Mathematical Biosciences and Engineering

http://www.aimspress.com/journal/mbe

MBE, 19(6): 6296–6316.
DOI: 10.3934/mbe.2022294
Received: 29 January 2022
Revised: 31 March 2022
Accepted: 31 March 2022
Published: 19 April 2022

Research article

Mathematical modeling and stability analysis of the time-delayed $SAIM$ model for COVID-19 vaccination and media coverage

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Abstract: Since the COVID-19 outbreak began in early 2020, it has spread rapidly and threatened public health worldwide. Vaccination is an effective way to control the epidemic. In this paper, we model a $SAIM$ equation. Our model involves vaccination and the time delay for people to change their willingness to be vaccinated, which is influenced by media coverage. Second, we theoretically analyze the existence and stability of the equilibria of our model. Then, we study the existence of Hopf bifurcation related to the two equilibria and obtain the normal form near the Hopf bifurcating critical point. Third, numerical simulations based two groups of values for model parameters are carried out to verify our theoretical analysis and assess features such as stable equilibria and periodic solutions. To ensure the appropriateness of model parameters, we conduct a mathematical analysis of official data. Next, we study the effect of the media influence rate and attenuation rate of media coverage on vaccination and epidemic control. The analysis results are consistent with real-world conditions. Finally, we present conclusions and suggestions related to the impact of media coverage on vaccination and epidemic control.

Keywords: COVID-19 epidemic; media coverage; vaccination; time-delay; Hopf bifurcation; normal form; multiple time scales method

1. Introduction

A new viral infection, COVID-19 (Coronavirus Disease 2019) emerged in early 2020 and attracted widespread attention. The virus spread around the world at a very high speed and many studies have been carried out examining different epidemic patterns of COVID-19 based on official data [1–3]. There are many routes through which COVID-19 can spread, and it is possible that the virus may mutate at any time [4]. That is why the epidemic is difficult to control and was designated as a global pandemic by the World Health Organization (WHO). According to statistics from the WHO, more than 482 million people have been diagnosed with COVID-19, and more than 6 million people have died

due to this disease as of March 30, 2022. The situation remains severe even now. Therefore, many scholars are looking for ways to contain the epidemic.

Many epidemic models of COVID-19 have been developed [5–9]. Studying the effects of factors such as incubation periods and vaccines on the spread of COVID-19 by analyzing the dynamic property of the system has been an important area of research. Abdy et al. [10] constructed a COVID-19 SIR Model with fuzzy parameters, taking into account vaccination, treatment, compliance with health protocols, viral load, and other factors. Their simulation results showed that differences in coronavirus loads would also cause differences in the transmission of COVID-19. Likewise, the factors of vaccination and compliance with health protocols had the same effect in slowing or stopping the transmission of COVID-19 in Indonesia. In the work of Wang et al. [11], a $SVEIR$ epidemic model with media impact, age-dependent vaccination and latency was proposed, where the efficacy of vaccines depended on the time since vaccination.

Clearly, vaccines play a vital role in controlling infectious diseases. For COVID-19, a specific vaccine, which significantly reduces the risk of COVID-19 infection, has already been developed and has attracted much attention. In reference [12], Olorunsaiye et al. found that the global differences in the rates of immunization with the COVID-19 vaccine resulted in different levels of COVID-19 immunity in different countries. Based on the study of Anderson et al. [13] and the epidemic data available so far, we found that only a small number of people in Western developed countries have been immunized after paying a heavy toll of death due to COVID-19 infection, and they have had to use mass vaccination to achieve herd immunity. Therefore, it is necessary to study the effects of vaccines in models of COVID-19. Zhai et al. [14] studied vaccination control in an epidemic model with time delay and applied it to COVID-19.

These studies all point to the importance of vaccination against COVID-19. The development of COVID-19 also highlights the need to increase public awareness of disease prevention and promote proper understanding of vaccination. Vaccination rates are related to public opinion [15, 16]. In reference [15], Yang et al. show that communication channels are one of the basic factors affecting the level of COVID-19 vaccination awareness in society. Agaba et al. [16] studied vaccination in a time-delay epidemic model with consciousness. Their model accounted for the contribution of global information campaigns to overall awareness, direct contact between uninformed and informed individuals, and reported cases of infection.

Many studies have shown that media coverage is an effective way to raise awareness of the epidemic and increase people's willingness to be vaccinated. In the study of Kiss et al. [17] in 2010, a model was developed to study whether some people became consciously susceptible and others became unconsciously susceptible after recovering from the disease. They showed that if the dissemination of information is fast enough, infection will be eradicated. When this is not possible, information transmission has an important effect on reducing the prevalence of the infection. In reference [18], Kar et al. established a $SEIR$ model, analyzed the existence and stability of the disease-free equilibrium point of the model, and found that the system had the phenomenon of backward bifurcation, which considered the two important control measures of treatment and media coverage. Obviously, media coverage can affect the psychology of people and, thus, control the spread of infectious diseases.

According to the characteristics of the COVID-19 epidemic, time delay plays an important role in the epidemic models studied. Liu et al. [19] developed a differential equation with time delay to assess

the impact of the incubation period on the epidemiological dynamics of COVID-19 before an infected person was able to transmit the infection to others. In reference [20–23], it showed that the model with the time delay could better describe the epidemic trend of infectious diseases. In some cases, the original stable equilibrium became unstable and destroyed the stability of endemic disease equilibrium.

COVID-19 is an infectious disease that is not completely controlled and, therefore, has great research significance. Considering the effectiveness of vaccines, studying the impact of media coverage on people's willingness to get vaccinated against COVID-19 is key for analyzing the role of media in controlling the spread of COVID-19. Therefore, the main objectives of this paper include constructing a COVID-19 vaccination willingness model in the context of media coverage and analyzing the stability of the model and the existence and stability of Hopf bifurcation.

The rest of the content is arranged as follows. In Section 2, we establish a model based on the characteristics of media coverage and COVID-19 transmission. In Section 3, we analyse the existence and stability of equilibria and the existence of Hopf bifurcation for models with time delay. In Section 4, we derive the normal form of the Hopf bifurcation of the above model and analyse the stability of the periodic solution of the bifurcation. In Section 5, we present numerical simulations to verify the correctness of our analysis. Finally, the conclusion is drawn in Section 6.

2. Mathematical modeling

Media coverage has an impact on epidemic prevention and control. In reference [24], Misra et al. studied the time delay of media broadcasting and established the following model:

$$\begin{cases} \dfrac{dX}{dt} = A - \beta XY - \lambda X\dfrac{M}{k+M} - dX + \nu Y + \lambda_0 X_m, \\ \dfrac{dY}{dt} = \beta XY - \nu Y - \alpha Y - dY, \\ \dfrac{dX_m}{dt} = \lambda X\dfrac{M}{k+M} - dX_m - \lambda_0 X_m, \\ \dfrac{dM}{dt} = \mu Y(t-\tau) - \mu_0 M, \end{cases} \tag{2.1}$$

where X, Y, and X_m denote the numbers of susceptible, infected and conscious people, respectively, and M denotes the number of people affected by media coverage. $A = X + X_m + Y$ is the number of total population individuals. τ is the time delay of the media coverage effect. Misra et al. [24] analyzed the Hopf bifurcation of their model and concluded that although awareness programs cannot eradicate infection, they help in controlling the prevalence of disease.

People who are susceptible to COVID-19 can become infected, and vaccination is one of the effective ways to reduce the likelihood of the susceptible population becoming infected. We will improve the system (2.1) based on the work of Misra et al. [24] and assume that the country has sufficient vaccine resources. We study the influence of media coverage on vaccination and COVID-19 epidemic control.

We divide the people into three groups. One group includes susceptible people who do not have antibodies in their bodies (S); that is, they have not received the vaccine. Another group includes people who have antibodies (A). The third group includes infected people (I). Most people from A obtain antibodies through vaccination ($S \to A$). We assume these people are influenced by media

because they need to be informed about vaccines through media coverage. The remaining people in A were infected and developed antibodies after recovering since the vaccine is an inactivated virus [25] ($I \rightarrow A$). Both S and A are likely to suffer from COVID-19 after contact with infected people and become infected people I. First, this is because current vaccines reduce the infection rate but do not impart complete immunity. Second, recovered people can be reinfected [26] (see Figure 1).

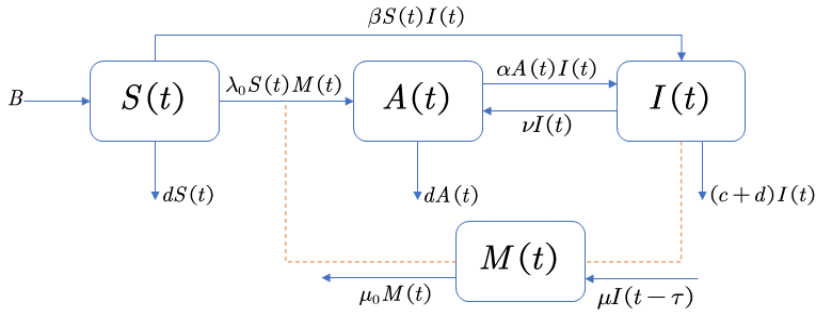

Figure 1. $SAIM$ Model diagram.

