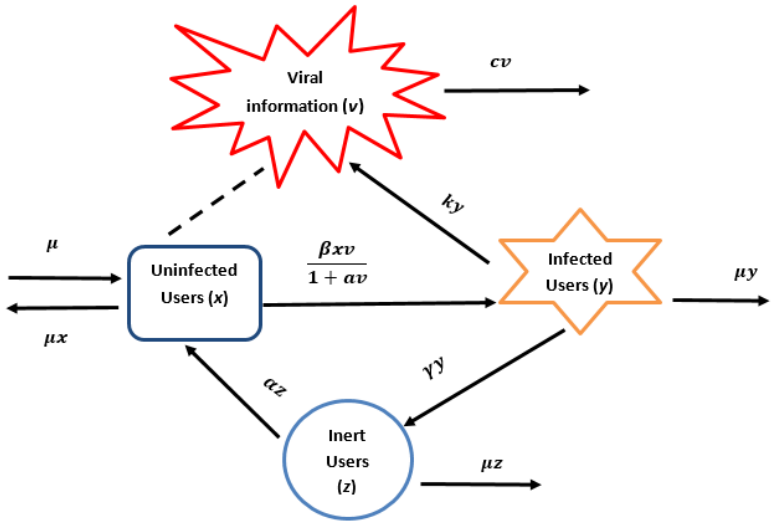


FIGURE 1. Diagram of the viral information dynamics on social media.

Let $(x(t),y(t),z(t))=\left(\frac{X(t)}{N},\frac{Y(t)}{N},\frac{Z(t)}{N}\right)$. The model under consideration consists of four ordinary differential equations illustrating the interaction between the susceptible users x, the

infected users y, the inert users z and the viral posts v

$$\left\{\begin{array}{l} \frac{dx(t)}{dt} = \mu - \frac{\beta x(t)v(t)}{1+av(t)} - \mu x(t) + \alpha z(t), \\ \frac{dy(t)}{dt} = \frac{\beta x(t)v(t)}{1+av(t)} - \mu y(t) - \gamma y(t), \\ \frac{dv(t)}{dt} = ky(t) - cv(t), \\ \frac{dz(t)}{dt} = \gamma y(t) - \mu z(t) - \alpha z(t). \end{array}\right. \tag{1}$$

Remark 2.1. *The proposed mathematical viral model can be useful to describe different forms of information diffusion on social media networks such as rumors, marketing campaigns, and all other types of messages or information that are largely published by users population of social media.*

3. Positivity and Boundedness of Solutions of Model (1)

It is well-known that the number of social media platforms users and its related viral posts remain nonnegative and bounded. Therefore, we will establish, in this section, the positivity and boundedness of solution of our model (1). To this end, and to retain the validity of our problem, we will assume first that the initial data satisfy:

$$x(0) \geq 0,\ y(0) \geq 0,\ v(0) \geq 0 \text{ and } z(0) \geq 0. \tag{2}$$

The well-posedness is proved in terms of positivity and boundedness of solution. Indeed, we have the following result

Theorem 3.1. *All solutions $(x(t), y(t), v(t), z(t))$ of model (1) starting from nonnegative initial conditions exist, remain nonnegative and bounded.*

Proof. From the functional differential equations theory (see for instance [8] and the references therein), we can show that there exists a unique local solution to model (1). Let $(x(t), y(t), v(t), z(t))$ be a solution of system (1) with positive initial condition. We have from the second equation of (1) that

$$\frac{dy}{dt} = \frac{\beta xv}{1+av} - \mu y - \gamma y \geq -\mu y - \gamma y.$$

Let $f : \mathbb{R}^+ \to \mathbb{R}$ where $f(t) = y(t)e^{(\mu+\gamma)t}$,

thus

$$\begin{aligned} f'(t) = &\quad y'(t)e^{(\mu+\gamma)t} + (\mu+\gamma)y(t)\exp(\mu+\gamma)t \\ = &\quad e^{(\mu+\gamma)t}[y'(t) + (\mu+\gamma)y(t)] \\ \geq &\quad 0. \end{aligned}$$

