An epidemic model coupled with environmental level: explore the impact of disease awareness on direct and indirect transmission

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Abstract

To study the impact of disease awareness on infectious diseases with direct and indirect transmission, we develop a mathematical model by coupling the transmission dynamics at the population level and the environmental level. The basic reproduction number R_0 of the coupled model is calculated, and the existence and stability of the disease-free and endemic equilibrium are analyzed in detail. By using center manifold theory, it is verified that the model undergoes backward bifurcation under certain conditions. Numerical simulations verify our theoretical results and indicate that enhancing disease awareness can help reduce both the risk of direct and indirect disease transmission. Interestingly, increasing disease awareness decreases the backward regime of the bifurcation curve, thereby the R_0 interval in which the endemic equilibrium and the disease-free equilibrium showed bistability becomes smaller, and the R_0 interval in which the disease-free equilibrium showed global stability becomes greater. If the disease cannot be eliminated, the number of infected persons at the steady state decreases with the increase in disease awareness. The findings have certain reference values for the development of effective non-pharmaceutical intervention policies.

Keywords: Coupled epidemic model; Disease awareness; Backward bifurcation; Global stability.

1. Introduction

Infectious diseases seriously threaten human health and economic development. Since the 21st century, there have been many large-scale outbreaks of infectious diseases, such as H1N1 in 2009 and COVID-19 in 2019, etc [1]. Some infectious diseases, like viral respiratory infections (VRIs), cholera, and hand, foot and mouth disease (HFMD) can be spread not only directly by contact with infected individuals but also indirectly by contact with the contaminated surfaces or objects [2–15]. For example, some respiratory viruses, such as human rhinovirus (HRV), respiratory syncytial virus (RSV), and influenza virus (IFV), can survive in the environment, especially in cold and dry environments, for a long time and

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remain infectious, thus presenting a risk of indirect transmission [2–5]. SARS-CoV-2 can survive in the environment, especially on plastic and stainless steel, for several days [6], thus indirect transmission has had a significant impact on the COVID-19 outbreak [7–9]. Cholera is an acute intestinal infectious disease caused by the bacteria Vibrio cholerae (V. cholerae), which can survive in contaminated environments, especially river water and seawater, for several weeks [10–12], and indirect transmission is one of the main transmission routes of cholera. The human enterovirus (EV) that causes HFMD can also survive for 2 to 12 days on the surfaces of a wide variety of household items [13–15], so indirect transmission cannot be ignored. In the process of disease transmission, people may get information about the disease gradually, and people who are aware of the disease may engage in protective behaviors to reduce the risk of infection, such as maintaining personal hygiene and maintaining social distancing. Thus, people's disease awareness has a great effect on disease transmission, especially in today's society, where the media is highly developed and information spreads rapidly. Hence, it is of great significance to study how disease awareness affects the direct and indirect transmission of infectious diseases.

Many mathematical models have been proposed to investigate the impact of disease awareness on the spread of infectious diseases [16–24]. Agaba et al. [17] have proposed a SIRS model considering the influence of public and private awareness on infectious diseases. The study found that the spread of awareness reduces disease transmission, and increases the recovery rate of infected people. Das et al. [20] have established a SEIR model by introducing a media awareness related infection rate function to research the influence of awareness on tuberculosis transmission. The research found that increasing media awareness can reduce the peak level of infected people. Sharmin et al. [21] have investigated how awareness affects disease transmission by introducing a media compartment to the classical SIR model. Their findings indicate continuous publicity is effective in preventing disease transmission. By supposing that the infection rate is a decreasing function and removal rate of mosquitoes is a non-decreasing saturation function of disease awareness, Basir et al. [22] have studied the influence of awareness on malaria transmission. The study found that increasing people's awareness would reduce the abundance of mosquitoes in the environment. Recently, Aldila [24] has studied how media awareness impacts dengue eradication by introducing the control variable media publicity and found that high-intensity media attention significantly reduced the scale of infection. However, to our knowledge, current studies have not examined the effects of awareness on diseases that can be transmitted both directly and indirectly.

To study the indirect transmission of infectious diseases, many researchers have introduced an environmental compartment into their models [25–31]. Feng et al. [25] have studied toxoplasmosis infectious diseases by explicitly linking epidemiology and immunology through an environmental compartment. Taking bacterial infection as an example, Xiao et al. [27] have established a model considering the pathogen concentration in the environment to study the impact of individual movement and spatial control measures on disease outbreaks. In this article, we shall establish a mathematical model with disease awareness, which takes into account both direct transmission between people and indirect transmission between people and the environment. It should be noted that the environmental time scale is slower than the epidemiological time scale [25], so our model couples two different time

scales and contains multiple transmission routes of the disease.

Based on the proposed multiscale model, we shall study how disease awareness affects both the direct and indirect transmission of infectious diseases. For this model, detailed theoretical analyses of the local and global dynamics are presented. Choosing the basic reproduction number as the bifurcation parameter, the emergence of backward bifurcation under certain conditions is proved. Moreover, by numerical simulations, we verify our theoretical conclusions, and it is found that disease awareness significantly affects both the direct and indirect transmission. A great reduction in the peak level of the number of exposed individuals, infected individuals, and the virus concentration is observed when disease awareness increases. Interestingly, increasing disease awareness decreases the backward regime of the bifurcation curve, so that the R_0 interval in which the endemic equilibrium and the disease-free equilibrium showed bistability becomes smaller, and the R_0 interval in which the disease-free equilibrium showed global stability becomes greater. If the disease cannot be eliminated, the number of infected persons at the steady state decreases with the increase in disease awareness.

The paper is organized as follows: the mathematical model is given in Section 2. In Section 3, the expression of the basic reproduction number is given, and the existence and stability of the disease-free equilibrium and the endemic equilibrium are analyzed. In Section 4, the possibility of backward bifurcation at $R_0 = 1$ is proved. Section 5 validates the theoretical results and assesses the impact of disease awareness on disease transmission through numerical simulations. In Section 6, some discussions and summaries are given.

2. Mathematical model

A mathematical model with disease awareness is formulated by coupling the transmission dynamics at the population level and the environmental level. The total population (N) is divided into five compartments, including unaware susceptible individuals (S_n), aware susceptible individuals (S_a), exposed individuals (E), infected individuals (I), and recovered individuals (R). The environmental contamination level is denoted by W. There is a constant recruitment rate A for the susceptible population, and a natural death rate ϵ for the whole population. It is assumed that all the newly recruited susceptibles are unaware of the disease. The unaware susceptible individuals could become aware susceptibles due to disease awareness at a rate of a. The awareness acquisition rate, denoted as a, can be regarded as a constant, as this rate is primarily influenced by stable external factors such as the efficiency of information dissemination and the level of public attention. Both unaware and aware susceptible individuals could be infected directly by contacting with exposed and infected individuals or indirectly through contacting the contaminated environment, and the infection rate of the aware susceptibles is decreased by proportion σ compared with unaware susceptibles, where σ $(0 \leq \sigma < 1)$ represents the effect of disease awareness on direct and indirect transmission. Since the viral dynamics in the environment is slow relative to the epidemic dynamics between hosts, two different time scales are coupled by a constant ζ

$(0 < \zeta < 1)$. The following equations can be used to describe the mathematical model:

$$\begin{cases} \frac{\mathrm{d}S_n}{\mathrm{d}t} = A - (\beta_1 E + \beta_2 I + \beta_3 W)S_n - aS_n + \theta S_a - \epsilon S_n, \\ \frac{\mathrm{d}S_a}{\mathrm{d}t} = aS_n - (1-\sigma)(\beta_1 E + \beta_2 I + \beta_3 W)S_a - \theta S_a - \epsilon S_a, \\ \frac{\mathrm{d}E}{\mathrm{d}t} = (\beta_1 E + \beta_2 I + \beta_3 W)S_n + (1-\sigma)(\beta_1 E + \beta_2 I + \beta_3 W)S_a - \delta E - \epsilon E, \\ \frac{\mathrm{d}I}{\mathrm{d}t} = \delta E - \gamma I - \epsilon I, \\ \frac{\mathrm{d}R}{\mathrm{d}t} = \gamma I - \epsilon R, \\ \frac{\mathrm{d}W}{\mathrm{d}t} = \zeta(\eta_1 E + \eta_2 I - \mu W), \end{cases} \tag{1}$$

with initial conditions

$$S_n(0) > 0,\ S_a(0) > 0,\ E(0) > 0,\ I(0) > 0,\ R(0) > 0,\ W(0) > 0.$$

Based on references [17, 18], and [27], we assign specific values to the parameters a, θ, and η_2. The remaining parameters are assigned ranges based on their biological relevance. Notably, as this paper employs a bilinear incidence rate, the three transmission rates must be scaled in proportion to the reciprocal of the total population. Furthermore, we assume that the virus release rate from individuals during the latent period is lower than that from infected individuals. Table 1 lists all the parameters involved in the model (1).

Table 1: Interpretation of the parameters in the model (1).

Param	Biological Meaning	Value	Source
A	Recruitment rate of susceptible individuals	$0.1 \sim 2$/day	Assumed
ϵ	Natural death rate	$0 \sim 1$/day	Assumed
a	Awareness acquisition rate	0.4/day	[17]
θ	Awareness lossing rate	0.2/day	[18]
β_1	Direct transmission rate of exposed individuals	$0.4\times10^{-3} \sim 0.045$/day	Assumed
β_2	Direct transmission rate of infected individuals	$0.5\times10^{-3} \sim 0.053$/day	Assumed
β_3	Environmental Indirect transmission rate	$0.2\times10^{-3} \sim 0.023$/day	Assumed
σ	The impact of disease awareness on both direct and indirect transmission	$0 \sim 1$/day	Assumed
δ	Probability of conversion of exposed persons to infected persons	$0.16 \sim 0.45$/day	Assumed
γ	Recovery rate for those with infection	$0.18 \sim 0.45$/day	Assumed
η_1	Viral shedding rate of exposed individuals	$0 \sim 0.6$/day	Assumed
η_2	Viral shedding rate of infected individuals	0.6/day	[27]
μ	Environmental clearance rate	$0 \sim 1$/day	Assumed
ζ	Scale parameter	$0 \sim 1$/day	Assumed

Adding the first five equations of the model (1), we obtain

$$\frac{\mathrm{d}N}{\mathrm{d}t} = A - \epsilon(S_n + S_a + E + I + R) = A - \epsilon N. \tag{2}$$

Therefore, we have

$$N(t) = N(0)e^{-\epsilon t} + \frac{A}{\epsilon}(1 - e^{-\epsilon t}) \text{ and } \lim_{t\to\infty} N(t) = \frac{A}{\epsilon}.$$

From the last equation of the model (1), we obtain

$$\begin{aligned}\frac{\mathrm{d}W}{\mathrm{d}t} &= \zeta(\eta_1 E + \eta_2 I - \mu W)\\ &\leq \zeta[(\eta_1+\eta_2)\frac{A}{\epsilon} - \mu W].\end{aligned}$$

Hence, the biological feasible region of model (1) is given as

$$\Omega = \left\{(S_n, S_a, E, I, R, W) \in \mathbb{R}_+^6 \mid 0 \leq S_n + S_a + E + I + R \leq \tfrac{A}{\epsilon}, 0 \leq W \leq \tfrac{A(\eta_1+\eta_2)}{\epsilon\mu}\right\}.$$

Throughout this paper, the dynamical behaviors of system (1) will be discussed in the region Ω.