As shown in Figure 1, we present a differential equation model with a time delay, and the relationships among the four populations are obtained. The solid line represents the flow relationship among the non-antibody population S, the antibody population A and the infected population I. The dotted line indicates that cabin (M) does not participate in the population flow, but it is affected by cabin I and affects the conversion rate from S to A. In this model, it is clear that people with antibodies are less likely to be infected, that is, the proportion of A converting to I is less than that of S, which means $\alpha < \beta$. Some infected people die of the disease, while others are cured after treatment and have antibodies. We define the cure rate as ν. After τ days since the epidemic was reported by the media, people realized the seriousness of the epidemic. We assume the people who don't exhibit vaccination willingness at the beginning, but accept the idea of vaccination after being influenced by the media are (M). Media broadcast has an attenuation rate μ_0. The number of people who follow the media will influence the vaccination rate with an influence coefficient λ_0. This is consistent with the fact that the greater the number of people who pay attention to the news and think it is necessary to get vaccinated is, the higher the vaccination rate, converting S to A. Thus, we construct the following model:

$$\begin{cases} \dfrac{dS(t)}{dt} = B - \beta S(t) I(t) - dS(t) - \lambda_0 M(t) S(t), \\ \dfrac{dA(t)}{dt} = \lambda_0 M(t) S(t) - dA(t) - \alpha A(t) I(t) + \nu I(t), \\ \dfrac{dI(t)}{dt} = \beta S(t) I(t) + \alpha A(t) I(t) - \nu I(t) - (c+d) I(t), \\ \dfrac{dM(t)}{dt} = \mu I(t-\tau) - \mu_0 M(t), \end{cases} \tag{2.2}$$

where B, β, d, λ_0, α, ν, c, μ, μ_0 are positive parameters; S, A, I, M are variables; and τ is the time delay for people to change their willingness to vaccinate. It is influenced by media coverage of the epidemic. The specific descriptions are given in Table 1.

Table 1. Descriptions of variables and parameters in the model (2.2).

Symbol	Description
S	Number of susceptible people without antibodies
A	Number of susceptible people with antibodies
I	Number of infected people
M	Number of people who are influenced by the media and accept the idea of vaccination
B	Natural increase rate of population
β	Transition rate from S to I
λ_0	Transition rate from S to A
α	Transition rate from A to I
ν	Transition rate from I to A, the cure rate of infected people
d	Natural death rate of population
c	National case fatality rate of COVID-19
μ	Media influence rate
μ_0	Attenuation rate of media coverage
τ	Time-delay for people to change their willingness to vaccinate

3. Stability analysis of equilibria and the existence of Hopf bifurcation

In this section, the system (2.2) is considered. Clearly, the system (2.2) has two equilibria:

$$P_1 = \left(\frac{B}{d}, 0, 0, 0\right),\ P_2 = (S^*, A^*, I^*, M^*), \tag{3.1}$$

where $S^* = \frac{B}{\beta I^* + d + \xi I^*}$, $A^* = \frac{B}{d} - \frac{B}{\beta I^* + d + \xi I^*} - \frac{c+d}{d} I^*$, $M^* = \frac{\mu}{\mu_0} I^*$,

$I^* = \frac{-\left[(c+d)\alpha d - (\alpha B - (\nu+c+d)d)(\beta+\xi)\right]}{2(c+d)\alpha(\beta+\xi)} + \frac{\sqrt{\left[(c+d)\alpha d - (\alpha B - (\nu+c+d)d)(\beta+\xi)\right]^2 - 4(c+d)\alpha(\beta+\xi)\left[(\nu+c+d)d^2 - Bd\beta\right]}}{2(c+d)\alpha(\beta+\xi)}$, with $\xi = \frac{\lambda_0 \mu}{\mu_0}$.

It is straightforward to find that the basic regeneration number of the system (2.2) is

$$R_0 = \frac{\beta B}{d\,(\nu + c + d)}.$$

When $R_0 < 1$, there is only one semitrivial equilibrium $P_1 = \left(\frac{B}{d}, 0, 0, 0\right)$. Transferring the equilibrium P_1 to the origin and linearizing the surrounding system (2.2), we obtain the characteristic equation of the linearized system as follows:

$$(\lambda + d)^2 \left(\lambda - \frac{\beta B}{d} + \nu + c + d\right)(\lambda + \mu_0) = 0. \tag{3.2}$$

The characteristic equation Eq (3.2) of equilibrium P_1 is independent of τ. Equation (3.2) has four roots: $\lambda_1 = \lambda_2 = -d,\ \lambda_3 = \frac{\beta B}{d} - \nu - c - d,\ \lambda_4 = -\mu_0$. Thus the equilibrium P_1 is locally asymptotically stable for any $\tau \geq 0$ due to $d > 0, \mu_0 > 0, R_0 < 1$.

When $R_0 = 1$, there is also only one semitrivial equilibrium $P_1 = \left(\frac{B}{d}, 0, 0, 0\right)$. The Eq (3.2) has a root: $\lambda = 0$. Therefore, the equilibrium P_1 undergoes a fixed point bifurcation.

When $R_0 > 1$, the equilibrium P_1 is unstable, and the other equilibrium P_2=(S^*, A^*, I^*, M^*) for the system (2.2) exists and is positive.

Similarly, transferring the equilibrium $P_2 = (S^*, A^*, I^*, M^*)$ to the origin and linearizing the system (2.2) around it, we obtain the characteristic equation of the linearized system as follows:

$$\mathrm{e}^{-\lambda\tau}(A_1\lambda + B_1) + \lambda^4 + C_1\lambda^3 + D_1\lambda^2 + E_1\lambda + F_1 = 0, \tag{3.3}$$

where

$$\begin{aligned}
A_1 &= \mu(-\alpha\lambda_0 I^* S^* + \lambda_0\beta S^* I^*),\\
B_1 &= \mu\left[(-\alpha\lambda_0 I^* S^*)(\beta I^* + d + \lambda_0 M^*) + \lambda_0^2\alpha S^* I^* + \lambda_0\beta S^* I^*(d + \alpha I^*)\right],\\
C_1 &= \mu_0 + \beta I^* + 3d + \lambda_0 M^* + c + \nu + \alpha I^* - \beta S^* - \alpha A^*,\\
D_1 &= (\beta I^* + d + \lambda_0 M^*)(\mu_0 + d + \alpha I^* - \beta S^* - \alpha A^* + \nu + c + d)\\
&\quad + (-\beta S^* - \alpha A^* + \nu + c + d)(\mu_0 + d + \alpha I^*) + \mu_0(d + \alpha I^*) + \alpha I^*(\alpha A^* - \nu) + \beta^2 S^* I^*,\\
E_1 &= \mu_0\left[(\beta I^* + d + \lambda_0 M^*)(d + \alpha I^* - \beta S^* - \alpha A^* + \nu + c + d)\right.\\
&\quad \left. + (d + \alpha I^*)(-\beta S^* - \alpha A^* + \nu + c + d) + \alpha I^*(\alpha A^* - \nu) + \beta^2 S^* I^*\right]\\
&\quad + (\beta I^* + d + \lambda_0 M^*)\left[(d + \alpha I^*)(-\beta S^* - \alpha A^* + \nu + c + d) + \alpha I^*(\alpha A^* - \nu)\right]\\
&\quad + \beta^2 S^* I^*(d + \alpha I^*) + \beta\alpha\lambda_0 S^* I^* M^*,\\
F_1 &= \mu_0\left[(\beta I^* + d + \lambda_0 M^*)\left[(d + \alpha I^*)(-\beta S^* - \alpha A^* + \nu + c + d) + \alpha I^*(\alpha A^* - \nu)\right]\right.\\
&\quad \left. + \beta\alpha\lambda_0 S^* I^* M^* + \beta^2 S^* I^*(d + \alpha I^*)\right].
\end{aligned}$$

When $\tau = 0$, Eq (3.3) becomes

$$\lambda^4 + C_1\lambda^3 + D_1\lambda^2 + (A_1 + E_1)\lambda + B_1 + F_1 = 0. \tag{3.4}$$

We consider the following assumption obtained by the Routh-Hurwitz criterion:

(H1) $C_1 > 0,\ C_1D_1 - A_1 - E_1 > 0,\ C_1[D_1(A_1 + E_1) - C_1(B_1 + F_1)] - (A_1 + E_1)^2 > 0,\ B_1 + F_1 > 0.$

Under the assumption **(H1)**, all the roots of Eq (3.4) have negative real parts, and the equilibrium $P_2 = (S^*, A^*, I^*, M^*)$ is locally asymptotically stable when $\tau = 0$.

When $\tau > 0$, we try to discuss the existence of Hopf bifurcation. We assume that $\lambda = \mathrm{i}\omega$ $(\omega > 0)$ is a pure imaginary root of Eq (3.3). Substituting it into Eq (3.3) and separating the real and imaginary parts, we obtain:

$$\begin{cases} C_1\omega^3 - E_1\omega = A_1\omega\cos(\omega\tau) - B_1\sin(\omega\tau), \\ -\omega^4 + D_1\omega^2 - F_1 = B_1\cos(\omega\tau) + A_1\omega\sin(\omega\tau). \end{cases} \tag{3.5}$$

Eq (3.5) derives the following:

$$\begin{cases} \sin(\omega\tau) = \frac{-A_1\omega^5 + (A_1D_1 - B_1C_1)\omega^3 - (A_1F_1 - B_1E_1)\omega}{A_1^2\omega^2 + B_1^2}, \\ \cos(\omega\tau) = \frac{(A_1C_1 - B_1)\omega^4 + (B_1D_1 - A_1E_1)\omega^2 - B_1F_1}{A_1^2\omega^2 + B_1^2}. \end{cases} \tag{3.6}$$

Adding the square of the two equations in Eq (3.5), letting $z = \omega^2$, we obtain

$$h(z) = z^4 + c_3z^3 + c_2z^2 + c_1z + c_0 = 0, \tag{3.7}$$

where $c_3 = C_1^2 - 2D_1,\ c_2 = 2F_1 - 2C_1E_1 + D_1^2,\ c_1 = E_1^2 - 2D_1F_1 - A_1^2,\ c_0 = F_1^2 - B_1^2.$

