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A mathematical model on the impact of awareness and traditional medicine in the control of Ebola: case study of the 2014–2016 outbreaks in Sierra Leone and Liberia

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In the control of infectious diseases worldwide, awareness of the population occupies a prominent place. In Africa, there has been a long standing rivalry between traditional medicine and modern medicine. Any disease control strategy must take into account disease-oriented education, as this has a direct influence on the choice of treatment type to follow. In this work, we present a mathematical model that takes into consideration not only public health awareness but also the significant contribution of traditional medicine to the Ebola treatment effort. This study uses data from the 2014–2016 Ebola outbreaks in Sierra Leone and Liberia. Theoretically, we show that our model exhibits a trans-critical bifurcation at $\mathscr{R}_c = 1$ and a backward bifurcation phenomenon whenever $\mathscr{R}_c^c < \mathscr{R}_c < 1$. While the disease persists when $\mathscr{R}_c > 1$. In addition, a threshold number $\mathscr{T}_0$ is obtained, which ensures the global asymptotic stability of the disease-free equilibrium when its value is less than 1. Numerically, it is shown that the number of hospitalized infected cases increases more rapidly than the number of infected cases treated by traditional healers in both countries, suggesting that people have a high tendency to visit hospitals than visiting traditional healers. Our analysis reveals that during an Ebola outbreak, awareness messages should target the susceptible population for behaviour change in order to mitigate the spread of the disease.

Calibrating the model, it fits well the weekly cumulative cases in Sierra Leone and Liberia, and their corresponding estimated control reproduction numbers are 0.5725 and 0.8340, respectively.

Keywords: Ebola; Awareness; Traditional medicine; Modern medicine; Stability; sensitivity analysis.

1. Introduction

The Ebola virus is a filovirus that causes severe haemorrhagic fever in humans. It can be transmitted through close contacts with susceptible and bodily fluids of infected individuals (dead or alive) Mouanguissa *et al.* (2021). Once infected, individuals usually exhibit symptoms such as fever, severe headaches, muscle aches, weakness, vomiting, bloody diarrhea, stomach pains, loss of appetite and sometimes bleeding, which are often fatal to humans Wilkinson & Leach (2015); Agusto (2017). The Ebola virus disease (EVD) fatality rates range from 25% to 90% Pan *et al.* (2021), depending on the strain of the virus and the response measures put in place. No protocol treatment against EVD is approved by the World Health Organization (despite some protocol treatments approved by the US Food and Drug Administration El Ayoubi *et al.* (2024)), but early supportive care with rehydration and symptomatic treatments improve survival rate Conrad *et al.* (2016). A vaccine against this pathology is still in an experimental phase.

About 30 outbreaks of EVD have occurred in Africa since the virus was first discovered in the Democratic Republic of Congo (DRC) in 1976. However, several studies indicate that up to now, many people deny the existence of Ebola virus Buli *et al.* (2015); Njankou (2015) and some associate the disease to witchcraft, anger of the ancestors, and God's punishment for sins committed. Thus, many infected individuals seek care from traditional healers who use a combination of herbs and sometimes some rituals to heal their aches and pains Onyeneho et al. (2023). So, the place of traditional medicine, misconceptions and ignorance about EVD should not be neglected as far as EVD prevention and control is concerned. During EVD outbreaks, the choice between traditional medicine and modern medicine depends on the community and media resources available within the community. By media resources, we mean broadcast media (radio and tv), print media (newspapers and magazines), out of home media (public sensitization meeting) and social media (internet).

Awareness of the disease is contingent on the adherence of community members to information disseminated through the above media resources. This leads to behavioural change and consequently an informed choice on where to seek appropriate treatment in case of infection and how to handle Ebola-deceased individuals as they remain infectious even after death. Positive media outlets have the duty to inform people on EVD symptoms and the different modes of contracting the disease.

Since the devastating 2014–2016 EVD outbreak, an outfit of mathematical models have been developed to investigate the impact of the control strategies implemented against EVD Njankou (2015); Djiomba Njankou & Nyabadza (2017); Levy *et al.* (2017); Berge *et al.* (2018); Dautel & Agyingi (2021); Juga *et al.* (2021). In Djiomba Njankou & Nyabadza (2017), for instance, the authors developed a model on the role of media campaigns in the Ebola transmission dynamics. This role is represented by a linear decreasing function which acts on the force of infection. In this model, individuals of the different compartments circulate messages to the population. The authors numerically show the reducing effect of media campaigns on the number of infected individuals. The authors in Juga *et al.* (2021) propose a model with a fear dependent transmission rate as well as direct and indirect transmission of EVD through a polluted environment, in which the carrying capacity of the environment is taken into account. They show the existence of a forward and a backward bifurcations of their model and numerically determine the direction of these bifurcations. In addition, they fit their model to reported data for the 2018–2020 EVD outbreak in DR Congo and prove the decrease of the number of infected individuals as the level of fear from this pathology increases.

In Levy *et al.* (2017), the authors propose a model with the role of public health education during EVD outbreaks. In this model, the transmission rates of infected individuals depend on whether they are aware or not. These authors compare the number of cases reported during two consecutive outbreaks in Sudan and posit that the second outbreak produced far fewer cases than the first because the population of this country learned from the first outbreak and changed her behaviour. Their results highlight the importance of public health education in mitigating EVD outbreaks.

In light of their paper, we want to make a contribution by taking into consideration the role of traditional medicine in the treatment of EVD. Our motivation lies in the fact that many infected individuals who are not well educated (unaware) about the disease would prefer traditional medicine. We present a mathematical model which takes into account the following:

(a) the use of traditional and modern medicines in the treatment of EVD patients;

(b) the fact that some unaware susceptible and infected individuals become aware and choose between traditional medicine (seeing a traditional healer) and modern medicine (going to hospital);

(c) the increase in transmission rate due to unawareness.

Our model focuses on addressing the following key research questions.

i How to assess the effect of the awareness of the population on the dynamics of EVD.

ii Which kind of medicine (traditional or modern medicine) is able to eliminate the disease.

iii Does traditional medicine contribute to the mitigation of EVD outbreaks ?

We consider data reported in Sierra Leone and Liberia on the outbreaks of the EVD epidemic between 2014 and 2016 WHO (2015). The data provided by the Centers for Disease Control and Prevention, is a daily cumulative number of infected cases. Due to error in reporting on the data in Sierra Leone, we took 27 May 2014 as the starting date in the model fitting process, because as of that date, there was at least one infected case reported in the two countries.

The rest of this paper is organized as follows: in Section 2, the model is formulated with details. The theoretical and in-depth analyses of the model are presented in Section 3. Section 4 focuses on model calibration and parameters estimation. The sensitivity analysis of the model is performed in Section 6. The discussion on the results of the paper is given in Section 7.

2. Model formulation

2.1 *Main assumptions and model variables*

Throughout this paper, we assume the following: The hospitals (Ebola treatment units (ETUs)) are isolation centres and all the hospitalized people stay in hospital until they recover or die. Ebola-infected corpses in ETUs are properly handled by well-trained personnel and the risk of transmission is negligible. Ebola-deceased individuals out of hospitals and ETUs remain highly infectious. The population is divided into two groups: aware/educated and unaware/uneducated about EVD, subscripts 1 and 2 represent the population groups, respectively. We consider the following mutually exclusive classes:

- $S_1(t)$: susceptible aware/educated category. The individuals in this category are well informed on the natural existence of Ebola virus and are able to identify EVD symptoms. This knowledge helps them to avoid close contacts with suspected EVD cases.
- $S_2(t)$: susceptible who are unaware/uneducated. These individuals have mitigated opinions about EVD. This class includes those who are ignorant of how the Ebola virus is transmitted. In addition,

some of the members here think that EVD is due to witchcraft or is spiritual and hence only traditional healers can cure the disease Cénat *et al.* (2021).

- $I_1(t)$: infected aware/educated individuals. It is assumed that this category of infected people will prefer modern treatment.
- $I_2(t)$: infected unaware/uneducated individuals. Since they have mitigated opinions on EVD, some of them will prefer traditional medicine while others will choose modern medicine.
- $H(t)$: infected individuals who are in hospital, receiving modern medicine for their treatment.
- $U(t)$: infected individuals who are followed up by traditional healers.
- $D(t)$: Ebola-deceased individuals buried without safety measures. The corpses can transmit EVD during traditional funeral and mourning ceremonies.
- $R(t)$: recovered individuals. The people who recover from EVD and are assumed immune to the disease.
- $N(t) = S_1(t) + I_1(t) + S_2(t) + I_2(t) + U(t) + H(t) + R(t)$ is the total population alive at time t.

2.2 *Equations of the model*

The susceptible population is recruited at a rate π. Among these recruited individuals, a proportion p are aware about EVD. The susceptible individuals can be infected by people of classes I_1, I_2, H, U and D at constant rates β, $\nu_2\beta$, $\nu_1\beta$, $\nu_3\beta$ and $\nu_4\beta$, respectively. However, people having more comprehensive knowledge on EVD, limit contacts with suspected Ebola individuals since they can easily identify symptoms of the disease. This mitigates their effective transmission rate by a factor $(1 - \varepsilon)$, where ε measures the efficiency of awareness campaigns. Due to these campaigns, a proportion σ_s of susceptible individuals of compartment S_2 becomes aware about the disease and leaves that compartment to compartment S_1. Once infected, they enter in compartment I_1 and I_2, depending on whether they are aware or not. Individuals in compartment I_1, can either go to hospital at rate η_1, recover at rate γ_1 (those who present benign symptoms) or decease at rate $(\mu + \delta_1)$, where μ is the natural mortality rate and δ_1, the mortality rate due to EVD. As individuals in compartment I_2 have some doubts about the existence or non-existence of EVD, a proportion $q\eta_2$ will choose hospital, while the remaining proportion $(1 - q)\eta_2$ will choose traditional medicine. In compartment I_2, one can recover at rate γ_2, decease at rate $(\mu + \delta_2)$ (where δ_2 is the death rate due to EVD) or become aware of the existence of EVD at rate σ_i due to sensitization campaigns. In fact, we assume that the spread of information will lead to awareness of some uneducated infected persons who will change their behaviours first (that is move from compartment I_2 to compartment I_1) before going to hospital. Infected individuals receiving traditional medicine recovers at rate γ_u, decease at rate $(\mu + \delta_u)$ (where δ_u is the death rate due to EVD in compartment U) or become aware and therefore go to hospital at rate σ_u. In hospital, an infected patient recovers at rate γ_h or succumbs to the disease at rate $(\mu + \delta_h)$ (where δ_h is the death rate due to EVD in compartment H). The Ebola deceased persons are buried at rate b.

Based on the main assumptions presented in Section 2.1, infected individuals in compartments I_1, H, I_2, U and D contribute to the force of infection. The infected individuals in compartment I_1 mix freely with the population, while those in compartments H, I_2, U and D have some degree of restrictions in mixing with the population measured by the parameters (contact rates) ν_1, ν_2, ν_3 and ν_4, respectively. For I_1 in particular, the corresponding parameter is assumed to be $\nu_0 = 1$. The probability that a contact between a susceptible and an infectious individual results in an infection is β. Therefore, the force of infection is given by $\lambda(t) = \dfrac{\beta(I_1(t) + \nu_1 H(t) + \nu_2 I_2(t) + \nu_3 U(t) + \nu_4 D(t))}{N(t)}$.

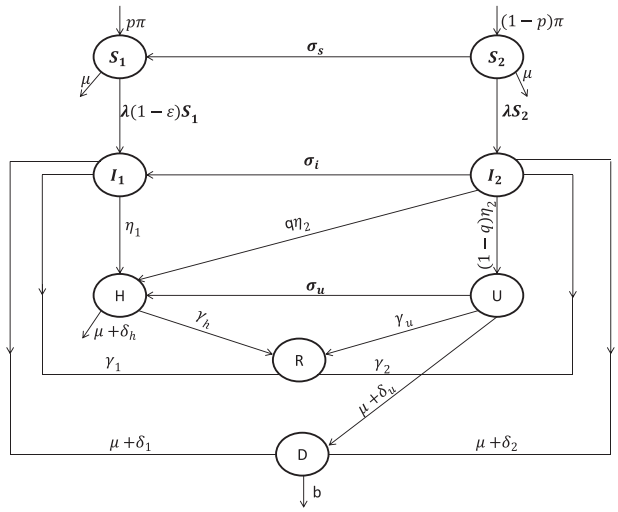

FIG. 1. Flow diagram for Model (2.1).

This information is summarized in the flow diagram for the model given in Fig. 1.

$$\begin{cases} \dot{S}_1(t) &= p\pi + \sigma_s S_2 - \lambda(1-\varepsilon)S_1 - \mu S_1, \\ \dot{S}_2(t) &= (1-p)\pi - \lambda S_2 - (\mu+\sigma_s)S_2, \\ \dot{I}_1(t) &= \lambda(1-\varepsilon)S_1 + \sigma_i I_2 - (\mu+\delta_1+\gamma_1+\eta_1)I_1, \\ \dot{I}_2(t) &= \lambda S_2 - (\mu+\sigma_i+\delta_2+\gamma_2+\eta_2)I_2, \\ \dot{H}(t) &= \eta_1 I_1 + q\eta_2 I_2 + \sigma_u U - (\mu+\delta_h+\gamma_h)H, \\ \dot{U}(t) &= (1-q)\eta_2 I_2 - (\mu+\sigma_u+\delta_u+\gamma_u)U, \\ \dot{D}(t) &= (\mu+\delta_1)I_1 + (\mu+\delta_2)I_2 + (\mu+\delta_u)U - bD, \\ \dot{R}(t) &= \gamma_1 I_1 + \gamma_2 I_2 + \gamma_u U + \gamma_h H - \mu R. \end{cases} \tag{2.1}$$

3. Analytical results

In this section, we study the well-posedness, the existence and stability of equilibria, the uniform persistence and the bifurcations of Model (2.1). To simplify our notations, we define the following parameters:

$$\begin{cases} \theta_1 &= p\pi, \quad \theta_2 = (1-p)\pi, \quad \theta_3 = 1-\varepsilon \\ \tau_s &= (\mu+\sigma_s), \quad \tau_u = \mu+\delta_u, \quad \tau_1 = \mu+\delta_1, \quad \tau_2 = \mu+\delta_2 \\ \phi_1 &= (\mu+\delta_1+\gamma_1+\eta_1), \quad \phi_u = \mu+\sigma_u+\delta_u+\gamma_u \\ \phi_2 &= (\mu+\sigma_i+\delta_2+\gamma_2+\eta_2), \quad \phi_h = \mu+\delta_h+\gamma_h \end{cases} \tag{3.1}$$

3.1 *Well-posedness and existence of equilibria for the model*

The following propositions ensure the well-posedness of Model (2.1), in the sense that, for any positive initial condition, there exists a unique global positive solution. The proofs of propositions 1 and 2 are given in the Appendix.