3. Model Analysis

In this section, we shall calculate the basic reproduction number and analyze the existence and stability of the disease-free equilibrium and endemic equilibrium of model (1).

Letting the right-hand sides of model (1) be 0, it is obvious that the disease-free equilibrium $E_0 = (S_n^0, S_a^0, 0, 0, 0, 0) = (\frac{A(\theta+\epsilon)}{\epsilon(a+\theta+\epsilon)}, \frac{Aa}{\epsilon(a+\theta+\epsilon)}, 0, 0, 0, 0)$ always exists. The basic reproduction number R_0 gives the average number of secondary infection caused by a single infected individual in a whole susceptible population [32]. According to the next-generation matrix method illustrated by Van den Driessche et al. [33], the transmission (F) and transition (V) matrix of system (1) evaluated in E_0 as follows

$$F = \begin{pmatrix} \beta_1 S_n^0 + (1-\sigma)\beta_1 S_a^0 & \beta_2 S_n^0 + (1-\sigma)\beta_2 S_a^0 & \beta_3 S_n^0 + (1-\sigma)\beta_3 S_a^0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix},$$

and

$$V = \begin{pmatrix} \delta+\epsilon & 0 & 0 \\ -\delta & \gamma+\epsilon & 0 \\ -\zeta\eta_1 & -\zeta\eta_2 & \zeta\mu \end{pmatrix}.$$

Then, R_0 can be calculated by

$$R_0 = \rho(FV^{-1}) = R_{0E} + R_{0I} + R_{0W} = \frac{Ah\delta[\theta+\epsilon+(1-\sigma)a]}{\epsilon(\delta+\epsilon)(\gamma+\epsilon)(a+\theta+\epsilon)}, \tag{3}$$

where

$$\begin{aligned} h &= \beta_1\frac{\gamma+\epsilon}{\delta} + \beta_2 + \beta_3\frac{\eta_1(\gamma+\epsilon)+\eta_2\delta}{\delta\mu},\\ R_{0E} &= [\beta_1 S_n^0 + (1-\sigma)\beta_1 S_a^0]\frac{1}{\delta+\epsilon},\\ R_{0I} &= [\beta_2 S_n^0 + (1-\sigma)\beta_2 S_a^0]\frac{\delta}{(\delta+\epsilon)(\gamma+\epsilon)},\\ R_{0W} &= [\beta_3 S_n^0 + (1-\sigma)\beta_3 S_a^0]\frac{\eta_1(\gamma+\epsilon)+\delta\eta_2}{\mu(\delta+\epsilon)(\gamma+\epsilon)}. \end{aligned}$$

Here, R_{0E} and R_{0I} are the average number of secondary infections caused by the exposed and infected individuals, respectively. And R_{0W} measures the contribution of indirect transmission by contacting the contaminated environment.

3.1. Existence of endemic equilibria

Let $Q^*(S_n^*, S_a^*, E^*, I^*, R^*, W^*)$ be an arbitrary endemic equilibrium of system (1), we have

$$\begin{aligned}
S_n^* &= \frac{A[(1-\sigma)hI^* + \theta + \epsilon]}{(hI^* + a + \epsilon)[(1-\sigma)hI^* + \theta + \epsilon] - a\theta},\\
S_a^* &= \frac{Aa}{(hI^* + a + \epsilon)[(1-\sigma)hI^* + \theta + \epsilon] - a\theta},\\
E^* &= \frac{\gamma + \epsilon}{\delta} I^*, \quad R^* = \frac{\gamma}{\epsilon} I^*, \quad W^* = \frac{\eta_1(\gamma + \epsilon) + \eta_2\delta}{\delta\mu} I^*.
\end{aligned}$$

Here, I^* satisfies

$$m(I^*)^2 + nI^* + c = 0, \tag{4}$$

where

$$\begin{aligned}
m &= (\delta + \epsilon)(\gamma + \epsilon)(1-\sigma)h^2,\\
n &= (\gamma + \epsilon)(\delta + \epsilon)[(1-\sigma)(a + \epsilon) + \theta + \epsilon]h - A\delta(1-\sigma)h^2,\\
c &= \epsilon(a + \epsilon + \theta)(\delta + \epsilon)(\gamma + \epsilon)(1 - R_0).
\end{aligned} \tag{5}$$

Note that $m > 0$ and

$$c < 0 \Leftrightarrow R_0 > 1;\ c = 0 \Leftrightarrow R_0 = 1;\ c > 0 \Leftrightarrow R_0 < 1.$$

According to the discriminant of equation (4) that $\triangle = n^2 - 4mc = n^2 - 4m\epsilon(a + \epsilon + \theta)(\delta + \epsilon)(\gamma + \epsilon)(1 - R_0)$, solving for $\triangle = 0$ by R_0, we have $R_0 = R_c$, where

$$R_c = 1 - \frac{n^2}{4m\epsilon(a + \epsilon + \theta)(\delta + \epsilon)(\gamma + \epsilon)}.$$

Clearly, $R_c < 1$, and the following equivalent relations are true

$$R_0 < R_c \Leftrightarrow \triangle < 0;\ R_0 = R_c \Leftrightarrow \triangle = 0;\ R_0 > R_c \Leftrightarrow \triangle > 0.$$

Through detailed analyses, the following conclusions on the existence of endemic equilibria are obtained.

Theorem 1. *System (1) has*

(1)If $n > 0$, system (1) has one unique endemic equilibrium $Q_1^*(S_{n1}^*, S_{a1}^*, E_1^*, I_1^*, R_1^*, W_1^*)$ for $R_0 > 1$, but no endemic equilibrium for $R_0 \le 1$.

(2)If $n < 0$, system (1) has one endemic equilibrium $Q_1^*(S_{n1}^*, S_{a1}^*, E_1^*, I_1^*, R_1^*, W_1^*)$ for $R_0 \ge 1$, two unequal endemic equilibria $Q_1^*(S_{n1}^*, S_{a1}^*, E_1^*, I_1^*, R_1^*, W_1^*)$ and $Q_2^*(S_{n2}^*, S_{a2}^*, E_2^*, I_2^*, R_2^*, W_2^*)$ for $R_c < R_0 < 1$, and the two equilibria degenerates to one unique endemic equilibrium $Q_3(S_{n3}^*, S_{a3}^*, E_3^*, I_3^*, R_3^*, W_3^*)$ for $R_c = R_0 < 1$, where

$$I_1^* = \frac{-n + \sqrt{\triangle}}{2m}, \quad I_2^* = \frac{-n - \sqrt{\triangle}}{2m}, \quad I_3^* = \frac{-n}{2m}.$$

Table 2: Existence of endemic equilibria for the system (1).

Cases	Ranges of Threshold	Existence of Endemic Equilibria
(1)	$n > 0$ and $R_0 \leq 1$	-
(2)	$n > 0$ and $R_0 > 1$	Q_1^*
(3)	$n < 0$ and $R_0 \geq 1$	Q_1^*
(4)	$n < 0$ and $R_c < R_0 < 1$	Q_1^*, Q_2^*
(5)	$n < 0$ and $R_c = R_0 < 1$	Q_3^*
(6)	$n < 0$ and $R_0 < R_c$	-

By Theorem 1, we summarize the conditions for the existence of endemic equilibria in Table 2 to facilitate subsequent analysis and discussion.

Due to the complexity of the n form, we further simplify it to (6) in order to observe its biological significance, where (6) is as follows

$$\begin{aligned} n > 0 &\Leftrightarrow (\gamma+\epsilon)(\delta+\epsilon)[(1-\sigma)(a+\epsilon)+\theta+\epsilon]h - A\delta(1-\sigma)h^2 > 0, \\ &\Leftrightarrow (\gamma+\epsilon)(\delta+\epsilon)[(1-\sigma)(a+\epsilon)+\theta+\epsilon] - A\delta(1-\sigma)h > 0, \\ &\Leftrightarrow \frac{A\delta(1-\sigma)h}{(\gamma+\epsilon)(\delta+\epsilon)[(1-\sigma)(a+\epsilon)+\theta+\epsilon]} < 1. \end{aligned} \tag{6}$$

It is evident that as h increases, the left-hand side of the last equation of (6) becomes larger, making it more likely that the value of n is greater than 0. Since h reflects the transmission intensity of the disease per unit time to a certain extent, it follows that the weaker the transmission intensity per unit time, the more likely it is for the coexistence of a disease-free equilibrium and an endemic equilibrium, presenting a bistable phenomenon.

3.2. Stability of the disease-free equilibrium

Theorem 2. *The disease-free equilibrium E_0 of system(1) is locally asymptotically stable if $R_0 < 1$ and is unstable if $R_0 > 1$.*

Proof. Let $a_1 = \beta_1[S_n^0+(1-\sigma)S_a^0]-\delta-\epsilon$, $a_2 = \beta_2[S_n^0+(1-\sigma)S_a^0]$, $a_3 = \beta_3[S_n^0+(1-\sigma)S_a^0]$. The Jacobian matrix of the system (1) at E_0 is calculated as follows

$$J(E_0) = \begin{pmatrix} -a-\epsilon & \theta & -\beta_1 S_n^0 & -\beta_2 S_n^0 & 0 & -\beta_3 S_n^0 \\ a & -\theta-\epsilon & -(1-\sigma)\beta_1 S_a^0 & -(1-\sigma)\beta_2 S_a^0 & 0 & -(1-\sigma)\beta_3 S_a^0 \\ 0 & 0 & a_1 & a_2 & 0 & a_3 \\ 0 & 0 & \delta & -\gamma-\epsilon & 0 & 0 \\ 0 & 0 & 0 & \gamma & -\epsilon & 0 \\ 0 & 0 & \zeta\eta_1 & \zeta\eta_2 & 0 & -\zeta\mu \end{pmatrix}.$$

Its characteristic equation is

$$\lambda^3 + b_1\lambda^2 + b_2\lambda + b_3 = 0.$$

Here, $b_1 = \zeta\mu + \gamma + \epsilon + (\delta+\epsilon)(1-R_{0E}), b_2 = \zeta\mu(\gamma+\epsilon) + (\gamma+\epsilon)(\delta+\epsilon)(1-R_{0E}-R_{0I})$ $+\zeta\mu(\delta+\epsilon)(1-R_{0E}-\frac{\eta_1(\gamma+\epsilon)}{\eta_1(\gamma+\epsilon)+\eta_2\delta}R_{0W}), b_3 = \zeta\mu(\delta+\epsilon)(\gamma+\epsilon)(1-R_0)$. Clearly, when $R_0 < 1$, we have $b_1 > 0$, $b_2 > 0$, $b_3 > 0$ and $b_1b_2 - b_3 > 0$. According to the Routh-Hurwitz criteria, we conclude that all eigenvalues of the Jacobian matrix $J(E_0)$ have negative real parts, which means E_0 is locally asymptotically stable when $R_0 < 1$. If $R_0 > 1$, then E_0 is unstable as $b_3 < 0$ holds.

