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Investigating the Impact of Media Alerts on an Epidemiological Model Involving Asymptomatic Cases.

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Abstract

The significance of media alerts in influencing public perception and response during disease outbreaks is of paramount importance. This research investigates how media alerts affect the epidemiological model by taking into account both asymptomatic and symptomatic cases. Media alerts serve as a powerful tool for raising awareness about the disease, driving people to undergo testing and diagnosis, and promoting adherence to public health measures. This, in turn, contributes to better reporting and surveillance of cases, leading to a more accurate representation of disease spread in the model. The proposed model introduces a five-dimensional compartment system that includes susceptible, asymptomatic, symptomatic, recovered populations and media. This research addresses various aspects of the model, such as its uniqueness, boundedness, and existence of solutions. The determination of all possible model equilibrium points is thoroughly explored. To study the global dynamics of the proposed models, appropriate Lyapunov functions are employed. Additionally, numerical simulation is also carried out to investigate the influence of parameters affecting the dynamics of the model and to support the gathered analytical findings of the model.

Keywords: Asymptomatic , Symptomatic ,Media, Mathematical model, Stability.

دراسة تأثير التنبيهات الإعلامية على النموذج الوبائي المتضمن للحالات التي لا تظهر عليها أعراض.

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الخلاصة

إن أهمية تنبيهات وسائل الإعلام في التأثير على الإدراك العام والاستجابة أثناء تفشي الأمراض لها أهمية قصوى. يبحث هذا البحث في كيفية تأثير تنبيهات وسائل الإعلام على النموذج الوبائي من خلال مراعاة الحالات التي لا تظهر عليها الأعراض والحالات التي تظهر عليها الأعراض. تعمل التنبيهات الإعلامية كأداة قوية لرفع مستوى الوعي حول المرض، ودفع الناس للخضوع للاختبار والتشخيص، وتعزيز الالتزام بتدابير الصحة العامة. وهذا بدوره يساهم في تحسين الإبلاغ عن الحالات ومراقبتها، مما يؤدي إلى تمثيل أكثر دقة لانتشار المرض في النموذج. يقدم هذا البحث نظام من خمسة مكونات تدعى الافراد المعرضين للإصابة , الافراد المصابين الذين لا تظهر عليهم اعراض , الافراد المصابين الذين تظهر عليهم اعراض, المتعافين واخيرا

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وسائل الاعلام. تمت مناقشة وحدانية ووجود الحل . تم ايجاد جميع نقاط التوازن المحتملة للنموذج . تم دراسة الديناميات العالمية للنموذج المقترح من خلال استخدام وظائف لابونوف المناسبة. وأخيرا ,تم إجراء المحاكاة العددية لدراسة تأثير المعلمات على ديناميات النموذج المقترح ودعم النتائج التحليلية .

1. Introduction

An epidemiological model serves as a mathematical representation of how infectious diseases spread and evolve within a population. Through this model, researchers and public health experts can gain insights into the dynamics of disease transmission, forecast its progression over time, and assess the potential impact of different interventions or control measures. The influence of media alerts on the epidemiological model is noteworthy, considering both asymptomatic and symptomatic cases. Such alerts can have a complex and multifaceted impact on various aspects of disease transmission and public health response. Media alerts can significantly influence public behavior and compliance with guidelines. When the media consistently emphasizes the importance of adhering to guidelines, people are more likely to follow them, potentially influencing the model's projections and outcomes. Conversely, if media alerts spread misinformation or induce panic, there can be adverse consequences. Misinformation may lead to ineffective or harmful responses, while panic can disrupt public health efforts and worsen disease spread. Both of these factors introduce biases and inaccuracies into the epidemiological model, affecting its predictive capabilities. Hence, it is crucial to handle media alerts carefully to ensure they contribute positively to disease management and control. Pathogens have a variety of life history methods, from acute disorders like syphilis to chronic conditions like influenza A viruses [1–2]. Understanding infections' evolutionarily stable strategies (ESSs), which are constantly subject to selective pressures, might provide important information for the prevention and treatment of disease. Researchers have studied ESSs concerning aspects like virulence, persistence, and recovery. The understanding of pathogen pathogenicity evolution has significantly advanced because of classic research by Anderson and May [3] and Dwyer et al. [4-8]. Regarding various characteristics of pathogens, King et al. [9] investigated pathogen evolution by considering a trade-off between persistence and invasion. On the other hand, Alizon [10] explored the evolutionary consequences of pathogens when there is a trade-off between transmission and host recovery, incorporating explicit immune dynamics into the study. Another key life history strategy for a pathogen is its capacity to display milder symptoms or a time of latency at the beginning of an infection, in addition to virulence, persistence, and recovery. Such "hidden" infections provide the pathogen with a number of benefits in a variety of settings. Additionally, in both animals and humans, symptoms often act as a cue for susceptible individuals to avoid infectious hosts [11–12], and a hidden infection would reduce this avoidance. Fraser et al. [13] found that the effectiveness of disease control strategies depends significantly on the number of asymptomatic transmissions before the full development of symptoms. Hence, these less symptomatic stages of infection are critical and present significant epidemiological challenges.An infection's ability to spread from an infected person to an unknowing host is strongly influenced by the severity of the sickness. Having fewer symptoms lowers transmission in situations where symptoms themselves are frequently connected to enhanced transmission (e.g., through coughing, sneezing, or rashes) [14]. Lower pathogen loads can also result in milder symptoms and less shedding, which lowers transmissibility. Several epidemiological models have been studied, and each model focuses on an infectious epidemiological disease. Among these studies, Watheq Ibrahim Jasim et al. [15]. They suggested a study of the shigellosis bacteria disease model with awareness effects. Chadi M. Saad-Roy et al.[16] studied the dynamics in a simple evolutionary-epidemiological model for the evolution of an initial asymptomatic infection stage. Nourridine Siewe et al.[17] studied the mathematical model of the role of asymptomatic infection in outbreaks of some

emerging pathogens. Anirban Ghatak et al.[18] they suggested a generalized epidemiological model with dynamic and asymptomatic population. Ahmed A. Mohsen et al.[19] studied the awareness effect of the dynamical behavior of SIS epidemic model with Crowley-Martin incidence rate and holling type III treatment function. Shurowq K. Shafeeq et al.[20] studied the bifurcation analysis of a vaccination mathematical model with application to COVID-19 pandemic. Ahmed A. Mohsen et al. [21] conducted a study on the dynamics of Coronavirus pandemic disease model in the existence of a curfew strategy. Similar studies have also been conducted by other researchers [22–24].The main focus of this research is to develop a mathematical model that incorporates the influence of media alerts on epidemiological dynamics, considering both asymptomatic and symptomatic cases. This article is organized as follows:

- Section 2 presents the mathematical modeling of the novel media alert, taking into account asymptomatic and symptomatic cases.
- In section 3, various essential characteristics of the model are discussed, including the boundedness of solutions and the existence of equilibrium points.
- The local stability analysis using Gersgorin's theorem is covered in Section 4.
- In section 5, the Lyapunov function is used to examine the proposed model's overall stability at all equilibrium locations.
- Finally, numerical simulations are carried in section 6 to examine the effects of changing every system parameter.