Therefore $f(t) \geq f(0)$, multiplying each side by $e^{-(\mu+\gamma)t}$, we get

$$y(t) \geq y(0)e^{-(\mu+\alpha)t} \geq 0.$$

Following a similar reasoning for the other cases, we can straightforward prove that

$$\begin{aligned} v(t) &\geq v(0)e^{-ct} \geq 0, \qquad z(t) \geq z(0)e^{-(\mu+\alpha)t} \geq 0, \\ x(t) &\geq x(0)e^{-\mu t - \beta \int_0^t \frac{v(u)}{1+av(u)}du} \geq 0. \end{aligned}$$

Therefore, for all $t \geq 0$, we have that all the variables of our problem are nonnegative. Hence, the positivity of all solutions is satisfied.

Now, we will prove that the solutions are bounded. Since the population is constant for all $t \geq 0$, we have

$$x(t) \leq 1, \quad y(t) \leq 1 \text{ and } \quad z(t) \leq 1. \tag{3}$$

Therefore, from the third equation of system (1) we obtain

$$\frac{dv}{dt} \leq k - cv. \tag{4}$$

By comparison principle we get

$$v(t) \leq v(0)e^{-ct} + \frac{k}{c}, \quad \text{and for } t \to \infty, \text{ we obtain} \quad v(t) \leq \frac{k}{c}.$$

Thus, each local solution can be prolonged to any positive time, which means that the unique solution exists globally. This complete the proof. □

4. Equilibria and Basic Reproduction Number

4.1. Basic reproduction number. The basic reproduction number R_0 is the number of secondary cases which one case would produce in the entire population supposed susceptible. In order to determine the basic reproduction number expression, we apply the next generation matrix method (see [11]).

Consider the next generation matrix G. It is comprised of two parts: F and V^{-1}, where:

$$F = \left(\frac{\partial F_i(x_0)}{\partial x_j}\right), \qquad V = \left(\frac{\partial V_i(x_0)}{\partial x_j}\right)$$

The F_i are the new infections, while V_i represent the infection transfer from a compartment to another. x_0 is the disease-free equilibrium state. R_0 is the dominant eigenvalue of the matrix $G = FV^{-1}$. From the model we have

$$F = \begin{pmatrix} 0 & \beta \\ 0 & 0 \end{pmatrix}, \qquad V = \begin{pmatrix} \gamma+\mu & 0 \\ -k & c \end{pmatrix}, \qquad FV^{-1} = \begin{pmatrix} \frac{\beta k}{c(\gamma+\mu)} & \frac{\beta}{c} \\ 0 & 0 \end{pmatrix},$$

thus

$$R_0 = \frac{\beta k}{c(\gamma+\mu)}.$$

4.2. Existence of equilibria. In this subsection, we show that there exist two equilibria, namely:

(1) The disease-free equilibrium $E_0 = (1,0,0,0)$ which corresponds to the infection free state. From biological point of view, the disease-free equilibrium corresponds to the maximum of healthy cells and the infection can not invade the cell population. In this case of viral information modeling viewpoint, it corresponds to the maximum of uninfected social media users and the viral information infection can not invade the population of users.

(2) The endemic equilibrium $E_e = (x^*, y^*, v^*, z^*)$, where:

$$v^* = \frac{(R_0-1)(\mu+\alpha)(\mu+\gamma)}{[(\mu+\alpha)\beta+\gamma\beta+a(\mu+\alpha)(\mu+\gamma)]}, \qquad x^* = \frac{(1+av^*)}{R_0},$$

$$y^* = \frac{c}{k}v^*, \qquad z^* = \frac{c\gamma}{k(\mu+\alpha)}v^*.$$

Here the endemic equilibrium point E_e represents the equilibrium case when the viral information can invade the total studied population of social media users. If $R_0 > 1$, then we have the existence of the endemic equilibrium E_e.