Theorem 3. *The disease-free equilibrium E_0 of system (1) is globally asymptotically stable if $R_0^* < 1$, where $R_0^* = \frac{A\delta h}{\epsilon(\delta+\epsilon)(\gamma+\epsilon)}$.*

Proof. Define the Lyapunov function $V(t)$ as follows

$$V(t) = E(t) + \frac{A(\beta_2\mu+\eta_2\beta_3)}{\epsilon\mu(\gamma+\epsilon)}I(t) + \frac{A\beta_3}{\epsilon\zeta\mu}W(t).$$

Clearly, $V(t) \geq 0$, and $V(t) = 0$ holds only at E_0. Along the solution of the model (1), the derivative of $V(t)$ is given as

$$\begin{aligned}
\frac{\mathrm{d}V(t)}{\mathrm{d}t} &= \frac{\mathrm{d}E}{\mathrm{d}t} + \frac{A(\beta_2\mu+\eta_2\beta_3)}{\epsilon\mu(\gamma+\epsilon)}\frac{\mathrm{d}I}{\mathrm{d}t} + \frac{A\beta_3}{\epsilon\zeta\mu}\frac{\mathrm{d}W}{\mathrm{d}t} \\
&= [(\beta_1E + \beta_2I + \beta_3W)(S_n + (1-\sigma)S_a) - (\delta+\epsilon)E] \\
&\quad + \frac{A(\beta_2\mu+\eta_2\beta_3)}{\epsilon\mu(\gamma+\epsilon)}[\delta E - (\gamma+\epsilon)I] + \frac{A\beta_3}{\epsilon\mu}(\eta_1E + \eta_2I - \mu W) \\
&= [\beta_1(S_n + (1-\sigma)S_a) + \frac{A(\beta_2\mu+\eta_2\beta_3)}{\epsilon\mu(\gamma+\epsilon)}\delta + \frac{A\beta_3\eta_1}{\epsilon\mu} - (\delta+\epsilon)]E \\
&\quad + [\beta_2(S_n + (1-\sigma)S_a) + \frac{A\beta_3\eta_2}{\epsilon\mu} - \frac{A(\beta_2\mu+\eta_2\beta_3)}{\epsilon\mu}]I \\
&\quad + [\beta_3(S_n + (1-\sigma)S_a) - \frac{A\beta_3}{\epsilon}]W \\
&\leq [\beta_1\frac{A}{\epsilon} + \frac{A(\beta_2\mu+\eta_2\beta_3)}{\epsilon\mu(\gamma+\epsilon)}\delta + \frac{A\beta_3\eta_1}{\epsilon\mu} - (\delta+\epsilon)]E + [\beta_2\frac{A}{\epsilon} \\
&\quad + \frac{A\beta_3\eta_2}{\epsilon\mu} - \frac{A(\beta_2\mu+\eta_2\beta_3)}{\epsilon\mu}]I + [\beta_3\frac{A}{\epsilon} - \frac{A\beta_3}{\epsilon}]W \\
&= [\frac{A}{\epsilon}(\beta_1 + \beta_2\frac{\delta}{(\gamma+\epsilon)} + \beta_3\frac{\eta_1(\gamma+\epsilon)+\eta_2\delta}{\mu(\gamma+\epsilon)}) - (\delta+\epsilon)]E \\
&= (\delta+\epsilon)[\frac{A\delta h}{\epsilon(\delta+\epsilon)(\gamma+\epsilon)} - 1]E \\
&= (\delta+\epsilon)(R_0^* - 1)E.
\end{aligned}$$

It is clear that if $R_0^* < 1$, $\frac{\mathrm{d}V(t)}{\mathrm{d}t} \leq 0$ holds, and $\frac{\mathrm{d}V(t)}{\mathrm{d}t} = 0$ if and only if $E = I = W = 0$. Consequently, E_0 is globally asymptotically stable for $R_0^* < 1$ by using LaSalle's invariance principle [34]. It needs to mention that $n = (\gamma+\epsilon)(\delta+\epsilon)h[(1-\sigma)a+\epsilon+\theta+\epsilon(1-\sigma)(1-R_0^*)]$, thus $n > 0$ holds if $R_0^* < 1$. Meanwhile, it is simple to calculate that $R_0 < R_0^*$ always holds. Hence, if $R_0^* < 1$, then $n > 0$ and $R_0 < 1$ hold, and E_0 is globally asymptotically stable.

3.3. Stability of the endemic equilibrium

Firstly, by studying the Jacobian matrix of the system (1) at the endemic equilibrium Q_1^*, we can get the local stability of the equilibrium by Routh-Hurwitz criteria, as given in the following theorem.

Theorem 4. *The endemic equilibrium Q_1^* of system (1) is locally asymptotically stable if $B_3(B_1B_2 - B_3) > B_1(B_1B_4 - B_5)$ and $(B_4B_3 - B_2B_5)(B_1B_2 - B_3) > (B_1B_4 - B_5)^2$.*

See the appendix A for the proof and expressions of B_i $(i = 1, 2, \cdots, 5)$.

Note that by Theorem 2, E_0 is locally asymptotically stable when $R_0 < 1$. Therefore, if $n < 0$, then E_0 and Q_1^* may be bistable for $R_c < R_0 < 1$. This bistable phenomenon is caused by the backward bifurcation at $R_0 = 1$, which we will investigate later. In the following, we shall construct a Lyapunov function to analyze the global stability of equilibrium Q_1^* for $R_0 > 1$.

Theorem 5. *The unique endemic equilibrium Q_1^* of system (1) is globally asymptotically stable if $R_0 > 1$.*

Proof. Define the Lyapunov function $\mathcal{L}(t)$ as follows

$$\begin{aligned}\mathcal{L}(t) = {} & S_n(t) - S_{n1}^* - S_{n1}^* \ln \frac{S_n(t)}{S_{n1}^*} + S_a(t) - S_{a1}^* - S_{a1}^* \ln \frac{S_a(t)}{S_{a1}^*} + E(t) - E_1^* - E_1^* \ln \frac{E(t)}{E_1^*} \\ & + \ell_1(I(t) - I_1^* - I_1^* \ln \frac{I(t)}{I_1^*}) + \ell_2(W(t) - W_1^* - W_1^* \ln \frac{W(t)}{W_1^*}),\end{aligned}$$

where

$$\ell_1 = \frac{(\mu\beta_2 + \eta_2\beta_3)[S_{n1}^* + (1-\sigma)S_{a1}^*]}{\mu(\gamma+\epsilon)}, \quad \ell_2 = \frac{\beta_3[S_{n1}^* + (1-\sigma)S_{a1}^*]}{\zeta\mu}.$$

Then, the derivative of $\mathcal{L}(t)$ is given as

$$\begin{aligned}\frac{\mathrm{d}\mathcal{L}(t)}{\mathrm{d}t} = {} & \frac{\mathrm{d}S_n}{\mathrm{d}t}(1 - \frac{S_{n1}^*}{S_n}) + \frac{\mathrm{d}S_a}{\mathrm{d}t}(1 - \frac{S_{a1}^*}{S_a}) + \frac{\mathrm{d}E}{\mathrm{d}t}(1 - \frac{E_1^*}{E}) + \ell_1\frac{\mathrm{d}I}{\mathrm{d}t}(1 - \frac{I_1^*}{I}) + \ell_2\frac{\mathrm{d}W}{\mathrm{d}t}(1 - \frac{W_1^*}{W}) \\ = {} & (1 - \frac{S_{n1}^*}{S_n})[A - (\beta_1E + \beta_2I + \beta_3W)S_n - aS_n + \theta S_a - \epsilon S_n] + (1 - \frac{S_{a1}^*}{S_a})[aS_n \\ & - (1-\sigma)(\beta_1E + \beta_2I + \beta_3W)S_a - \theta S_a - \epsilon S_a] + (1 - \frac{E_1^*}{E})[(\beta_1E + \beta_2I + \beta_3W)S_n \\ & + (1-\sigma)(\beta_1E + \beta_2I + \beta_3W)S_a - \delta E - \epsilon E] + \ell_1(1 - \frac{I_1^*}{I})[\delta E - \gamma I - \epsilon I] \\ & + \ell_2(1 - \frac{W_1^*}{W})\zeta(\eta_1E + \eta_2I - \mu W).\end{aligned}$$

By denoting $\frac{S_n}{S_{n1}^*}=y_1$, $\frac{S_a}{S_{a1}^*}=y_2$, $\frac{E}{E_1^*}=z_1$, $\frac{I}{I_1^*}=z_2$, $\frac{W}{W_1^*}=z_3$, we obtain

$$\begin{aligned}
\frac{\mathrm{d}\mathcal{L}(t)}{\mathrm{d}t} &= A+(\epsilon+a)S_{n1}^*+(\theta+\epsilon)S_{a1}^*+(\delta+\epsilon)E_1^*+\ell_1(\gamma+\epsilon)I_1^*+\ell_2\zeta\mu W_1^* \\
&\quad -y_1(\epsilon S_{n1}^*+\beta_1E_1^*S_{n1}^*)-\frac{1}{y_1}A-\frac{y_2}{y_1}\theta S_{a1}^*-\frac{y_1}{y_2}aS_{n1}^*-y_2[\epsilon S_{a1}^*+(1-\sigma)\beta_1E_1^*S_{a1}^*] \\
&\quad -\frac{y_1z_2}{z_1}\beta_2I_1^*S_{n1}^*-\frac{y_1z_3}{z_1}\beta_3W_1^*S_{n1}^*-\frac{y_2z_2}{z_1}(1-\sigma)\beta_2I_1^*S_{a1}^*-\frac{y_2z_3}{z_1}(1-\sigma)\beta_3W_1^*S_{a1}^* \\
&\quad -\ell_1\frac{z_1}{z_2}\delta E_1^*-\frac{z_1}{z_3}\ell_2\zeta\eta_1E_1^*-\frac{z_2}{z_3}\ell_2\zeta\eta_2I_1^* \\
&= (\epsilon S_{n1}^*+\beta_1E_1^*S_{n1}^*)(2-y_1-\frac{1}{y_1})+[\epsilon S_{a1}^*+(1-\sigma)\beta_1E_1^*S_{a1}^*](3-y_2-\frac{y_1}{y_2}-\frac{1}{y_1}) \\
&\quad +\theta S_{a1}^*(2-\frac{y_2}{y_1}-\frac{y_1}{y_2})+\beta_2I_1^*S_{n1}^*(3-\frac{y_1z_2}{z_1}-\frac{z_1}{z_2}-\frac{1}{y_1})+\frac{\eta_1(\gamma+\epsilon)}{\mu\delta}\beta_3S_{n1}^*I_1^*(3-\frac{y_1z_3}{z_1} \\
&\quad -\frac{z_1}{z_3}-\frac{1}{y_1})+(1-\sigma)\beta_2I_1^*S_{a1}^*(4-\frac{y_2z_2}{z_1}-\frac{y_1}{y_2}-\frac{z_1}{z_2}-\frac{1}{y_1})+(1-\sigma)\frac{\eta_1(\gamma+\epsilon)}{\mu\delta}\beta_3S_{a1}^*I_1^*(4 \\
&\quad -\frac{y_2z_3}{z_1}-\frac{z_1}{z_3}-\frac{y_1}{y_2}-\frac{1}{y_1})+\frac{\eta_2}{\mu}\beta_3S_{n1}^*I_1^*(4-\frac{y_1z_3}{z_1}+\frac{z_2}{z_3}-\frac{z_1}{z_2}-\frac{1}{y_1}) \\
&\quad +(1-\sigma)\frac{\eta_2}{\mu}\beta_3S_{a1}^*I_1^*(5-\frac{y_2z_3}{z_1}-\frac{z_2}{z_3}-\frac{z_1}{z_2}-\frac{y_1}{y_2}-\frac{1}{y_1}).
\end{aligned}$$

Since the arithmetic mean is greater than the geometric mean, according to the above analysis, we have $\frac{\mathrm{d}\mathcal{L}(t)}{\mathrm{d}t}\leq 0$. And $\frac{\mathrm{d}\mathcal{L}(t)}{\mathrm{d}t}=0$ if and only if $y_i=1$ $(i=1,2)$, $z_1=1$, $z_2=z_3=z_1$ that is, $\frac{\mathrm{d}\mathcal{L}(t)}{\mathrm{d}t}=0$ if and only if $S_n=S_{n1}^*$, $S_a=S_{a1}^*$, $E={E_1}^*$, $I=I_1^*$, $W=W_1^*$. Substituting relations $I=I_1^*$ into the fifth equation of system (1), we get $\gamma I_1^*-\epsilon R=0$, then we have $R=R_1^*=\frac{\gamma}{\epsilon}I_1^*$. Consequently, we obtain that Q_1^* is globally asymptotically stable if $R_0>1$ by using LaSalle's invariance principle [34].