2. Mathematical model

In this section, we create a mathematical representation to study the impact of media alerts on an epidemiological model that considers both asymptomatic and symptomatic cases. The remaining parameters can be found in Table 1.

Table 1: Parameters description utilized in the system(1).

Parameters	Interpretation
$S(t)$	Susceptible population.
$I_a(t)$	Individuals asymptomatic infected.
$I_s(t)$	Individuals symptomatic infected.
$R(t)$	Recovered population.
$M(t)$	Media.
Λ	Birth rate.
β	The contacts rate between the susceptible and symptomatic infected population.
h	The amount disease control due to the media.
μ	Natural death rate.
p	Fractional rate, $0 \leq p \leq 1$.
σ	Rate of progression from asymptomatic infected to symptomatic infected stages.
$\alpha_i, i = 1,2.$	Recovery rates .
$\rho\ , \theta$	The influence rate of media campaigns.
δ	The reduction rate of media campaigns.

Hence, the behavior of the system suggested above can be described using the following set of 1st. non-linear differential equations, and the graphical representation of this system is depicted in Figure 1.

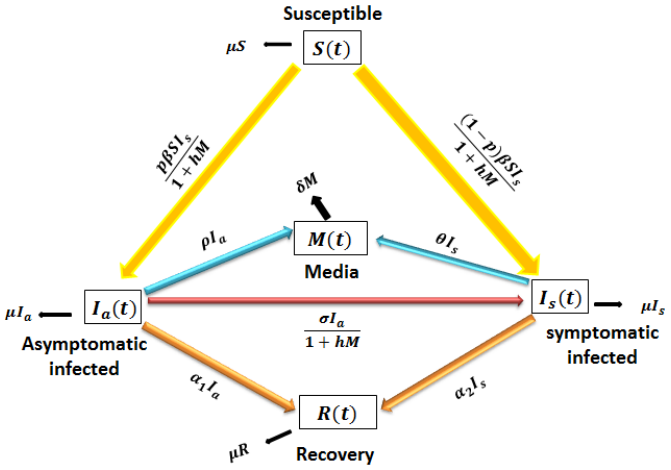

Figure 1: The diagram illustrating the system (1).

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \frac{\beta SI_s}{1+hM} - \mu S \\
\frac{dI_a}{dt} &= \frac{p\beta SI_s}{1+hM} - \frac{\sigma I_a}{1+hM} - (\mu+\alpha_1)I_a \\
\frac{dI_s}{dt} &= \frac{(1-p)\beta SI_s}{1+hM} + \frac{\sigma I_a}{1+hM} - (\mu+\alpha_2)I_s \\
\frac{dR}{dt} &= \alpha_1 I_a + \alpha_2 I_s - \mu R \\
\frac{dM}{dt} &= \rho I_a + \theta I_s - \delta M
\end{aligned} \tag{1}$$

With the initial conditions ,

$$S(0) > 0 \ , \quad I_a(0) \geq 0 \ , \quad I_s(0) \geq 0 \ , R(0) \geq 0 \ , M(0) \geq 0$$

Where $(S(t), I_a(t), I_s(t), R(t), M(t)) \in R_+^5$.It is suppose that for all $t \geq 0$ the functions $S(t), I_a(t), I_s(t), R(t), M(t)$ and their derivatives are continuous, making them Lipschitzian. As a result, the system (1) has a unique and existing solution. Furthermore, the theorem presented below demonstrates the bounded nature of the solution for the system (1).

Theorem (1) : Every solution to the system (1) of equations that begins with R_+^5 as the initial value remains uniformly bounded.

Proof:

The solution $(S(t), I_a(t), I_s(t), R(t), M(t))$ to system (1) is obtained from non-negative initial values $(S(0), I_a(0), I_s(0), R(0), M(0))$, and let

$H(t) = S(t) + I_a(t) + I_s(t) + R(t) + M(t)$, then

$$\frac{dH}{dt} = \frac{dS}{dt} + \frac{dI_a}{dt} + \frac{dI_s}{dt} + \frac{dR}{dt} + \frac{dM}{dt}$$

Next, by deriving H with regard to time, we obtain:

$$\frac{dH}{dt} \leq \Lambda - \mu H$$

The Gronwall lemma [25] is now used to prove that

$$H(t) \leq \frac{\Lambda}{\mu} + \left(H_0 - \frac{\Lambda}{\mu}\right) e^{-\mu t},$$

Where $H_0 = (S(0), I_a(0), I_s(0), R(0), M(0))$.

So, $(t) \leq \frac{\Lambda}{\mu}$, as $t \to \infty$.

3. Existence of the equilibrium points

It has been noted that the first three equations and the fifth equation of system (1) do not contain the variable R, which stands for the recovery rate. Hence, an alternative system can be

solved instead of the original system (1). By solving this alternative system and substituting the obtained values of I_a and I_s into the second and third equations of system (1), we can treat them as linear differential equations with respect to the variable R. Consequently, we obtain the solution for the fourth equation as t approaches infinity, which can be expressed as follows:

$$R(t) = \frac{\alpha_1 \check{I}_a + \alpha_2 \check{I}_s}{\mu}, \tag{2}$$

The solution values for the following system (3) are represented by $\check{I}_a$ and $\check{I}_s$.Therefore, we will focus on studying the system (3) instead of the original system (1).