TABLE 1 *Parameters and their epidemiological interpretations*

Parameter	Epidemiological interpretation
β	Probability that a contact between a susceptible and an infective leads to an infection
$\nu_1, \nu_2, \nu_3, \nu_4$	Contact rates for infected individuals in compartments H, I_2, U and D, respectively.
ε	Measure of precaution taken by a susceptible in compartment S_1 due to awareness.
π	Recruitment rate in the susceptible population.
p	Proportion of informed susceptible individuals recruited in the population.
η_1	Hospitalization rate of informed infected individuals.
η_2	Exit rate from compartment I_2 to compartment H or U.
$q\eta_2$	Rate of infected unaware persons who go to hospital.
σ_u	Exit rate from compartment U to compartment H.
σ_i	Impact of awareness on the infected persons in compartment I_2.
σ_s	Impact of awareness on the susceptible in class S_2.
$1/b$	Average time from death to burial.
π	Recruitment rate in the susceptible population.
δ_1, δ_2	Death rates due to EVD in compartments I_1 and I_2, respectively.
δ_u, δ_h	Death rates due to EVD in compartments U and H, respectively.
γ_1, γ_2	Recovery rates in compartments I_1 and I_2, respectively.
γ_u, γ_h	Recovery rates, receiving traditional and modern treatments, respectively.
μ	Natural mortality rate.

PROPOSITION 1. The orthant $\mathbb{R}^8_+$ is positively invariant under the flow of Model (2.1). That is, if

$$S_1(0) > 0, \quad S_2(0) > 0, \quad I_1(0) \geq 0, \quad I_2(0) \geq 0, H(0) \geq 0, \quad U(0) \geq 0, \quad D(0) \geq 0, \quad R(0) \geq 0,$$

then for all $t > 0$,

$$S_1(t) > 0, \quad S_2(t), \quad I_1(t) \geq 0, \quad I_2(t) \geq 0, \quad H(t) \geq 0, \quad U(t) \geq 0, \quad D(t) \geq 0, \quad R(t) \geq 0.$$

PROPOSITION 2. Suppose the initial conditions for System (2.1) are non-negative.

If

$$N(0) \leq \frac{\pi}{\mu} \quad \text{and} \quad D(0) \leq \frac{\pi(3\mu + \delta_1 + \delta_2 + \delta_u)}{\mu b},$$

then

$$\forall\, t > 0,\ N(t) \leq \frac{\pi}{\mu} \quad \text{and} \quad D(t) \leq \frac{\pi(3\mu + \delta_1 + \delta_2 + \delta_u)}{\mu b}.$$

Furthermore, Model (2.1) is a dynamical system on

$$\Omega : = \left\{(S_1, S_2, I_1(t), I_2, H, U, D, R) \in \mathbb{R}^8_+ / N \leq \frac{\pi}{\mu}; \quad D \leq \frac{\pi(3\mu + \delta_1 + \delta_2 + \delta_u)}{\mu b}\right\}.$$

In the study of the existence of equilibria for Model (2.1), again we define the following parameters:

$$\begin{cases} A_1 = \theta_1\theta_3\phi_2\tau_s + \sigma_s\theta_2\theta_3\phi_2 + \sigma_i\theta_2\mu, \quad A_2 = \theta_1\theta_3\phi_2 + \sigma_i\theta_2\theta_3, \\ B_1 = \eta_1\theta_3\phi_2\theta_1\tau_s\phi_u + \eta_1\theta_3\theta_2\phi_2\phi_u\sigma_s + \eta_1\sigma_i\theta_2\phi_u\mu + q\eta_2\theta_2\phi_1\phi_u\mu + \sigma_u(1-q)\theta_2\phi_1\mu\eta_2, \\ B_2 = \eta_1\theta_3\theta_1\phi_2\phi_u + \eta_1\sigma_i\theta_2\theta_3\phi_u + q\eta_2\theta_2\theta_3\phi_1\phi_u + \sigma_u(1-q)\theta_2\theta_3\phi_1\eta_2, \\ C_1 = \tau_1\tau_s\theta_1\theta_3\phi_2\phi_u + \tau_1\theta_2\theta_3\phi_2\phi_u\sigma_s + \tau_1\sigma_i\theta_2\phi_u\mu + \tau_2\theta_2\phi_1\phi_u\mu + \phi_1\tau_u(1-q)\eta_2\theta_2\mu, \\ C_2 = \tau_1\theta_1\theta_3\phi_2\phi_u + \tau_1\sigma_i\theta_2\theta_3\phi_u + \tau_2\theta_2\theta_3\phi_1\phi_u + \phi_1\tau_u(1-q)\eta_2\theta_2\theta_3, \\ E_1 = \gamma_1A_1\phi_h\phi_u + \gamma_2\theta_2\phi_1\phi_u\phi_h\mu + \gamma_u(1-q)\eta_2\theta_2\phi_1\phi_h\mu + \gamma_hB_1, \\ E_2 = \gamma_1A_2\phi_h\phi_u + \gamma_hB_2 + \gamma_2\theta_2\theta_3\phi_1\phi_u\phi_h + \gamma_u(1-q)\eta_2\theta_2\theta_3\phi_1\phi_h, \\ F_0 = (\sigma_s\theta_2 + \theta_1\tau_s)\mu\phi_1\phi_2\phi_h\phi_u + \theta_2\mu^2\phi_1\phi_2\phi_u\phi_h, \\ F_1 = \theta_1\mu\phi_1\phi_2\phi_h\phi_u + A_1\mu\phi_h\phi_u + B_1\mu + E_1 + \theta_2\theta_3\mu\phi_1\phi_2\phi_u\phi_h + \theta_2\mu^2\phi_1\phi_h\phi_u \\ \qquad +(1-q)\eta_2\theta_2\mu^2\phi_1\phi_h, \\ F_2 = A_2\mu\phi_u\phi_h + B_2\mu + E_2 + \theta_2\theta_3\mu\phi_1\phi_h\phi_u + (1-q)\eta_2\theta_2\theta_3\phi_1\phi_h\mu, \\ G_0 = bF_0 - \beta(A_1\phi_u\phi_h\mu b + \nu_1bB_1\mu + \nu_4\phi_hC_1\mu + b\nu_2\theta_2\phi_1\phi_h\phi_u\mu^2 + \eta_2\nu_3(1-q)\theta_2\phi_1\phi_h\mu^2b), \\ G_1 = bF_1 - \beta(A_2\phi_u\phi_h\mu b + \nu_1bB_2\mu + \nu_4\phi_hC_2\mu + b\nu_2\theta_2\theta_3\phi_1\phi_h\phi_u\mu + \eta_2\nu_3(1-q)\theta_2\theta_3\phi_1\phi_h\mu b), \\ G_2 = bF_2. \end{cases} \tag{3.2}$$

An equilibrium $E^* = (S_1^*, S_2^*, I_1^*, I_2^*, H^*, U^*, D^*, R^*)$ of System (2.1) satisfies the following relations:

$$\begin{cases} \theta_1 + \sigma_sS_2^* - \lambda^*\theta_3S_1^* - \mu S_1^* = 0, \\ \theta_2 - \lambda^*S_2^* - \tau_sS_2^* = 0, \\ \lambda^*\theta_3S_1^* + \sigma_iI_2^* - \phi_1I_1^* = 0, \\ \lambda^*S_2^* - \phi_2I_2^* = 0, \\ \eta_1I_1^* + q\eta_2I_2^* + \sigma_uU^* - \phi_hH^* = 0, \\ (1-q)\eta_2I_2^* - \phi_uU^* = 0, \\ \tau_1I_1^* + \tau_2I_2^* + \tau_uU^* - bD^* = 0, \\ \gamma_1I_1^* + \gamma_2I_2^* + \gamma_uU^* + \gamma_hH^* - \mu R^* = 0, \end{cases} \tag{3.3}$$

with

$$\lambda^* = \frac{\beta(I_1^* + \nu_1H^* + \nu_2I_2^* + \nu_3U^* + \nu_4D^*)}{N^*}. \tag{3.4}$$

From System (3.3), we get

$$\begin{cases} S_2^* = \dfrac{\theta_2}{\tau_s + \lambda^*}, \quad S_1^* = \dfrac{\theta_1(\tau_s + \lambda^*) + \sigma_s\theta_2}{(\theta_3\lambda^* + \mu)(\tau_s + \lambda^*)}, \quad I_2^* = \dfrac{\lambda^*\theta_2}{\phi_2(\tau_s + \lambda^*)} \\ U^* = \dfrac{(1-q)\eta_2\theta_2\lambda^*}{\phi_u\phi_2(\tau_s + \lambda^*)}, \quad I_1^* = \dfrac{A_1\lambda^* + A_2\lambda^{*2}}{\phi_1\phi_2(\theta_3\lambda^* + \mu)(\tau_s + \lambda^*)} \\ H^* = \dfrac{B_1\lambda^* + B_2\lambda^{*2}}{\phi_1\phi_2\phi_h\phi_u(\theta_3\lambda^* + \mu)(\tau_s + \lambda^*)}, \quad D^* = \dfrac{C_1\lambda^* + C_2\lambda^{*2}}{b\phi_1\phi_2\phi_u(\theta_3\lambda^* + \mu)(\tau_s + \lambda^*)} \\ R^* = \dfrac{E_1\lambda^* + E_2\lambda^{*2}}{\mu\phi_1\phi_2\phi_h\phi_u(\theta_3\lambda^* + \mu)(\tau_s + \lambda^*)}. \end{cases} \tag{3.5}$$

Moreover,

$$N^* = \frac{F_0 + F_1\lambda^* + F_2\lambda^{*2}}{\mu\phi_1\phi_2\phi_h\phi_u(\theta_3\lambda^* + \mu)(\tau_s + \lambda^*)}.$$

Thus, from Equation (3.4), we have

$$N^*\lambda^* = \beta\left[\frac{A_1\lambda^* + A_2\lambda^{*2}}{\phi_1\phi_2(\theta_3\lambda^* + \mu)(\tau_s + \lambda^*)} + \frac{\nu_1(B_1\lambda^* + B_2\lambda^{*2})}{\phi_1\phi_2\phi_h\phi_u(\theta_3\lambda^* + \mu)(\tau_s + \lambda^*)} + \frac{\nu_2\lambda^*\theta_2}{\phi_2(\tau_s + \lambda^*)}\right.$$
$$\left. + \frac{\nu_3(1-q)\eta_2\theta_2\lambda^*}{\phi_u\phi_2(\tau_s + \lambda^*)} + \frac{\nu_4(C_1\lambda^* + C_2\lambda^{*2})}{b\phi_1\phi_2\phi_u(\theta_3\lambda^* + \mu)(\tau_s + \lambda^*)}\right].$$

which is equivalent to

$$\lambda^*[G_2\lambda^{*2} + G_1\lambda^* - G_0] = 0. \tag{3.6}$$

Therefore, $\lambda^* = 0$ is a trivial solution of Equation (3.6), which corresponds to the disease–free equilibrium. Any endemic equilibrium (EE) corresponds to the positive solution λ^* of the quadratic equation $G_2\lambda^{*2} + G_1\lambda^* - G_0 = 0$. In order to investigate its existence, we set

$$\mathscr{R}_c = \frac{\beta(A_1\phi_u\phi_h\mu b + \nu_1 bB_1\mu + \nu_4\phi_h C_1\mu + b\nu_2\theta_2\phi_1\phi_h\phi_u\mu^2 + \eta_2\nu_3(1-q)\theta_2\phi_1\phi_h\mu^2 b)}{bF_0}, \tag{3.7}$$

so that G_0 can be written as

$$G_0 = bF_0\left[\mathscr{R}_c - 1\right].$$

When $\mathscr{R}_c > 1$, there exists exactly one positive root, which corresponds to the unique EE of System (2.1). When $\mathscr{R}_c < 1$, we can either have no EE or two endemic equilibria.

Remark 1. The quantity $\mathscr{R}_c$ defined in (3.7) is actually the control reproduction number of System (2.1). This can be readily verified by applying the method in Van den Driessche & Watmough (2002), in which $\mathscr{R}_c$ is the spectral radius of the matrix FV^{-1}, where F and V are given later in (3.13).

In order to verify the existence of endemic equilibria, we define the following parameters:

$$F_3 = A_2\phi_u\phi_h\mu b + \nu_1 bB_2\mu + \nu_4\phi_h C_2\mu + b\nu_2\theta_2\theta_3\phi_1\phi_h\phi_u\mu + \eta_2\nu_3(1-q)\theta_2\theta_3\phi_1\phi_h\mu b$$
$$F_4 = A_1\phi_u\phi_h\mu b + \nu_1 bB_1\mu + \nu_4\phi_h C_1\mu + b\nu_2\theta_2\phi_1\phi_h\phi_u\mu^2 + \eta_2\nu_3(1-q)\theta_2\phi_1\phi_h\mu^2 b.$$

so that $\mathscr{R}_c = \dfrac{\beta F_4}{bF_0}$, $G_0 = \beta F_4 - bF_0$ and $G_1 = bF_1 - \beta F_3$.