We hypothesize that Eq (3.7) has k($k = 1, 2, 3, 4$) positive roots and denote them as $z_1 < z_2 < z_3 < z_4$. Substituting $\omega_k = \sqrt{z_k}$ ($k = 1, 2, 3, 4$) into Eq (3.6), we obtain the expression of τ:

$$\tau_k^{(j)} = \begin{cases} \frac{1}{\omega_k}[\arccos(P_k) + 2j\pi], & Q_k \geq 0, \\ \frac{1}{\omega_k}[2\pi - \arccos(P_k) + 2j\pi], & Q_k < 0, \ k = 1, 2, 3, 4, \ j = 0, 1, 2, \cdots, \end{cases} \tag{3.8}$$

where

$$Q_k = \sin(\omega_k \tau_k^{(j)}) = \frac{-A_1\omega_k^5 + (A_1D_1 - B_1C_1)\,\omega_k^3 - (A_1F_1 - B_1E_1)\,\omega_k}{A_1^2\omega_k^2 + B_1^2},$$
$$P_k = \cos(\omega_k \tau_k^{(j)}) = \frac{(A_1C_1 - B_1)\,\omega_k^4 + (B_1D_1 - A_1E_1)\,\omega_k^2 - B_1F_1}{A_1^2\omega_k^2 + B_1^2}.$$

Lemma 3.1. *If $R_0 > 1$ and* **(H1)** *holds, when $\tau = \tau_k^{(j)}$ ($k = 1, 2, 3, 4$; $j = 0, 1, 2, \cdots$), then Eq (3.3) has a pair of pure imaginary roots $\pm i\omega_k$, and all the other roots of Eq (3.3) have nonzero real parts.*

Furthermore, let $\lambda(\tau) = \alpha(\tau) + i\omega(\tau)$ be the root of Eq (3.3) satisfying $\alpha(\tau_k^{(j)}) = 0$, $\omega(\tau_k^{(j)}) = \omega_k$ ($k = 1, 2, 3, 4$; $j = 0, 1, 2, \cdots$).

Lemma 3.2. *If $R_0 > 1$ and* **(H1)** *holds, and $z_k = \omega_k^2$, $h'(z_k) \neq 0$, where $h'(z)$ is the derivative of $h(z)$ with respect to z. Then, we have the following transversality condition:*
$\mathrm{Re}(\frac{d\tau}{d\lambda})\Big|_{\tau=\tau_k^{(j)}} = \mathrm{Re}(\frac{d\lambda}{d\tau})^{-1}\Big|_{\tau=\tau_k^{(j)}} = \frac{h'(z_k)}{A_1^2 z_k + B_1^2} \neq 0$, $k = 1, 2, 3, 4$, $j = 0, 1, 2, \cdots$.

Theorem 3.1. *Considering system (2.2), we draw the following conclusions.*

(1) If $R_0 < 1$ holds, there is only one semitrivial equilibrium P_1 for the system (2.2), and it is locally asymptotically stable for any $\tau \geq 0$.

(2) If $R_0 = 1$ holds, the only semitrivial equilibrium P_1 undergoes a fixed point bifurcation.

(3) If $R_0 > 1$ holds, the equilibrium P_1 is unstable, and the other equilibrium P_2=(S^*, A^*, I^*, M^*) for the system (2.2) exists and is positive. When **(H1)** *holds as well, the equilibrium P_2 of the system (2.2) undergoes Hopf bifurcation at $\tau = \tau_k^{(j)}$ ($k = 1, 2, 3, 4$; $j = 0, 1, 2, \cdots$), where $\tau_k^{(j)}$ is given by Eq (3.8), and*

(a) If $h(z)$ has one positive root z_1, then when $\tau \in [0, \tau_1^{(0)})$, the equilibrium P_2 is locally asymptotically stable and unstable when $\tau > \tau_1^{(0)}$.

(b) If $h(z)$ has two positive roots z_1 and z_2, we suppose $z_1 < z_2$; then, $h'(z_1) < 0, h'(z_2) > 0$; note that $\tau_1^{(0)} > \tau_2^{(0)}$. Then, $\exists\, m \in N$ makes $0 < \tau_2^{(0)} < \tau_1^{(0)} < \tau_2^{(1)} < \tau_1^{(1)} < \cdots < \tau_1^{(m-1)} < \tau_2^{(m)} < \tau_2^{(m+1)}$. When $\tau \in [0, \tau_2^{(0)}) \cup \bigcup_{l=1}^{m} (\tau_1^{(l-1)}, \tau_2^{(l)})$, the equilibrium P_2 of the system (2.2) is locally asymptotically stable, and when $\tau \in \bigcup_{l=0}^{m-1} (\tau_2^{(l)}, \tau_1^{(l)}) \cup (\tau_2^{(m)}, +\infty)$, the equilibrium P_2 is unstable.

(c) If $h(z)$ has three or four positive roots, the phenomenon of stability switching similar to the case of (b) will occur.

4. Normal form of Hopf bifurcation

In this section, we derive the normal form of Hopf bifurcation for the system (2.2) by using the multiple time scales method. To reflect the actual situation, we focus on the delay in people formulating

ideas about COVID-19 vaccination and study the impact of the delay on epidemic control. Therefore, we consider the time-delay τ as a bifurcation parameter. Let $\tau{=}\tau_c + \varepsilon\tau_\varepsilon$, where τ_c is the critical value of Hopf bifurcation given in Eq (3.8), τ_ε is the disturbance parameter, and ε is the dimensionless scale parameter. When $\tau{=}\tau_c$, the characteristic Eq (3.3) has eigenvalue $\lambda = \mathrm{i}\omega^{(k)}$ ($k = 1, 2, 3, 4$), at which system (2.2) undergoes a Hopf bifurcation at equilibrium $P_2 = (S^*, A^*, I^*, M^*)$.

System (2.2) can be written as $\dot{X}(t) = A_2X(t) + B_2X(t-\tau) + F[X(t), X(t-\tau)]$, we let $t \to t/\tau$, then the system (2.2) turns to

$$\dot{X} = \tau A_2 X + B_2 \tau X(t-1) + \tau F(X, X(t-1)), \tag{4.1}$$

where $X(t) = (S, A, I, M)^{\mathrm{T}}$, $X(t-1) = (S(t-1), A(t-1), I(t-1), M(t-1))^{\mathrm{T}}$,

$$A_2 \triangleq \begin{pmatrix} a_{11} & 0 & a_{13} & a_{14} \\ a_{21} & a_{22} & a_{23} & a_{24} \\ a_{31} & a_{32} & a_{33} & 0 \\ 0 & 0 & 0 & a_{44} \end{pmatrix} = \begin{pmatrix} -\beta I^* - d - \lambda_0 M^* & 0 & -\beta S^* & -\lambda_0 S^* \\ \lambda_0 M^* & -d - \alpha I^* & -\alpha A^* + \nu & \lambda_0 S^* \\ \beta I^* & \alpha I^* & \beta S^* + \alpha A^* - \nu - c - d & 0 \\ 0 & 0 & 0 & -\mu_0 \end{pmatrix},$$

$$B_2 = \begin{pmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & \mu & 0 \end{pmatrix}, F(X(t), X(t-1)) \triangleq \begin{pmatrix} F_S \\ F_A \\ F_I \\ F_M \end{pmatrix} = \begin{pmatrix} -\beta S I - \lambda_0 M S \\ \lambda_0 M S - \alpha A I \\ \beta S I + \alpha A I \\ 0 \end{pmatrix}.$$

We suppose h is the eigenvector of the linear operator corresponding to the eigenvalue $\mathrm{i}\omega^{(k)}\tau$ of Eq (4.1) for equilibrium P_2, and h^* is the normalized eigenvector of the adjoint operator of the linear operator corresponding to the eigenvalue $-\mathrm{i}\omega^{(k)}\tau$ and satisfies $\langle h^*, h\rangle = \overline{h^*}^{\mathrm{T}} h = 1$. By simple calculation, we obtain:

$$\begin{aligned} h &= \begin{pmatrix} h_1 \\ h_2 \\ h_3 \\ h_4 \end{pmatrix} = \begin{pmatrix} \frac{-\beta S^*\left(\mathrm{i}\omega^{(k)}+\mu_0\right)-\mathrm{i}\omega^{(k)}\mu\tau S^*\mathrm{e}^{-\mathrm{i}\omega^{(k)}\tau}}{\left(\mathrm{i}\omega^{(k)}+\mu_0\right)\left(-\mathrm{i}\omega^{(k)}+\beta I^*+d+\lambda_0 M^*\right)} \\ \frac{\beta^2 S^*\left(\mathrm{i}\omega^{(k)}+\mu_0\right)+\mathrm{i}\omega^{(k)}\tau\mu\beta S^*\mathrm{e}^{-\mathrm{i}\omega^{(k)}\tau}}{\left(\mathrm{i}\omega^{(k)}+\mu_0\right)\left(\mathrm{i}\omega^{(k)}+\beta I^*+d+\lambda_0 M^*\right)\alpha} + \frac{\left[\mathrm{i}\omega^{(k)}-(\beta S^*+\alpha A^*-\nu-c-d)\right]}{\alpha I^*} \\ 1 \\ \frac{\mu\mathrm{e}^{-\mathrm{i}\omega^{(k)}\tau}}{\mathrm{i}\omega^{(k)}+\mu_0} \end{pmatrix}, \\ h^* &= d\begin{pmatrix} h_1^* \\ h_2^* \\ h_3^* \\ h_4^* \end{pmatrix} = d\begin{pmatrix} \frac{\lambda_0\alpha M^* I^*+\beta I^*\left(-\mathrm{i}\omega^{(k)}+d+\alpha I^*\right)}{\left(-\mathrm{i}\omega^{(k)}+d+\alpha I^*\right)\left(-\mathrm{i}\omega^{(k)}+\beta I^*+d+\lambda_0 M^*\right)} \\ \frac{\alpha I^*}{-\mathrm{i}\omega^{(k)}+(d+\alpha I^*)} \\ 1 \\ \frac{(-\lambda_0 S^*)\left[\lambda_0\alpha M^* I^*+\beta I^*\left[-\mathrm{i}\omega^{(k)}+(d+\alpha I^*)\right]\right]}{\left(-\mathrm{i}\omega^{(k)}+\mu_0\right)\left(-\mathrm{i}\omega^{(k)}+d+\alpha I^*\right)\left[-\mathrm{i}\omega^{(k)}+(\beta I^*+d+\lambda_0 M^*)\right]} + \frac{\lambda_0\alpha S^* I^*}{\left(-\mathrm{i}\omega^{(k)}+\mu_0\right)\left(-\mathrm{i}\omega^{(k)}+d+\alpha I^*\right)} \end{pmatrix}. \end{aligned} \tag{4.2}$$

where $d = \frac{1}{h_1\overline{h_1^*}+h_2\overline{h_2^*}+h_3\overline{h_3^*}+h_4\overline{h_4^*}}$.