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \frac{\beta S I_s}{1+hM} - \mu S \\
\frac{dI_a}{dt} &= \frac{p\beta S I_s}{1+hM} - \frac{\sigma I_a}{1+hM} - (\mu + \alpha_1) I_a \\
\frac{dI_s}{dt} &= \frac{(1-p)\beta S I_s}{1+hM} + \frac{\sigma I_a}{1+hM} - (\mu + \alpha_2) I_s \\
\frac{dM}{dt} &= \rho I_a + \theta I_s - \delta M
\end{aligned} \tag{3}$$

There are three equilibrium points in system (3), and they are as follows:

- The first equilibrium point (FEP) is $F_0 = (\bar{S}, 0,0,0)$,

where

$$\bar{S} = \Lambda/\mu \quad . \tag{4}$$

- The second equilibrium point (SEP) is $F_1 = (\tilde{S}, 0, \tilde{I}_s, \tilde{M})$ when $p = 0$,

where

$$\tilde{S} = \frac{(1+h\tilde{M})(\mu+\alpha_2)}{\beta} \ , \tag{5}$$

$$\tilde{I}_s = \frac{(1+h\tilde{M})(\Lambda-\mu\tilde{S})}{\beta\tilde{S}} \ , \tag{6}$$

$$\tilde{M} = \frac{\theta \tilde{I}_s}{\delta} , \tag{7}$$

exists, if:

$$\mu\tilde{S} < \Lambda \ . \tag{8}$$

- The third equilibrium point (TEP) is $F_2 = (S^*, I_a^*, I_s^*, M^*)$,

where

$$S^* = \frac{\Lambda(1+hM^*)}{\beta I_s^* + \mu(1+hM^*)} \ , \tag{9}$$

$$I_a^* = \frac{p\beta S^* I_s^*}{\sigma + (1+hM^*)(\mu+\alpha_1)} \ , \tag{10}$$

$$I_s^* = \frac{\sigma I_a^*}{p\beta S^* + (1+hM^*)(\mu+\alpha_2) - \beta S^*} \ , \tag{11}$$

$$M^* = \frac{\rho I_a^* + \theta I_s^*}{\delta} , \tag{12}$$

exists, if:

$$\beta S^* < p\beta S^* + (1 + hM^*)(\mu + \alpha_2). \tag{13}$$

4. Local stability analysis

In this section, the linearization method is used to examine the local stability of the system (3).The Jacobian matrix (J.M.) of system (3), at $F = (S, I_a, I_s, M)$ is $J = (a_{ij})_{4\times4}$; $i, j = 1,2,3,4,$

Here

$$a_{11} = -\left(\frac{\beta I_s}{1+hM} + \mu\right) \ , \quad a_{13} = \frac{-\beta S}{1+hM} \ , a_{14} = \frac{\beta S I_s h}{(1+hM)^2} \ , \quad a_{21} = \frac{p\beta I_s}{1+hM} \ ,$$
$$a_{22} = -\left(\frac{\sigma}{1+hM} + \mu + \alpha_1\right) \ , a_{23} = \frac{p\beta S}{1+hM} \ , \quad a_{24} = \frac{-h}{(1+hM)^2}(p\beta S I_s - \sigma I_a) \ ,$$
$$a_{31} = \frac{(1-p)\beta I_s}{1+hM} , a_{32} = \frac{\sigma}{1+hM} \ , \quad a_{33} = \frac{(1-p)\beta S}{1+hM} - (\mu + \alpha_2) \ , a_{12} = a_{41} = 0 \ ,$$
$$a_{34} = \frac{-h}{(1+hM)^2}\left((1-p)\beta S I_s + \sigma I_a\right) , a_{42} = \rho \ , a_{43} = \theta \ , a_{44} = -\delta .$$

……..(14)

Theorem 2: F_0 is locally asymptotically stable (L.A.S.) if the conditions are satisfied:

$$\beta\bar{S} < p\beta\bar{S} + \sigma + 2\mu + \alpha_1 + \alpha_2, \tag{15a}$$
$$\beta\bar{S}(\sigma + \mu + \alpha_1) < p\beta\bar{S}(\mu + \alpha_1) + \mu(\sigma + \mu + \alpha_1 + \alpha_2) + \alpha_2(\sigma + \alpha_1). \tag{15b}$$

Proof: the (J.M) at F_0 is:

$$J(F_0) = \begin{bmatrix} -\mu & 0 & -\beta\bar{S} & 0 \\ 0 & -(\sigma+\mu+\alpha_1) & p\beta\bar{S} & 0 \\ 0 & \sigma & (1-p)\beta\bar{S} - (\mu+\alpha_2) & 0 \\ 0 & \rho & \theta & -\delta \end{bmatrix} \tag{16}$$

The characteristic equation (C.E.) of $J(F_0)$ is :

$$[-\mu - \lambda][-\delta - \lambda][\lambda^2 + A_1\lambda + A_2] = 0 \tag{17a}$$

Here

$$A_1 = p\beta\bar{S} + \sigma + 2\mu + \alpha_1 + \alpha_2 - \beta\bar{S},$$
$$A_2 = p\beta\bar{S}(\mu + \alpha_1) + \mu(\sigma + \mu + \alpha_1 + \alpha_2) + \alpha_2(\sigma + \alpha_1) - \beta\bar{S}(\sigma + \mu + \alpha_1).$$

The equation (17a) has four roots, representing the eigenvalues of $J(F_0)$:

$$\left.\begin{aligned} \lambda_{2,3} &= -\tfrac{A_1}{2} \mp \tfrac{1}{2}\sqrt{A_1^2 - 4A_2} \\ \lambda_1 &= -\mu < 0 \\ \lambda_4 &= -\sigma < 0 \end{aligned}\right\} \tag{17b}$$

Then, all the eigenvalues will be non-positive and hence F_0 is (L.A.S.) , if the conditions (15a) - (15b) holds.

Theorem 3: F_1 becomes (L.A.S.) when the specified conditions are met:

$$\beta\tilde{S} < \left(1 + h\tilde{M}\right)(\mu + \alpha_2) \tag{18a}$$
$$\tilde{I}_s h\theta < \left(1 + h\tilde{M}\right)\delta \tag{18b}$$

Proof: the (J.M.) at F_1 is

$J(F_1) = \left(d_{ij}\right)_{4\times4}$; $i, j = 1,2,3,4$

here

$$d_{11} = -\left(\frac{\beta\tilde{I}_s}{1+h\tilde{M}} + \mu\right) \ , d_{13} = -\frac{\beta\tilde{S}}{1+h\tilde{M}} \ , d_{14} = \frac{\beta h\tilde{S}\tilde{I}_s}{(1+h\tilde{M})^2} \ ,$$
$$d_{22} = -\left(\frac{\sigma}{1+h\tilde{M}} + \mu + \alpha_1\right) \ , \ d_{31} = \frac{\beta\tilde{I}_s}{1+h\tilde{M}} \ , \ d_{32} = \frac{\sigma}{1+h\tilde{M}} \ ,$$
$$d_{33} = \frac{\beta\tilde{S}}{1+h\tilde{M}} - (\mu + \alpha_2) \ , d_{34} = -\frac{\beta h\tilde{S}\tilde{I}_s}{(1+h\tilde{M})^2} \ , d_{42} = \rho \ , d_{43} = \theta \ ,$$
$$d_{44} = -\delta \ , \ d_{12} = d_{21} = d_{23} = d_{24} = d_{41} = 0 \ .$$

……(19)

The (C.E.) of $J(F_1)$ is :

$$\left[-\left(\frac{\sigma}{1+h\widetilde{M}}+\mu+\alpha_1\right)-\lambda\right][\lambda^3+D_1\lambda^2+D_2\lambda+D_3]=0 \quad (20)$$