5. Stability Analysis and Uniform Persistence

5.1. Stability of the model. Stability analysis is an essential tool to predict the long-time behavior of dynamical model solutions. Generally, there are two main types of stability analysis, local and global which are widely used in the literature. Local stability is related with the behavior of solutions near an equilibrium point, while global stability can describe solution behavior in the whole domain. In order to study the local stability of the disease-free equilibrium E_0 and the endemic one E_e, let us first give the problem Jacobian matrix at an arbitrary point $\bar{E} = (\bar{x}, \bar{y}, \bar{v}, \bar{z})$

$$J(\bar{E}) = \begin{pmatrix} -\frac{\beta\bar{v}}{1+a\bar{v}} - \mu & 0 & -\frac{\beta\bar{x}}{(1+a\bar{v})^2} & \alpha \\ \frac{\beta\bar{v}}{1+a\bar{v}} & -(\mu+\gamma) & \frac{\beta\bar{v}}{(1+a\bar{v})^2} & 0 \\ 0 & k & -c & 0 \\ 0 & \gamma & 0 & -(\mu+\alpha) \end{pmatrix}. \tag{5}$$

To determine the local asymptotic stability of our system, we analyze the eigenvalues of (5) at each equilibrium point. The equilibrium is said to be stable if all eigenvalues of its corresponding Jacobian matrix have a negative real part.

Theorem 5.1. *The disease-free equilibrium E_0 is locally asymptotically stable if $R_0 < 1$, otherwise it is unstable.*

Proof. The Jacobian matrix of system (1) at E_0 is given as follows

$$J(E_0) = \begin{pmatrix} -\mu & 0 & -\beta & \alpha \\ 0 & -(\mu+\gamma) & \beta & 0 \\ 0 & k & -c & 0 \\ 0 & \gamma & 0 & -(\mu+\alpha) \end{pmatrix}.$$

The characteristic equation of $J(E_0)$ is given by

$$(\lambda+\mu)(\lambda+(\mu+\alpha))A(\lambda) = 0, \tag{6}$$

with

$$A(\lambda) = \lambda^2 + \lambda(\mu+\gamma+c) - \beta k + c(\mu+\gamma). \tag{7}$$

The equation $A(\lambda) = 0$ has all roots with negative real parts if $-\beta k + c(\mu+\gamma) > 0$, according to Routh Hurwitz criterion. Then disease-free state E_0 is locally asymptotically stable if $R_0 < 1$ and unstable when $R_0 > 1$. □

Theorem 5.2. *The disease-free equilibrium E_0 is globally asymptotically stable if $R_0 < 1$.*

Proof. Let define a Lyapunov function V_0 as

$$V_0(x,y,v,z) = (x-1) + 2y + \frac{\mu+\gamma}{k}v + z.$$

Tacking the derivative of V_0, we get

$$\begin{aligned}
\frac{dV_0}{dt} &= \mu - \frac{\beta xv}{1+av} - \mu x + \alpha z + 2\frac{\beta xv}{1+av} - 2(\mu+\gamma)y + (\mu+\gamma)y \\
&\quad - \frac{cv(\mu+\gamma)}{k} + \gamma y - \mu x - \alpha z \\
&= \mu(1-x-y-z) + \frac{\beta xv}{(1+av)} - \frac{cv(\mu+\gamma)}{k},
\end{aligned}$$

since $x+y+z = 1$ we obtain

$$\frac{dV_0}{dt} = \frac{\beta xv}{(1+av)} - \frac{cv(\mu+\gamma)}{k},$$

it follows that

$$\begin{aligned}
\frac{dV_0}{dt} &\leq v(\beta - \frac{c(\mu+\gamma)}{k}) \\
&\leq v(1 - \frac{1}{R_0}).
\end{aligned} \tag{8}$$

If $R_0 \leq 1$, then $\frac{dV_0}{dt} \leq 0$. Moreover $\frac{dV_0}{dt} \leq 0$ holds when $v = 0$. The largest compact invariant is

$$E = \{(x,y,v,z) | v = 0\}. \tag{9}$$

Therefore, by the LaSalle invariance principle, we have $\lim_{t\to\infty} v(t) = 0$. The limit system of equations is:

$$\begin{cases} \frac{dx(t)}{dt} = \mu - \mu x(t) + \alpha z(t), \\ \frac{dy(t)}{dt} = -\mu y(t) - \gamma y(t), \\ \frac{dz(t)}{dt} = \gamma y(t) - \mu z(t) - \alpha z(t). \end{cases} \tag{10}$$