We suppose the solution of Eq (4.1) is as follows:

$$X(t) = X(T_0, T_1, T_2, \cdots) = \sum_{k=1}^{\infty} \varepsilon^k X_k(T_0, T_1, T_2, \cdots), \tag{4.3}$$

where $X(T_0, T_1, T_2, \cdots) = [S(T_0, T_1, T_2, \cdots), A(T_0, T_1, T_2, \cdots), I(T_0, T_1, T_2, \cdots), M(T_0, T_1, T_2, \cdots)]^{\mathrm{T}}$, $X_k(T_0, T_1, T_2, \cdots) = [S_k(T_0, T_1, T_2, \cdots), A_k(T_0, T_1, T_2, \cdots), I_k(T_0, T_1, T_2, \cdots), M_k(T_0, T_1, T_2, \cdots)]^{\mathrm{T}}$.

The derivative with respect to t is transformed:

$$\frac{d}{dt} = \frac{\partial}{\partial T_0} + \varepsilon\frac{\partial}{\partial T_1} + \varepsilon^2\frac{\partial}{\partial T_2} + \cdots = D_0 + \varepsilon D_1 + \varepsilon^2 D_2 + \cdots,$$

where $D_i = \frac{\partial}{\partial T_i}$, $i = 0, 1, 2, \cdots$.

Note that

$$X_i = (S_i, A_i, I_i, M_i)^T = X_i\left(t, \varepsilon t, \varepsilon^2 t, \cdots\right),$$
$$X_{i1} = (S_i, A_i, I_i, M_i)^T = X_i\left(t-1, \varepsilon t, \varepsilon^2 t, \cdots\right), i = 1, 2, \cdots.$$

Then, we obtain

$$\dot{X}(t) = \varepsilon D_0 X_1 + \varepsilon^2 D_1 X_1 + \varepsilon^3 D_2 X_1 + \varepsilon^2 D_0 X_2 + \varepsilon^3 D_1 X_2 + \varepsilon^3 D_0 X_3 + \cdots. \tag{4.4}$$

Using a Taylor series expansion of $X(t-1)$, we obtain that

$$X(t-1) = \varepsilon X_{11} + \varepsilon^2 (X_{21} - D_1 X_{11}) + \varepsilon^3 (X_{31} - D_1 X_{21} - D_2 X_{11}) + \cdots, \tag{4.5}$$

where $X_{i1} = X_i(T_0 - 1, T_1, T_2, \cdots)$, $i = 1, 2, 3, \cdots$.

As we stated, τ is the bifurcation parameter, and $\tau = \tau_c + \varepsilon\tau_\varepsilon$. Substituting Eqs (4.3)–(4.5) into Eq (4.1) and balancing the coefficients before ε on both sides of the equation, the following expression is obtained:

$$\begin{cases} D_0 S_1 - \tau_c (a_{11} S_1 + a_{13} I_1 + a_{14} M_1) = 0, \\ D_0 A_1 - \tau_c (a_{21} S_1 + a_{22} A_1 + a_{23} I_1 + a_{24} M_1) = 0, \\ D_0 I_1 - \tau_c (a_{31} S_1 + a_{32} A_1 + a_{33} I_1) = 0, \\ D_0 M_1 - \tau_c (a_{44} M_1 + \mu I_{11}) = 0. \end{cases} \tag{4.6}$$

Thus, Eq (4.6) has the following solution form:

$$X_1(T_1, T_2, T_3, \cdots) = G(T_1, T_2, T_3, \cdots)\mathrm{e}^{\mathrm{i}\omega^{(k)}\tau_c T_0} h + \bar{G}(T_1, T_2, T_3, \cdots)\mathrm{e}^{-\mathrm{i}\omega^{(k)}\tau_c T_0}\bar{h}. \tag{4.7}$$

The expression of the coefficient before ε^2 is as follows:

$$\begin{cases} D_0 S_2 - \tau_c (a_{11} S_2 + a_{13} I_2 + a_{14} M_2) \\ = -D_1 S_{k1} - \tau_c (\beta S_1 I_1 + \lambda_0 S_1 M_1) + \tau_\varepsilon (a_{11} S_1 + a_{13} I_1 + a_{14} M_1), \\ D_0 A_2 - \tau_c (a_{21} S_2 + a_{22} A_2 + a_{23} I_2 + a_{24} M_2) \\ = -D_1 A_1 + \tau_\varepsilon (a_{21} S_1 + a_{22} A_1 + a_{23} I_1 + a_{24} M_1) + \tau_c (-\alpha A_1 I_1 + \lambda_0 S_1 M_1), \\ D_0 I_2 - \tau_c (a_{31} S_2 + a_{32} A_2 + a_{33} I_2) \\ = -D_1 I_1 + \tau_c (\beta S_1 I_1 + \alpha A_1 I_1) + \tau_\varepsilon (a_{31} S_1 + a_{32} A_1 + a_{33} I_1), \\ D_0 M_2 - \tau_c (a_{44} M_2 + \mu I_{21}) \\ = -D_1 M_1 - \mu\tau_c D_1 I_{11} + \tau_\varepsilon (a_{44} M_1 + \mu I_{11}). \end{cases} \tag{4.8}$$

Substituting Eq (4.7) into the right-hand side of Eq (4.8), and the coefficient vector of $e^{i\omega^{(k)}\tau_c T_0}$ is denoted by m_1. According to the solvability condition $\langle h^*, m_1\rangle = 0$, the expression of $\frac{\partial G}{\partial T_1}$ is obtained as follows:

$$\frac{\partial G}{\partial T_1} = N_k \tau_\varepsilon G, \tag{4.9}$$

where $N_k = \frac{i\omega^{(k)}}{1+\mu h_3 \overline{h_4^*} e^{-i\omega^{(k)}\tau_c d}}$, $k = 1, 2, 3, 4$.

Since τ_ε is a disturbance parameter, we only consider its effect on the linear part. It has little effect on the high order, so it can be ignored. Therefore, we ignore the part containing τ_ε in the higher order. We suppose the solutions of Eq (4.8) are given as follows:

$$\begin{aligned}
S_2 &= g_1 e^{2i\omega^{(k)}\tau_c T_0} G^2 + \bar{g}_1 e^{-2i\omega^{(k)}\tau_c T_0} \bar{G}^2 + l_1 G\bar{G},\\
A_2 &= g_2 e^{2i\omega^{(k)}\tau_c T_0} G^2 + \bar{g}_2 e^{-2i\omega^{(k)}\tau_c T_0} \bar{G}^2 + l_2 G\bar{G},\\
I_2 &= g_3 e^{2i\omega^{(k)}\tau_c T_0} G^2 + \bar{g}_3 e^{-2i\omega^{(k)}\tau_c T_0} \bar{G}^2 + l_3 G\bar{G},\\
M_2 &= g_4 e^{2i\omega^{(k)}\tau_c T_0} G^2 + \bar{g}_4 e^{-2i\omega^{(k)}\tau_c T_0} \bar{G}^2 + l_4 G\bar{G},
\end{aligned} \tag{4.10}$$

where

$$\begin{pmatrix} g_1\\ g_2\\ g_3\\ g_4 \end{pmatrix} = \begin{pmatrix} 2i\omega^{(k)} + \beta I^* + d + \lambda_0 M^* & 0 & \beta S^* & \lambda_0 S^*\\ -\lambda_0 M^* & 2i\omega^{(k)} + d + \alpha I^* & \alpha A^* - \nu & -\lambda_0 S^*\\ -\beta I^* & -\alpha I^* & 2i\omega^{(k)} - \beta S^* - \alpha A^* + \nu + c + d & 0\\ 0 & 0 & -\mu e^{-2i\omega^{(k)}\tau_c} & 2i\omega^{(k)} + \mu_0 \end{pmatrix}^{-1} \begin{pmatrix} b_1\\ b_2\\ b_3\\ b_4 \end{pmatrix},$$

$$\begin{pmatrix} l_1\\ l_2\\ l_3\\ l_4 \end{pmatrix} = \begin{pmatrix} \beta I^* + d + \lambda_0 M^* & 0 & \beta S^* & \lambda_0 S^*\\ -\lambda_0 M^* & d + \alpha I^* & \alpha A^* - \nu & -\lambda_0 S^*\\ -\beta I^* & -\alpha I^* & -\beta S^* - \alpha A^* + \nu + c + d & 0\\ 0 & 0 & -\mu & \mu_0 \end{pmatrix}^{-1} \begin{pmatrix} c_1\\ c_2\\ c_3\\ c_4 \end{pmatrix}, \tag{4.11}$$

with h_1, h_2, h_3, h_4 are given in Eq (4.2) and

$$\begin{pmatrix} b_1\\ b_2\\ b_3\\ b_4 \end{pmatrix} = \begin{pmatrix} -\beta h_1 h_3 - \lambda_0 h_1 h_4\\ -\alpha h_2 h_3 + \lambda_0 h_1 h_4\\ \beta h_1 h_3 + \alpha h_2 h_3\\ 0 \end{pmatrix}, \begin{pmatrix} c_1\\ c_2\\ c_3\\ c_4 \end{pmatrix} = \begin{pmatrix} -\beta h_1 \overline{h_3} - \beta \overline{h_1} h_3 - \lambda_0 h_1 \overline{h_4} - \lambda_0 h_4 \overline{h_1}\\ \lambda_0 h_1 \overline{h_4} + \lambda_0 h_4 \overline{h_1} - \alpha h_2 \overline{h_3} - \alpha h_3 \overline{h_2}\\ \beta h_1 \overline{h_3} + \beta h_1 \overline{h_3} + \alpha h_2 \overline{h_3} + \alpha h_3 \overline{h_2}\\ 0 \end{pmatrix}.$$