Hear

$$D_1=-(d_{11}+d_{33}+d_{44}),$$
$$D_2=(d_{11}d_{33}-d_{13}d_{31})+(d_{33}d_{44}-d_{34}d_{43}),$$
$$D_3=d_{11}(d_{34}d_{43}-d_{33}d_{44})+d_{31}(d_{13}d_{44}-d_{14}d_{43}).$$

While

$\Delta= D_1D_2-D_3$, that is

$$\Delta= d_{11}(d_{13}d_{31}-d_{11}d_{33}-d_{11}d_{44})+d_{33}(d_{13}d_{31}-2d_{11}d_{44}-d_{33}d_{44}+d_{34}d_{43})+$$
$$d_{44}(d_{13}d_{31}-d_{11}d_{44}-d_{33}d_{44}++d_{34}d_{43})-(d_{11}d_{33}^2+d_{13}d_{31}d_{44})+d_{14}d_{31}d_{43}.$$

So, either

$$\left[-\left(\frac{\sigma}{1+h\widetilde{M}}+\mu+\alpha_1\right)-\lambda\right], \quad (21a)$$

or

$$[\lambda^3+D_1\lambda^2+D_2\lambda+D_3]=0, \quad (21b)$$

from equation (21a) we obtain that

$$\lambda_2=-\left(\frac{\sigma}{1+h\widetilde{M}}+\mu+\alpha_1\right)<0$$

The eigenvalue of the Jacobian matrix is consistently negative.

However, it is easy to confirm that $D_1>0\ and\ D_3>0$ under the conditions (18a) - (18b) . While $\Delta>0$ under the condition (18a). So, all the eigenvalues λ_1 , $\lambda_3 and\ \lambda_4$ of Eq.(21b) have negative real parts. So, F_1 is (L.A.S.) , if the conditions (18a-18b) are holds.

Theorem 4: F_2 is (L.A.S.) in the subregion $\Omega\in R_+^4$ which meets the conditions:

$$\rho<\mu+\alpha_1, \quad (22a)$$
$$\frac{2\beta S^*}{1+hM^*}+\theta<\mu+\alpha_2, \quad (22b)$$
$$h(p\beta S^*I_s^*+2\beta S^*I_s^*+\sigma I_a^*)<\ \delta(1+hM^*)^2+h\sigma I_a^*(1+hM^*)+hp\beta S^*I_s^* \quad . \quad (22c)$$

Proof: the (J.M.) at F_2 is

$$J(F_2)=\left(r_{ij}\right)_{4\times4}\ ;\ i,j=1,2,3,4$$

here

$$r_{11}=-\left(\frac{\beta I_s^*}{1+hM^*}+\mu\right)\ ,\ r_{13}=\frac{-\beta S^*}{1+hM^*}\ ,r_{14}=\frac{\beta S^*I_s^*h}{(1+hM)^2}\ ,\ r_{21}=\frac{p\beta I_s^*}{1+hM^*},$$
$$r_{22}=-\left(\frac{\sigma}{1+hM^*}+\mu+\alpha_1\right)\ \ ,r_{23}=\frac{p\beta S^*}{1+hM^*}\ ,\ r_{24}=\frac{-h}{(1+hM^*)^2}(p\beta S^*I_s^*-\sigma I_a^*)\ ,$$
$$r_{31}=\frac{(1-p)\beta I_s^*}{1+hM^*},r_{32}=\frac{\sigma}{1+hM^*}\ ,\ r_{33}=\frac{(1-p)\beta S^*}{1+hM^*}-(\mu+\alpha_2)\ ,r_{12}=r_{41}=0\ ,$$
$$r_{34}=\frac{-h}{(1+hM^*)^2}\left((1-p)\beta S^*I_s^*+\sigma I_a^*\right),r_{42}=\rho\ ,r_{43}=\theta\ ,r_{44}=-\delta.$$

……(23)

If the requirement is met, the Gersgorin theorem [26] can be used,

$$|r_{ii}|>\sum_{\substack{i=1\\ i\neq j}}^{4}|r_{ij}|.$$

So, all the eigenvalues of (J.M.) at (F_2) exists in Ω , where

$$\Omega=\cup\left\{U^*\in C:\left|U^*-r_{ij}\right|<\sum_{\substack{i=1\\ i\neq j}}^{4}|r_{ij}|\right\}$$

Consequently, every eigenvalue of $J(F_2)$ lies within the disk centered at r_{ii}. Hence, when the diagonal elements are negative and conditions (22a)-(22c) are satisfied, all the eigenvalues will reside in the left half-plane, resulting in the (L.A.S.) of the (TEP).

5. Global stability analysis

In this section, the theorems that demonstrate the global stability of all (E.Ps.) in system (3) have been presented.

Theorem 5: Assume that F_0 is (L.A.S.),then it is a globally asymptotically stable (G.A.S.) if:

$$\rho < \mu + \alpha_1, \tag{24a}$$

$$\beta S + (1 + hM)\theta < (\mu + \alpha_2)(1 + hM). \tag{24b}$$

Proof: we define:

$$W_1(S, I_a, I_s, M) = \frac{(S-\bar{S})^2}{2} + I_a + I_s + M,$$

It is evident that W_1 is a positive definite function and $W_1: R_+^4 \longrightarrow R$ is a continuously differentiable function,

$W_1(\bar{S}, 0,0,0) = 0$ and $W_1(S, I_a, I_s, M) > 0$, $\forall (S, I_a, I_s, M) \neq (\bar{S}, 0,0,0)$.

Further,

$$\frac{dW_1}{dt} = (S - \bar{S})\left[\Lambda - \frac{\beta S I_s}{1+hM} - \mu S\right] - (\mu + \alpha_1 - \rho) I_a - \left[\mu + \alpha_2 - \left(\frac{\beta S}{1+hM} + \theta\right)\right] I_s - \delta M ,$$

Hence we obtain

$$\frac{dW_1}{dt} \leq (S - \bar{S})\left[\frac{\beta S I_s}{1+hM} + \mu\right] - \delta M.$$

Consequently, due to the conditions above , we get $\frac{dW_1}{dt} \leq 0$, hence W_1 is Lyapunov function with respect to F_0. Thus ,(FEP) is a (G.A.S.).