We define the function:

$$V_1(x,y,z) = x - 1 - \ln(x) + y + z.$$

Since $x+y+z=1$, it follows

$$\frac{dV_1}{dt} = \frac{-\mu}{x}(1-x) - \frac{\alpha z}{x}.$$

Therefore $\frac{dV_1}{dt} \leq 0$ and the equality holds if $x=1$ and $z=0$, which complete the proof. □

Theorem 5.3. *The endemic equilibrium is locally asymptotically stable if $R_0 > 1$.*

Proof. The Jacobian matrix of system (1) at E_e is given as follows

$$J(E_e) = \begin{pmatrix} -\frac{\beta v^*}{1+av^*} - \mu & 0 & -\frac{\beta x^*}{(1+av^*)^2} & \alpha \\ \frac{\beta v^*}{1+av^*} & -(\mu+\gamma) & \frac{\beta x^*}{(1+av^*)^2} & 0 \\ 0 & k & -c & 0 \\ 0 & \gamma & 0 & -(\mu+\alpha) \end{pmatrix}.$$

In order to investigate the local stability of the endemic equilibrium we make an elementary row-transformation for the matrix $J(E_e)$ to obtain the following matrix:

$$J^\star(E_e) = \begin{pmatrix} H & 0 & 0 & 0 \\ \mu & G & 0 & 0 \\ 0 & k & -c & 0 \\ 0 & \gamma & 0 & -(\mu+\alpha) \end{pmatrix},$$

where

$$H = -\frac{\beta v^*}{1+av^*} + \mu \left(\frac{(\mu+\gamma)}{-(\mu+\gamma) + \frac{\gamma\alpha}{(\alpha+\mu)} - \frac{\beta k x^*}{c(1+av^*)^2}} \right), \quad \text{and}$$

$$G = -(\mu+\gamma) + \frac{\gamma\alpha}{(\alpha+\mu)} - \frac{\beta k x^*}{c(1+av^*)^2}.$$

Thus, the eigenvalues are

$$\begin{aligned}
\lambda_1 &= -(\mu+\alpha) < 0, \qquad \lambda_2 = -c < 0,\\
\lambda_3 &= G = -(\mu+\gamma) + \frac{\gamma\alpha}{(\alpha+\mu)} - \frac{\beta k x^*}{c(1+av^*)^2},\\
\lambda_4 &= H = -\frac{\beta v^*}{1+av^*} + \mu\left(\frac{(\mu+\gamma)}{-(\mu+\gamma)+\frac{\gamma\alpha}{(\alpha+\mu)}-\frac{\beta k x^*}{c(1+av^*)^2}}\right).
\end{aligned}$$

Since $-(\mu+\gamma)+\frac{\gamma\alpha}{(\alpha+\mu)} \leq 0$, hence

$$(11)\quad G = -(\mu+\gamma) + \frac{\gamma\alpha}{(\alpha+\mu)} - \frac{\beta k x^*}{c(1+av^*)^2} < 0, \quad \text{and} \quad H = -\frac{\beta v^*}{1+av^*} + \mu\left(\frac{(\mu+\gamma)}{G}\right) < 0,$$

which implies that all the eigenvalues are negatives. Therefore E_e is asymptotically stable when $R_0 > 1$. □

5.2. Uniform persistence. Uniform persistence is an crucial concept in population biology. It captures the long-time survival of species, even when the size of the species is quite low at time. From persistence theory in population dynamics (see for instance [22, 26] and the references therein), and by using the same argument as in ([3, 24, 15]), we investigate the uniform persistence of the model (1). Indeed, we have the following result

Theorem 5.4. *If $R_0 > 1$ then model (1) is uniformly persistent. In other words, there exists $\varepsilon > 0$ such that, for any positive solution $(x(t), y(t), v(t), z(t))$ of the model (1)*

$$\liminf_{t\to\infty} x(t) \geq \varepsilon, \qquad \liminf_{t\to\infty} y(t) \geq \varepsilon, \qquad \liminf_{t\to\infty} v(t) \geq \varepsilon, \qquad \liminf_{t\to\infty} z(t) \geq \varepsilon.$$