Based on the detailed analysis of the dynamics of system (1), the stability of the relevant equilibria is summarized in Table 3. The asterisk (*) in Table 3 indicates that the result is numerically verified.

4. Backward bifurcation analysis

In this section, combined with the previous analysis of the existence of endemic equilibria, we shall discuss the backward bifurcation problem of system (1). In addition, the influence of disease awareness on backward bifurcation is investigated.

From Theorem (1), it shows that if $n<0$, there exists a unique endemic equilibrium of the system (1) for $R_0\geq 1$, and there are two endemic equilibria for $R_c<R_0<1$, which indicates that the system (1) may occur backward bifurcation. Thus, based on the general center manifold theory proposed by Castillo-Chavez and Song, the threshold conditions under which backward bifurcation may exhibit in the system (1) are investigated.

Table 3: Stability of equilibria for the system (1).

Ranges of Threshold	E_0	Q_1^*	Q_2^*	Q_3^*
$n > 0$ and $R_0 < 1$	LAS	-	-	-
$n > 0$ and $R_0^* < 1$	GAS	-	-	-
$n > 0$ and $R_0 > 1$	Unstable	GAS	-	-
$n < 0$ and $R_0 > 1$	Unstable	GAS	-	-
$n < 0$ and $R_c < R_0 < 1$	LAS	LAS*	Unstable*	-
$n < 0$ and $R_c = R_0 < 1$	LAS	-	-	LAS*

• LAS: Locally asymptotically stable; GAS: Globally asymptotically stable.

Firstly, we consider the following ordinary system with a parameter ψ:

$$\frac{\mathrm{d}X}{\mathrm{d}t} = f(X, \psi), \tag{7}$$

where $f : \mathbb{R}^n \times \mathbb{R} \to \mathbb{R}$ and $f \in C^2(\mathbb{R}^n \times \mathbb{R})$. Without loss of generality, we assume that 0 is an equilibrium of system (7)) with the parameter ψ,that is, $f(0, \psi) = 0$, for all ψ.

Lemma 1. *Assume that (Castillo-Chavez and Song [35]) :*

(H_1) $G = D_X f(0,0) = \frac{\partial f_i}{\partial X_i}(0,0)$ is the Jacobian matrix of system (7) around the equilibrium X=0. 0 is a simple eigenvalue of G and all other eigenvalues of G have negative real parts;

(H_2) A right eigenvector w and a left eigenvector v of matrix G , corresponding to zero eigenvalues, respectively.

Let f_k be the k^{th} component of f and

$$\alpha = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0),$$

$$b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \psi}(0,0).$$

Then, the local dynamics of system (7) around $X = 0$ are totally determined by α and b.

(1) $\alpha > 0$, $b > 0$. If $\psi < 0$ with $|\psi| \ll 1$, $X = 0$ is locally asymptotically stable and there exists a positive unstable equilibrium; if $0 < \psi \ll 1$, $X = 0$ is unstable and there exists a negative and locally asymptotically stable equilibrium;

(2) $\alpha < 0$, $b < 0$. If $\psi < 0$ with $|\psi| \ll 1$, $X = 0$ is unstable; if $0 < \psi \ll 1$, $X = 0$ is locally asymptotically stable and there exists a positive unstable equilibrium;

(3) $\alpha > 0$, $b < 0$. If $\psi < 0$ with $|\psi| \ll 1$, $X = 0$ is unstable and there exists a locally asymptotically stable negative equilibrium; if $0 < \psi \ll 1$, $X = 0$ is stable and a

positive unstable equilibrium appears;

(4) $\alpha < 0$, $b > 0$. When ψ changes from negative to positive, $X = 0$ changes its stability from stable to unstable. Correspondently, a negative unstable equilibrium becomes a locally asymptotically stable positive equilibrium.

Next, by using Lemma 1, we shall discuss the conditions under which system (1) undergoes backward bifurcation.

Theorem 6. *If $n < 0$, system (1) exhibits a backward bifurcation at $R_0 = 1$.*

Proof. Introducing change of variables: $S_n = x_1$, $S_a = x_2$, $E = x_3$, $I = x_4$, $R = x_5$, $W = x_6$. Further, using the vector notation $X = (x_1, x_2, x_3, x_4, x_5, x_6)^{\mathrm{T}}$, our system (1) can be rewritten in the form as $\frac{\mathrm{d}X}{\mathrm{d}t} = f(X)$ with $f(X) = (f_1, f_2, f_3, f_4, f_5, f_6)^{\mathrm{T}}$, where

$$
\begin{aligned}
f_1 &= A - (\beta_1 x_3 + \beta_2 x_4 + \beta_3 x_6)x_1 - a x_1 + \theta x_2 - \epsilon x_1,\\
f_2 &= a x_1 - (1-\sigma)(\beta_1 x_3 + \beta_2 x_4 + \beta_3 x_6)x_2 - (\theta + \epsilon)x_2,\\
f_3 &= (\beta_1 x_3 + \beta_2 x_4 + \beta_3 x_6)x_1 + (1-\sigma)(\beta_1 x_3 + \beta_2 x_4 + \beta_3 x_6)x_2 - (\delta + \epsilon)x_3,\\
f_4 &= \delta x_3 - (\gamma + \epsilon)x_4,\\
f_5 &= \gamma x_4 - \epsilon x_5,\\
f_6 &= \zeta(\eta_1 x_3 + \eta_2 x_4 - \mu x_6).
\end{aligned}
$$

We focus on the case where $R_0 = 1$, choosing β_2 as a bifurcation parameter. Solving for β_2 from the formula (3), we obtain

$$
\beta_2 = \beta_2^* = \frac{\epsilon(\delta+\epsilon)(\gamma+\epsilon)(a+\theta+\epsilon)}{\delta A[\theta+\epsilon+(1-\sigma)a]} - \beta_1\frac{(\gamma+\epsilon)}{\delta} - \beta_3\frac{\eta_1(\gamma+\epsilon)+\delta\eta_2}{\delta\mu}.
$$

Further, the Jacobian matrix $J(E_0, \beta_2^*)$ at E_0 is given as

$$
J(E_0,\beta_2^*) = \begin{pmatrix}
-a-\epsilon & \theta & -\beta_1 S_n^0 & -\beta_2^* S_n^0 & 0 & -\beta_3 S_n^0\\
a & -\theta-\epsilon & -(1-\sigma)\beta_1 S_a^0 & -(1-\sigma)\beta_2^* S_a^0 & 0 & -(1-\sigma)\beta_3 S_a^0\\
0 & 0 & a_1 & \beta_2^*[S_n^0 + (1-\sigma)S_a^0] & 0 & a_3\\
0 & 0 & \delta & -\gamma-\epsilon & 0 & 0\\
0 & 0 & 0 & \gamma & -\epsilon & 0\\
0 & 0 & \zeta\eta_1 & \zeta\eta_2 & 0 & -\zeta\mu
\end{pmatrix}.
$$

So we obtain that the Jacobian matrix $J(E_0, \beta_2^*)$ has a simple zero eigenvalue if and only if $R_0 = 1$ and all the other eigenvalues have negative real parts. Thus, E_0 is a nonhyperbolic equilibrium when $\beta_2 = \beta_2^*$. It is evident that model (1) satisfies assumption (H_1) in Lemma 1.

Assume that the right eigenvector of the matrix $J(E_0, \beta_2^*)$ is $w = (w_1, w_2, w_3, w_4, w_5, w_6)^{\mathrm{T}}$, which is given by

$$\begin{cases} (-a-\epsilon)w_1 + \theta w_2 - \beta_1 S_n^0 w_3 - \beta_2^* S_n^0 w_4 - \beta_3 S_n^0 w_6 = 0, \\ a w_1 - (\theta+\epsilon)w_2 - (1-\sigma)\beta_1 S_a^0 w_3 - (1-\sigma)\beta_2^* S_a^0 w_4 - (1-\sigma)\beta_3 S_a^0 w_6 = 0, \\ [\beta_1(S_n^0 + (1-\sigma)S_a^0) - \delta - \epsilon]w_3 + \beta_2^*(S_n^0 + (1-\sigma)S_a^0)w_4 + \beta_3(S_n^0 + (1-\sigma)S_a^0)w_6 = 0, \\ \delta w_3 - (\gamma+\epsilon)w_4 = 0, \\ \gamma w_4 - \epsilon w_5 = 0, \\ \zeta(\eta_1 w_3 + \eta_2 w_4 - \mu w_6) = 0. \end{cases} \tag{8}$$

Solving the equation (8), we get

$$\begin{aligned} w_1 &= \frac{(1-\sigma)\delta\epsilon h_1 S_a^0 - (\gamma+\epsilon)(\delta+\epsilon)(\theta+\epsilon)}{\delta\epsilon(a+\epsilon+\theta)} w_4, \\ w_2 &= \frac{\delta\epsilon h_1 S_n^0 - (\gamma+\epsilon)(\delta+\epsilon)(a+\epsilon)}{\delta\epsilon(a+\epsilon+\theta)} w_4, \\ w_3 &= \frac{(\gamma+\epsilon)}{\delta} w_4, \quad w_4 = w_4, \quad w_5 = \frac{\gamma}{\epsilon} w_4, \quad w_6 = \frac{\eta_1(\gamma+\epsilon) + \eta_2\delta}{\delta\mu} w_4, \end{aligned} \tag{9}$$

where

$$h_1 = \beta_1 \frac{(\gamma+\epsilon)}{\delta} + \beta_2^* + \beta_3 \frac{\eta_1(\gamma+\epsilon) + \eta_2\delta}{\delta\mu}.$$

By calculation, we have $h_1 = \frac{h}{R_0}$, then $h_1 = h$ when $R_0 = 1$. Additionally, the left eigenvector $v = (v_1, v_2, v_3, v_4, v_5, v_6)$ of the matrix $J(E_0, \beta_2^*)$, which satisfies $v \cdot w = 1$, is given by

$$\begin{cases} (-a-\epsilon)v_1 + a v_2 = 0, \\ \theta v_1 + (-\theta-\epsilon)v_2 = 0, \\ -\beta_1 S_n^0 v_1 - (1-\sigma)\beta_1 S_a^0 v_2 + [\beta_1(S_n^0 + (1-\sigma)S_a^0) - \delta - \epsilon]v_3 + \delta v_4 + \zeta\eta_1 v_6 = 0, \\ -\beta_2^* S_n^0 v_1 - (1-\sigma)\beta_2^* S_a^0 v_2 + \beta_2^*[S_n^0 + (1-\sigma)S_a^0]v_3 - (\gamma+\epsilon)v_4 + \gamma v_5 + \zeta\eta_2 v_6 = 0, \\ -\epsilon v_5 = 0, \\ -\beta_3 S_n^0 v_1 - (1-\sigma)\beta_3 S_a^0 v_2 + \beta_3[S_n^0 + (1-\sigma)S_a^0]v_3 - \zeta\mu v_6 = 0. \end{cases} \tag{10}$$