Theorem 6: Assume that F_1 is (L.A.S.), then it is a (G.A.S.) , if:

$$\tilde{S}M(S + 1) + S\widetilde{M}\left(\tilde{S} + I_s\right) < S\widetilde{M}(S + \tilde{I}_s) + \tilde{S}M\left(\tilde{S} + I_s\right), \tag{25a}$$

$$I_s < \tilde{I}_s + 1, \tag{25b}$$

$$M\rho < \mu + \alpha_2 + \widetilde{M}\rho + \theta M, \tag{25c}$$

$$M + \widetilde{M} < \widetilde{M}, \tag{25d}$$

$$\alpha_2 k + \beta S + \beta h \widetilde{M} S < \mu\, k, \tag{25e}$$

$$P_{12}^2 < 4 P_{11} P_{22}. \tag{25f}$$

Where, P_{ij} , $i, j = 1,2$ is given in the proof.

Proof: we define:

$$W_2(S, I_a, I_s, M) = \frac{(S-\tilde{S})^2}{2} + I_a + \frac{(I_s-\tilde{I}_s)^2}{2} + \frac{(M-\widetilde{M})^2}{2}$$

It is evident that W_2 is a positive definite function and $W_2: R_+^4 \longrightarrow R$ is a continuously differentiable function,

$W_2\left(\tilde{S}, 0, \tilde{I}_s, \widetilde{M}\right) = 0$ and $W_2(S, I_a, I_s, M) > 0$, $\forall (S, I_a, I_s, M) \neq \left(\tilde{S}, 0, \tilde{I}_s, \widetilde{M}\right)$.

Furthermore, by differentiating W_2 with respect to time and simplifying the resulting expressions, we obtain the desired outcome as follows:

$$\frac{dW_2}{dt} = \left(S - \tilde{S}\right)\left[\Lambda - \frac{\beta S I_s}{1 + hM} - \mu S\right] - \frac{\sigma I_a}{1 + hM} - \mu I_a - \alpha_1 I_a + (I_s - \tilde{I}_s)\left[\frac{\beta S I_s}{1 + hM} + \frac{\sigma I_a}{1 + hM} - (\mu + \alpha_2) I_s\right] + \left(M - \widetilde{M}\right)[\rho I_a + \theta I_s - \delta M]$$

$$\frac{dW_2}{dt} = -\left[P_{11}\left(S-\tilde{S}\right)^2 - P_{12}\left(S-\tilde{S}\right)(I_s-\tilde{I}_s) + P_{22}(I_s-\tilde{I}_s)^2\right]$$
$$-\frac{\beta h\tilde{I}_s}{k}\left[S^2\widetilde{M} + \tilde{S}^2M + \tilde{S}MI_s + \tilde{I}_s\widetilde{M}S - \left(\tilde{S}\left(SM + S\widetilde{M} + M\right) + S\widetilde{M}I_s\right)\right]$$
$$-\frac{\sigma I_a}{1+hM}\left[\tilde{I}_s + 1 - I_s\right] - I_a\left[\mu + \alpha_2 + \widetilde{M}\rho + \theta M - M\rho\right]$$
$$-\theta\left[\widetilde{M}I_s - \left(MI_s + \widetilde{M}\tilde{I}_s\right)\right],$$

Where, $k = (1+hM)\left(1+h\widetilde{M}\right)$.

Consequently, by using the conditions (25a – 25f) it follows that:

$$\frac{dW_2}{dt} \le -\left[\sqrt{P_{11}}\left(S-\tilde{S}\right) - \sqrt{P_{22}}(I_s-\tilde{I}_s)\right]^2.$$

where ,

$$P_{11} = \frac{\beta\,\tilde{I}_s}{k} + \mu\ , P_{12} = \frac{\beta}{k}\left[I_s - S - hS\widetilde{M}\right]\ , P_{22} = \left[\mu - \left(\alpha_2 + \frac{\beta S}{k} + \frac{\beta h\widetilde{M}S}{k}\right)\right]$$

Obviously, $\frac{dW_2}{dt} \le 0$, hence W_2 is Lyapunov function. Thus, (SEP) is a (G.A.S.).

Theorem 7: Assume that F_2 is (L.A.S.),then it is a (G.A.S.) if:

$$C_1 < C_2, \tag{26a}$$
$$(1-p)\beta(S+I_s^*) < k_1(\mu+\alpha_2) \tag{26b}$$
$$D_{12}^2 < \frac{4}{6}D_{11}D_{22}, \tag{26c}$$
$$D_{13}^2 < \frac{4}{6}D_{11}D_{33}, \tag{26d}$$
$$D_{23}^2 < \frac{4}{9}D_{22}D_{33}, \tag{26e}$$
$$D_{24}^2 < \frac{4}{6}D_{22}D_{44}, \tag{26f}$$
$$D_{34}^2 < \frac{4}{6}D_{33}D_{44}. \tag{26g}$$

Where ,

$$C_1 = SM^*(S + pI_a^* + (1-p)I_s^*) + S^*M(S^* + pI_a + (1-p)I_s),$$
$$C_2 = SM^*(S^* + pI_a + (1-p)I_s) + S^*M(S + pI_a^* + (1-p)I_s^*).$$
$$D_{11} = \frac{\beta I_s^*}{k_1} + \mu\ , D_{12} = \frac{p\beta I_s^*}{k_1}\ , D_{22} = \frac{\sigma}{k_1}(1+hM^*) + (\mu+\alpha_1)\ ,$$
$$D_{13} = \frac{\beta S}{k_1}(1+hM^*) - \frac{(1-p)\beta I_s^*}{k_1},\ D_{23} = \frac{p\beta S}{k_1}, D_{44} = \delta, D_{34} = \theta,$$
$$D_{33} = (\mu+\alpha_1) - \frac{(1-p)\beta S}{k_1} - \frac{(1-p)\beta I_s^*}{k_1}, D_{24} = \frac{\sigma h I_a^*}{k_1} + \rho.$$

Proof: we define:

$$W_3(S, I_a, I_s, M) = \frac{(S-S^*)^2}{2} + \frac{(I_a-I_a^*)^2}{2} + \frac{(I_s-I_s^*)^2}{2} + \frac{(M-M^*)^2}{2}$$

It is evident that W_3 is a positive definite function and $W_3: R_+^4 \longrightarrow R$ is a continuously differentiable function,

$W_3(S^*, I_a^*, I_s^*, M^*) = 0$ and $W_3(S, I_a, I_s, M) > 0\ , \forall (S, I_a, I_s, M) \neq (S^*, I_a^*, I_s^*, M^*)$.

