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Optimal control of an epidemic model with treatment in the presence of media coverage

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ABSTRACT

During large scale outbreaks of infectious diseases, it is imperative that media report about the potential risks. Because media reporting plays a vital role in disseminating crucial information about diseases and its associated risk, understanding how media reports could influence individuals' behavior and its potential impact on disease transmission dynamics is important. A mathematical model within an optimal control framework of a generic disease, accounting for treatment and media reporting of disease-induced deaths is formulated. Due to the complexity of choosing the best media function, our goal is to attempt to address the following research question: what is the effect of the media-induced functional response on mitigating the spread of the disease? Connecting the functional forms to the control problem is an approach that is not very developed in the literature. Thus, this study analyses the effect of different incidence functions on disease transmission, and the qualitative nature of epidemic dynamics by carrying out optimal control analysis using three different contact rates and a media function that is dependent on the number of deaths. Theoretical analyses show that the functional forms of the effective contact rate have no effect on initial disease transmission. Time-dependent controls for treatment and vaccination with a constant effective contact rate are incorporated to determine optimal control strategies. Numerical simulations show the short-term impact of media coverage on mitigating the spread of the disease, and it is observed that with three incidence functions used, the qualitative nature of the controls remains the same. The effective contact rates are graphically shown to have a population-level effect on the disease dynamics as the number of treated and recovered individuals could be significantly different. Finally, it is shown that treatment of infectives should be at its maximum rate for a longer period compared to vaccination, while concurrent implementation of vaccination and treatment is more impactful in mitigating the spread of the disease. Thus, it is imperative that media reports and health policy decision making on infectious diseases are contextualized.

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Introduction

Infectious diseases have for centuries ranked with wars and famine as major challenges to human progress and survival [1], and they are responsible for a quarter of all deaths in the world annually [2]. Some diseases exhibit distinct features such as rapid spatial spread and self-control [3]. These features, associated with the increasing trend of globalization and the development of information technology, are expected to be shared by other emerging/re-emerging infectious diseases. It is therefore important to refine classical mathematical models to reflect these features by adding the new dimensions of massive news coverage that have great influence not only on the individual behaviors but also on the formation and implementation of public health interventions and control policies [3]. There is great potential for thinking about informational asymmetries in the context of disease as individuals living in an area of low prevalence of West Nile virus for instance are relatively less likely to protect themselves from mosquitoes [4].

Against a constant background of established infections, outbreaks of new and old infectious diseases periodically emerge, greatly magnifying the global burden of infections [1]. While mathematical models play a major role in guiding policy decisions during an epidemic, media reports could help in shaping opinions, attitudes and perspectives [5]. Several mathematical models of infectious diseases have largely ignored the impact of media on the disease transmission, however, the importance of media has gradually been recognized and incorporated in mathematical models [3,6–12], to name a few and the references therein. Aldila [13] analyzed an optimal control problem by considering media campaign and rapid testing as time-dependent control. Misra et al. [14] investigated the extent to which the provision of awareness and behavioral changes affect the epidemic trajectory by including awareness campaigns explicitly as a separate dynamic variable in their model. Using optimal control theory, they obtained an optimal implementation rate of awareness campaigns to mitigate the spread of the disease.

Awareness through media and education play a tremendous role in mounting prevention of infectious disease [15,16]. The spread of information and its impact on epidemic outbreaks has been discussed in [15]. Public health educational/information campaigns can help in slowing down epidemics [17,18], while public awareness can play a key role in limiting disease outbreaks [15,16,19]. Even though the media coverage/news reporting is not the intrinsic factor that decides if the disease will break out, it has great impact on the pattern and scale of the transmission [3]. What factors influence people to change their behavior and how the provision of information influences individual's risk perception and shapes the evolution of epidemics are difficult and complex. An example of the complexity of such dynamics is the 1994 outbreak of plague in India which after the announcement of the disease, many people fled the state of Surat in an effort to escape the disease, potentially carrying it to other parts of the country [20]. In recent years, there has been a great deal of attention on the potential impact of media coverage on disease transmission dynamics, see [21,22] and the references therein. Media reporting does not impact initial disease transmission [21], but could affect the disease prevalence [8]. It has been shown that the contact rate is the most sensitive parameter with regards to the basic reproduction number (which is independent of the media function since at the onset of the epidemic, there is no media report), while the recovery rate is crucial to the disease prevalence [21]. Also, several individuals could be infected when there is no media report, that is, accounting for media function has a potential implication on the long-term dynamics of the disease. Thus, the choice of the media function in a modeling framework is critical in the long run after the disease outbreak [21]. Hamid and Sinha [23] showed that the effects of media coverage on the transmission of a vector-borne disease may decrease the peak value of the infectives or the average number of the infectives, while parameters describing media coverage have a significant influence on the spatial pattern of the disease [24].