The expression of the coefficient before ε^3 is:

$$\begin{cases} D_0S_3 - \tau_c\left(a_{11}S_3 + a_{13}I_3 + a_{14}M_3\right) \\ = -D_2S_1 - D_1S_2 - \tau_c\left(\beta S_2I_1 + \beta S_1I_2 + \lambda_0 S_1M_2 + \lambda_0 S_2M_1\right) \\ \quad +\tau_\varepsilon\left(a_{11}S_2 + a_{13}I_2 + a_{14}M_2 - \beta S_1I_1 - \lambda_0 S_1M_1\right), \\ \\ D_0A_3 - \tau_c\left(a_{21}S_3 + a_{22}A_3 + a_{23}I_3 + a_{24}M_3\right) \\ = -D_2A_1 - D_1A_2 + \tau_c\left(-\alpha A_1I_2 - \alpha A_2I_1 + \lambda_0 S_1M_2 + \lambda_0 S_2M_2\right) \\ \quad +\tau_\varepsilon\left(a_{21}S_2 + a_{22}A_2 + a_{23}I_2 + a_{24}M_2 - \alpha A_1I_1 + \lambda_0 S_1M_1\right), \\ \\ D_0I_3 - \tau_c\left(a_{31}S_3 + a_{32}A_3 + a_{33}I_3\right) \\ = -D_2I_1 - D_1I_2 + \tau_c\left(\beta S_1I_2 + \alpha A_1I_2 + \beta S_2I_1 + \alpha A_2I_1\right) \\ \quad +\tau_\varepsilon\left(a_{31}S_2 + a_{32}A_2 + a_{33}I_2 + \beta S_1I_1 + \alpha A_1I_1\right), \\ \\ D_0M_3 - \tau_c\left(a_{44}M_3 + \mu I_{31}\right) \\ = -D_2M_1 - D_1M_2 - \tau_c\left(\mu D_1I_{21} + \mu D_2I_{11}\right) + \tau_\varepsilon\left(a_{44}M_2 + \mu I_{21} - \mu D_1I_{11}\right). \end{cases} \tag{4.12}$$

Substitute Eqs (4.6), (4.10) and (4.11) into the right-hand side of Eq (4.12), and the coefficient vector of $e^{i\omega^{(k)}\tau_c T_0}$ is denoted by m_2. According to the solvability condition $\langle h^*, m_2\rangle = 0$, the expression of $\frac{\partial G}{\partial T_2}$ can be obtained as follows:

$$\frac{\partial G}{\partial T_2} = M_kN_k\tau_cG^2\bar{G}, \tag{4.13}$$

where

$$\begin{aligned} M_k = \frac{1}{i\omega^{(k)}}\{&\left[-\beta\left(l_1h_3 + g_1h_3 + l_3h_1 + g_3h_1\right) - \lambda_0\left(l_1h_4 + g_1h_4 + l_4h_1 + g_4h_1\right)\right]\overline{h_1^*} \\ &+\left[-\alpha\left(l_2h_3 + g_2h_3 + l_3h_2 + g_3h_2\right) + \lambda_0\left(l_1h_4 + g_1h_4 + l_4h_1 + g_4h_1\right)\right]\overline{h_2^*} \\ &+\left[\beta\left(l_1h_3 + g_1h_3 + l_3h_1 + g_3h_1\right) + \alpha\left(l_2h_3 + g_2h_3 + l_3h_2 + g_3h_2\right)\right]\overline{h_3^*}\}, \end{aligned}$$

with h_k given in Eq (4.2), g_k, l_k given in Eq (4.11), and N_k given in Eq (4.9).

Let $G \to G/\varepsilon$; then, the deduced third-order normal form of Hopf bifurcation of system (2.2) is:

$$\dot{G} = N_k\tau_\varepsilon G + M_kN_k\tau_cG^2\bar{G}, \tag{4.14}$$

where N_k is given in Eq (4.9), and M_k is given in Eq (4.13).

Substituting $G = re^{i\theta}$ into Eq (4.14), the following normal form of Hopf bifurcation in polar coordinates is obtained:

$$\begin{cases} \dot{r} = \mathrm{Re}(N_k)\tau_\varepsilon r + \mathrm{Re}(M_kN_k)\tau_c r^3, \\ \dot{\theta} = \mathrm{Im}(N_k)\tau_\varepsilon + \mathrm{Im}(M_kN_k)\tau_c r^2. \end{cases} \tag{4.15}$$

According to the normal form of Hopf bifurcation in polar coordinates, we only need to consider the first equation in system (4.15). Thus, the following theorem holds:

Theorem 4.1. *For the system (4.15), when* $\frac{\mathrm{Re}(N_k)\tau_\varepsilon}{\mathrm{Re}(M_kN_k)\tau_c} < 0$ *, there is a semitrivial fixed point* $r = \sqrt{-\frac{\mathrm{Re}(N_k)\tau_\varepsilon}{\mathrm{Re}(M_kN_k)\tau_c}}$*, and system (2.2) has periodic solution.*

(1) If $\mathrm{Re}(N_k)\tau_\varepsilon < 0$*, then the periodic solution reduced on the center manifold is unstable.*

(2) If $\mathrm{Re}(N_k)\tau_\varepsilon > 0$*, then the periodic solution reduced on the center manifold is stable.*

5. Numerical simulations

In this section, we carry out numerical simulations to verify our theoretical analysis. Then, we study two important parameters, that is, the media influence rate μ and attenuation rate of media μ_0, and simulate the impact of these two parameters on the time required for people to be willing to vaccinate. Finally, we explore the influence of timely media coverage and the propaganda efforts of media coverage on the epidemic, and we propose reasonable suggestions for effectively controlling the COVID-19 epidemic.

5.1. Determination of parameter values

Based on official statistics ($https://github.com/CSSEGISandData/COVID-19; https://voice.baidu.com/act/newpneumonia/newpneumonia/?from=osari_pc_3\#tab4$), we obtain data on national case fatality rates and cure rates for different countries. To ensure that the data can relect the average, we retain representative data and eliminate outliers. Finally, we screen the death rates due to disease for 29 countries and cure rates for 30 countries. According to the data, we generate bar charts, which are presented in Figures 2 and 3.

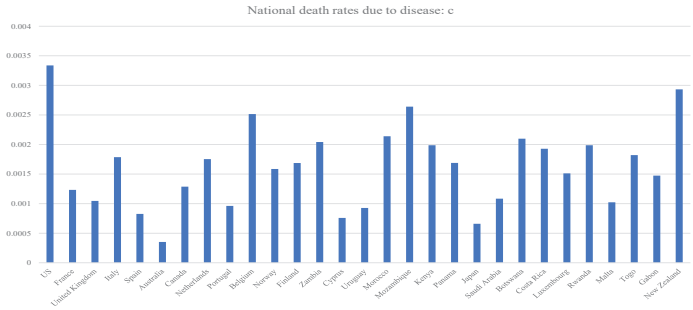

Figure 2. Values of national case fatality rates c in 29 countries.

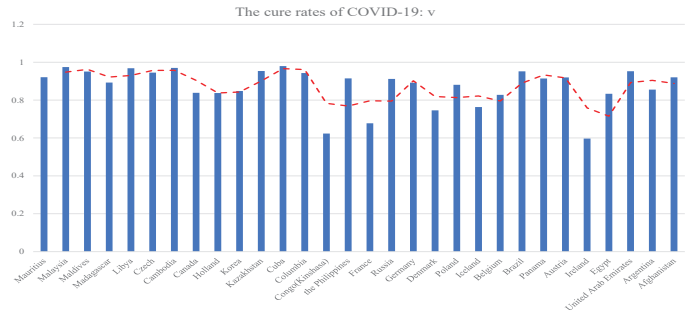

Figure 3. Values of cure rates v in 30 countries.

Figure 2 shows the national case fatality rates of these countries are mostly in the range of 0.001 to 0.002, so we calculate the mean value 0.0016 and choose it as the value of c. Cure rates v are almost at the same level through the red dotted line in Figure 3, so we calculate the average rate for 30 countries and set 0.861 as the value of v. To find the value of the natural mortality rate d, we select population

data from the National Bureau of Statistics ($http://www.stats.gov.cn/enGliSH/$) over the last 20 years and obtain a relatively stable natural mortality rate $d = 0.00707$.

Based on the above consideration and values, we take two groups of parameters as follows:

(1) $B = 28, \beta = 0.00021, d = 0.00707, \lambda_0 = 0.00073, \mu = 100, \mu_0 = 0.25, v = 0.861, c = 0.0016, \alpha = 0.000051$;

(2) $B = 28, \beta = 0.00816, d = 0.00707, \lambda_0 = 0.00051, \mu = 100, \mu_0 = 0.25, v = 0.861, c = 0.0016, \alpha = 0.00011$.