Furthermore, it can be shown that by differentiating W_3 with respect to time and simplifying the resulting expressions, we obtain the desired outcome.

$$\frac{dW_3}{dt} = (S-S^*)\left[\Lambda - \frac{\beta SI_s}{1+hM} - \mu S\right] + (I_a - I_a^*)\left[\frac{p\beta SI_s}{1+hM} - \frac{\sigma I_a}{1+hM} - (\mu+\alpha_1)I_a\right]$$
$$+(I_s - I_s^*)\left[\frac{(1-p)\beta SI_s}{1+hM} + \frac{\sigma I_a}{1+hM} - (\mu+\alpha_2)I_s\right] + (M-M^*)[\rho I_a + \theta I_s - \delta M]$$

$$\frac{dW_3}{dt} = -\left[\frac{D_{11}}{2}(S-S^*)^2 - D_{12}(S-S^*)(I_a-I_a^*) + \frac{D_{22}}{3}(I_a-I_a^*)^2\right]$$
$$-\left[\frac{D_{11}}{2}(S-S^*)^2 + D_{13}(S-S^*)(I_s-I_s^*) + \frac{D_{33}}{3}(I_s-I_s^*)^2\right]$$
$$-\left[\frac{D_{22}}{3}(I_a-I_a^*)^2 - D_{23}(I_a-I_a^*)(I_s-I_s^*) + \frac{D_{33}}{2}(I_s-I_s^*)^2\right]$$
$$-\left[\frac{D_{22}}{3}(I_a-I_a^*)^2 - D_{24}(I_a-I_a^*)(M-M^*) + \frac{D_{44}}{2}(M-M^*)^2\right]$$
$$-\left[\frac{D_{33}}{3}(I_s-I_s^*)^2 - D_{34}(I_s-I_s^*)(M-M^*) + \frac{D_{44}}{3}(M-M^*)^2\right]$$
$$-\frac{\beta h I_s^*}{k_1}\left[SM^*(S+pI_a^*+(1-p)I_s^*) + S^*M(S^*+pI_a+(1-p)I_s) - \right.$$
$$\left.\left(SM^*(S^*+pI_a+(1-p)I_s) + S^*M(S+pI_a^*+(1-p)I_s^*)\right)\right]$$

Where, $k_1 = (1+hM)(1+hM^*)$.

Consequently, by using the conditions (26a – 26g) it follows that:

$$\frac{dW_3}{dt} \leq -\left[\sqrt{\frac{D_{11}}{2}}(S-S^*) - \sqrt{\frac{D_{22}}{3}}(I_a-I_a^*)\right]^2 - \left[\sqrt{\frac{D_{11}}{2}}(S-S^*) + \sqrt{\frac{D_{44}}{3}}(I_s-I_s^*)\right]^2$$
$$-\left[\sqrt{\frac{D_{22}}{3}}(I_a-I_a^*) - \sqrt{\frac{D_{33}}{2}}(I_s-I_s^*)\right]^2 - \left[\sqrt{\frac{D_{22}}{3}}(I_a-I_a^*) - \sqrt{\frac{D_{44}}{2}}(M-M^*)\right]^2$$
$$-\left[\sqrt{\frac{D_{33}}{3}}(I_s-I_s^*) - \sqrt{\frac{D_{44}}{2}}(M-M^*)\right]^2.$$

Obviously, $\frac{dW_3}{dt} \leq 0$, hence W_3 is Lyapunov function. Thus, (TEP) is a (G.A.S.).

6. Numerical simulation

To validate our findings and gain a better understanding of how altering parameter values affects the system's dynamics, we conducted numerical simulations in this section. We initiated the numerical solution of the system using different initial conditions, following the application of hypothetical parameter values. The trajectories generated were illustrated using MATLAB 2014a.

$$\Lambda = 5000\,, \beta = 0.0003\,, h = 0.00002\,, p = 0.02\,, \alpha_1 = 0.005\,,$$
$$\sigma = 0.01\,,\ \theta = 0.003\,,\ \alpha_2 = 0.4\,, \rho = 0.03\,, \delta = 0.01,\ \mu = 0.01. \qquad (27)$$

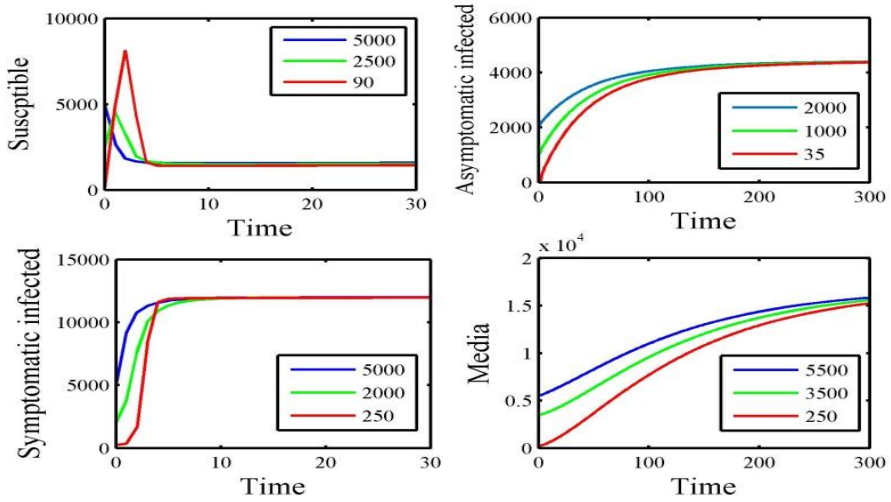

Figure 2: Trajectories of the system (3) for (27) which approaches to $F_2 = (0.1807\,, 0.4369\,, 1.1989\,, 1.5240)$.

The results of our analytical effort are validated by the phase diagram provided by Figure 2,which clearly demonstrates that the (TEP) of the system (3) indicated by $F_2 = (0.1807\ , 0.4369\ ,\ 1.1989\ , 1.5240)$ is a (G.A.S.). Now, in order to illustrate the effect of the parameter values of system (3) on the dynamical behavior of the system, the system is numerically solved for the data supplied in (27) while changing one or more parameters each time. It is noted that when the parameter $\beta = 0.0000003$ is changed for the remaining data in Eq. (27), the trajectories of system (3) approach asymptotically to $F_0 = (4.7511\ , 0\ , 0\ , 0)$ as illustrated in Figure 3.

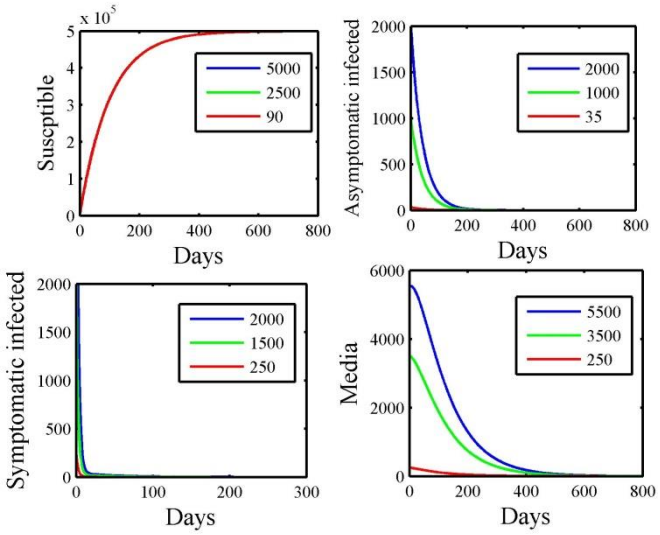

Figure 3: The trajectories of the system (3) for (27) with changing the parameter $\beta = 0.0000003$, which approaches to $F_0 = (4.7511\ , 0\ , 0\ , 0)$.