Proof. Let

$$A = \left\{(x,y,v,z) \in \mathbb{R}^4_+ : x \geq 0, y > 0, v > 0, z > 0\right\},$$

then the boundary of A is

$$\partial A = \left\{(x,y,v,z) \in \mathbb{R}^4_+ : x \geq 0, y = 0 \text{ or } v = 0 \text{ or } z = 0\right\}.$$

For any $T_0 = (x_0, y_0, v_0, z_0) \in \mathbb{R}^4_+$, define $u(t, T_0) = (x(t,T_0), y(t,T_0), v(t,T_0), z(t,T_0))$ be the solution of model (1) with the initial condition $u(0,T_0) = u(t,T_0)$. It's clear that $\{u(t)\}_{t\geq 0}$ is a C_0-semigroup generated by system (1). We have u(t) is dissipative in A and that by theorem

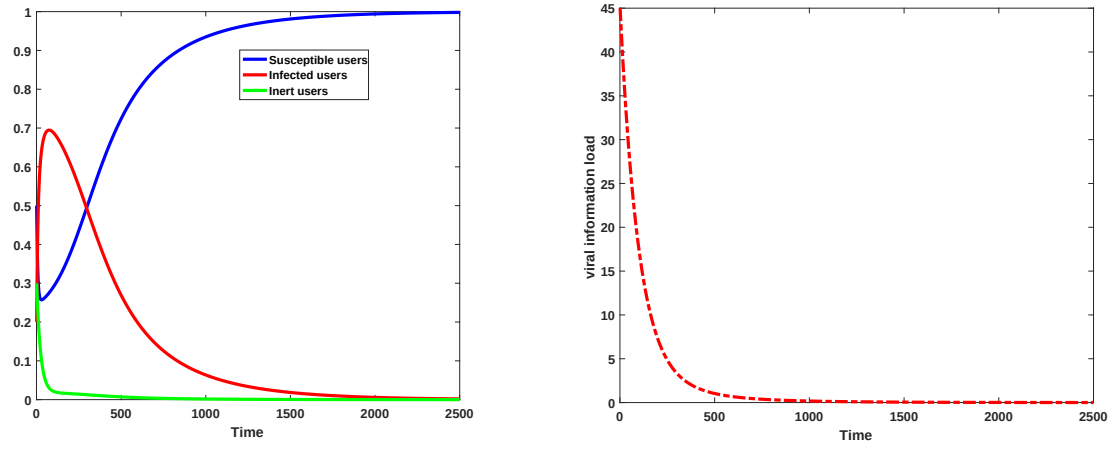
3.1, thus conditions (1) and (2) of lemma 3.7 in [24] are satisfied. Notice that the model (1) admits one free equilibrium $E_0 = (1,0,0,0)$. Denote $N_\partial = \left\{T_0 \in \mathbb{R}^4_+ : u(t,T_0) \in \partial A, \forall t \geq 0\right\}$ and let $\omega(T_0)$ be the ω-limit set of the solution $u(t,T_0)$, its clear that $\{E_0\} \subset \bigcup_{T_0 \in N_\partial} \omega(T_0)$. Furthermore, for any solution $u(t,T_0) \in \partial A$, then $x(t) \to 1$, $y(t) \to 0$, $v(t) \to 0$, $z(t) \to 0$ as $t \to \infty$ (the approach is similar to the proof of theorem 5 in [3]). Hence $\{E_0\}$ contains all ω-limit set in ∂A. From Theorem 5.1, we have E_0 is unstable if $R_0 \geq 1$, then E_0 is isolated and hence the third condition of lemma 3.7 in [24] is satisfied. Moreover, as $W^s(E_0) \bigcap A = \emptyset$, the condition (4) of lemma 3.7 in [24] is satisfied. Based on the theory of uniform persistence in dynamical systems, there is $\varepsilon > 0$ such that, for any T_0 in A, we have

$$\liminf_{t\to\infty} x(t) \geq \varepsilon, \qquad \liminf_{t\to\infty} y(t) \geq \varepsilon, \qquad \liminf_{t\to\infty} v(t) \geq \varepsilon, \qquad \liminf_{t\to\infty} z(t) \geq \varepsilon.$$