The discriminant $\Delta(\mathscr{R}_c)$ of the polynomial $G_2\lambda^{*2} + G_1\lambda^* - G_0$ is given by

$$\Delta(\mathscr{R}_c) = G_1^2 + 4G_2G_0 = G_1^2 + 4G_2bF_0(\mathscr{R}_c - 1).$$

It follows that the unique solution of $\Delta(\mathscr{R}_c) = 0$ is

$$\mathscr{R}_c = 1 - \frac{G_1^2}{4G_2 bF_0} := \mathscr{R}_c^c.$$

Therefore, $\Delta(\mathscr{R}_c)$ is negative for $\mathscr{R}_c < \mathscr{R}_c^c$ and positive for $\mathscr{R}_c^c < \mathscr{R}_c < 1$. This can be summarized in Theorem 1 that follows.

THEOREM 1. The following statements hold:

(a) If $\mathscr{R}_c < \mathscr{R}_c^c < 1$, the Model (2.1) has only one equilibrium denoted by $E_0 = (S_{10}, S_{20}, 0, 0, 0, 0, 0, 0)$. This corresponds to the disease-free equilibrium, where

$$S_{10} = \frac{\pi(p(\mu + \sigma_s) + \sigma_s(1-p))}{\mu(\mu + \sigma_s)}, \quad S_{20} = \frac{(1-p)\pi}{\mu + \sigma_s}.$$

(b) If $\mathscr{R}_c^c < \mathscr{R}_c < 1$, we have the DFE and two endemic equilibria, which can be denoted by E_1^* and E_2^*.

(c) If either $\mathscr{R}_c = 1$ and $bF_1 < \beta F_3$ or $\mathscr{R}_c = \mathscr{R}_c^c$ and $bF_1 < \beta F_3$, Model (2.1) has the DFE and a unique EE denoted by E_3^*.

(d) If either $\mathscr{R}_c = 1$ and $bF_1 < \beta F_3$ or $\mathscr{R}_c = \mathscr{R}_c^c$ and $bF_1 > \beta F_3$, Model (2.1) has only the DFE E_0.

(e) If $\mathscr{R}_c > 1$, there are two equilibria: the DFE and a unique EE denoted by E_4^*.

3.2 *Bifurcation analysis*

Bifurcation is a change of the topological structure of a system when its parameters pass through a critical value Juga *et al.* (2021). Since the behaviour of our system changes as the control reproduction number crosses the value 1, $\mathscr{R}_c = 1$ is a critical point. Expressing equation $G_2\lambda^{*2} + G_1\lambda^* - G_0 = 0$ as a function of β and λ^* gives the equation

$$F(\beta; \lambda^*) := G_2\lambda^{*2} + (bF_1 - \beta F_3)\lambda^* - \beta F_4 + bF_0 = 0. \tag{3.8}$$

When $\lambda^* = 0$, we obtain the solution β^* from Eq. (3.8) given by

$$\beta^* := \beta = \frac{bF_0}{F_4} = \frac{1}{\mathscr{R}_c}.$$

The direction (forward or backward) of the bifurcation is given by the sign of

$$\frac{\partial \lambda^*}{\partial \beta}(\beta^*, 0) = -\frac{F_{\beta^*}(\beta^*, 0)}{F_{\lambda^*}(\beta^*, 0)}.$$

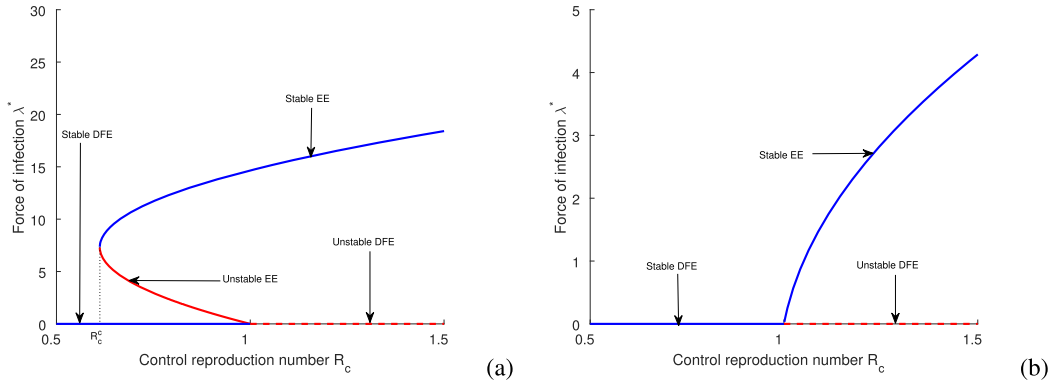

FIG. 2. (a): backward bifurcation for $\beta = 0.011, \nu_3 = 0.065, \nu_2 = 0.05, \nu_4 = 10\,000.05, \nu_1 = 0.0385, b = 4.5, \mu = 60.0004, \eta_1 = 100.3, \eta_2 = 50.05, \varepsilon = 0.5, \gamma_1 = 0.01, \gamma_2 = 0.02, \gamma_u = 0.02, \gamma_h = 0.025, \delta_2 = 100.1, \sigma_i = 0.025, \sigma_s = 0.3$ and $, \sigma_u = 0.3$. (b): forward bifurcation for $p = 0.0003, \beta = 0.15, \nu_3 = 0.65, \mu = 50.0004, \gamma_1 = 0.01, \gamma_2 = 0.02, \gamma_u = 0.02, \gamma_h = 0.025, b = 0.5, \sigma_i = 0.0025, \sigma_s = 0.003, \sigma_u = 0.003$.

We have $F_{\beta^*}(\beta^*, 0) = -F_4 < 0, \quad F_{\lambda^*}(\beta^*, 0) = bF_1 - \beta^* F_3 = bF_1 - \frac{F_3}{\mathscr{R}_c}$. The sign of $\frac{\partial \lambda^*}{\partial \beta}(\beta^*, 0)$ depends on the sign of $F_{\lambda^*}(\beta^*, 0)$.

If $\mathscr{R}_c < \frac{F_3}{bF_1}$, then $\frac{\partial \lambda^*}{\partial \beta}(\beta^*, 0) < 0$ and the bifurcation is backward. On the other hand, if $\mathscr{R}_c > \frac{F_3}{bF_1}$, then $\frac{\partial \lambda^*}{\partial \beta}(\beta^*, 0) > 0$ and the bifurcation is forward in this case. Similarly, it is straightforward that the bifurcation is forward whenever $\mathscr{R}_c > \frac{F_3}{bF_1}$. The epidemiological consequence of a backward bifurcation is that the requirement $\mathscr{R}_c < 1$, although necessary is no longer sufficient for global elimination of EVD Safi & Gumel (2015). Fig. 2(a) gives an example of the construction of the backward bifurcation. We observe that a locally asymptotically stable (LAS) DFE co-exists with an LAS EE when the value of $\mathscr{R}_c$ is greater than $\mathscr{R}_c^c$ but less than 1. This makes the effective control of EVD difficult since the value of $\mathscr{R}_c$ needs to be less than $\mathscr{R}_c^c$. Note that the existence of a backward bifurcation in our model could likely be due to the standard incidence transmission considered in this article Agusto (2017); Ouemba Tasse *et al.* (2022) and the recruitment in the two groups of our population (aware and unaware individuals) Malik *et al.* (2013); Ouemba Tassé *et al.* (2024). In particular, when $\theta_3 = 0$ or $\theta_1 = \theta_2 = 0$, $G_1 = bF_1 > 0$, the model does not exhibit the backward bifurcation phenomenon because no EE exists for $\mathscr{R}_c < 1$. The case where $\theta_3 = 0$ refers to the situation where aware susceptible do not contract the disease, while the case $\theta_1 = \theta_2 = 0$ leads to an epidemic model rather than the endemic one proposed in this paper.

Figure 2(b) gives an example of a construction of the forward bifurcation, which means that the disease will persist in the community for a value of $\mathscr{R}_c > 1$.

3.3 *Global stability of the DFE*

In this section, we check the conditions for the global asymptotic stability of the disease-free equilibrium. This will be done using the method of Kamgang and Sallet described in Kamgang & Sallet (2008).

Using the method of Kamgang and Sallet, let us define the following parameters:

$$L_1 = \frac{\beta}{\phi_1}\left(1 + \frac{\nu_1\eta_1}{\phi_h} + \frac{\tau_1\nu_4}{b}\right),$$
$$L_2 = \frac{\beta}{\phi_2}\left(\frac{\sigma_i}{\phi_1} + \nu_2 + \nu_1\left(\frac{\eta_1\sigma_i}{\phi_1\phi_h} + \frac{q\eta_2}{\phi_h} + \frac{\sigma_u(1-q)\eta_2}{\phi_u}\right) + \frac{\nu_3(1-q)\eta_2}{\phi_u} + \nu_4\left(\frac{\tau_1\sigma_i}{b\phi_1} + \frac{\tau_2}{b} + \frac{\tau_u(1-q)\eta_2}{b\phi_u}\right)\right).$$

THEOREM 2. The disease-free equilibrium E_0 for Model (2.1) is globally asymptotically stable in Ω whenever $\mathscr{T}_0 \leq 1$, where

$$\mathscr{T}_0 := \frac{(1-\varepsilon)L_1 + L_2 + \sqrt{((1-\varepsilon)L_1 + L_2)^2 + 4(1-\varepsilon)L_1L_2}}{2}.$$

Proof. Following the method in Kamgang & Sallet (2008), let's define $x_1 = (S_1, S_2, R); x_2 = (I_1, I_2, H, U, D)$ the non-infected components and the infected components, respectively, and $x_1^* = (S_{10}, S_{20}, 0)$ the disease-free equilibrium. We can express the sub-system $\dot{x} = A_1(x_1, 0)(x_1 - x_1^*)$ by

$$\begin{cases} \dot{S}_1 = p\pi + \sigma_s S_2 - \mu S_1 \\ \dot{S}_2 = (1-p)\pi - (\mu + \sigma_s)S_2 \\ \dot{R} = -\mu R. \end{cases} \tag{3.9}$$

It is straightforward that the linear System (3.9) is GAS at the equilibrium x_1^*, corresponding to the disease-free equilibrium.

Moreover the matrix $A_2(x)$ can be written as

$$A_2(x) = \begin{pmatrix} \frac{\beta(1-\varepsilon)S_1}{N} - \phi_1 & \frac{\beta\nu_2(1-\varepsilon)S_1}{N} + \sigma_i & \frac{\beta\nu_1(1-\varepsilon)S_1}{N} & \frac{\beta\nu_3(1-\varepsilon)S_1}{N} & \frac{\beta\nu_4(1-\varepsilon)S_1}{N} \\ \frac{\beta S_2}{N} & \frac{\beta\nu_2 S_2}{N} - \phi_2 & \frac{\beta\nu_1 S_2}{N} & \frac{\beta\nu_3 S_2}{N} & \frac{\beta\nu_4 S_2}{N} \\ \eta_1 & q\eta_2 & -\phi_h & \sigma_u & 0 \\ 0 & (1-q)\eta_2 & 0 & -\phi_u & 0 \\ \tau_1 & \tau_2 & 0 & \tau_u & -b \end{pmatrix}$$

Clearly, $A_2(x)$ is irreducible and has a maximum uniquely realized in Ω for $S_1 = S_2 = N$. This maximum leads to the matrix J_2 defined by

$$J_2 = \begin{pmatrix} \beta(1-\varepsilon)-\phi_1 & \beta\nu_2(1-\varepsilon)+\sigma_i & \beta\nu_1(1-\varepsilon) & \beta\nu_3(1-\varepsilon) & \beta\nu_4(1-\varepsilon) \\ \beta & \beta\nu_2-\phi_2 & \beta\nu_1 & \beta\nu_3 & \beta\nu_4 \\ \eta_1 & q\eta_2 & -\phi_h & \sigma_u & 0 \\ 0 & (1-q)\eta_2 & 0 & -\phi_u & 0 \\ \tau_1 & \tau_2 & 0 & \tau_u & -b \end{pmatrix}$$

According to Kamgang & Sallet (2008), the DFE for Model (2.1) is GAS if $\alpha(J_2) \leq 0$, where $\alpha(J_2)$ denotes the stability modulus of the matrix J_2. Simple computations show that $\alpha(J_2) \leq 0$ is equivalent to $\mathscr{T}_0 \leq 1$. This completes the proof. □

3.4 *Local stability of the EE and uniform persistence of the disease*

THEOREM 3. If $\mathscr{R}_c > 1$ but close to 1, then the unique EE E_4^* of System (2.1) is locally asymptotically stable. In addition, System (2.1) undergoes a trans-critical bifurcation at $\mathscr{R}_c = 1$.

Proof. We prove the two statements of Theorem 3 simultaneously using the centre manifold theory approach Castillo-Chavez & Song (2004).