Solving the equation (10), we get

$$\begin{aligned} v_1 &= v_2 = v_5 = 0, \quad v_3 = v_3, \\ v_4 &= \frac{\delta\zeta\mu^2 - v_3 w_4[\zeta\mu^2(\gamma+\epsilon) + \beta_3(\eta_1(\gamma+\epsilon) + \eta_2\delta)(S_n^0 + (1-\sigma)S_a^0)]}{\delta\zeta w_4\mu^2}, \\ v_6 &= \frac{\beta_3[S_n^0 + (1-\sigma)S_a^0]}{\zeta\mu} v_3. \end{aligned} \tag{11}$$

Because v_1, v_2, and v_5 are zero and the second-order partial derivatives of f_4 and f_6 are zero, only nonzero partial derivatives of f_3 need to be calculated. Then, we obtain

$$\frac{\partial^2 f_3}{\partial x_1 \partial x_3}(E_0, \beta_2^*) = \beta_1, \quad \frac{\partial^2 f_3}{\partial x_1 \partial x_4}(E_0, \beta_2^*) = \beta_2^*, \quad \frac{\partial^2 f_3}{\partial x_1 \partial x_6}(E_0, \beta_2^*) = \beta_3,$$
$$\frac{\partial^2 f_3}{\partial x_2 \partial x_3}(E_0, \beta_2^*) = (1-\sigma)\beta_1, \quad \frac{\partial^2 f_3}{\partial x_2 \partial x_4}(E_0, \beta_2^*) = (1-\sigma)\beta_2^*,$$
$$\frac{\partial^2 f_3}{\partial x_2 \partial x_6}(E_0, \beta_2^*) = (1-\sigma)\beta_3, \quad \frac{\partial^2 f_3}{\partial x_4 \partial \beta_2}(E_0, \beta_2^*) = S_n^0 + (1-\sigma)S_a^0.$$

Next, we calculate the bifurcation coefficients α and b, it follows that

$$\begin{aligned}
\alpha &= \sum_{k,i,j=1}^{6} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(E_0, \beta_2^*) \\
&= 2v_3 w_1 w_3 \frac{\partial^2 f_3}{\partial x_1 \partial x_3}(E_0, \beta_2^*) + 2v_3 w_1 w_4 \frac{\partial^2 f_3}{\partial x_1 \partial x_4}(E_0, \beta_2^*) + 2v_3 w_1 w_6 \frac{\partial^2 f_3}{\partial x_1 \partial x_6}(E_0, \beta_2^*) \\
&\quad + 2v_3 w_2 w_3 \frac{\partial^2 f_3}{\partial x_2 \partial x_3}(E_0, \beta_2^*) + 2v_3 w_2 w_4 \frac{\partial^2 f_3}{\partial x_2 \partial x_4}(E_0, \beta_2^*) + 2v_3 w_2 w_6 \frac{\partial^2 f_3}{\partial x_2 \partial x_6}(E_0, \beta_2^*),
\end{aligned}$$

and

$$b = \sum_{k,i=1}^{6} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta_2}(E_0, \beta_2^*) = v_3 w_4 \frac{\partial^2 f_3}{\partial x_4 \partial \beta_2}(E_0, \beta_2^*).$$

In view of (9) and (11), we obtain

$$\begin{aligned}
\alpha &= 2v_3[\beta_1 w_1 w_3 + \beta_2^* w_1 w_4 + \beta_3 w_1 w_6 + (1-\sigma)(\beta_1 w_2 w_3 + \beta_2^* w_2 w_4 + \beta_3 w_2 w_6)] \\
&= 2v_3(\beta_1 w_3 + \beta_2^* w_4 + \beta_3 w_6)[w_1 + (1-\sigma)w_2] \\
&= \frac{2h}{\delta\epsilon(a+\epsilon+\theta)}[\frac{(1-\sigma)\delta\epsilon h(S_n^0 + S_a^0)}{(\gamma+\epsilon)(\delta+\epsilon)[\theta+\epsilon+(1-\sigma)(a+\epsilon)]} - 1] \\
&= \frac{2h}{\delta\epsilon(a+\epsilon+\theta)}[\frac{(1-\sigma)\delta A h^2}{(1-\sigma)\delta A h^2 + n} - 1],
\end{aligned} \tag{12}$$

and

$$b = S_n^0 + (1-\sigma)S_a^0 = \frac{A[\theta+\epsilon+(1-\sigma)a]}{\epsilon(\theta+\epsilon+a)}. \tag{13}$$

Obviously, b is positive. And from the expression (12), we can observe that if $n < 0$, we have $\alpha > 0$. Hence, it follows from Lemma 1 that system (1) occurs backward bifurcation at $R_0 = 1$ if $n < 0$.

The emergence of backward bifurcation makes the disease control strategy more challenging, the elimination of epidemics cannot be guaranteed even if R_0 is below 1. From the expressions (12) of the bifurcation coefficient α, we find that α decreases with the increase of the disease awareness impact factor σ, indicating that α is a decreasing function of σ. Hence, the possibility of backward bifurcation in system (1) is influenced by disease awareness, that is, it decreases with the increase of disease awareness.

5. Numerical simulation

In this section, we numerically verify the theoretical results and analyze the impact of disease awareness on disease spread. The values of parameters are provided in Table 1.

5.1. The influence of disease awareness on disease transmission

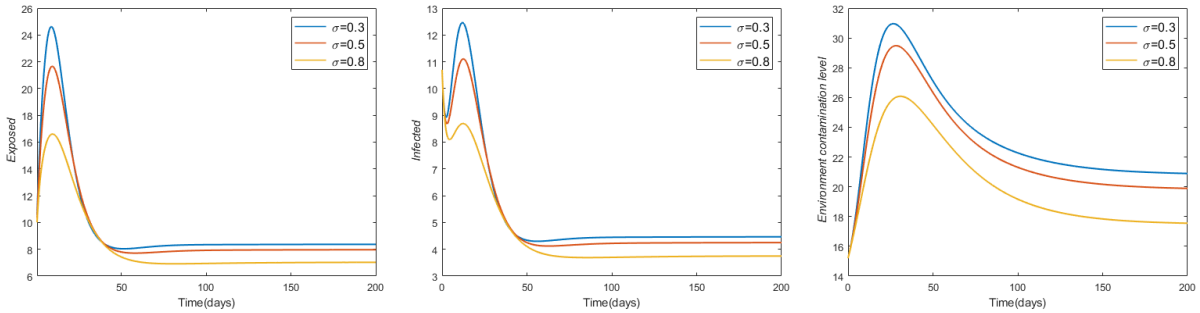


Figure 1: Variation in the number of exposed and infected people and virus concentration in the environment under different values of σ.

To assess the effect of disease awareness on both direct and indirect transmission, we set $A = 2$, $\beta_1 = 3 \times 10^{-3}$, $\beta_2 = 3.6 \times 10^{-3}$, $\beta_3 = 1.5 \times 10^{-3}$, $\delta = 0.16$, $\theta = 0.3$, $\gamma = 0.28$ and $\mu = 0.33$. The curves of the number of exposed and infected individuals and the virus concentration in the environment under different values of the disease awareness impact factor σ over time are plotted in Figure 1. As shown in Figure 1, the number of exposed individuals and infected individuals and the virus concentration in the environment all decrease significantly when the value of σ changes from 0.3 to 0.8. Moreover, the increased disease awareness impact factor σ decreases the peak level of the number of exposed individuals, infected individuals, and virus concentration. This illustrates that when people have a higher level of awareness, they are motivated to take preventive measures, thereby slowing virus transmission. Thus, increasing disease awareness helps prevent and control direct and indirect disease transmission.

5.2. The effect of environmental clearance and viral shedding on R_0

Set $A = 1$, $\beta_1 = 0.4 \times 10^{-3}$, $\beta_2 = 0.5 \times 10^{-3}$, $\beta_3 = 0.2 \times 10^{-3}$, $\delta = 0.35$, $\gamma = 0.26$ and $\sigma = 0.36$, the change of the basic reproduction number R_0 under various environmental clearance rates μ and various viral shedding rates η_i is drawn, as shown in Figure 2. It shows that as the environmental clearance rate μ increases and the viral shedding rate η_i $(i = 1, 2)$ decreases, R_0 decreases significantly (as shown in Figure 2(c-e)). This illustrates that strengthening environmental health management and improving the personal hygiene of exposed and infected people can effectively reduce disease transmission.

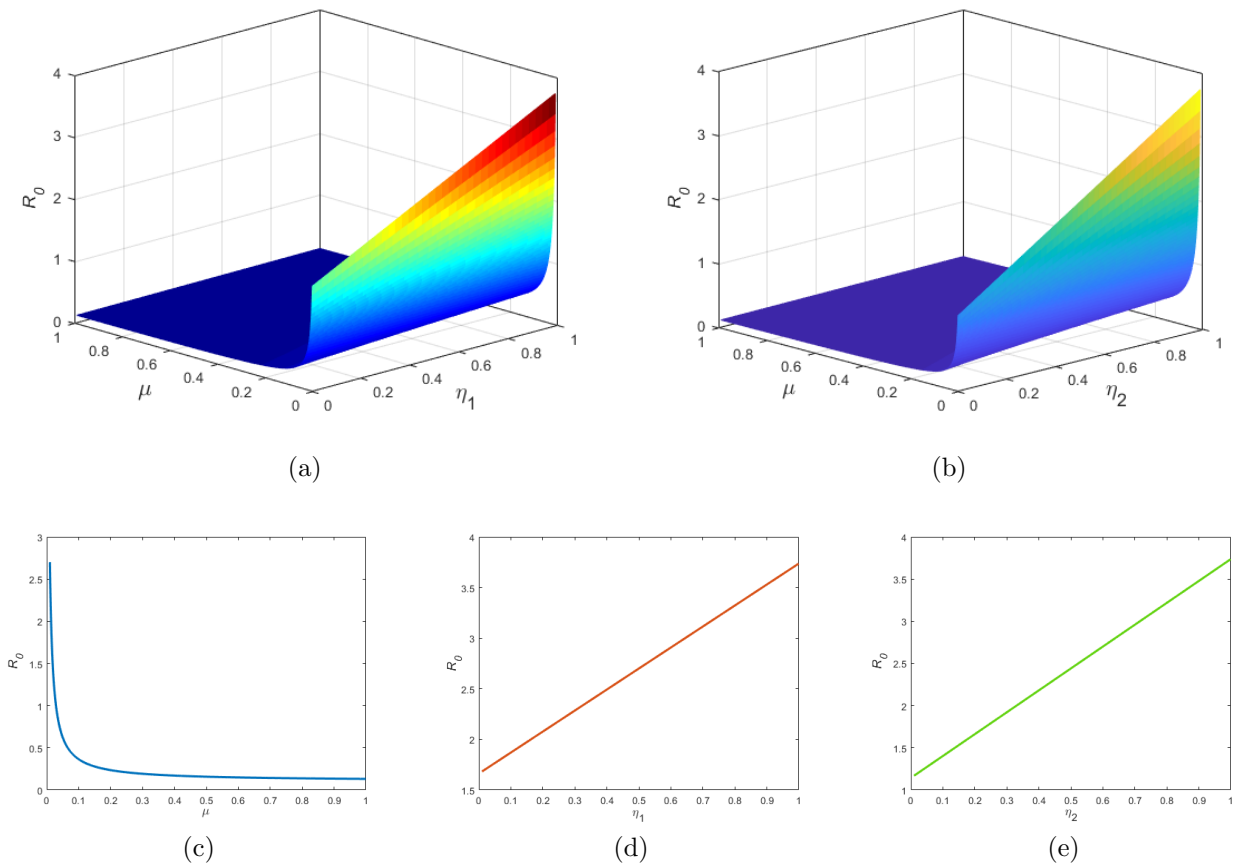
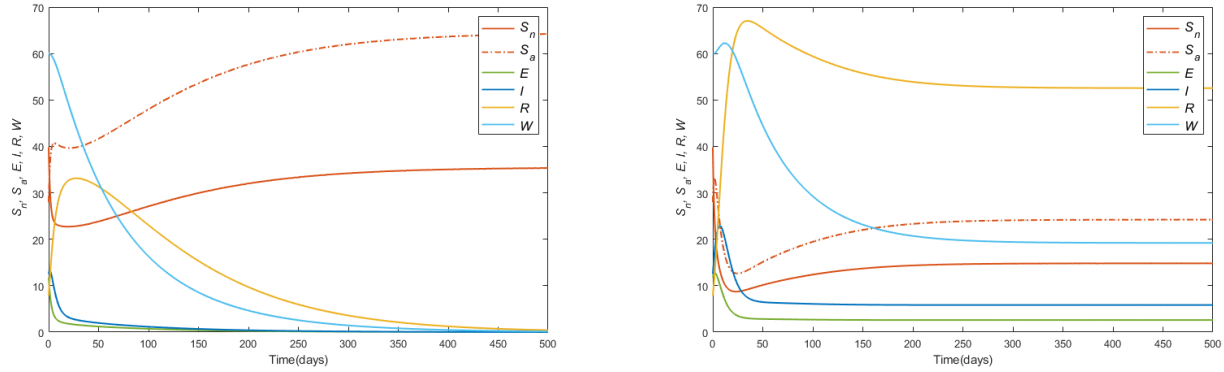