This complete the proof. □

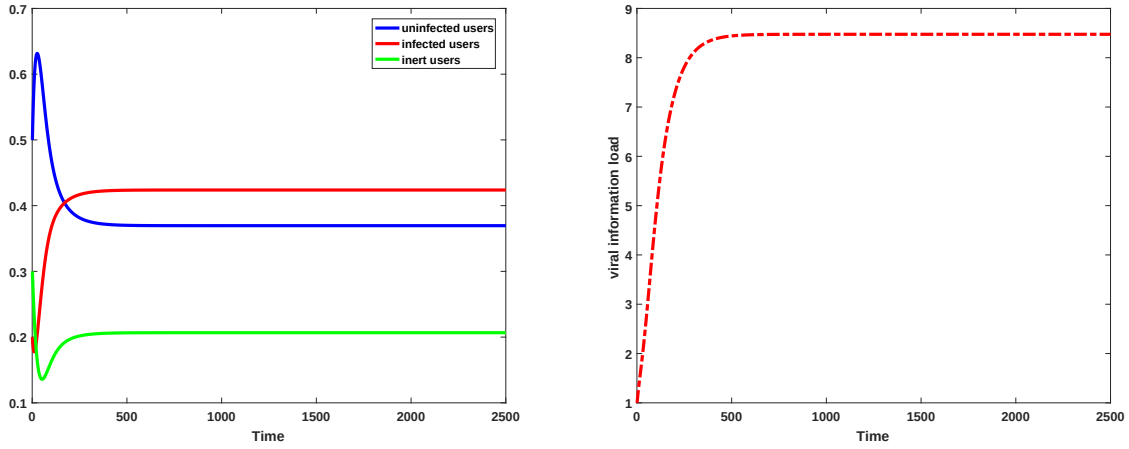
6. Numerical Simulation

In this section some numerical simulations are performed in order to check the time evolution of our model. We have used the Euler finite-difference scheme method to numerically resolve the four equations.



(A) (B)

FIGURE 2. Time evolution of the viral information dynamics for $\mu = 0.04$, $\alpha = 0.001$, $\gamma = 0.001$, $\beta = 0.015$, $a = 0.1$, $k = 0.02$, $c = 0.01$ and $(x(0), y(0), v(0), z(0)) = (0.5, 0.2, 45, 0.3)$.



(A) (B)

FIGURE 3. Time evolution of the viral information dynamics for $\mu = 0.04, \alpha = 0.001, \gamma = 0.02, \beta = 0.015, a = 0.1, k = 0.3, c = 0.015$ and $(x(0), y(0), v(0), z(0)) = (0.5, 0.2, 1, 0.3)$.

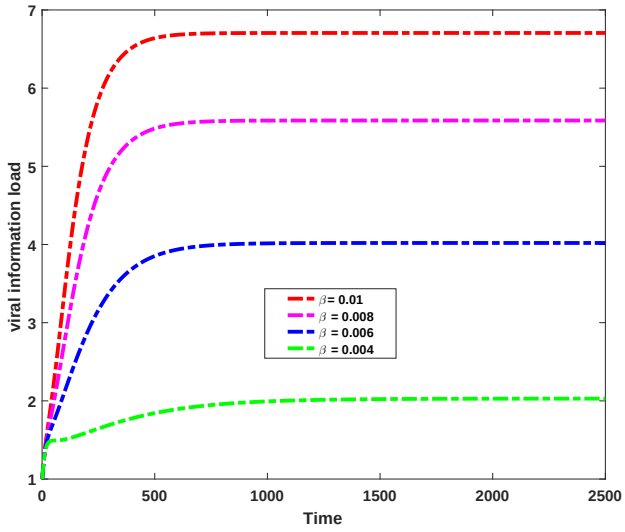


FIGURE 4. Time evolution of the viral information load for $\mu = 0.04, \alpha = 0.001, \gamma = 0.02, a = 0.1, k = 0.3, c = 0.015$ and $(x(0), y(0), v(0), z(0)) = (0.5, 0.2, 1, 0.3)$ with $R_0 > 1$.