Let β be the bifurcation parameter and β^* its critical value obtained by solving the equation $\mathscr{R}_c = 1$. Then β^* is given by

$$\beta^* = \frac{bF_0}{A_1\phi_u\phi_h\mu b + \nu_1 bB_1\mu + \nu_4\phi_h C_1\mu + b\nu_2\theta_2\phi_1\phi_h\phi_u\mu^2 + \eta_2\nu_3(1-q)\theta_2\phi_1\phi_h\mu^2 b}.$$

To establish the local asymptotic stability of E_4^*, we make the following change of variable;

$$x_1 = S_1, \quad x_2 = S_2, \quad x_3 = I_1, \quad x_4 = I_2, \quad x_5 = H, \quad x_6 = U, \quad x_7 = D, \quad x_8 = R.$$

Then with change of variables, System (2.1) takes the form

$$\frac{dx}{dt} = f(x),$$

where $f = (f_1, f_2, f_3, f_4, f_5, f_6, f_7, f_8)$, are defined as

$$\begin{cases} f_1 = \theta_1 + \sigma_s x_2 - \beta^* \theta_3 x_1 \left(\dfrac{x_3 + \nu_1 x_5 + \nu_2 x_4 + \nu_3 x_6 + \nu_4 x_7}{x_1 + x_2 + x_3 + x_4 + x_5 + x_6 + x_8} \right) - \mu x_1, \\ f_2 = \theta_2 - \beta^* x_2 \left(\dfrac{x_3 + \nu_1 x_5 + \nu_2 x_4 + \nu_3 x_6 + \nu_4 x_7}{x_1 + x_2 + x_3 + x_4 + x_5 + x_6 + x_8} \right) - \tau_s x_2, \\ f_3 = \beta^* \theta_3 x_1 \left(\dfrac{x_3 + \nu_1 x_5 + \nu_2 x_4 + \nu_3 x_6 + \nu_4 x_7}{x_1 + x_2 + x_3 + x_4 + x_5 + x_6 + x_8} \right) + \sigma_i x_4 - \phi_1 x_3, \\ f_4 = \beta^* x_2 \left(\dfrac{x_3 + \nu_1 x_5 + \nu_2 x_4 + \nu_3 x_6 + \nu_4 x_7}{x_1 + x_2 + x_3 + x_4 + x_5 + x_6 + x_8} \right) - \phi_2 x_4, \\ f_5 = \eta_1 x_3 + q\eta_2 x_4 + \sigma_u x_6 - \phi_h x_5, \\ f_6 = (1-q)\eta_2 x_4 - \phi_u x_6, \\ f_7 = \tau_1 x_3 + \tau_2 x_4 + \tau_u U - b x_7, \\ f_8 = \gamma_1 x_3 + \gamma_2 x_4 + \gamma_u x_6 + \gamma_h x_5. \end{cases} \tag{3.10}$$

The Jacobian matrix J_* of (3.10) at the DFE E_0 when $\beta = \beta^*$ is given by;

$$J_* = \begin{pmatrix} -\mu & \sigma_s & -\dfrac{\beta^*\theta_3 S_{10}}{N_0} & -\dfrac{\beta^*\theta_3\nu_2 S_{10}}{N_0} & -\dfrac{\beta^*\theta_3\nu_1 S_{10}}{N_0} & -\dfrac{\beta^*\theta_3\nu_3 S_{10}}{N_0} & -\dfrac{\beta^*\theta_3\nu_4 S_{10}}{N_0} & 0 \\ 0 & -\tau_s & -\dfrac{\beta^* S_{20}}{N_0} & -\dfrac{\beta^*\nu_2 S_{20}}{N_0} & -\dfrac{\beta^*\nu_1 S_{20}}{N_0} & -\dfrac{\beta^*\nu_3 S_{20}}{N_0} & -\dfrac{\beta^*\nu_4 S_{20}}{N_0} & 0 \\ 0 & 0 & \dfrac{\beta^*\theta_3 S_{10}}{N_0} - \phi_1 & \dfrac{\beta^*\theta_3\nu_2 S_{10}}{N_0} + \sigma_i & \dfrac{\beta^*\theta_3\nu_1 S_{10}}{N_0} & \dfrac{\beta^*\theta_3\nu_3 S_{10}}{N_0} & \dfrac{\beta^*\theta_3\nu_4 S_{10}}{N_0} & 0 \\ 0 & 0 & \dfrac{\beta^* S_{20}}{N_0} & \dfrac{\beta^*\nu_2 S_{20}}{N_0} - \phi_2 & \dfrac{\beta^*\nu_1 S_{20}}{N_0} & \dfrac{\beta^*\nu_3 S_{20}}{N_0} & \dfrac{\beta^*\nu_4 S_{20}}{N_0} & 0 \\ 0 & 0 & \eta_1 & q\eta_2 & -\phi_h & \sigma_i & 0 & 0 \\ 0 & 0 & 0 & (1-q)\eta_2 & 0 & -\phi_u & 0 & 0 \\ 0 & 0 & \tau_1 & \tau_2 & 0 & \tau_u & -b & 0 \\ 0 & 0 & \gamma_1 & \gamma_2 & \gamma_h & \gamma_u & 0 & -\mu \end{pmatrix}$$

It is straightforward to prove that the matrix J_* has zero as a simple eigenvalue while the remaining eigenvalues have negative real parts. Therefore, using Castillo-Chavez & Song (2004), we prove the local stability of the EE E_4^* when $\mathscr{R}_c > 1$ but close to 1 and the trans-critical bifurcation of System (2.1) at $\mathscr{R}_c = 1$.

In order to apply the centre manifold result in Castillo-Chavez & Song (2004), we compute the right-eigenvector $w = (w_1, w_2, w_3, w_4, w_5, w_6, w_7, w_8)^T$ and the left-eigenvector $u =$

$(u_1, u_2, u_3, u_4, u_5, u_6, u_7, u_8)^T$ of J_* as follows:

$$\begin{cases} w_1 = -\dfrac{1}{\mu N_0}(k_{10}w_3 + k_{11}w_4), \quad w_2 = -\dfrac{k_8 w_3 + k_9 w_4}{N_0 \tau_s b \phi_u \phi_h} \\ w_5 = \dfrac{k_4 w_3 + k_5 w_4}{\phi_u \phi_h}, \quad w_6 = \dfrac{k_1 w_4}{\phi_u}, \quad w_3, w_4 > 0 \\ w_7 = \dfrac{k_2 w_3 + k_3 w_4}{b\phi_u} \quad w_8 = \dfrac{k_6 w_3 + k_7 w_4}{\mu \phi_u \phi_h} \end{cases}$$

and

$$\begin{cases} u_1 = u_2 = u_8 = 0, \quad u_3, u_4 > 0, \quad u_5 = \dfrac{\beta^* \nu_1}{N_0 \phi_h}(\theta_3 S_{10} u_3 + S_{20} u_4) \\ u_6 = \dfrac{k_{12}(S_{10}\theta_3 u_3 + S_{20} u_4)}{N_0 \phi_u \phi_h b}, \quad u_7 = \dfrac{\beta^* \nu_4}{N_0 b}(\theta_3 S_{10} u_3 + S_{20} u_4) \end{cases}$$

where the positive constants $k_1, k_2, \cdots, k_{12}$ are given by

$$\begin{cases} k_1 &= (1-q)\eta_2, \quad k_2 = \tau_1 \phi_u, \quad k_3 = \tau_2 \phi_u + \tau_u (1-q)\eta_2 \\ k_4 &= \eta_1 \phi_u, \quad k_5 = q\eta_2 \phi_u + \sigma_u k_1, \quad k_6 = \gamma_1 \phi_u \phi_h + \gamma_h k_4, \\ k_7 &= \gamma_2 \phi_u \phi_h + \gamma_u k_1 \phi_h + \gamma_h k_5 \\ k_8 &= \beta^* S_{20}(b\phi_u \phi_h + \nu_1 k_4 b + \nu_4 k_2 \phi_h), \\ k_9 &= \beta^* S_{20}(\nu_2 b \phi_u \phi_h + \nu_1 k_5 b + \nu_3 k_1 b \phi_h + \nu_4 k_3 \phi_h) \\ k_{10} &= \left[\dfrac{\sigma_s k_8}{\tau_s b \phi_u \phi_h} + \beta^* \theta_3 S_{10}\left(1 + \dfrac{\nu_1 k_4}{\phi_u \phi_h} + \dfrac{\nu_4 k_2}{\phi_u b}\right)\right], \\ k_{11} &= \left[\dfrac{\sigma_s k_9}{\tau_s b \phi_u \phi_h} + \beta^* \theta_3 S_{10}\left(\nu_2 + \dfrac{\nu_1 k_5}{\phi_u \phi_h} + \dfrac{\nu_3 k_1}{\phi_u} + \dfrac{\nu_4 k_3}{\phi_u b}\right)\right], \\ k_{12} &= \beta^*(\nu_3 \phi_h b + \sigma_u \nu_1 b + \tau_u \nu_4 \phi_h). \end{cases} \tag{3.11}$$

Next, we calculate the following coefficients:

$$a = \sum_{k,i,j=1}^{8} u_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0) \quad \text{and} \quad b = \sum_{k,i=1}^{8} u_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta^*}(0,0).$$

After some simple but lengthy algebraic calculations, we have

$$\begin{aligned} a &= 2N_0^{-2}\beta^*\theta_3 u_3[w_1 S_{20}(w_3+\nu_2 w_4+\nu_1 w_5+\nu_3 w_6+\nu_4 w_7)-S_{10}(w_3^2+\nu_2 w_4^2+\nu_1 w_5^2\\ &\quad +\nu_3 w_6^2+\nu_4 w_7^2+(1+\nu_1)w_3 w_5+(1+\nu_2)w_3 w_4+(1+\nu_3)w_3 w_6+(1+\nu_4)w_3 w_7+w_3 w_8\\ &\quad +(\nu_1+\nu_2)w_4 w_5+(\nu_2+\nu_3)w_4 w_6+(\nu_2+\nu_4)w_4 w_7+\nu_2 w_4 w_8+(\nu_1+\nu_3)w_5 w_6\\ &\quad +(\nu_1+\nu_4)w_5 w_7+\nu_1 w_5 w_8+(\nu_3+\nu_4)w_6 w_7+\nu_3 w_6 w_8+\nu_4 w_7 w_8)]\\ &\quad +2N_0^{-2}\beta^* u_4[w_2 S_{10}(w_3+\nu_2 w_4+\nu_1 w_5+\nu_3 w_6+\nu_4 w_7)-S_{20}(w_3^2+\nu_2 w_4^2+\nu_1 w_5^2\\ &\quad +\nu_3 w_6^2+\nu_4 w_7^2+(1+\nu_1)w_3 w_5+(1+\nu_2)w_3 w_4+(1+\nu_3)w_3 w_6+(1+\nu_4)w_3 w_7+w_3 w_8\\ &\quad +(\nu_1+\nu_2)w_4 w_5+(\nu_2+\nu_3)w_4 w_6+(\nu_2+\nu_4)w_4 w_7+\nu_2 w_4 w_8+(\nu_1+\nu_3)w_5 w_6\\ &\quad +(\nu_1+\nu_4)w_5 w_7+\nu_1 w_5 w_8+(\nu_3+\nu_4)w_6 w_7+\nu_3 w_6 w_8+\nu_4 w_7 w_8)] \end{aligned}$$

$$\begin{aligned} b &= u_3 S_{10} N_0^{-1}\theta_3(w_3+\nu_2 w_4+\nu_1 w_5+\nu_3 w_6+\nu_4 w_7)\\ &\quad +u_4 S_{20} N_0^{-1}(w_3+\nu_2 w_4+\nu_1 w_5+\nu_3 w_6+\nu_4 w_7). \end{aligned}$$

Through inspection of the signs of the components of the vectors ω and u, we see that $a<0$ and $b>0$. Hence, the unique EE of E_4^* is locally asymptotically stable when $\mathscr{R}_c>1$, but close to 1 and Model (2.1) undergoes a trans-critical bifurcation at $\mathscr{R}_c=1$. □

The epidemiological interpretation of Theorem 3 is that EVD persists and is endemic in the population when the control reproduction number $\mathscr{R}_c>1$. Hence, it is important to identify influential parameters of our model (2.1) that have direct impact on the dynamics of EVD in order to apply appropriate control strategies.

3.5 *Uniform persistence of the disease*

THEOREM 4. Assume $\mathscr{R}_c>1$, then EVD is uniformly persistent. That is, there exists an $\alpha>0$ such that for every positive solution of System (2.1), the following expressions are true:

$$\liminf_{t\longrightarrow+\infty} I_1(t)>\alpha,\ \liminf_{t\longrightarrow+\infty} I_2(t)>\alpha,\ \liminf_{t\longrightarrow+\infty} H(t)>\alpha,\ \liminf_{t\longrightarrow+\infty} U(t)>\alpha,\ \liminf_{t\longrightarrow+\infty} D(t)>\alpha.$$

Proof. To prove the uniform persistence of EVD when $\mathscr{R}_c>1$, let

$$\begin{aligned} &X=\{(S_1,S_2,I_1,I_2,H,U,D,R)\in\mathbb{R}_+^8\}\\ &X_0=\{(S_1,S_2,I_1,I_2,H,U,D,R)\in X: I_1,I_2,H,U,D>0\}\\ &Y=X\setminus X_0=\{(S_1,S_2,I_1,I_2,H,U,D,R)\in X:\ I_1=0\ \text{or}\ I_2=0\ \text{or}\ H=0\ \text{or}\ U=0\ \text{or}\ D=0\}.\\ &M=(S_1,S_2,I_1,I_2,H,U,D,R)\in\mathbb{R}^8 \text{ and } \overline{M}=(I_1,I_2,H,U,D). \end{aligned}$$

Since Y contains only the equilibrium E_0, to establish the uniform persistence of Model (2.1), all we have to do is to show that $W^s(E_0)\cap X_0=\emptyset$, where $W^s(E_0)$ denotes the stable manifold of E_0.