Figure 2: The impact of μ and η_i $(i = 1, 2)$ on R_0.

5.3. The stability of the equilibrium

Firstly, the stability of the disease-free equilibrium E_0 is verified. We take the parameter values $A = 2$, $\beta_1 = 0.4 \times 10^{-3}$, $\beta_2 = 0.5 \times 10^{-3}$, $\beta_3 = 0.3 \times 10^{-3}$, $\delta = 0.42$, $\gamma = 0.25$, $\sigma = 0.45$, $\theta = 0.2$ and $\mu = 0.23$, then we have $R_0^* = 0.6926 < 1$, $R_0 = 0.4915 < 1$ and $n = 1.0319 \times 10^{-4} > 0$, which obviously satisfies the condition that equilibrium E_0 global stability in Theorem 3. The simulation result is shown in Figure 3(a), where the number of exposed and infected individuals and the virus concentration decrease over time and eventually approach to 0. Thus, E_0 is globally asymptotically stable and the disease will become extinct when the condition $R_0^* < 1$ is satisfied.

Next, we verify the globally stability of the endemic equilibrium Q_1^* when $R_0 > 1$. Set $A = 2$, $\beta_1 = 2 \times 10^{-3}$, $\beta_2 = 3 \times 10^{-3}$, $\beta_3 = 1 \times 10^{-3}$, $\delta = 0.45$, $\gamma = 0.18$, $\sigma = 0.41$, $\theta = 0.2$ and $\mu = 0.25$, then we get $R_0 = 2.5272 > 1$, which satisfies the condition that equilibrium Q_1^* global stability in Theorem 5. As illustrated in Figure 3(b), exposed people, infected people, and viruses in the environment persist, while the number of unaware susceptible and aware susceptible decreases rapidly over time and then increases slowly. Finally, system (1) is stabilized to the unique endemic equilibrium Q_1^*, which verifies Theorem 5.



(a) (b)

Figure 3: (a) shows that E_0 is globally asymptotically stable if $n > 0$ and $R_0^* < 1$. (b) shows that Q_1^* is globally asymptotically stable if $R_0 > 1$.

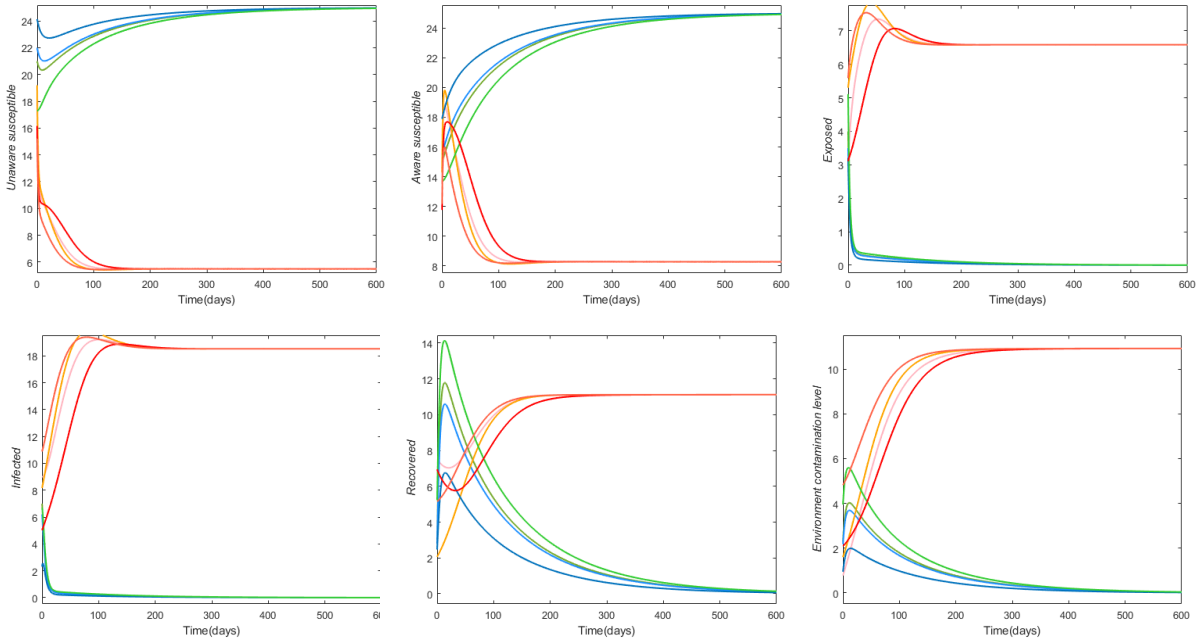
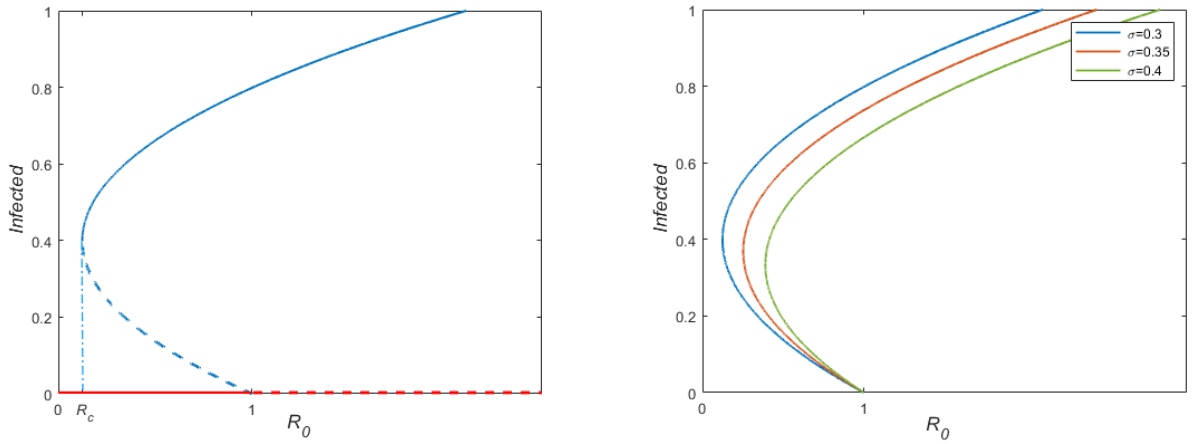


Figure 4: Taking different initial values, E_0 and Q_1^* are bi-stable when $n < 0$ and $R_c < R_0 < 1$.

Further, we verify that E_0 and Q_1^* are bistable for $R_c < R_0 < 1$. Set $A = 1$, $\beta_1 = 0.815 \times 10^{-3}$, $\beta_2 = 0.91 \times 10^{-3}$, $\beta_3 = 0.76 \times 10^{-3}$, $\sigma = 0.3$, $\delta = 0.36$, $\gamma = 0.28$ and $\mu = 0.23$, then we have $n = -4.0088 \times 10^{-5} < 0$, $R_0 = 0.6642 < 1$, $R_c = -0.1272 < 0$, which obviously satisfies the condition (2) in Theorem 1. Thus, we obtain two edemic equilibria $Q_1^*(5.4881, 8.2816, 6.5868, 18.5254, 11.1152, 10.9183)$ and $Q_2^*(19.7058, 17.2252, 1.3857, 1.6629, 10.0204, 7.3504)$. For Q_1^*, we have $B_1 = 0.9499$, $B_5 = 3.2117 \times 10^{-5}$, $B_1B_2 - B_3 = 0.2557$, $B_3(B_1B_2 - B_3) - B_1(B_1B_4 - B_5) = 0.0091$ and $(B_4B_3 - B_2B_5)(B_1B_2 - B_3) - B_1B_4(B_1B_4 - B_5) = 2.0024 \times 10^{-5}$. For Q_2^*, we have $B_1 = 0.7865$, $B_5 = 5.6351 \times 10^{-6}$, $B_1B_2 - B_3 = 0.1313$,

$B_3(B_1B_2 - B_3) - B_1(B_1B_4 - B_5) = -0.0017$ and $(B_4B_3 - B_2B_5)(B_1B_2 - B_3) - B_1B_4(B_1B_4 - B_5) = -7.5077 \times 10^{-7}$. Consequently, we conclude that system (1) possesses a locally asymptotically stable endemic equilibrium Q_1^* and an unstable endemic equilibrium Q_2^* if $n < 0$ and $R_c < R_0 < 1$. And as illustrated in Figure 4, the system (1) stabilizes to equilibrium Q_1^* and equilibrium E_0, which verifies Theorem 2 and Theorem 4 and shows that E_0 and Q_1^* are bistable when $n < 0$ and $R_c < R_0 < 1$.

5.4. Backward bifurcation



(a) (b)

Figure 5: Bifurcation diagram for different awareness impact factors. (a) shows the backward bifurcation diagram of system (1), where the dash curve represents unstable equilibrium while the solid curve represents stable equilibrium. (b) shows the influence of the value of different awareness impact factor σ on the backward bifurcation curve.