The trajectories of system (3) approach asymptotically to $F_1 = (0.1462\ , 0\ , 1.2159\ , 0.3477)$ as illustrated in Figure 4 by changing the parameter $p = 0$ while maintaining the rest of the parameter values as in Eq. (27).

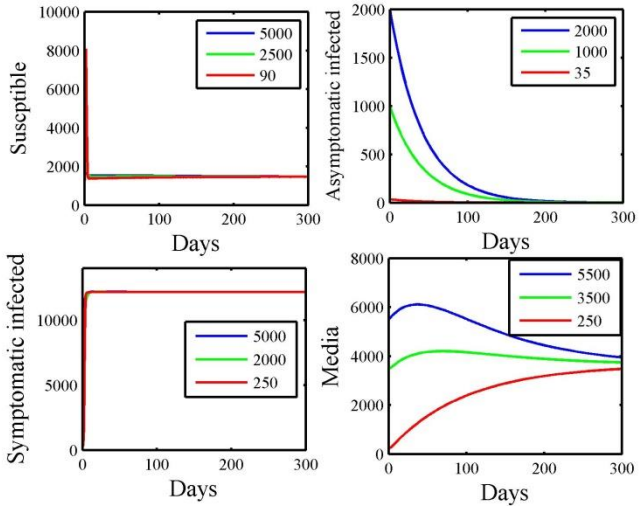

Figure 4: Trajectories of the system (3) for (27) with changing the parameter $p = 0$, which approaches to $F_1 = (0.1462\ , 0\ , 1.2159\ , 0.3477)$.

Now, we discuss the effect of the contacts rate between the susceptible and symptomatic infected β, for the Eq.(27) with different values of $\beta < 0.00003$, the trajectories of system (3) approaches to (FEP) as shown in Figure 5.

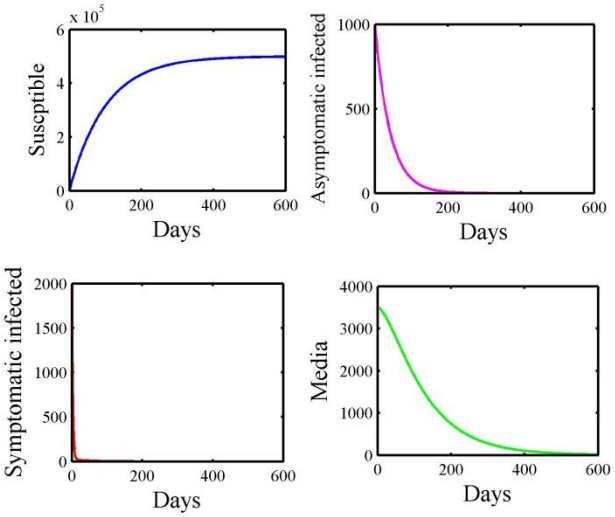

Figure 5: Trajectories of the system (3) for(27) with changing the parameters $\beta < 0.00003$, which approaches to (FEP).

However for $0.00003 \leq \beta \leq 0.003$ it is observed that system (3) as shown in Figure 6.

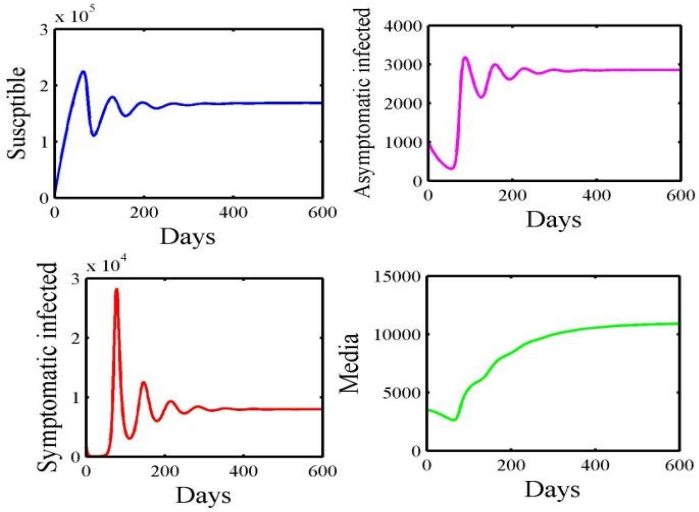

Figure 6: Trajectories of the system (3) for (27) with $0.00003 \leq \beta \leq 0.003$.

Now, the effect of the amount disease control due to the media h, for the Eq.(27) with different values of $h < 0.009$, the trajectories of system (3) approaches to (TEP) as shown in Figure 7.

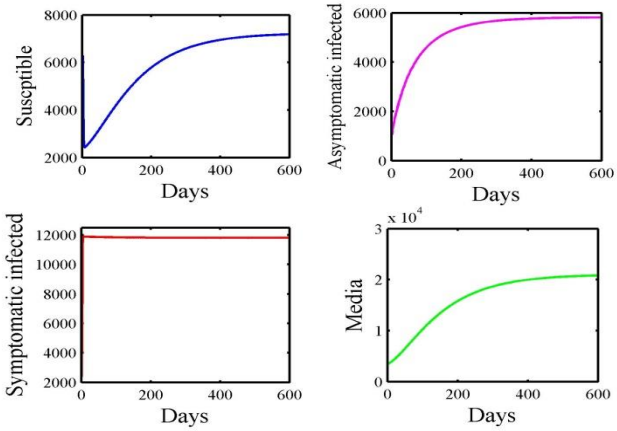

Figure 7: Trajectories of the system (3) for (27) with $h < 0.009$, which approaches to (TEP).

However for $0.009 \leq h \leq 0.02$ it is observed that system (3) as shown in Figure 8.

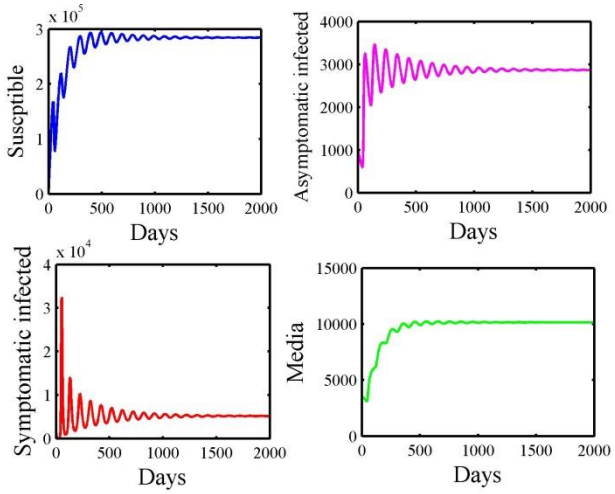

Figure 8: Trajectories of the system (3) for (27) with $0.009 \leq h \leq 0.02$.