Suppose this is not true, that is, $W^s(E_0)\cap X_0\neq\emptyset$. Then there exists a solution $(S_1,S_2,I_1,I_2,H,U,D,R)$ of System (2.1) in X_0 such that

$$\lim_{t\longrightarrow+\infty}(S_1,S_2,I_1,I_2,H,U,D,R)\longrightarrow E_0=\left(S_{10},S_{20},0,0,0,0,0,0\right).$$

Thus, for any $c > 0$, and for sufficiency large values of t, we have

$$\begin{cases} S_{10} - c \le S_1(t) \le S_{10} + c, \quad S_{20} - c \le S_2(t) \le S_{20} + c, \quad 0 \le I_1(t) \le c, \\ 0 \le I_2(t) \le c, \quad 0 \le H(t) \le c, \quad 0 \le U(t) \le c, \quad 0 \le D(t) \le c, \quad 0 \le R(t) \le c. \end{cases} \tag{3.12}$$

Using Equation Carter (2014), we obtain the following lower bound of System (2.1):

$$\begin{pmatrix} \dot{I}_1(t) \\ \dot{I}_2(t) \\ \dot{H}(t) \\ \dot{U}(t) \\ \dot{D}(t) \end{pmatrix} = \begin{pmatrix} \dfrac{\beta\theta_3 S_1(I_1 + \nu_1 H + \nu_2 I_2 + \nu_3 U + \nu_4 D)}{N} \\ \dfrac{\beta S_2(I_1 + \nu_1 H + \nu_2 I_2 + \nu_3 U + \nu_4 D)}{N} \\ 0 \\ 0 \\ 0 \end{pmatrix} + \begin{pmatrix} -\phi_1 & \sigma_i & 0 & 0 & 0 \\ 0 & -\phi_2 & 0 & 0 & 0 \\ \eta_1 & q\eta_2 & -\phi_h & \sigma_u & 0 \\ 0 & (1-q)\eta_2 & 0 & -\phi_u & 0 \\ \tau_1 & \tau_2 & 0 & \tau_3 & -b \end{pmatrix} \begin{pmatrix} I_1(t) \\ I_2(t) \\ H(t) \\ U(t) \\ D(t) \end{pmatrix}$$

$$\begin{pmatrix} \dot{I}_1(t) \\ \dot{I}_2(t) \\ \dot{H}(t) \\ \dot{U}(t) \\ \dot{D}(t) \end{pmatrix} \ge \widetilde{J(c)} \begin{pmatrix} I_1(t) \\ I_2(t) \\ H(t) \\ U(t) \\ D(t) \end{pmatrix}$$

where $N_0 = \pi/\mu$,

$$\widetilde{J(c)} = \begin{pmatrix} \beta\theta_3\widetilde{S}_{10}(c) - \phi_1 & \beta\nu_2\theta_3\widetilde{S}_{10}(c) + \sigma_i & \beta\theta_3\nu_1\widetilde{S}_{10}(c) & \beta\theta_3\nu_3\widetilde{S}_{10}(c) & \beta\theta_3\nu_4\widetilde{S}_{10}(c) \\ \beta\widetilde{S}_{20}(c) & \beta\nu_2\widetilde{S}_{20}(c) - \phi_2 & \beta\nu_1\widetilde{S}_{20}(c) & \beta\nu_3\widetilde{S}_{20}(c) & \beta\nu_4\widetilde{S}_{20}(c) \\ \eta_1 & q\eta_2 & -\phi_h & \sigma_u & 0 \\ 0 & (1-q)\eta_2 & 0 & -\phi_u & 0 \\ \tau_1 & \tau_2 & 0 & \tau_3 & -b \end{pmatrix}$$

and

$$\widetilde{S}_{10}(c) = \frac{S_{10} - c}{N_0 + 5c}, \quad \widetilde{S}_{20}(c) = \frac{S_{20} - c}{N_0 + 5c}.$$

Clearly,

$$\widetilde{J(0)} = \begin{pmatrix} \beta\theta_3\dfrac{S_{10}}{N_0} - \phi_1 & \beta\nu_2\theta_3\dfrac{S_{10}}{N_0} + \sigma_i & \beta\theta_3\nu_1\dfrac{S_{10}}{N_0} & \beta\theta_3\nu_3\dfrac{S_{10}}{N_0} & \beta\theta_3\nu_4\dfrac{S_{10}}{N_0} \\ \beta\dfrac{S_{20}}{N_0} & \beta\nu_2\dfrac{S_{20}}{N_0} - \phi_2 & \beta\nu_1\dfrac{S_{20}}{N_0} & \beta\nu_3\dfrac{S_{20}}{N_0} & \beta\nu_4\dfrac{S_{20}}{N_0} \\ \eta_1 & q\eta_2 & -\phi_h & \sigma_u & 0 \\ 0 & (1-q)\eta_2 & 0 & -\phi_u & 0 \\ \tau_1 & \tau_2 & 0 & \tau_3 & -b \end{pmatrix} = F - V,$$

with F and V given by

$$F = \begin{pmatrix} \beta\theta_3\frac{S_{10}}{N_0} & \beta\nu_2\theta_3\frac{S_{10}}{N_0} & \beta\theta_3\nu_1\frac{S_{10}}{N_0} & \beta\theta_3\nu_3\frac{S_{10}}{N_0} & \beta\theta_3\nu_4\frac{S_{10}}{N_0} \\ \beta\frac{S_{20}}{N_0} & \beta\nu_2\frac{S_{20}}{N_0} & \beta\nu_1\frac{S_{20}}{N_0} & \beta\nu_3\frac{S_{20}}{N_0} & \beta\nu_4\frac{S_{20}}{N_0} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

$$V = \begin{pmatrix} \phi_1 & -\sigma_i & 0 & 0 & 0 \\ 0 & \phi_2 & 0 & 0 & 0 \\ -\eta_1 & -q\eta_2 & \phi_h & -\sigma_u & 0 \\ 0 & -(1-q)\eta_2 & 0 & \phi_u & 0 \\ -\tau_1 & -\tau_2 & 0 & -\tau_3 & b \end{pmatrix} \tag{3.13}$$

$\widetilde{J(0)}$ has at least one eigenvalue with positive real part when $\mathscr{R}_c = \rho(FV^{-1}) > 1$ (see Berman & Plemmons (1994)). That is, $s(F-V) = s(\widetilde{J(0)}) > 0$, where $s(A)$ is the largest real part of the eigenvalues of the matrix A. Moreover, since $c > 0$ is arbitrary, one can choose c small enough so that $s(\widetilde{J(c)})$ is positive. Therefore, there exist solutions of the linear system $\dot{\overline{M}} = \widetilde{J(c)}\overline{M}$ that grows exponentially near $\overline{M} = 0$. By comparison, the solution $\overline{M}(t)$ of the inequality above become unbounded as $t \longrightarrow +\infty$. This contradicts the fact that solutions of System (2.1) are ultimately bounded. Therefore, $W^s(E_0) \cap X_0 = \emptyset$. Applying Theorem 4.6 in Thieme (1993), one concludes that System (2.1) is uniformly persistent with respect to (X_0, Y). □

4. Model calibration

We now consider calibrating our model. The data for this study is from weekly cumulative reported cases of EVD in Sierra Leone and Liberia WHO (2015). Model (2.1) is calibrated for 42 weeks, using the weekly cumulative cases reported. We made use of the Nonlinear Least Squares fitting routine **lsqnonlin** function in the optimization tool box of MATLAB.

For the sake of simplicity, we assume that in both countries, half of the population is aware and the other half unaware about the EVD. The population of Sierra Leone is estimated to be 8 million while that of Liberia is 5 million. We assume that $S_1(0) = S_2(0) = 4\,000\,000$ for Sierra Leone and $S_1(0) = S_2(0) = 2\,500\,000$ for Liberia. On 27 May 2014, there was one case in Sierra Leone. We assume this person was unaware about EVD. The initial condition for the infected classes is $I_1(0) = 0$; $I_2(0) = 1$; $H(0) = 0$; $U(0) = 0$; $D(0) = 0$; $R(0) = 0$. On the other hand, there were 12 cases reported in Liberia on this same day. We divide the 12 cases equally (2 each) into the 6 infected compartments of the model. That is, $I_1(0) = 2$; $I_2(0) = 2$; $H(0) = 2$; $U(0) = 2$; $D(0) = 2$; $R(0) = 2$ in Liberia.

The fitted curves are plotted in Fig. 3(a), for Sierra Leone and Fig. 3(b), for Liberia, and the estimated parameters values are displayed in Table 2. In order to evaluate the goodness of the calibrations Ouemba Tasse *et al.* (2022), we compute the Mean Absolute Error (MAE) and the Root Mean Square Error

TABLE 2 *Parameter values for the calibration of the model*

Parameter	Sierra Leone	Liberia	Source
μ	0.00004	0.00004	Agusto *et al.* (2015)
π	15.0384	10.0471	Fitted
ε	0.0416	0.4202	Fitted
η_1	0.8267	0.5046	Fitted
η_2	0.0706	0.0528	Fitted
β	0.5003	0.5015	Fitted
p	0.3324	0.3297	Fitted
q	0.9252	0.5151	Fitted
δ_1	0.3518	0.3883	Fitted
δ_2	0.1293	0.2322	Fitted
δ_u	0.0902	0.2	Fitted
δ_h	0.1198081	0.1198081	Grigorieva & Khailov (2018)
ν_1	0.3875	0.3875	Ouemba Tasse *et al.* (2022)
ν_2	3.0013	3.0226	Fitted
ν_3	2.0809	2.1616	Fitted
ν_4	1.5681	1.2	Fitted
σ_s	0.0717	0.0838	Fitted
σ_i	0.0797	0.0366	Fitted
σ_u	0.0041	0.0093	Fitted
γ_1	0.6221	0.1651	Fitted
γ_2	0.0654	0.0622	Fitted
γ_h	0.5099	0.5023	Fitted
γ_u	0.0053	0.0534	Fitted
b	0.8687	0.6769	Fitted

TABLE 3 *Initial conditions and metrics of the model calibrations.*

Country	Initial condition value	$\mathscr{R}_c$ value	MAE	RMSE
Sierra Leone	$S_1(0) = S_2(0) = 4,000,000; I_1(0) = 0; I_2(0) = 1; H(0) = 0; U(0) = 0; D(0) = 0; R(0) = 0$	0.5725	1.4199	9.2021
Liberia	$S_1(0) = 2,500,000;\ S_2(0) = 2,500,000;\ I_1(0) = I_2(0) = R(0) = U(0) = H(0) = D(0) = 2$	0.8340	0.9826	6.3681

(RMSE) for each fitted curve, defined by

$$MAE = \frac{1}{N_p}\sum_{i=1}^{N_p}|Y(i) - \hat{Y}(i)| \text{ and } RMSE = \sqrt{\sum_{i=1}^{N_p}(Y(i) - \hat{Y}(i))^2/N_p},$$

where $Y(i)$ represents original cases, $\hat{Y}(i)$, the predicted values and N_p is the size of the data.

The values of these metrics, as well as the estimated values of the control reproduction number in these two countries are presented in Table 3. The small values of MAE and RMSE for each country show that our model gives a good fit to observed data.

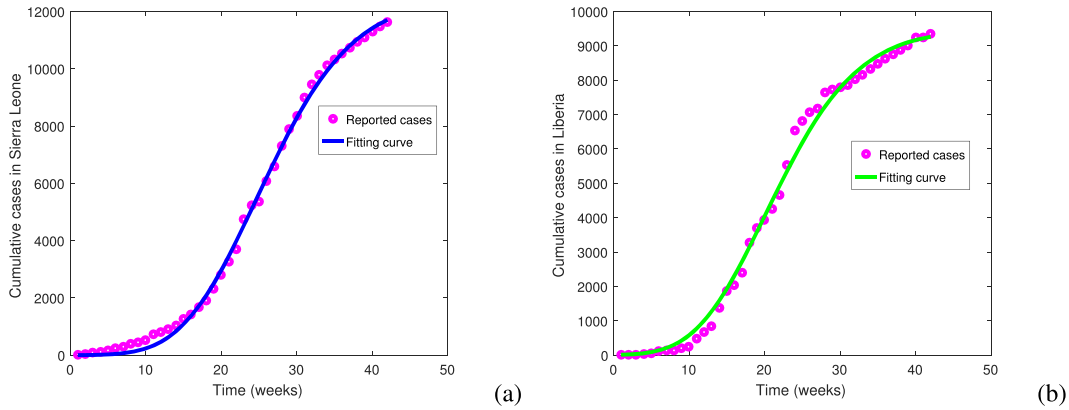

FIG. 3. Model calibration: fitted curves for the cumulative reported cases for 42 weeks in (a) Sierra Leone and (b) Liberia.

5. Assessment of the role of awareness and traditional medicine

In this section, we carry out some simulations in order to assess the effect of awareness on the behaviour of the population and its influence on the choice of a suitable treatment (traditional medicine or modern medicine) in controlling the Ebola outbreaks in Sierra Leone and Liberia.

5.1 *Traditional medicine versus modern medicine*

Traditional medicine occupies a prominent place in Africa, due principally to poverty, lack of means, lack of healthcare facilities or the misconceptions about diseases (which can be generally attributed to witchcraft) Cénat *et al.* (2021). Many infected individuals seek care from traditional healers who most of the time do not respect preventive measures against the EVD. Therefore, the contribution of traditional medicine in the treatment of EVD needs to be investigated. In this paper, this is done in two steps. The first step is to know how the proportion, $\omega = 1 - q$, of infected people who seek traditional treatment influences the dynamics of the model. In the second step, we compare the two treatment approaches.

We begin with the first step. Figure 4(a), which represents the control reproduction number $\mathscr{R}_c$ versus ω, shows that the control reproduction number in Sierra Leone is an increasing function. This suggests that the more the infected will choose traditional medicine, the more EVD will be spread. A similar result is found in Liberia because the algebraic expression for $\mathscr{R}_c$ is the same in both countries.