5.2. *The numerical simulations*

First, we show the simulation results under the first group of parameters (1):

$$B = 28, \beta = 0.00021, d = 0.00707, \lambda_0 = 0.00073,$$
$$\mu = 100, \mu_0 = 0.25, v = 0.861, c = 0.0016, \alpha = 0.000051.$$

It is easy to find that the basic regeneration number of the system (2.2) $R_0 = \frac{\beta B}{d(v+c+d)} < 1$, so there is only one semitrivial equilibrium $P_1 = \left(S_1^*, A_1^*, I_1^*, M_1^*\right) = (3960.39, 0, 0, 0)$ of system (2.2) according to expression (3.1). The equilibrium P_1 is locally asymptotically stable for any $\tau \geq 0$ by Theorem 3.1. This means that P_1 is a disease-free equilibrium.

For the initial values [4000,10,10,10], we choose $\tau = 0$ and $\tau = 5$ for the simulations. Clearly, the equilibrium P_1 is locally asymptotically stable, as shown in Figure 4.

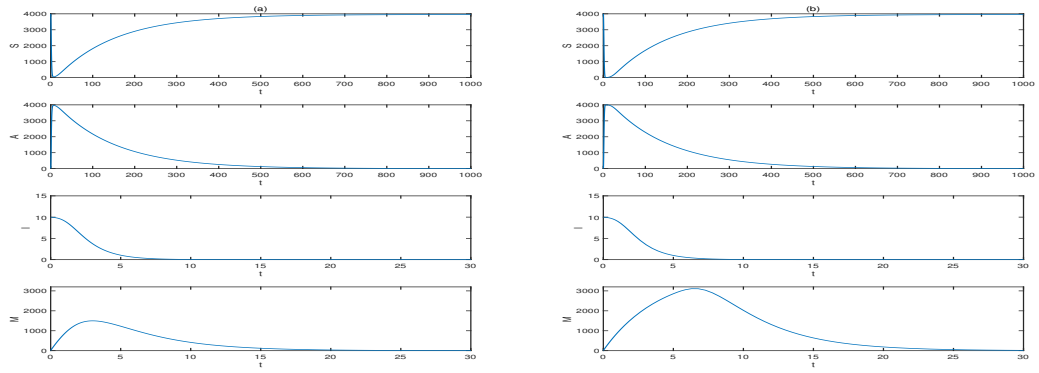

Figure 4. Equilibrium P_1 of the system (2.2) is locally asymptotically stable, (a) the solution for $\tau = 0$, (b) the solution for $\tau = 5$.

When $\tau = 0$, the solution is shown in Figure 4(a). Although there are fluctuations in 0–500 days, it tends to be stable after the 500th day. There will be no infected people, and the COVID-19 epidemic will be eliminated completely. This means that in this case, people pay attention to media reports regarding the epidemic and the epidemic will not develop, which verifies our theoretical analysis.

When $\tau = 5$, people's willingness to be vaccinated will change after 5 days of media coverage. It represents the situation in which people are unable to pay timely attention to news reported by the media and they do not want to get vaccinated immediately. As we can see in Figure 4(b), compared with $\tau = 0$, it takes a bit longer for equilibrium P_1 to stabilize. However, eventually, there will be no infected people, and the disease will still disappear in this case.

***Remark* 1**: According to the numerical simulations of parameters (1), we find that for any $\tau \geq 0$,

the equilibrium P_1 of the system (2.2) is locally asymptotically stable, and the COVID-19 epidemic is completely eliminated. The smaller τ is, the faster the equilibrium stabilizes. This suggests that the influence of media on people's willingness to vaccinate does not change the stability of the disease-free equilibrium, and no matter how long it takes people to accept the idea of vaccination after viewing media coverage, the disease will eventually disappear. However, the less time it takes for people to change their vaccination intentions after viewing media coverage, the more it helps to contain the epidemic. This corresponds to the actual situation.

For the group of parameters (2):

$$B = 28, \beta = 0.00861, d = 0.00707, \lambda_0 = 0.00051,$$
$$\mu = 100, \mu_0 = 0.25, v = 0.861, c = 0.0016, \alpha = 0.00011,$$

we find that $R_0 > 1$ and **(H1)** hold. Then, we calculate the equilibria $P_1 = \left(S_1^*, A_1^*, I_1^*, M_1^*\right) = (3960.39, 0, 0, 0)$ and $P_2 = \left(S_2^*, A_2^*, I_2^*, M_2^*\right) = (53.96, 3903.48, 2.41, 965.06)$ by expression (3.1). Due to Theorem 3.1, the equilibrium P_1 is unstable for any $\tau \geq 0$, and P_2 is locally asymptotically stable when $\tau = 0$. Substituting parameters (2) into Eq (3.6), we obtain $\tau_1^{(0)} = 1.4448, \sin(\omega_1\tau) = 0.3680, \cos(\omega_1\tau) = 0.9298$ by MATLAB. We know that P_2 is locally asymptotically stable for any $0 < \tau < \tau_1^{(0)} = 1.4448$ and unstable for any $\tau > \tau_1^{(0)} = 1.4448$. Then, we calculate the normal form of Hopf bifurcation and obtain $\mathrm{Re}(N_k) > 0, \mathrm{Re}(M_k N_k) < 0$ from Eq (4.15). The periodic solution is stable when $\tau_\varepsilon > 0$ according to Theorem 4.1.

When $\tau = 0$, it means that as soon as the media report the situation of the epidemic, it will receive widespread attention from the public, and many people will change their willingness to vaccinate. We choose the initial values [100,3000,10,1000], and the equilibrium is locally asymptotically stable, as shown in Figure 5.

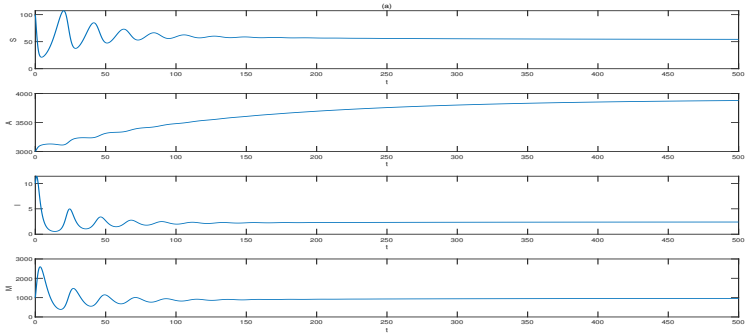

Figure 5. When $\tau = 0$, equilibrium P_2 of the system (2.2) is locally asymptotically stable.

As seen in Figure 5, although there are fluctuations of S, I, M in 0–100 days, it tends to be stable after the 100th day. In contrast, A tends to stabilize more slowly. This suggests that although the epidemic has almost stabilized and the number of infected people has stopped rising, people still want to protect themselves by getting vaccinated. Therefore, the number of people with antibodies continues to rise until a long time after the epidemic has stabilized. The number of people with antibodies is significantly greater than that of people who do not have antibodies, as shown in Figure 5. This means that in this case, the efficiency with which people consistently monitor the epidemic situation is meaningful, but this situation is under perfect conditions. Considering that it takes a certain amount of time for people to pay attention to media coverage, this situation is basically impossible.

When $\tau = 1 \in \left(0, \tau_1^{(0)}\right)$ with $\tau_1^{(0)} = 1.4448$, which means that people respond to media reports of the epidemic one day later, we still choose the initial values [100,3000,10,1000], and the solution of numerical simulations is shown in Figure 6.

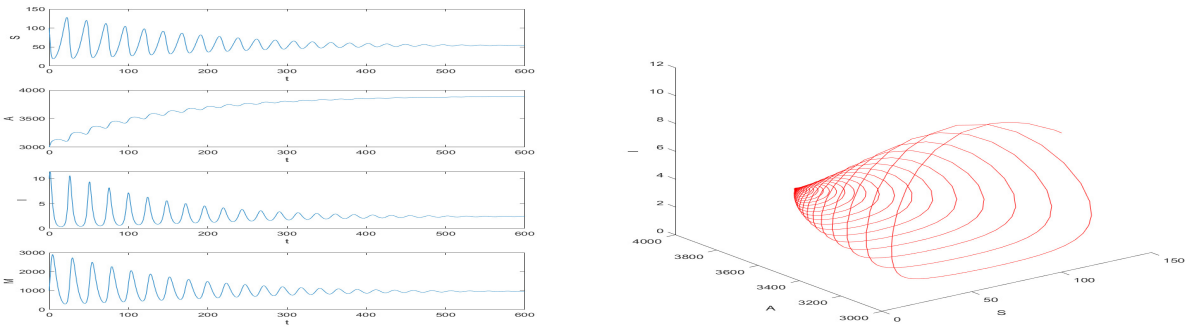

Figure 6. When $\tau = 1$, equilibrium P_2 of the system (2.2) is locally asymptotically stable.

In Figure 6, we find that equilibrium P_2 is also locally asymptotically stable. The fluctuation in the first 400 days is obvious, but it gradually stabilizes after the 400th day, and the disease can also be controlled. Although its regional stability speed is significantly less than when $\tau = 0$, it will still reach stability in a short time. That is, if people respond to the epidemic and become willing to vaccinate within 1 day after receiving media reports, the rate of vaccination will increase to effectively control the epidemic.

Through the analysis of Hopf bifurcation, we obtain that when $\tau > \tau_1^{(0)} = 1.4448$, which means that people respond to media reports of the epidemic after τ days later, the equilibrium P_2 is unstable. Since $\mathrm{Re}\,(N_k) > 0, \mathrm{Re}\,(M_k N_k) < 0, \tau_\varepsilon > 0$, system (2.2) has a forward periodic solution, and the bifurcating periodic solutions near $\tau_1^{(0)}$ are locally asymptotically stable. We still choose the initial values [100,3000,10,1000], and the solution of the numerical simulations when $\tau = 2$ is shown in Figure 7.