Our objective therefore is to formulate an epidemic model that includes treatment and relevant media detail, and use optimal control methods to assess the impact of media coverage when deaths are reported. We begin with a description of the model and underlying assumptions in Section "Model Framework". The proposed model is analyzed in Section "Analysis of the model". The optimal control is investigated in Section "The Optimal Control Problem", with numerical simulations carried out to support the analytical findings.

Model framework

The active population is subdivided into various classes according to individuals' disease status, namely: susceptible $S(t)$, infectious $I(t)$, individuals receiving treatment $T(t)$, recovered individuals $R(t)$. The individuals who died from the disease are accounted for the purpose of news reporting and represented by $D(t)$. The total populations given by $N(t) = S + I + T + R$ is non-constant. Since we are interested in controlling the disease in the long-term, rather than preventing outbreaks altogether, we derive a model with recruitment. To describe short-term spread of diseases with a short course of infection and lifetime immunity (such as influenza), recruitment of infectives may be neglected. Susceptible individuals are recruited at a constant rate. We assume that recovered individuals lose immunity and re-enter the susceptible class at a fixed rate δ. Individuals under treatment recover at a constant rate κ (which includes both natural recovery and recovery due to treatment). The parameter λ accounts for treatment efficacy, and consequently, death due to the disease for individuals under treatment is reduced by a factor of $(1-\lambda)$. The natural and disease-induced death rates are μ and α, respectively. We note that when disease-induced deaths cannot be neglected, the population dynamics is coupled with the epidemic process [25]. Infected individuals are treated at a constant rate τ without media coverage effect. From the model flow diagram in Fig. 1 and the aforementioned, we establish the following system of non-linear ordinary

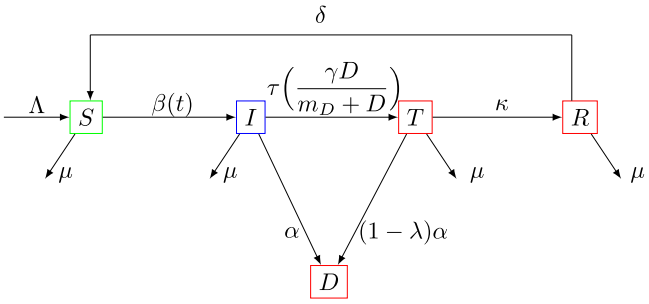

Fig. 1. The compartment flow diagram of the model.

differential equations for our caricature model.

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \beta(t)SI + \delta R - \mu S,\\
\frac{dI}{dt} &= \beta(t)SI - \tau\left(1 + \frac{\gamma D}{m_D + D}\right) I - (\mu + \alpha)I,\\
\frac{dT}{dt} &= \tau\left(1 + \frac{\gamma D}{m_D + D}\right) I - (\mu + \kappa)T - (1 - \lambda)\alpha T,\\
\frac{dR}{dt} &= \kappa T - (\mu + \delta)R,\\
\frac{dD}{dt} &= \alpha I + (1 - \lambda)\alpha T,
\end{aligned} \tag{1}$$

with the following non-negative initial conditions $S(0) \geq 0,\ I(0) \geq 0,\ T(0) \geq 0,\ R(0) \geq 0,\ D(0) \geq 0$. The increased treatment of infected individuals is modeled as a Holling type II functional response $\frac{\gamma D}{m_D + D}$, so that it achieves a saturation level, after which there is little benefit from treatment because the disease is almost under control. In fact, the term $\frac{\gamma D}{m_D + D}$ measures the increment of the treatment rate when deaths are reported (media coverage), while $\gamma = 0$ reflects the common situation when media coverage is not accounted for, m_D represents the half saturation constant for the reporting of deaths. The intuitive idea of considering deaths is that deaths are more news-worthy and media coverage in practice does not depend only on prevalence (infected). The potential effect of media covering the reported number of deaths will in a broad sense (implicitly) contribute to reducing the transmission rate $\beta(t)$, though $\beta(t)$ is not explicitly a function of the deaths. While the effect of media reporting is incorporated into the system (1), some authors have represented the media by its own specific ordinary differential equation [26].