This observation is supported by Fig. 4(b), (c), (d), (e) and (f), which show the number of infected individuals in compartments I_1, I_2, H, U and the total number of infected individuals $(I_1 + I_2 + H + U)$, respectively. We use three different values of ω ($\omega = 1; \omega = 0.5; \omega = 0.2$). We observe that, as ω increases, the number of infected individuals increases as well. The plots in Fig. 4 are obtained using the estimated parameters for Sierra Leone. Corresponding plots using data for Liberia are similar.

In the second step, a comparison between the two treatment approaches is made by plotting the number of cases treated per week for each treatment approach (modern medicine and traditional medicine). In other words, we investigate the propensity of the infected people in Sierra Leone and Liberia to go to hospital or to traditional healers during the course of EVD. Thus, we plot the two functions H and U against time as shown in Fig. 5. In order to have a reasonable comparison for these functions, we assume that the values for $H(0)$ and $U(0)$, though different for each country, are the same. Fig. 5 shows that as time evolves the number of hospitalized individuals increases more rapidly than the

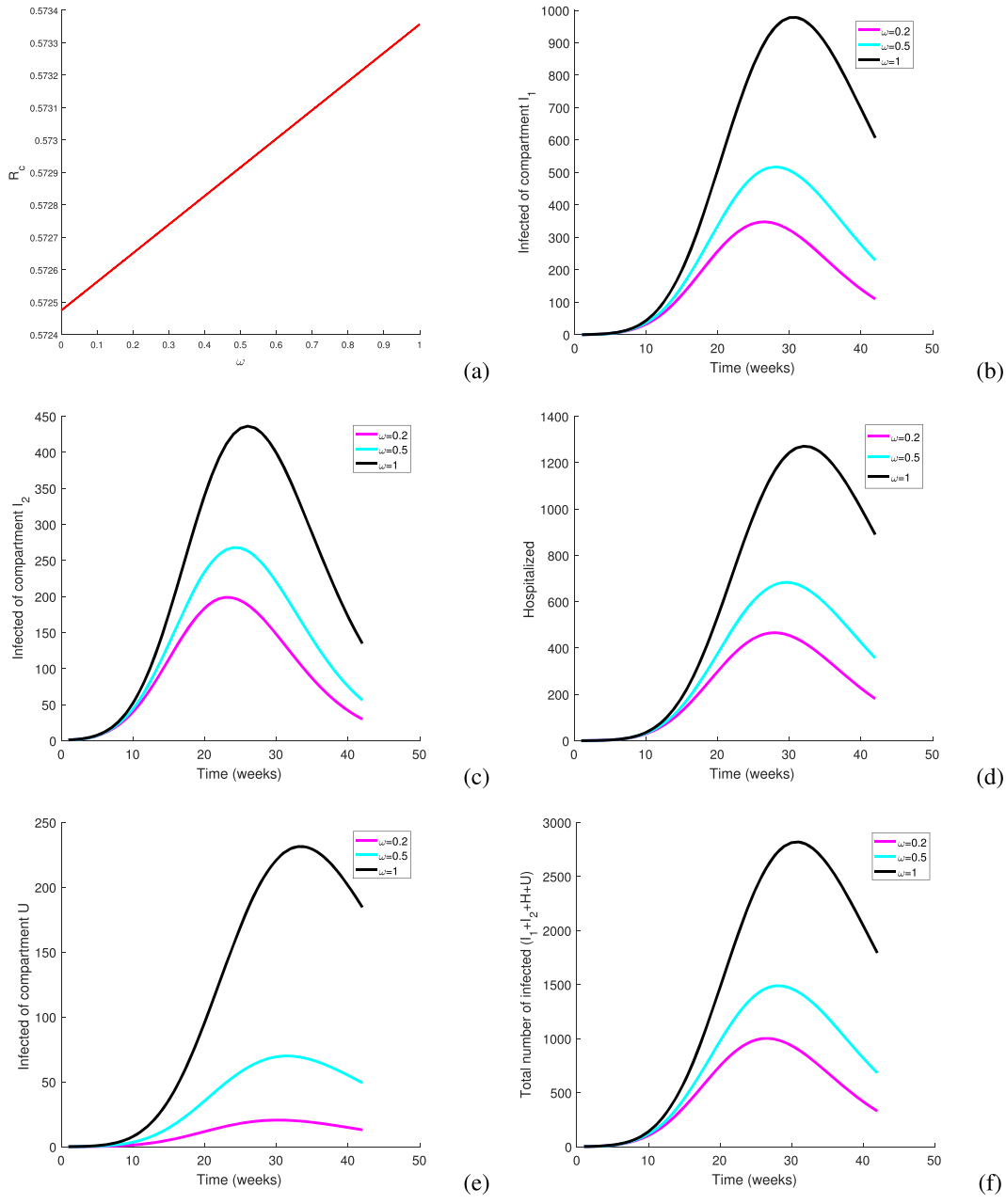

FIG. 4. Place of traditional medicine Figure 4 (a) shows a plot of $\mathscr{R}_c$ versus ω. The rest are plots of the number of infected in compartments I_1, I_2, H, U and the infection curve, for (i) $\omega = 1$; (ii) $\omega = 0.5$; (iii) $\omega = 0.2$. The other parameters for the simulations are those estimated for Sierra Leone.

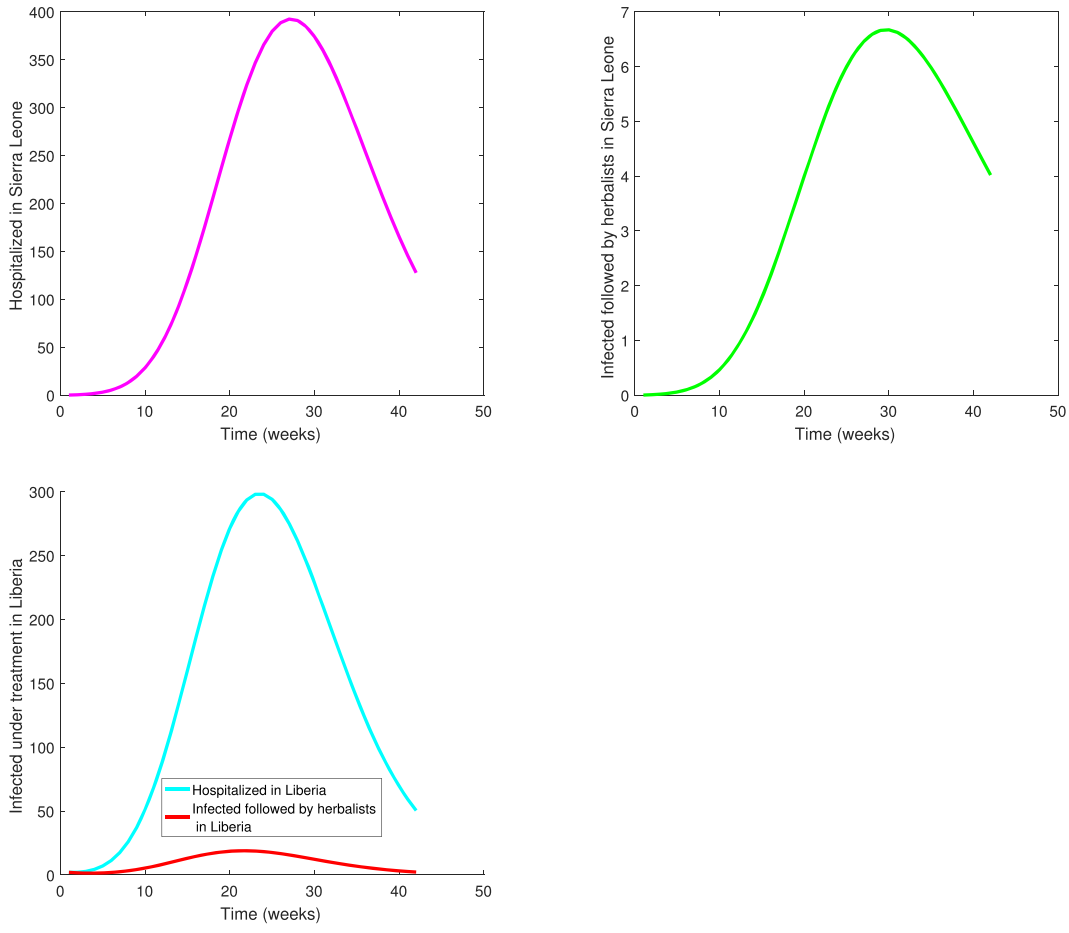

FIG. 5. Infected individuals in compartments H and U, in Sierra Leone and Liberia. Parameter values used are presented in Table 2.

number of infected cases treated by traditional healers in the two countries. In this figure, we plot H and U on the same graph for Liberia, while for Sierra Leone, we have separate graphs. The reason for these separate graphs for Sierra Leone being that the gap between the number of infected people who visited hospitals (H) and those who visited traditional healers (U) is too large. Fig. 5 suggests that the population will have more propensity to go to hospitals than to traditional healers. Moreover, it suggests that misconceptions about EVD were more pronounced in Liberia than in Sierra Leone. This is probably as a result of sensitization campaigns in favour of visiting modern health facilities than traditional healers homes.

5.2 *Awareness campaigns*

The aspect of awareness campaigns was incorporated into Model (2.1). We formulated our model in such a way that (i) unaware people are encouraged to go to hospitals. The parameters of interest here are σ_u, σ_i and σ_s. (ii) there is a reduction in the number of contacts between susceptible and infected

individuals. This was introduced with the parameter ε. We focus on the impact of the above awareness parameters on the control reproduction numbers, as well as their influence on the dynamics of the infected individuals. Figure 6(a), 7(a), 8(a), 9(a) shows plots of $\mathscr{R}_c$ against $\sigma_u, \sigma_i, \sigma_s$ and ε, respectively. They show the decreasing trend of $\mathscr{R}_c$. Moreover, the plots suggest that $\mathscr{R}_c$ is less sensitive to changes of $\sigma_u, \sigma_i, \sigma_s$ than to the parameter ε.

During an EVD outbreak, awareness campaigns are meant to fight against misconceptions and its attendant effect on human behaviour. Through these campaigns, the target population is not only assisted in making informed but right choices. The effect of awareness campaigns in our model on the dynamics of individuals in I_1, I_2, H, U classes and the total infected population $(I_1 + I_2 + H + U)$ in Sierra Leone are presented in Figs 6, 7, 8 and 9. We increase the estimated awareness parameters $\sigma_u^*, \sigma_i^*, \sigma_s^*$ and ε^* of $\sigma_u, \sigma_i, \sigma_s$ and ε, respectively, by 10% and 20%, to observe the effect of the change.

We see that among the three awareness parameters $\sigma_i, \sigma_s, \sigma_u$, σ_s does better in reducing the disease intensity. This suggests that in the event of an EVD outbreak, awareness messages should target the susceptible population for behaviour change in an attempt to treat and mitigate the spread of the disease.

REMARK 2. According to our flow chart, keeping all other parameters fixed, as σ_i increases the number of infected persons in I_2 should decrease while the number in I_1 should increase. But, Fig. 7 shows that during about 15 weeks, the change of the values of σ_i has almost no effect on the dynamics of I_1 and I_2, but after this time, the number of infected in these two compartments decrease as the value of σ_i increase. The decrease of the infected population of compartment I_1 may be explained by the fact that the number $\sigma_i I_2$ individuals who become aware, reach the compartment I_1, where they are three times less infectious than when they were unaware (ν_2=3.0013). Figures 7, 8 and 9 highlight that the number of infected in all infectious compartments decreases as the awareness parameters increase, that is: the number of infected individuals decreases as people become more aware of the disease. However, Fig. 6 shows that the change of the value of σ_u is less sensitive to the dynamics of infected individuals, compared to the other awareness parameters. All the curves plotted in this case are merged. This weak sensitivity of σ_u may perhaps be explained by its weak values estimated in Sierra Leone and Liberia. Note that these weak values are in accordance with African realities, since it is very difficult to convince people who generally link diseases to witchcraft.