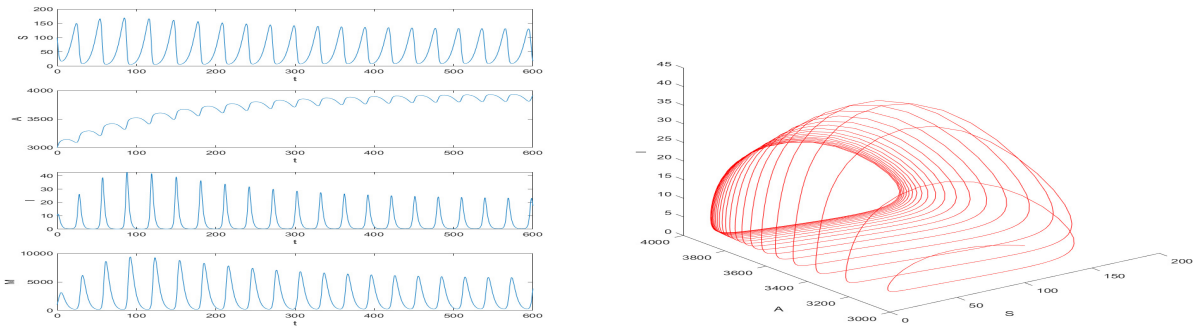

Figure 7. When $\tau = 2$, there exists a stable periodic solution near equilibrium P_2 of the system (2.2).

In Figure 7, we find that the bifurcating periodic solution is stable, which verifies our theoretical analysis. In fact, with the development of the epidemic, more people will be vaccinated, so the number of A tends to increase. On the other hand, people without antibodies and infected people will be largely affected whether the epidemic is severe or not, so periodic solutions of S and I fluctuate with the change in epidemic.

To study the situation in which people do not pay attention to the media and change their willingness to vaccinate in time, we carry out the numerical simulations when $\tau = 10$ with the same initial values [100,3000,10,1000] in Figure 8.

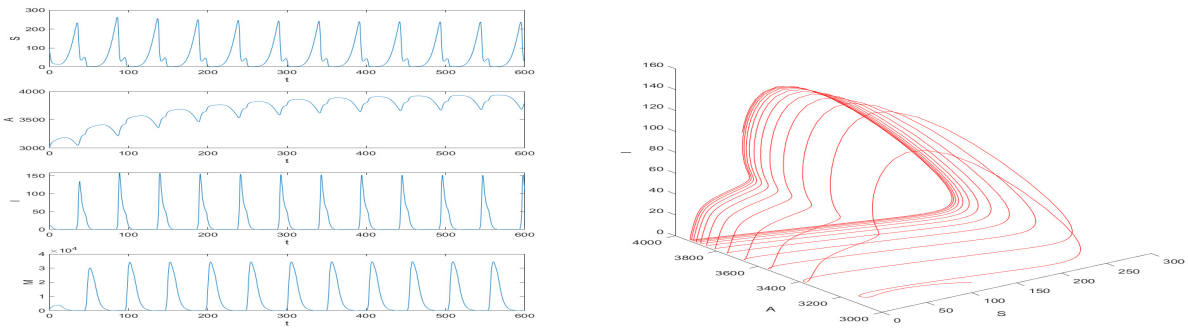

Figure 8. When $\tau = 10$, there exists a stable periodic solution near equilibrium P_2 of the system (2.2).

Figure 8 shows that when $\tau = 10$, the periodic solution of the system (2.2) is stable as well. However, compared with the situation of $\tau = 2$, the periodic solutions exhibite larger fluctuations and longer periods. In fact, the longer it takes people to change their willingness after media reports, the longer the epidemic is temporarily contained and the worse the situation is, which is consistent with our simulation results.

***Remark* 2**: According to the numerical simulations of parameters (2), we can obtain the following: when $0 < \tau < \tau_1^{(0)} = 1.4448$, the equilibrium P_2 of the system (2.2) is locally asymptotically stable, and the shorter the time that people spend changing their willingness, the better the epidemic can be controlled. However, if $\tau > \tau_1^{(0)} = 1.4448$, the equilibrium P_2 will be unstable. This means that under this group of parameters, if people do not respond to the epidemic situation within 1.5 days after the media report, the impact and timeliness of the media will be diminished. Therefore, the epidemic will be difficult to control. However, for any τ in the small neighborhood of $\tau > \tau_1^{(0)} = 1.4448$, the system (2.2) has a stable periodic solution, indicating that the timeliness and influence of media reports are not the main factors for the development of the epidemic. Under the circumstance that the governments of various countries take measures such as isolation treatment for the epidemic, there will not be a large-scale outbreak. However, we still hope to avoid this situation to effectively control the epidemic and prevent recurrence.

Then, we simulate the change in time that people required to respond to media under different media influence intensities μ and attenuation rates of media μ_0 (see Figure 9). The media's influence intensity μ here means that if the number of cases in a region exceeds a certain threshold, the media

coverage may result in a larger number of susceptibles becoming conscious. We find that when $\mu \in [40, 1000], \mu_0 \in [0.065, 0.8]$ and other parameters remain unchanged, the stability of the Hopf bifurcation is similar to the situation under the group of parameters (2). Moreover, we choose μ changes within [40,600] and $\mu_0 = 0.05, 0.15, 0.25, 0.35$ specifically (see Figure 10).

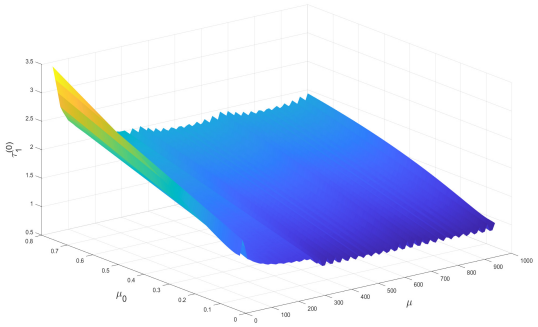

Figure 9. Critical time delay $\tau_1^{(0)}$ with respect to media influence rate μ and attenuation rate μ_0.

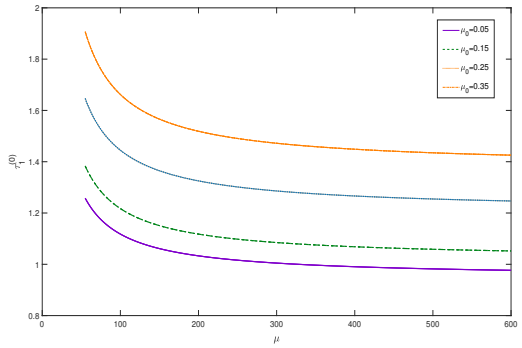

Figure 10. Critical time delay $\tau_1^{(0)}$ with respect to media influence rate μ for different attenuation rates μ_0.

According to Figures 9 and 10, we can clearly see the following:

(1) $\tau_1^{(0)}$ decreases as μ increases when the other parameters are fixed. This is consistent with the fact that when the media influence μ increases, it indicates that the epidemic is very serious, or the government attaches importance to the epidemic, so the media propaganda is increased. This requires people to respond to media coverage in a shorter time and develop a desire to be vaccinated to increase vaccination rates and achieve herd immunity to control the spread of the disease.

(2) $\tau_1^{(0)}$ decreases when μ_0 decreases from 0.35 to 0.05 and the other parameters are fixed. In fact, people are most alert when they first receive media reports about the severity of the epidemic and think it is necessary to be vaccinated. However, there is an attenuation rate of media μ_0, and the attenuation rate will be smaller if most people still believe that vaccination is needed to control the epidemic after they have calmed down. This means that the epidemic is serious, so the time needed for people to respond to the epidemic is shorter, which means that $\tau_1^{(0)}$ is smaller.

5.3. Analysis of simulations

Based on the above numerical simulations, we draw the following conclusions.

(*i*) For the first group of parameters (1), we find that for any $\tau \geq 0$, the equilibrium P_1 of the system (2.2) is locally asymptotically stable, and the COVID-19 epidemic will be completely eliminated. For the second group of parameters (2), the disease-free equilibrium P_1 is unstable, and although the epidemic may stabilize, it will not disappear. Therefore, we compare the two groups of parameters and find that the first group had low rates of infection and a high rate of vaccination. This suggests that if we can strengthen self-protection, such as wearing masks and other precautions to reduce the risk of infection, develop our vaccine, make it effective enough, and vaccinate the majority of people, then the COVID-19 epidemic will disappear completely. This corresponds to reality.

(*ii*) Through the simulations of parameters (2), we find that people's willingness to vaccinate will be improved and the epidemic will be effectively controlled if people respond to the epidemic situation and change their vaccination willingness within $\tau_1^{(0)}$ days after the media reported. The shorter the time that people spend changing their mind, the better the epidemic can be controlled. While the impact and timeliness of the media is diminished, people's willingness to be vaccinated will be even harder to change, and the epidemic will be difficult to control if the time people respond to media reports is larger than the critical time delay.

(*iii*) When the media influence intensity μ increases or the attenuation rate of media μ_0 decreases, this shows that the epidemic is very serious, and the government and the media attach great importance to it. Therefore, people need to react to media coverage in a shorter time. Therefore, to keep the epidemic under control, the media needs to step up their propaganda efforts according to the changes in the epidemic and make sure that people can respond within $\tau_1^{(0)}$.

(*iv*) Considering the limitations of the actual epidemic situation and the numerical simulations we have performed, we can better apply the model to real life.

Case 1: Since the vaccine is so new, it is unknown whether it will cause side effects many years later, so people may have doubts about the efficacy and safety of the COVID-19 vaccine. This means that people may be hesitant to get vaccinated. In this case, people may be initially motivated to be vaccinated by the media but become reluctant to be vaccinated after a while. This means that the impact factor of vaccination rate λ_0 decreases. According to the analysis of (*i*), when λ_0 decreases, the disease-free equilibrium will be unstable, and the epidemic will always exist, although it may become stable. This shows that vaccine hesitancy can have a negative effect on controlling the epidemic. Therefore, to effectively control the epidemic, people need to understand the COVID-19 vaccine. We recommend that the government use the media to disseminate more reports on the protective effects of vaccines and the importance of vaccination in the current situation and put forward policies that benefit the vaccinated population to guide people to dispel their fears about vaccines and actively get vaccinated against COVID-19.