Fig. 2 shows the behavior of the viral information dynamics during the period of observation for a set of feasible hypothetical parameters. In this figure, the basic reproduction number R_0 is less than unity ($R_0 = 0.73 < 1$). It can be seen that the curves converge to the disease-free steady state $E_0 = (1,0,0,0)$. The uninfected users reach their maximum, the infected users, inert users and the free viral information converge toward zero, this indicates that the viral campaign dies out which is consistent of the theoretical stability results.

Fig. 3 depicts the behavior of the viral information dynamics during the period of observation. In this figure, the basic reproduction number R_0 is greater than unity ($R_0 = 5 > 1$). It can be seen that the four curves converge to the endemic equilibrium $E_e = (0.37, 0.42, 8.48, 0.21)$. Indeed the free viral information posts reach their maximum which means that the viral social media campaign remain present in the population of users which is consistent with the analytical stability result of E_e.

Finally, Fig. 4 shows the dynamics of the free viral information load as function of time for different values of the infection rate β. We can clearly observe that by decreasing the infection rate, the number of viral information posts decreases also. This indicates that infection rate can control the viral campaign severity on social media. However, one can observe the persistence of the viral information when $R_0 > 1$.

Remark 6.1. *The proposed viral information model can be estimated by using datasets of a diffused viral information on Facebook platform. For this end, friendship networks, groups and pages can thus be analyzed quantitatively and qualitatively with regards to the viral characteristics of a studied viral information at regional and country levels. The diffused public information on Facebook pages can be used to explain the impact of a given boycott campaign or to control a viral marketing strategy.*

7. CONCLUSION

In this paper, a mathematical model of viral posts dynamic on social media network is proposed and studied. To this end, we have modeled the phenomenon by four differential equations that describe the interactions between ”Uninfected Users”, ”Infected Users”, ”Inert Users” and ”free viral information”. We have included to the model a saturated infection rate that reflects

the viral posts crowd near the uninfected users. The existence, positivity and boundedness of the model (1) have been proved and fulfilled as this is essential in any population dynamics models. Using the next generation matrix method, we have calculated the reproduction number R_0 related to the problem. Then we have proved the existence of two equilibrium points, namely free-disease state $E_0 = (1,0,0,0)$ which represents the absence of viral information infection and the total presence of uninfected users, endemic state $E_e = (x^\star, y^\star, v^\star, z^\star)$ which correspond to the existence of "free viral information" and the presence of uninfected and inert users population. Then we have investigated the stability analysis of both equilibria according to R_0. Furthermore, we have shown that our proposed model is uniformly persistent when $R_0 > 1$. Finally, we have illustrated the theoretical results by numerical simulations. This model may be conductive to understand some emergencies phenomena on social media networks such as boycott movements.

CRediT Authorship Contribution Statement

The authors have contributed equally to this work.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Conflict of Interests

The author(s) declare that there is no conflict of interests.

References

[1] S. Abdullah, X. Wu, An epidemic model for news spreading on twitter, in: 2011 IEEE 23rd International Conference on Tools with Artificial Intelligence, IEEE, Boca Raton, FL, USA, 2011: pp. 163–169.

[2] M. Bailey, R. Cao, T. Kuchler, J. Stroebel, The economic effects of social networks: Evidence from the housing market, J. Political Econ. 126 (2018), 2224–2276.

[3] W. Chen, N. Tuerxun, Z. Teng, The global dynamics in a wild-type and drug-resistant HIV infection model with saturated incidence, Adv. Differ. Equ. 2020 (2020), 25.

[4] D.J. Daley, D.G. Kendall, Stochastic Rumours, IMA J. Appl. Math. 1 (1965), 42–55.

[5] N. Eltantawy, J.B. Wiest, The Arab spring-social media in the Egyptian revolution: reconsidering resource mobilization theory, Int. J. Commun. 5 (2011), 1207–1224.

[6] S.A. Floria, F. Leon, D. Logofătu, A model of information diffusion in dynamic social networks based on evidence theory, J. Intell. Fuzzy Syst. 37 (2019), 7369–7381.