6. Sensitivity analysis

In order to evaluate appropriate control measures during EVD outbreaks, it is important to identify the parameters that are more influential or relevant and of practical significance in our model. To do so, we focus on the impact of parameter variation on the infected components assuming that each parameter is a random variable. In this process, a Latin Hypercube Sampling (LHS) scheme samples 1000 values for each input parameter using a uniform distribution. For each sample, System (2.1) is evaluated. Finally, Partial Rank Correlation Coefficients (PRCC) and corresponding p-values are computed, for the infected classes I_1, I_2, H, U and the total infected population (I_1+I_2+H+U). The PRCCs estimate are displayed in Table 4. We consider an estimated PRCC value significant if its value is either less than -0.05 or greater than $+0.05$, and the corresponding p-value is less than 5%. As seen from Table 4, the parameters (β, π, ν_2, ν_3), the disease-induced death rates (δ_1, δ_2, δ_u, δ_h) and the recovery rates (γ_1, γ_2, γ_h, γ_u) are the most sensitive parameters corresponding to the infected persons who are alive (I_1+I_2+H+U). The most significant parameter in terms of the impact of treatment choice here is treatment rate at hospital γ_h, with a PRCC estimate -0.1359, followed by the treatment rate at traditional healers homes γ_u with

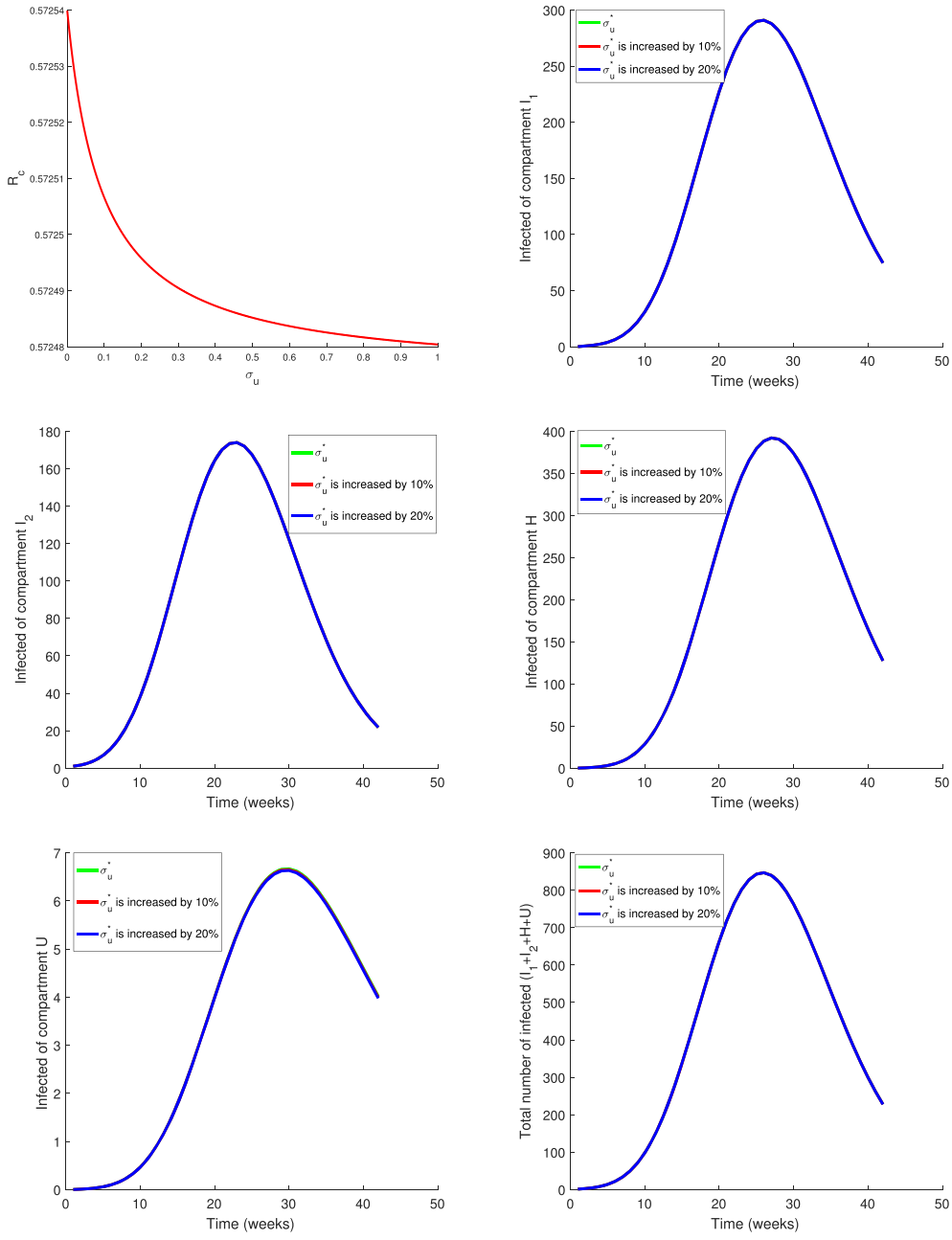

FIG. 6. Variation of σ_u Figure 6 (a) is a plot of $\mathscr{R}_c$ versus σ_u. The rest are curves of the infected in compartments I_1, I_2, H, U as well as the total number of infected $(I_1 + I_2 + H + U)$ when the parameter σ_u varies: (i) $\sigma_u = \sigma_u^*$; (ii) σ_u^* is increased by 10%; (iii) σ_u^* is increased by 20%. The other parameters are those estimated in Sierra Leone.

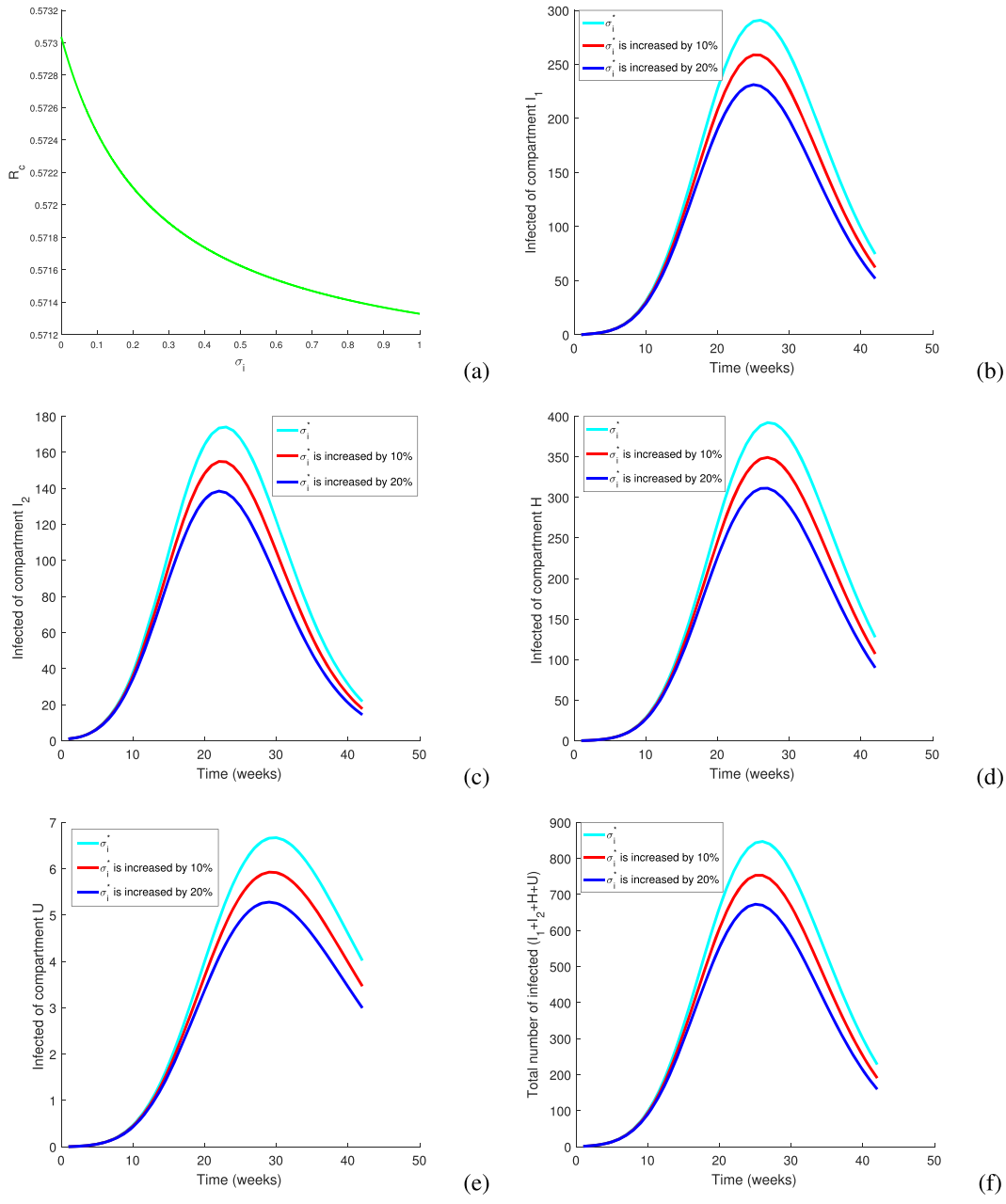

FIG. 7. Variation of σ_i The influence of σ_i on $\mathscr{R}_c$; I_1, I_2, H, U and $(I_1 + I_2 + H + U)$. (i) $\sigma_i = \sigma_i^*$; (ii) σ_i^* is increased by 10%; (iii) σ_i^* is increased by 20%. The other parameters are those estimated in Sierra Leone.

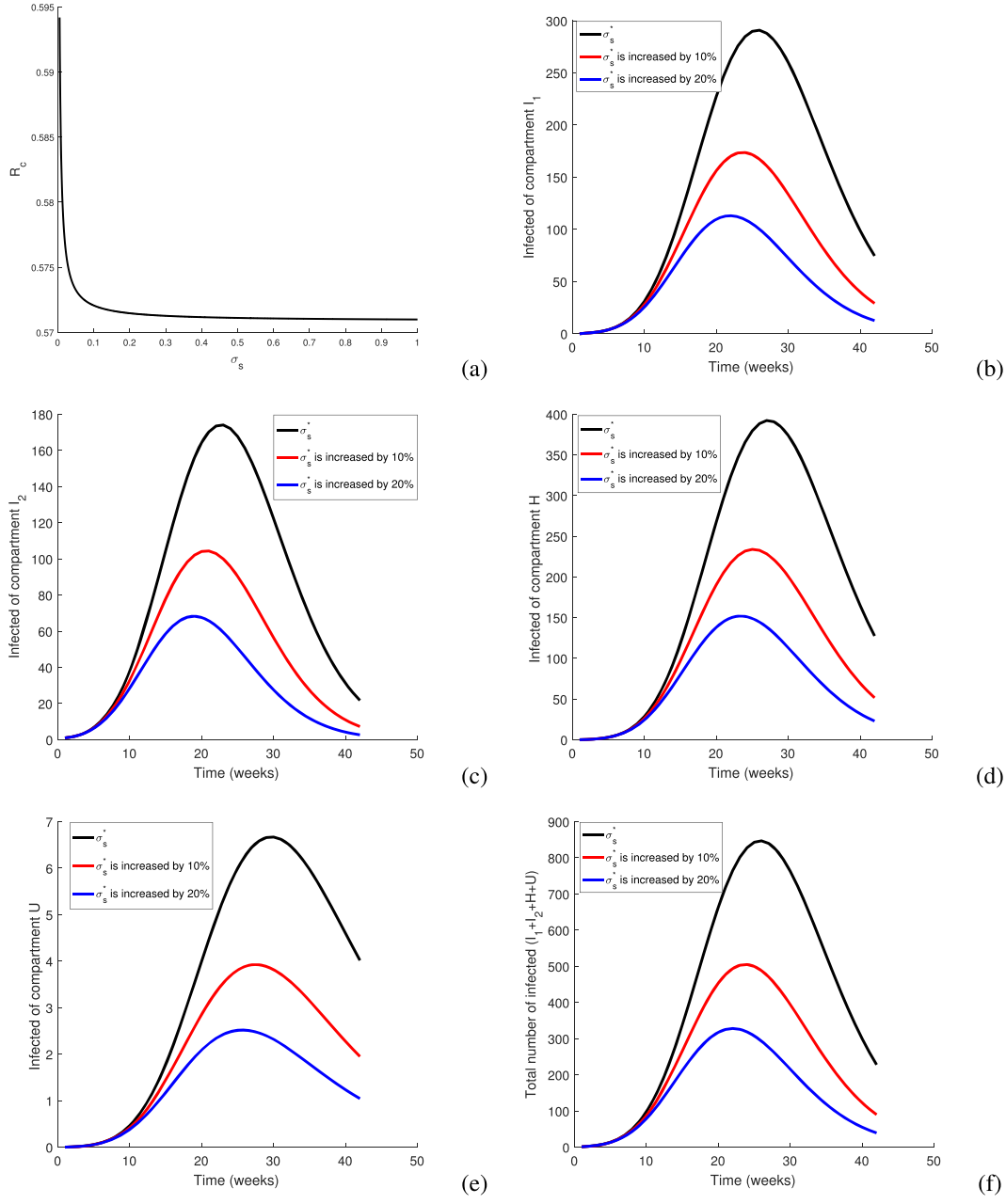

FIG. 8. Variation of σ_s The influence of σ_s on $\mathscr{R}_c$; I_1, I_2, H, U and $(I_1 + I_2 + H + U)$. (i) $\sigma_s = \sigma_s^*$; (ii) σ_s^* is increased by 10%; (iii) σ_s^* is increased by 20%. The other parameters are those estimated in Sierra Leone.