Now, the effect of the recovery rate α_1, for the Eq.(27) with different values of $\alpha_1 < 6.8$, the trajectories of system (3) approaches to (TEP) as shown in Figure 9.

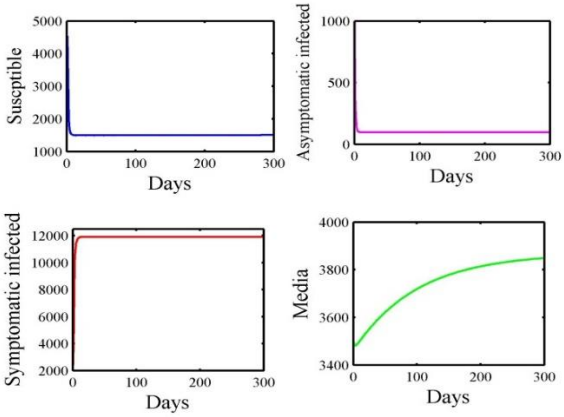

Figure 9: Trajectories of the system (3) for (27) with changing the parameters $\alpha_1 < 6.8$, which approaches to (TEP).

However for $6.8 \leq \alpha_1$ it is observed that system (3) approaches to (SEP) as shown in Figure 10.

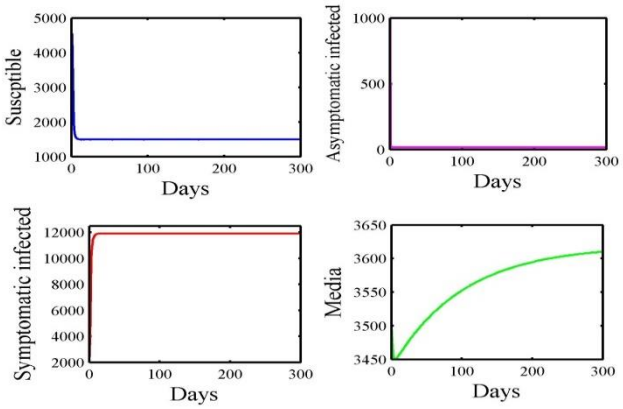

Figure 10: Trajectories of the system (3) for (27) with changing the parameters $6.8 \leq \alpha_1$, which approaches to (SEP).

Finally , we discuss the effect of the recovery rate α_2, for the Eq.(27) with different values of $\alpha_2 \leq 1.5$, the trajectories of system (3) approaches to (TEP) as shown in Figure 11.

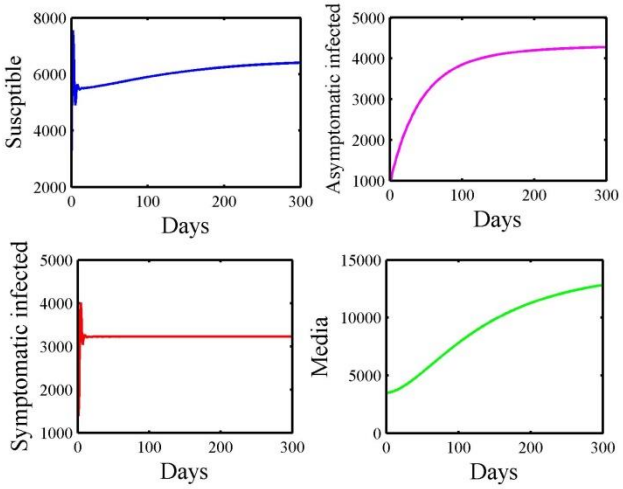

Figure 11: Trajectories of the system (3) for(27) with changing the parameters $\alpha_2 \leq 1.5$, which approaches to (TEP).

However for $1.6 \leq \alpha_2$ it is observed that system (3) as shown in Figure 12.

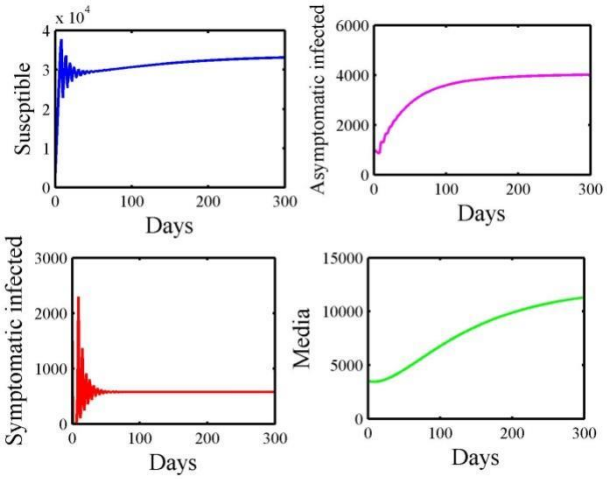

Figure 12: Trajectories of the system (3) for(27) with $1.6 \leq \alpha_2$.

7. Conclusion and discussion

In this article, we examined the impact of media alerts on an epidemiological model using a case with no symptoms. The study's objective is to comprehend how media influence is included in epidemiological models in order to gather knowledge on how it affects disease dynamics, transmission rates, and intervention tactics. The boundedness of the system has been investigated. The existence conditions of all potential system equilibrium points have been established. Both local and global stability are examined for all possible (E.Ps.). The qualitative dynamical behavior as a result of changing the parameter values is investigated using both analytical and numerical methods. Finally, the hypothetical data set that is physiologically plausible as given in (27) is numerically solved for the system (3), and the results are explained in a few standard visuals. The results are summed up as follows:

1- With the parameter values specified in (27), the system (3) demonstrates a state of (G.A.S.), denoted by $F_2 = (S^*, I_a^*, I_s^*, M^*)$.

2- Upon reducing the contact rate β between the susceptible and symptomatic infected populations to a certain threshold, the system (3) follows an asymptotic trajectory toward the (FEP).

3- When the fractional rate $p = 0$ as a condition of existence, the system(3) exhibits an asymptotic trajectory toward the (SEP).

4- Media alerts and public health messaging have the potential to enhance awareness and understanding within the general population regarding asymptomatic cases and their pivotal role in disease transmission. This, in turn, might lead to improved compliance with preventive measures and heightened rates of testing among individuals who could potentially carry the infection without showing symptoms.

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