[7] A. Guille, H. Hacid, C. Favre, D.A. Zighed, Information diffusion in online social networks: a survey, SIGMOD Rec. 42 (2013), 17–28.

[8] J.K. Hale, S.M.V. Lunel, L.S. Verduyn, Introduction to functional differential equations vol. 99, Springer Science & Business Media, New York, (1993).

[9] C. Jiang, Y. Chen, K.J.R. Liu, Modeling information diffusion dynamics over social networks, in: 2014 IEEE International Conference on Acoustics, Speech and Signal Processing (ICASSP), IEEE, Florence, Italy, 2014: pp. 1095–1099.

[10] F. Jin, E. Dougherty, P. Saraf, Y. Cao, N. Ramakrishnan, Epidemiological modeling of news and 282 rumors on twitter, In: Proceedings of the 7th workshop on social network mining and analysis (2013), pp. 1-9.

[11] J.H. Jones, Notes on R_0, Department of Anthropological Sciences, California, (2007), 1-19.

[12] K. Kandhway, J. Kuri, Accelerating information diffusion in social networks under the Susceptible-Infected-Susceptible epidemic model, in: 2014 International Conference on Advances in Computing, Communications and Informatics (ICACCI), IEEE, Delhi, India, 2014: pp. 1515–1519.

[13] S. Kumar, M. Saini, M. Goel, N. Aggarwal, Modeling information diffusion in online social networks using SEIR epidemic model, Procedia Computer Sci. 171 (2020), 672-678.

[14] D. Li, S. Zhang, X. Sun, H. Zhou, S. Li, X. Li, Modeling information diffusion over social networks for temporal dynamic prediction, IEEE Trans. Knowl. Data Eng. 29 (2017), 1985–1997.

[15] H. Li, S. Guo, Dynamics of a SIRC epidemiological model, Electron. J. Differ. Equ. 2017 (2017), 121.

[16] C. Liu, Z.-K. Zhang, Information spreading on dynamic social networks, Commun. Nonlinear Sci. Numer. Simul. 19 (2014), 896–904.

[17] C. Piccardi, Social networks and the spread of epidemics, Lett. Mat. 1(3) (2013), 119-126.

[18] V. Qazvinian, E. Rosengren, D. Radev, Q. Mei, Rumor has it: Identifying misinformation in microblogs, In: Proceedings of the 2011 Conference on Empirical Methods in Natural Language Processing (2011), pp. 1589-1599.

[19] H.S. Rodrigues, M.J. Fonseca, Viral marketing as epidemiological model, ArXiv:1507.06986 [Physics]. (2015).

[20] D. Shah, T. Zaman, Rumors in a network: Who's the culprit, IEEE Trans. Inform. Theory, 57(8) (2011), 5163-5181.

[21] J. Skaza, B. Blais, Modeling the infectiousness of Twitter hashtags, Physica A: Stat. Mech. Appl. 465 (2017) 289–296.

[22] H.L. Smith, H.R. Thieme, Dynamical systems and population persistence, vol. 118, Amer. Math. Soc. (2011).

[23] M. Taki, L. Coretti, The Role of Social Media in the Arab Uprisings–Past and Present. Westminster Papers in Communication and Culture, Sonderheft 9/2 (2013), 1–122.

[24] K. Wang, Y. Jin, A. Fan, The effect of immune responses in viral infections: A mathematical model view, Discr. Contin. Dyn. Syst. B. 19 (2014), 3379–3396.

[25] G. Wolfsfeld, E. Segev, T. Sheafer, Social Media and the Arab Spring: Politics Comes First, Int. J. Press/Politics. 18 (2013), 115–137.

[26] M. Li, An introduction to mathematical modeling of infectious diseases, Springer, New York, (2018).

[27] Z. Zhang, H. Wang, C. Wang, H. Fang, Modeling epidemics spreading on social contact networks, IEEE Trans. Emerg. Topics Comput. 3(3) (2015), 410-419.

[28] G. Zhu, G. Chen, X. Fu, Effects of active links on epidemic transmission over social networks, Physica A: Stat. Mech. Appl. 468 (2017), 614–621.