Case 2: There is false information in media reports about the epidemic in this case. This causes the media attenuation rate μ_0 to increase. That's because people are less willing to get vaccinated when they learn the epidemic is not as serious as reported. Therefore, the time needed for people to respond to the epidemic is longer, which is consistent with our analysis in (*iii*). However, we advise the media to ensure the accuracy of information so that people have a more accurate understanding of the epidemic situation, avoiding too little attention or too much attention, causing panic.

Case 3: The government can take preventive measures in a timely manner. For example, when an

infected person is identified, he or she is isolated immediately, or the government requires people to reduce social activities, wear masks when going out and other precautions. This means that in this case, the infection rates α and β will decrease. Then, the disease-free equilibrium may be stable, which means that these measures have a positive impact on epidemic control based on analysis (i). Thus, we consider it necessary for the government to take timely measures and advise people to strictly abide by relevant government measures and regulations.

6. Conclusions

In this paper, considering vaccination and the characteristics of the COVID-19 epidemic, we have constructed a new $SAIM$ model with a time delay for people to change their vaccination willingness, which is influenced by news reported in the media. We have studied the stability of the equilibria and the existence of Hopf bifurcation. Then, we have analyzed the stability and bifurcating direction of the Hopf bifurcating periodic solution by calculating the normal form with the multiple time scales method.

Based on the observed data, we have carried out numerical simulations. First, the change in the epidemic situation has been simulated through two groups of parameters to verify the theoretical analysis results. We have found that when media coverage is more effective and the vaccination rate will increase as people respond to the epidemic within the critical time τ after the media report, the epidemic will be effectively controlled. The shorter the time it takes for people to change their vaccination willingness, the better the epidemic can be controlled. Next, we have simulated the impact of media coverage and conclude that if media propaganda is increased and the attenuation rate decreases, that is, the epidemic is serious, this requires people to respond to media coverage in a shorter time and develop a desire to be vaccinated to increase vaccination rates and gain herd immunity to control the spread of the disease. In our analysis, we consider the limitations of the actual epidemic situation to better apply our model to the real-world conditions.

We also highlight the influence of the media based on our model; that is, to keep the epidemic under control and prevent a sustained outbreak, the media needs to step up or down their propaganda efforts according to the changes in the epidemic and ensure that people can respond within a critical time.

Acknowledgments

The authors wish to express their special gratitude to the editor and the reviewers for the helpful comments given for this paper. This study was funded by Heilongjiang Provincial Natural Science Foundation of China (Grant No. LH2019A001) and College Students Innovations Special Project funded by Northeast Forestry University of China (No. 202110225003).

Conflict of interest

All authors declare no conflicts of interest in this paper.

References

1. H. Guliyev, Determining the spatial effects of COVID-19 using the spatial panel data model, *Spatial Stat.*, **38** (2020), 100443. https://doi.org/10.1016/j.spasta.2020.100443
2. Y. Niu, J. Rui, Q. Wang, W. Zhang, Z. Chen, F. Xie, et al., Containing the transmission of COVID-19: a modeling study in 160 countries, *Front. Med.* , **8** (2021), 701836. https://doi.org/10.3389/fmed.2021.701836
3. M. Habadi, T. H. Balla Abdalla, N. Hamza, A. Al-Gedeei, COVID-19 Reinfection, *Cureus*, **13** (2021), e12730. https://dx.doi.org/10.7759%2Fcureus.12730
4. S. R. Kannan, A. N. Spratt, A. R. Cohen, S. H. Naqvi, H. S. Chand, T. P. Quinn, et al., Evolutionary analysis of the Delta and Delta plus variants of the SARS-CoV-2 viruses, *J. Autoimmun.*, **124** (2021), 102715. https://doi.org/10.1016/j.jaut.2021.102715
5. F. Wei, R. Xue, Stability and extinction of SEIR epidemic models with generalized nonlinear incidence, *Math. Comput. Simul.*, **170** (2020), 1–15. https://doi.org/10.1016/j.matcom.2018.09.029
6. O. Khyar, K. Allali, Global dynamics of a multi-strain SEIR epidemic model with general incidence rates: application to COVID-19 pandemic, *Nonlinear Dyn.*, **102** (2020), 489–509. https://doi.org/10.1007/s11071-020-05929-4
7. Z. Zhang, A. Zeb, O. F. Egbelowo, V. S. Erturk, Dynamics of a fractional order mathematical model for COVID-19 epidemic, *Adv. Differ. Equations*, **2020** (2020), 420. https://doi.org/10.1186/s13662-020-02873-w
8. S. Annas, M. I. Pratama, M. Rifandi, W. Sanusi, S. Side, Stability analysis and numerical simulation of SEIR model for pandemic COVID-19 spread in Indonesia, *Chaos Soliton Fract.*, **139** (2020), 110072. https://doi.org/10.1016/j.chaos.2020.110072
9. H. M. Youssef, N. A. Alghamdi, M. A. Ezzat, A. A. El-Bary, A. M. Shawky, A new dynamical modeling SEIR with global analysis applied to the real data of spreading COVID-19 in Saudi Arabia, *Math. Biosci. Eng.*, **17** (2020), 7018–7044. https://doi.org/10.3934/mbe.2020362
10. M. Abdy, S. Side, S. Annas, W. Nur, W. Sanusi, An SIR epidemic model for COVID-19 spread with fuzzy parameter: the case of Indonesia, *Adv. Differ. Equations*, **2021** (2021), 105. https://doi.org/10.1186/s13662-021-03263-6
11. L. Wang, Z. Liu, X. Zhang, Global dynamics for an age-structured epidemic model with media impact and incomplete vaccination, *Nonlinear Anal.*, **32** (2016), 136–158. https://doi.org/10.1016/j.nonrwa.2016.04.009
12. C. Z. Olorunsaiye, K. K. Yusuf, K. Reinhart, H. M. Salihu, COVID-19 and child vaccination: a systematic approach to closing the immunization gap, *Int. J. Matern. Child Health Aids*, **9** (2020), 381–385. http://orcid.org/0000-0003-4725-0448
13. R. M. Anderson, R. M. May, Vaccination and herd immunity to infectious diseases, *Nature*, **318** (1985), 323–329. https://doi.org/10.1038/318323a0
14. S. Zhai, G. Luo, T. Huang, X. Wang, J. Tao, P. Zhou, Vaccination control of an epidemic model with time delay and its application to COVID-19, *Nonlinear Dyn.*, **106** (2021), 1279–1292. https://doi.org/10.1007/s11071-021-06533-w

15. J. Yang, Q. Zhang, Z. Cao, J. Gao, D. Pfeiffer, L. Zhong, et al., The impact of non-pharmaceutical interventions on the prevention and control of COVID-19 in New York City, *Chaos*, **31** (2021), 021101. https://doi.org/10.1101/2020.12.01.20242347
16. G. O. Agaba, Y. N. Kyrychko, K. B. Blyuss, Dynamics of vaccination in a time-delayed epidemic model with awareness, *Math. Biosci.*, **294** (2017), 92–99. https://doi.org/10.1016/j.mbs.2017.09.007
17. I. Z. Kiss, J. Cassell, M. Recker, P. L. Simon, The impact of information transmission on epidemic outbreaks, *Math. Biosco.*, **225** (2010), 1–10. https://doi.org/10.1016/j.mbs.2009.11.009
18. T. K. Kar, S. K. Nandi, S. Jana, M. Mandal, Stability and bifurcation analysis of an epidemic model with the effect of media, *Chaos Soliton Fract.*, **120** (2019), 188–199. https://doi.org/10.1016/j.chaos.2019.01.025
19. Z. Liu, P. Magal, O. Seydi, G. Webb, A COVID-19 epidemic model with latency period, *Infect. Dis. Model.*, **5** (2020), 323–337. https://doi.org/10.1016/j.idm.2020.03.003
20. C. C. McCluskey, Global stability for an SIR epidemic model with delay and nonlinear incidence, *Nonlinear Anal.*, **11** (2010), 3106–3109. https://doi.org/10.1016/j.nonrwa.2009.11.005
21. X. Zhou, J. Cui, Stability and Hopf bifurcation of a delay eco-epidemiological model with nonlinear incidence rate, *Math. Model. Anal.*, **15** (2010), 547–569. https://doi.org/10.3846/1392-6292.2010.15.547-569
22. X. Zhou, Z. Guo, Analysis of stability and Hopf bifurcation for an eco-epidemiological model with distributed delay, *Electron. J. Qual. Theory*, **44** (2012), 1–22. https://doi.org/10.14232/ejqtde.2012.1.44
23. W. B. Ma, S. Mei, Y. Takeuchi, Global stability of an SIR epidemic model with time delay, *Appl. Math. Lett.*, **17** (2003), 1141–1145. https://doi.org/10.1016/j.aml.2003.11.005
24. A. K. Misra, A. Sharma, V. Singh, Effect of awareness programs in controlling the prevalence of an epidemic with time delay, *J. Biol. Syst.*, **19** (2011), 389–402. https://doi.org/10.1142/S0218339011004020
25. M. J. Mulligan, An inactivated virus candidate vaccine to prevent COVID-19, *J. Am. Med. Assoc.*, **324** (2020), 943–945. http://jamanetwork.com/article.aspx?doi=10.1001/jama.2020.15539
26. S. Rahman, M. M. Rahman, M. Rahman, M. N. Begum, M. Sarmin, M. Mahfuz, et al., COVID-19 reinfections among naturally infected and vaccinated individuals, *Sci. Rep.*, **12** (2022), 1438. https://doi.org/10.1038/s41598-022-05325-5

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