Let $A = 0.1$ $\beta_1 = 0.045$, $\beta_2 = 0.053$, $\beta_3 = 0.023$, $\sigma = 0.3$, $\delta = 0.42$, $\gamma = 0.45$, $\mu = 0.12$, we have $n = -0.0015 < 0$, $R_0 = 0.9248$, and $R_c = 0.1240$. It is evident that condition (2) in Theorem 1 and the condition that system (1) occurs backward bifurcation in Theorem 6 are satisfied, and the backward bifurcation diagram is depicted in Figure 5(a). We can observe that E_0 and Q_1^* are stable and Q_2^* is unstable when $n < 0$ and $R_c < R_0 < 1$ from Figure 5(a). Moreover, numerically, $E_0 = (1.4706, 3.5294, 0, 0, 0, 0)$, $Q_1^* = (0.5994, 0.4080, 0.8739, 0.7809, 2.3378, 0.2112)$ and $Q_2^* = (1.4151, 3.2141, 0.0196, 0.0175, 0.3337, 0.0047)$, where E_0 and Q_1^* satisfy the stability condition, while Q_2^* does not. Hence, E_0 and Q_1^* are bistable for $R_c < R_0 < 1$. As R_0 increases, when $R_0 > 1$, only the large endemic equilibrium Q_1^* exists and is stable, while the small endemic equilibrium Q_2^* does not exist and E_0 become unstable. The bifurcation diagram in Figure 5(a) verifies the conclusions of Theorem 1 and Theorem 6 and shows that when $R_c < R_0 < 1$, the disease will not be extinct.

In addition, Figure 5(b) shows that disease awareness has an impact on backward bifurcation of the model (1). Figure 5(b) shows that as awareness impact factor σ increases,

the interval of backward bifurcation decreases, thereby the R_0 interval in which the endemic equilibrium and the disease-free equilibrium showed bistability gets smaller, and the R_0 interval in which the disease-free equilibrium showed global stability gets greater. Moreover, when the awareness impact factor σ increases, the number of infected people at the stable endemic equilibrium also decreases. Therefore, it is necessary for society to strengthen health education and promote effective disease information to raise public disease awareness so that disease transmission can be effectively controlled.

6. Conclusion and discussion

In this paper, a coupled epidemic model with disease awareness is developed by considering both direct transmission between people and indirect transmission between people and the environment.

By analyzing the coupled model in detail, we find that it exhibits rich dynamic behaviors. The basic reproduction number R_0 is computed, which can be divided into three parts: secondary infections caused by direct transmission from exposed and infected individuals, and by indirect transmission from the contaminated environment. It is found that the disease-free equilibrium always exists, and system (1) has at most two endemic equilibria. By using the Routh-Hurwitz criterion and constructing Lyapunov functions, the local and global stability of the disease-free and endemic equilibrium under certain conditions are proved. By using center manifold theory, we verified that the system may undergo backward bifurcation, where the bi-stability phenomenon of disease-free equilibrium E_0 and endemic equilibrium Q_1^* can be observed.

Numerically, it is found that strengthening environmental clearance and reducing viral shedding from exposed and infected persons can reduce R_0 (as shown in Figure 2). This indicates that strengthening environmental health management, as well as regular environmental cleaning and disinfection, can reduce the spread and survival of viruses in the environment, thereby reducing new infections. Exposed and infected individuals could improve their personal hygiene and actively seek treatment to minimize environmental contamination, thereby reducing the risk of virus transmission. We also observed that the number of exposed and infected and the virus concentration in the environment show a decreasing trend with the increase of the disease awareness influence factor σ (as shown in Figure 1). Interestingly, increasing the value of σ decreases the backward regime of bifurcation curve so that the bistable interval decreases and the globally stable interval of disease-free equilibrium increases. Meanwhile, the number of infected people also decreases at the stable endemic equilibrium (as shown in Figure 5(b)). These findings indicate that enhancing the public's disease awareness can effectively reduce the spread of disease. Therefore, society should strengthen the publicity of disease information in various information channels to raise more people's disease awareness.

In summary, our findings show that disease awareness has a positive impact on controlling the direct and indirect transmission of infectious diseases. By enhancing disease awareness, the risk of direct and indirect transmission of diseases can be effectively reduced, which provides new insights into controlling disease transmission with multiple modes of transmission.

Meanwhile, this paper has shortcomings. Firstly, as the disease spreads, the intensity of media promotion may vary, and people may experience fatigue when facing the pandemic. Consequently, the rate of change in people's awareness, denoted as a, may no longer be a constant but rather a variable. However, if we treat a as a variable, we need to introduce an independent compartment in our model to represent the dynamic changes in disease information conveyed by media over time, allowing for a more accurate reflection of the impact of disease transmission on awareness acquisition. Therefore, such adjustments would significantly increase the complexity of the model and the difficulty of analysis. Secondly, random factors are common in disease transmission, which may also impact disease transmission and awareness acquisition. Therefore, it would be significant to develop a stochastic epidemic coupled model to further investigate how disease awareness affects both the direct and indirect spread of diseases. Based on these considerations, the model will become more complex, posing various challenges during the computation and analysis. We will further explore and address these issues in future research.

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References

[1] S. M. Levine and D. D. Marciniuk. Global impact of respiratory disease: What can we do, together, to make a difference? *Chest*, 161(5):1153–1154, 2022.

[2] J. D. Tamerius, J. Shaman, W. J. Alonso, et al. Environmental predictors of seasonal influenza epidemics across temperate and tropical climates. *PLoS Pathogens*, 9(3):e1003194, 2013.

[3] G. N. Sze-To, Y. Yang, J. K. C. Kwan, et al. Effects of surface material, ventilation, and human behavior on indirect contact transmission risk of respiratory infection. *Risk Analysis*, 34(5):818–830, 2014.

[4] N. H. L. Leung. Transmissibility and transmission of respiratory viruses. *Nature Reviews Microbiology*, 19(8):528–545, 2021.

[5] S. M. Weston and M. B. Frieman. Respiratory viruses. *Encyclopedia of Microbiology*, 2019.

[6] N. V. Doremalen, T. Bushmaker, D. H. Morris, et al. Aerosol and surface stability of SARS-CoV-2 as compared with SARS-CoV-1. *The New England Journal of Medicine*, 382(16):1564–1567, 2020.

[7] F. Xiao, M. W. Tang, X. B. Zheng, et al. Evidence for gastrointestinal infection of SARS-CoV-2. *Gastroenterology*, 158(6):1831–1833, 2020.

[8] J. C. Zhang, S. B. Wang, and Y. D. Xue. Fecal specimen diagnosis 2019 novel coronavirus–infected pneumonia. *Journal of Medical Virology*, 92(6):680–682, 2020.

[9] F. Colavita, D. Lapa, F. Carletti, et al. SARS-CoV-2 isolation from ocular secretions of a patient with COVID-19 in italy with prolonged viral RNA detection. *Annals of Internal Medicine*, 173(3):242–243, 2020.

[10] E. J. Nelson, J. B. Harris, J. Glenn Morris Jr, et al. Cholera transmission: the host, pathogen and bacteriophage dynamic. *Nature Reviews Microbiology*, 7(10):693–702, 2009.

[11] D. H. He, X. Y. Wang, D. Z. Gao, et al. Modeling the 2016–2017 Yemen Cholera outbreak with the impact of limited medical resources. *Journal of Theoretical Biology*, 451:80–85, 2018.

[12] D. Chac, C. N. Dunmire, J. Singh, et al. Update on environmental and host factors impacting the risk of Vibrio cholerae infection. *ACS Infectious Diseases*, 7(5):1010–1019, 2021.

[13] A. Rzeżutka and N. Cook. Survival of human enteric viruses in the environment and food. *FEMS Microbiology Reviews*, 28(4):441–453, 2004.

[14] Y. C. Bo, C. Song, J. F. Wang, et al. Using an autologistic regression model to identify spatial risk factors and spatial risk patterns of hand, foot and mouth disease (HFMD) in mainland China. *BMC Public Health*, 14:1–13, 2014.

[15] X. W. Li, X. Ni, S. Y. Qian, et al. Chinese guidelines for the diagnosis and treatment of hand, foot and mouth disease (2018 edition). *World Journal of Pediatrics*, 14(5):437–447, 2018.

[16] S. Funk, E. Gilad, C. Watkins, et al. The spread of awareness and its impact on epidemic outbreaks. *Proceedings of the National Academy of Sciences*, 106(16):6872–6877, 2009.

[17] G. O. Agaba, Y. N. Kyrychko, and K. B. Blyuss. Mathematical model for the impact of awareness on the dynamics of infectious diseases. *Mathematical Biosciences*, 286:22–30, 2017.

[18] S. R. Gani and S. V. Halawar. Optimal control for the spread of infectious disease: the role of awareness programs by media and antiviral treatment. *Optimal Control Applications and Methods*, 39(4):1407–1430, 2018.

[19] T. K. Kar, S. K. Nandi, S. Jana, et al. Stability and bifurcation analysis of an epidemic model with the effect of media. *Chaos, Solitons & Fractals*, 120:188–199, 2019.

[20] D. K. Das, S. Khajanchi, and T. K. Kar. The impact of the media awareness and optimal strategy on the prevalence of tuberculosis. *Applied Mathematics and Computation*, 366:124732, 2020.

[21] S. S. Shanta and M. H. A. Biswas. The impact of media awareness in controlling the spread of infectious diseases in terms of SIR model. *Mathematical Modelling of Engineering Problems*, 7(3), 2020.

[22] F. A. Basir, A. Banerjee, and S. Ray. Exploring the effects of awareness and time delay in controlling malaria disease propagation. *International Journal of Nonlinear Sciences and Numerical Simulation*, 22(6):665–683, 2021.

[23] A. A. Anteneh, Y. M. Bazezew, S. Palanisamy, et al. Mathematical model and analysis on the impact of awareness campaign and asymptomatic human immigrants in the transmission of COVID-19. *BioMed Research International*, 2022, 2022.

[24] D. Aldila. Optimal control for dengue eradication program under the media awareness effect. *International Journal of Nonlinear Sciences and Numerical Simulation*, 24(1):95–122, 2023.

[25] Z. L. Feng, J. Velasco-Hernandez, and B. Tapia-Santos. A mathematical model for coupling within-host and between-host dynamics in an environmentally-driven infectious disease. *Mathematical Biosciences*, 241(1):49–55, 2013.

[26] X. D. Sun and Y. N. Xiao. Multiscale system for environmentally-driven infectious disease with threshold control strategy. *International Journal of Bifurcation and Chaos*, 28(05):1850064:1–30, 2018.

[27] Y. N. Xiao, C. C. Xiang, R. A. Cheke, et al. Coupling the macroscale to the microscale in a spatiotemporal context to examine effects of spatial diffusion on disease transmission. *Bulletin of Mathematical Biology*, 82(5):58, 2020.

[28] Y. F. Xing, L. Zhang, and X. H. Wang. Modelling and stability of epidemic model with free-living pathogens growing in the environment. *Journal of Applied Analysis Computation*, 10(1):55–70, 2020.

[29] B. Musundi, J. Müller, and Z. L. Feng. A multi-scale model for Cholera outbreaks. *Mathematics*, 10(17):3114, 2022.

[30] X. Y. Wang, S. P. Wang, J. Wang, et al. A multiscale model of COVID-19 dynamics. *Bulletin of Mathematical Biology*, 84(9):99, 2022.

[31] A. Aili, Z. D. Teng, and L. Zhang. Dynamics in a disease transmission model coupled virus infection in host with incubation delay and environmental effects. *Journal of Applied Mathematics and Computing*, 68(6):4331–4359, 2022.