Because news coverage gets diluted, and the signal triggered by mass reporting at the onset of an epidemic fades away after some time, we consider the potential beneficial effect of media reporting case fatalities on disease dynamics, Fig. 2(a), and the treated class, Fig. 2(b). From these two Figs. 2(a) and 2(b) generated using $\beta(t) = \beta_0$, positive media coverage to inform the public could have a short term benefit in mitigating the spread of infectious disease.

Analysis of the model

The system (1) is non-autonomous, since β is time-dependent, its long-time average exists and is given by (see [25,27])

$$\langle\beta\rangle = \lim_{t\to\infty} \frac{1}{t}\int_0^t \beta(s)ds$$

provided that $\beta \sim \mathcal{O}(t)$ for $t \gg 1$. Having a β value that can change implies ambiguity in the definition of an epidemic. For instance, if β is initially very high but then declines dramatically, then one might see an initial increase in infected persons followed by a long-term decline. The question here then becomes, is it an epidemic or not? It is an epidemic in the sense that infections increase deterministically and then decrease, but it is not an epidemic in the sense that an elevated value of β through the outbreak would provide a much larger epidemic curve. To address the latter, because the impact of very large values of β could potentially be misleading, we consider $\langle\beta\rangle = \beta_0$ to be a constant (because its long-term average is a limiting value as $t \to \infty$). We shall show analytically in Subsection "Incorporating functional forms for $\beta(t)$" that the expression of the basic reproduction number is unchanged irrespective of the functional form of the disease effective time-dependent contact rate $\beta(t)$, a bounded and continuous function. In Subsection "Connecting $\beta(t)$ functional forms to the control problem", these results are then connected to the optimal control problem by graphically showing that the shape of the figures are almost unchanged when any of these forms of the contact rate is used. It is however important to note that this simplistic averaging of $\beta(t)$ to obtain some interesting results is quite different from having $\beta(D(t))$ or $\beta(S(t), I(t))$ and doing some averaging. The latter two cases will not be discussed further. Also, it is important to note that the contact rate $\beta(t)$ does not explicitly reflect the media effect, but it is time varying due to some potential changes in the effective contact rate due to the effect of media reporting. While modeling factors that influence people to change their behavior