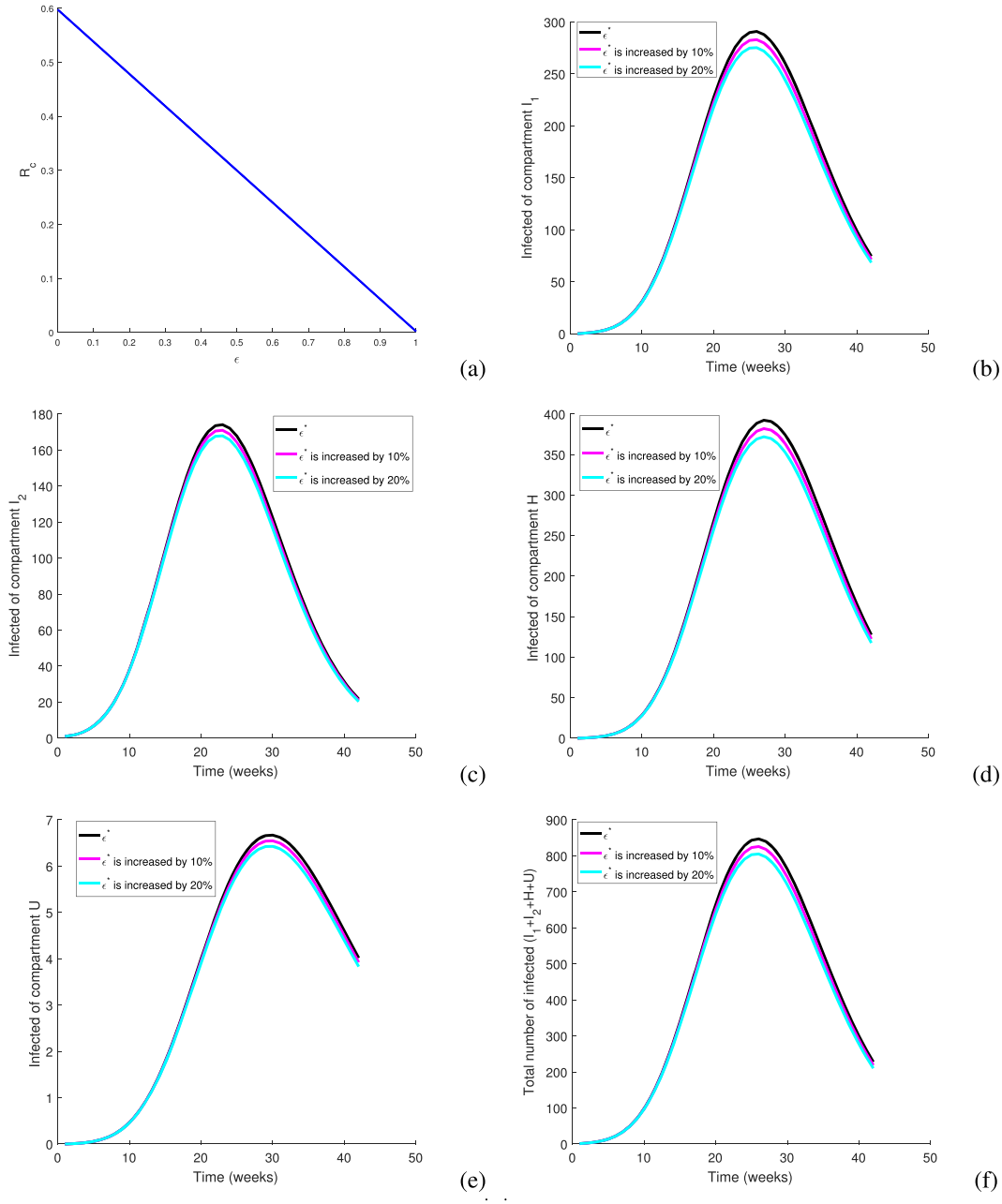

FIG. 9. Variation of ε The influence of ε on $\mathscr{R}_c$; I_1, I_2, H, U and $(I_1 + I_2 + H + U)$. (i) $\varepsilon = \varepsilon^*$; (ii) ε^* is increased by 10%; (iii) ε^* is increased by 20%. The other parameters are those estimated in Sierra Leone.

TABLE 4 *Partial rank correlation coefficient values for the model*

Parameters	Range	I_1	I_2	H	U	I_1+I_2+H+U
μ	0–1	−0.2036*	−0.1836*	−0.1700*	−0.1756*	−0.1470*
π	0–1	0.0370	−0.0300	0.0208	0.0092	0.0942*
ε	0–1	0.0466	−0.0696*	0.0183	0.0290	−0.0402
η_1	0–1	−0.1240*	−0.0042	−0.2070*	−0.0384	−0.0192
η_2	0–1	−0.0427	−0.1908*	−0.0011	−0.1727*	−0.0471
β	0–1	0.1174*	0.1897*	0.0038	0.0405	0.1259*
p	0–1	−0.0155	0.0560*	−0.0479	0.0438	0.0306
q	0–1	0.0310	−0.0025	0.0158	−0.0122	−0.0134
δ_1	0–1	−0.1787*	−0.0304	0.0389	0.0326	−0.1236*
δ_2	0–1	−0.0589*	−0.1955*	0.0507*	0.0108	−0.1257*
δ_u	0–1	0.0060	−0.0046	0.0193	−0.1701*	−0.0866*
δ_h	0–1	0.0092	−0.0203	−0.1728*	−0.0183	−0.0826*
ν_1	0–1	0.0038	0.0311	0.0263	0.0272	0.0020
ν_2	1–2	0.0147	0.0435	0.0394	0.0287	0.0524*
ν_3	1–2	0.0687*	0.0085	0.0188	0.0118	0.0657*
ν_4	1–2	0.0668*	0.0050	0.0353	0.0069	0.0278
σ_s	0–1	−0.0303	−0.0681*	0.0333	−0.0429	−0.0236
σ_i	0–1	−0.1628*	−0.2246*	0.0112	−0.0966*	−0.0495
σ_u	0–1	−0.0140	0.0036	0.1867*	−0.1771*	−0.0146
γ_1	0–1	−0.1849*	−0.0280	−0.0181	−0.0210	−0.0990*
γ_2	0–1	−0.0367	−0.1316*	−0.0461	−0.0029	−0.1573*
γ_h	0–1	−0.0435	0.0104	−0.1987*	−0.0014	−0.1359*
γ_u	0–1	−0.0258	−0.0294	−0.0302	−0.1917*	−0.1229*
b	0–1	−0.0103	−0.0366	−0.0375	−0.0275	−0.0036

*Indicates a most sensitive parameter.

the estimate −0.1229. This highlights the importance of awareness campaigns aiming at encouraging people to seek modern treatment at hospitals as formulated in our model.

In our model, the parameters q and σ_u represent the proportion of unaware infected individuals moving to hospital and the rate at which unaware infected persons treated by traditional healers move to the hospital, respectively. Awareness/educational campaigns will potentially increase the value of these parameters. It is important to have an idea of how large the region where $\mathscr{R}_c < 1$ is in the (σ_u, q) space. This is obtained by plotting the level curves of $\mathscr{R}_c$ in the (σ_u, q) space as shown in Fig. 10.

We see that for $\sigma_u \geq 0.13$, $\mathscr{R}_c < 1$ irrespective of the value of q. This suggests that the EVD will die out of the population. Comparing this value (0.13) to the fitted value (0.0041) in Sierra Leone indicates that, as a control measure, the awareness/educational campaigns aiming at advising people to abandon traditional healers homes and visit hospitals have to be intensified.

7. Discussion

Some studies have underscored the role of education in the control of the EVD outbreaks Djiomba Njankou & Nyabadza (2017); Levy *et al.* (2017); Dautel & Agyingi (2021). In this paper, we have formulated a mathematical model that does not only take into account the importance of awareness about the disease but more importantly the role of traditional medicine in the treatment of the EVD.

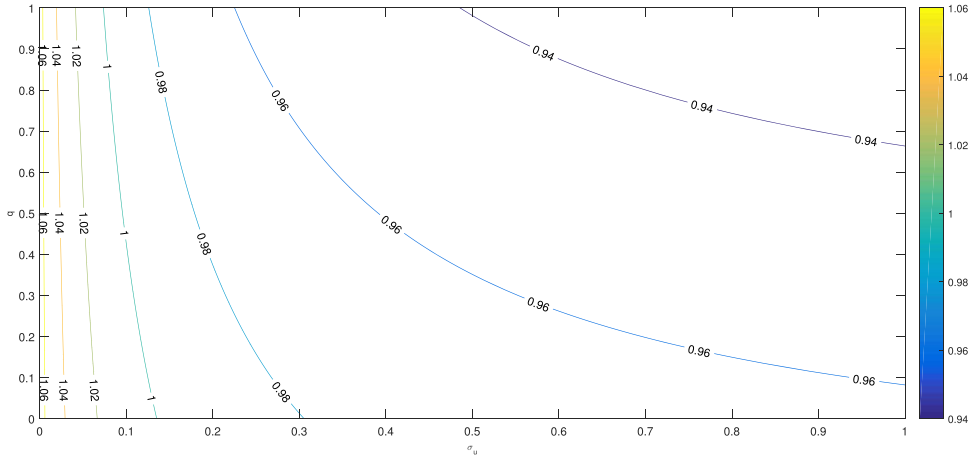

FIG. 10. Level curve of $\mathscr{R}_c$ in the (σ_u, q) space, with $\beta = 0.8$ for Sierra Leone. The other parameters are as in Table 2.

Through our model, we are able to assess the effect of awareness of the population on the dynamics of EVD. We found out that as time evolves the number of hospitalized individuals increases more rapidly than the number of infected cases treated by traditional healers in Sierra Leone and Liberia (the two countries chosen for the study). In addition, the population had high tendency to go to hospitals than to traditional healers. This is most likely because sensitization campaigns are in favour of visiting modern health facilities than traditional healers homes. Moreover, we prove that the more infected persons will choose traditional medicine, the more EVD will be spread, since people who seek care from traditional healers are neither isolated, nor educated on preventive measures.

Finding the target population for sensitization during an EVD outbreak is fundamental. Sending out awareness messages to the target audience may lead to a significant reduction in the spread of the disease. Results from our analysis of the model suggest that in the event of an EVD outbreak, awareness messages should target the susceptible population for behaviour change in an attempt to treat and mitigate the spread of the disease. As a control measure, awareness campaigns aiming at advising people to abandon traditional healers homes and visit hospitals have to be intensified for Sierra Leone.

On the theoretical perspective of our model, we showed that when the control reproduction number $\mathscr{R}_c$ is less than a threshold value $\mathscr{R}_c^c$, the unique equilibrium of the model is the disease-free equilibrium. Moreover, we derived a threshold value $\mathscr{T}_0$ which ensures the global asymptotic stability of the disease-free equilibrium in some cases when it is less than 1. When $\mathscr{R}_c$ is between $\mathscr{R}_c^c$ and 1, the model exhibits a backward bifurcation phenomenon, which may be as a result of having two groups of individuals and different modes of treatment Ouemba Tassé *et al.* (2024). In the case of a backward bifurcation, it is not enough to reduce $\mathscr{R}_c$ to a value less than 1 to ensure the global elimination of the disease. Furthermore, when the control reproduction number is greater than 1, the disease-free equilibrium is unstable, the disease is persistent and there exists a unique locally asymptotically stable EE.

Through sensitivity analysis, we showed that the EVD transmission rates are the most influential parameters and that awareness campaigns can substantially mitigate the intensity of EVD. Numerical simulations are carried out in order to validate our model using the reported data for the 2014–2016 outbreak in Sierra Leone and Liberia. The model gives a good fit, and a control reproduction number equal to 0.5725 in Sierra Leone and 0.8340 in Liberia.

However, the role of traditional medicine together with traditional practices is important and should not be underestimated in the fight against several diseases in Africa Manguvo & Mafuvadze (2015). In order to mitigate the negative impact of this kind of medicine for the treatment of EVD, there is a need for collaboration between traditional healers and modern medical officials in order to reduce suspicion and lack of trust.

Despite the relevance of this study, which helps to understand the impact of the awareness of the population, as well as the potential impact of the traditional medicine on the dynamics of EVD, the model has some limitations. Some major limitations are that it has not taken into account the difference in levels of education, sex, religion and age. In fact, the study in Cénat *et al.* (2021) revealed that men have better knowledge of EVD than women, people aged between 25 and 34 years have better knowledge than those aged from 18 to 24 years. This may help to better target the groups of people for whom awareness programs should be intensified and how to address them.

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Conflict of interest

The authors declare no conflict of interest.

Data availability statement

The data used in this study is provided by WHO and is available at: https://apps.who.int/gho/data/node.ebola-sitrep.quickdownloads.

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A. Appendices

A.1 *Appendix A: proof of Proposition 1*

Let's suppose that $S_2(0) > 0$. The integration from 0 to t of the first equation of (2.1) gives

$$S_2(t) = \left[S_2(0) + \int_0^t (1-p)\pi exp\left(\int_0^s (\lambda(u) + \tau_s)du\right) ds\right] \times exp\left(\int_0^t -(\lambda(u) + \tau_s)du\right).$$

Thus,

$$S_2(t) > 0, \quad \forall\, t > 0.$$

One can show similarly that $S_1(t) > 0, \forall\, t > 0$ if, in addition, $S_1(0) > 0$.

Assume that $I_1(0) \geq 0, I_2(0) \geq 0, H(0) \geq 0, U(0) \geq 0, D(0) \geq 0$ and $R(0) \geq 0$. If, for instance, I_2 becomes zero at a time t_2 before I_1, H, U, D and R become zero, from the fourth equation of (2.1), we get $\dot{I}_2(t) = \lambda(t)S_1(t) \geq 0$ at t_2. Thus, $I_2(t)$ is a non-decreasing function of t at t_2. Hence, $I_2(t)$ stays non-negative. Similarly, it can be shown that I_1, H, U, D and R remain non-negative for non-negative initial conditions.

A.2 *Appendix B: proof of Proposition 2*

Suppose $N(0) \leq \dfrac{\pi}{\mu}$ and $D(0) \leq \dfrac{\pi(3\mu + \delta_1 + \delta_2 + \delta_u)}{\mu b}$.

Simple computations show that

$$\begin{aligned} \dot{N}(t) &= \pi - \mu N(t) - \delta_1 I_1(t) - \delta_2 I_2(t) - \delta_u U(t) - \delta_h H(t) \\ \dot{N(t)} &\leq \pi - \mu N(t). \end{aligned}$$

Following Gronwall lemma, one has

$$N(t) \leq \frac{\pi}{\mu} + \left(N(0) - \frac{\pi}{\mu}\right) e^{-\mu t}.$$

Hence

$$N(t) \leq \frac{\pi}{\mu}.$$

Moreover, by the seventh equation of (2.1), since $I_1(t) \leq \dfrac{\pi}{\mu}, I_2(t) \leq \dfrac{\pi}{\mu}$ and $U(t) \leq \dfrac{\pi}{\mu}$, one has

$$\dot{D}(t) \leq \frac{\pi(3\mu + \delta_1 + \delta_2 + \delta_u)}{\mu} - bD(t).$$

Using the Gronwall lemma once more,

$$D(t) \leq \frac{\pi(3\mu + \delta_1 + \delta_2 + \delta_u)}{\mu b} + \left(D(0) - \frac{\pi(3\mu + \delta_1 + \delta_2 + \delta_u)}{\mu b}\right) e^{-bt}$$

Therefore,

$$D(t)) \leq \frac{\pi(3\mu + \delta_1 + \delta_2 + \delta_u)}{\mu b}, \quad \forall\, t > 0.$$