[32] O. Diekmann and J. A. P. Heesterbeek. Mathematical epidemiology of infectious diseases: model building, analysis and interpretation. *John Wiley Sons*, 2000.

[33] P. V. D. Driessche and J. Watmough. Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Mathematical Biosciences*, 180(1-2):29–48, 2002.

[34] J. P. La Salle. The stability of dynamical systems. *Regional Conference Series in Applied Mathematics*, 1976.

[35] C. Castillo-Chavez and B. J. Song. Dynamical models of tuberculosis and their applications. *Mathematical Biosciences and Engineering*, 1(2):361–404, 2004.

Appendix A. Locally stability of endemic equilibrium of system (1)

Appendix A.1. The endemic equilibrium Q_1^* of system (1) is locally asymptotically stable if $B_3(B_1B_2 - B_3) > B_1(B_1B_4 - B_5)$ and $(B_4B_3 - B_2B_5)(B_1B_2 - B_3) > (B_1B_4 - B_5)^2$.

Proof. Let $c_1 = -(1-\sigma)hI^* - \theta - \epsilon$, $c_2 = \beta_1[S_{n1}^* + (1-\sigma)S_{a1}^*] - \delta - \epsilon$, the Jacobian matrix of system (1) at the endemic equilibrium Q_1^* is given by

$$J(Q_1^*) = \begin{pmatrix} -hI^* - a - \epsilon & \theta & -\beta_1 S_{n1}^* & -\beta_2 S_{n1}^* & 0 & -\beta_3 S_{n1}^* \\ a & c_1 & -(1-\sigma)\beta_1 S_{a1}^* & -(1-\sigma)\beta_2 S_{a1}^* & 0 & -(1-\sigma)\beta_3 S_{a1}^* \\ hI_1^* & (1-\sigma)hI_1^* & c_2 & \beta_2[S_{n1}^* + (1-\sigma)S_{a1}^*] & 0 & \beta_3[S_{n1}^* + (1-\sigma)S_{a1}^*] \\ 0 & 0 & \delta & -\gamma - \epsilon & 0 & 0 \\ 0 & 0 & 0 & \gamma & -\epsilon & 0 \\ 0 & 0 & \zeta\eta_1 & \zeta\eta_2 & 0 & -\zeta\mu \end{pmatrix}.$$

Then, let

$$J(Q_1^*) = \begin{pmatrix} k_{11} & \theta & k_{13} & k_{14} & 0 & k_{15} \\ a & c_1 & k_{23} & k_{24} & 0 & k_{25} \\ hI_1^* & (1-\sigma)hI_1^* & c_2 & k_{34} & 0 & k_{35} \\ 0 & 0 & \delta & -\gamma - \epsilon & 0 & 0 \\ 0 & 0 & 0 & \gamma & -\epsilon & 0 \\ 0 & 0 & \zeta\eta_1 & \zeta\eta_2 & 0 & -\zeta\mu \end{pmatrix},$$

its characteristic equation is

$$(\lambda + \epsilon)(\lambda^5 + B_1\lambda^4 + B_2\lambda^3 + B_3\lambda^2 + B_4\lambda + B_5) = 0,$$

where

$$\begin{aligned} B_1 &= \zeta\mu + \gamma + \epsilon - (k_{11} + c_1 + k_{33}) \\ &= \zeta\mu + \gamma + \epsilon - (k_{11} + c_1) + (\delta + \epsilon)[1 - \beta_1\frac{(\gamma+\epsilon)}{\delta h}] > 0, \\ B_2 &= s_1 + \zeta\mu(\gamma+\epsilon) - (\zeta\mu + \gamma + \epsilon)(k_{11} + c_1 + c_2) - \zeta\eta_1 k_{35} - \delta k_{34} \\ &= s_1 + \zeta\mu(\gamma+\epsilon) - (\zeta\mu + \gamma + \epsilon)(k_{11} + c_1) + \zeta\mu(\delta+\epsilon)[1 - \beta_1\frac{(\gamma+\epsilon)}{\delta h} \\ &\quad - \beta_3\frac{\eta_1(\gamma+\epsilon)}{\delta h\mu}] + (\gamma+\epsilon)(\delta+\epsilon)[1 - \beta_1\frac{(\gamma+\epsilon)}{\delta h} - \frac{\beta_2}{h}] > 0, \end{aligned}$$

$$
\begin{aligned}
B_3 &= s_1(\zeta\mu+\gamma+\epsilon)+s_2+\zeta\eta_1 q_1+q_3\delta-k_{35}\zeta[\delta\eta_2+(\gamma+\epsilon)\eta_1]-k_{34}\delta\zeta\mu\\
&\quad-\zeta\mu(k_{11}+c_1+c_2)(\gamma+\epsilon)\\
&= s_2+(\zeta\mu+\gamma+\epsilon)[(hI_1^*+\epsilon)((1-\sigma)hI_1^*+\theta+\epsilon)+a((1-\sigma)hI_1^*+\epsilon)]\\
&\quad+\zeta\mu(a+\theta+2\epsilon)[(\delta+\epsilon)-(\beta_1+\beta_3\frac{\eta_1}{\mu})(S_{n1}^*+(1-\sigma)S_{a1}^*)]+[(\delta+\epsilon)\\
&\quad-(\beta_1+\beta_3\frac{\eta_1}{\mu})S_{n1}^*]\zeta\mu(1-\sigma)hI_1^*+\zeta\mu hI_1^*[(\delta+\epsilon)-(\beta_1+\beta_3\frac{\eta_1}{\mu})(1-\sigma)S_{a1}^*]\\
&\quad+(\gamma+\epsilon)(a+\theta+2\epsilon)[(\delta+\epsilon)-(\beta_1+\beta_2\frac{\delta}{(\gamma+\epsilon)})(S_{n1}^*+(1-\sigma)S_{a1}^*)]\\
&\quad+(\gamma+\epsilon)(1-\sigma)hI_1^*[(\delta+\epsilon)-(\beta_1+\beta_2\frac{\delta}{(\gamma+\epsilon)})S_{n1}^*]+(\gamma+\epsilon)hI_1^*[(\delta+\epsilon)\\
&\quad-(\beta_1+\beta_2\frac{\delta}{(\gamma+\epsilon)})(1-\sigma)S_{a1}^*]-\zeta\mu(k_{11}+c_1)(\gamma+\epsilon)>0,\\
B_4 &= s_1\zeta\mu(\gamma+\epsilon)+s_2(\zeta\mu+\gamma+\epsilon)+q_1\zeta[\delta\eta_2+(\gamma+\epsilon)\eta_1]+q_2\eta_1\zeta+\delta q_3\zeta\mu+\delta q_4\\
&= \zeta\mu(\gamma+\epsilon)[(hI_1^*+\epsilon)((1-\sigma)hI_1^*+\theta+\epsilon)+a((1-\sigma)hI_1^*+\epsilon)]+\zeta\mu(\gamma+\epsilon)hI_1^*[(\delta\\
&\quad+\epsilon)-\frac{\delta h}{(\gamma+\epsilon)}(1-\sigma)S_{a1}^*]+\zeta\mu(\gamma+\epsilon)(1-\sigma)[(\delta+\epsilon)-\frac{\delta h}{(\gamma+\epsilon)}S_{n1}^*]hI_1^*\\
&\quad+(\delta+\epsilon)hI_1^*[(1-\sigma)a+\theta+hI_1^*(1-\sigma)](\zeta\mu+\gamma+\epsilon)+\zeta\epsilon\mu(a+\theta+\epsilon)[(\delta+\epsilon)\\
&\quad-(\beta_1+\beta_3\frac{\eta_1}{\mu})(S_{n1}^*+(1-\sigma)S_{a1}^*)]+\zeta\epsilon\mu(1-\sigma)hI_1^*[(\delta+\epsilon)-(\beta_1+\beta_3\frac{\eta_1}{\mu})S_{n1}^*]\\
&\quad+\zeta\epsilon\mu hI_1^*[(\delta+\epsilon)-(\beta_1+\beta_3\frac{\eta_1}{\mu})(1-\sigma)S_{a1}^*]+\epsilon(\gamma+\epsilon)(a+\theta+\epsilon)[(\delta+\epsilon)\\
&\quad-(\beta_1+\beta_2\frac{\delta}{(\gamma+\epsilon)})(S_{n1}^*+(1-\sigma)S_{a1}^*)]+\epsilon(\gamma+\epsilon)(1-\sigma)hI_1^*[(\delta+\epsilon)\\
&\quad-(\beta_1+\beta_2\frac{\delta}{(\gamma+\epsilon)})S_{n1}^*]+\epsilon(\gamma+\epsilon)hI_1^*[(\delta+\epsilon)-(\beta_1+\beta_2\frac{\delta}{(\gamma+\epsilon)})(1-\sigma)S_{a1}^*]>0\\
B_5 &= s_2\zeta\mu(\gamma+\epsilon)+q_2\zeta[\delta\eta_2+(\gamma+\epsilon)\eta_1]+\delta q_4\zeta\mu\\
&= \zeta\mu(\gamma+\epsilon)(\delta+\epsilon)hI_1^*[(1-\sigma)a+\theta+hI_1^*(1-\sigma)]+\zeta\epsilon\mu(\gamma+\epsilon)hI_1^*[(\delta+\epsilon)\\
&\quad-\frac{\delta h}{(\gamma+\epsilon)}(1-\sigma)S_{a1}^*]+\zeta\epsilon\mu(\gamma+\epsilon)(1-\sigma)hI_1^*[(\delta+\epsilon)-\frac{\delta h}{(\gamma+\epsilon)}S_{n1}^*]>0,\\
s_1 &= k_{11}c_1+(k_{11}+c_1)c_2-k_{13}hI_1^*-k_{23}(1-\sigma)hI_1^*-a\theta,\\
s_2 &= c_1k_{13}hI_1^*+c_2\theta a+k_{11}k_{23}(1-\sigma)hI_1^*-\theta k_{23}hI_1^*-(1-\sigma)hI_1^*ak_{13}-k_{11}c_1c_2,\\
q_1 &= -\beta_3[S_{n1}^*+(1-\sigma)S_{a1}^*](a+\theta+2\epsilon)-(1-\sigma)\beta_3hI_1^*(S_{n1}^*+S_{a1}^*),\\
q_2 &= -\beta_3\epsilon[S_{n1}^*+(1-\sigma)S_{a1}^*](a+\theta+\epsilon)-(1-\sigma)\beta_3\epsilon hI_1^*(S_{n1}^*+S_{a1}^*),\\
q_3 &= -\beta_2[S_{n1}^*+(1-\sigma)S_{a1}^*](a+\theta+2\epsilon)-(1-\sigma)\beta_2hI_1^*(S_{n1}^*+S_{a1}^*),\\
q_4 &= -\beta_2\epsilon[S_{n1}^*+(1-\sigma)S_{a1}^*](a+\theta+\epsilon)-(1-\sigma)\beta_2\epsilon hI_1^*(S_{n1}^*+S_{a1}^*).
\end{aligned}
$$

By detailed calculation, we have $B_1B_2-B_3>0$. Thus, if the conditions $B_3(B_1B_2-B_3)>B_1(B_1B_4-B_5)$ and $(B_4B_3-B_2B_5)(B_1B_2-B_3)>(B_1B_4-B_5)^2$ are satisfied, then Q_1^* is locally asymptotically stable according to the Routh-Hurwitz criteria.