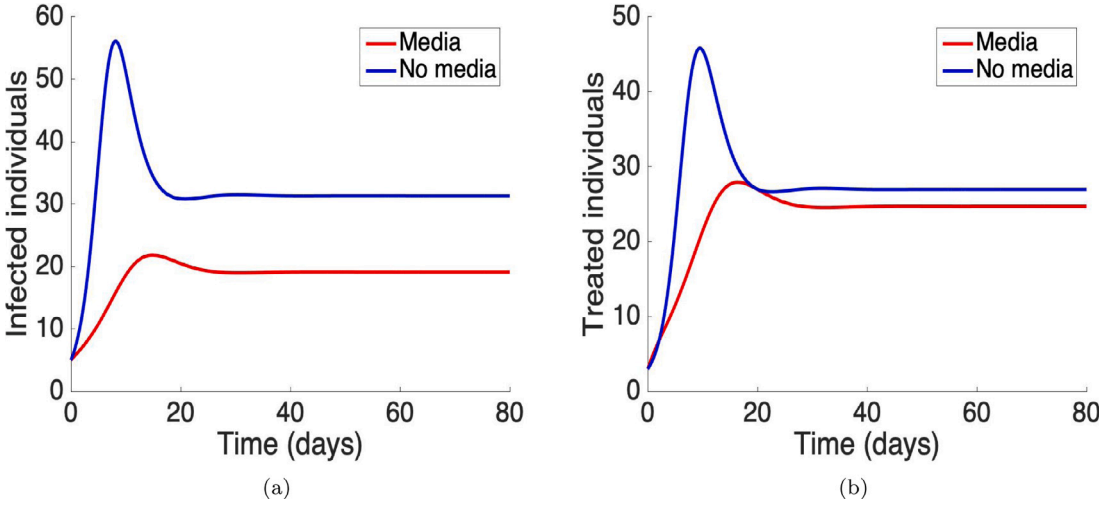

Fig. 2. Impact of media coverage on case fatalities and the treated class. (a) Impact of media coverage on $I(t)$; and (b) Impact of media coverage on $T(t)$.

is a daunting task, the time-dependent contact rate can be interpreted as an individual's reaction to media (behavioral change in response to media reporting of an outbreak, and/or seasonality) [9,28]. While all disease prevalence levels decrease or approach an endemic equilibrium with recruitment, the long-term behavior is also important even though a primary concern is when $\beta(t) \ll 1$, and then $\beta(t)$ later increases to induce an epidemic, then time should just be shifted so that $t = 0$ coincides with the epidemic onset.

The solution of the underlying deterministic model is always within the positive and invariant octant because either the boundary hyperplane is invariant ($S = 0$), or the trajectories point inside on the boundary ($I = 0, T = 0, R = 0, D = 0$). The model is epidemiologically and mathematically well-posed in the compact set (which is positively invariant and attracting)

$$\Omega = \left\{ (S, I, T, R) \in \mathbb{R}^4_+ \, : \, N \leq \frac{\Lambda}{\mu} \right\}.$$

The disease-free equilibrium (DFE) is given by

$$E^0 = (S^0, I^0, T^0, R^0, D^0) = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0 \right).$$

From the study of most autonomous epidemic models, it is well-known that the disease can cause an epidemic if and only if the basic reproduction number (that is, the expected number of secondary cases generated by a primary case in a fully susceptible/naive population) is greater than unity [29]. For the non-autonomous system where the time-varying parameter $\beta(t)$ is replaced by its long-term average, the basic reproduction number is given by

$$\mathcal{R}_0 = \frac{\langle \beta \rangle \Lambda}{\mu(\tau + \mu + \alpha)}. \tag{2}$$

$\mathcal{R}_0$ is the mean basic reproduction of the time-average system because the long-term average $\langle \beta \rangle$ is a constant. The following result holds (cf. Theorem 2 in [29]).

Lemma 1. *The disease-free equilibrium E^0 is locally asymptotically stable if $\mathcal{R}_0 < 1$, and unstable otherwise.*

The proof is immediate since the eigenvalues of the system (1) evaluated at E^0 are 0, $-\mu$, $-(\mu + \delta)$, $-(\mu + \kappa + \alpha(1 - \lambda))$, and $\langle \beta \rangle \Lambda - \mu \left(\tau_1 + \mu + \alpha \right)$. Next, for $\gamma = 0$ (i.e., when media coverage is not accounted for), we show that $I(\infty) \to 0$ whenever $\mathcal{R}_0 < 1$. The explicit solution of the second equation of system (1) when $\gamma = 0$ is given by

$$\begin{aligned} I(t) &= I(0) e^{\left(\frac{\langle \beta \rangle \Lambda}{\mu} - (\tau_1 + \mu + \alpha) \right) t}, \\ &= I(0) e^{\left(\frac{\langle \beta \rangle \Lambda}{\mu(\tau_1 + \mu + \alpha)} - 1 \right) (\tau_1 + \mu + \alpha) t}, \\ &= I(0) e^{(\mathcal{R}_0 - 1)(\tau_1 + \mu + \alpha) t}. \end{aligned} \tag{3}$$

From Lemma 1, whenever $\mathcal{R}_0 < 1$, $\lim_{t \to +\infty} I(t) = 0$. Hence, the disease-free equilibrium E^0 is locally asymptotically stable. The stability of the DFE implies that $\langle \beta \rangle < \frac{\mu\tau + \mu + \alpha}{\Lambda}$. However, if $\beta > \langle \beta \rangle$ for some large interval of time, then, the reproduction number could be significantly larger than one, and consequently, the DFE becomes unstable.

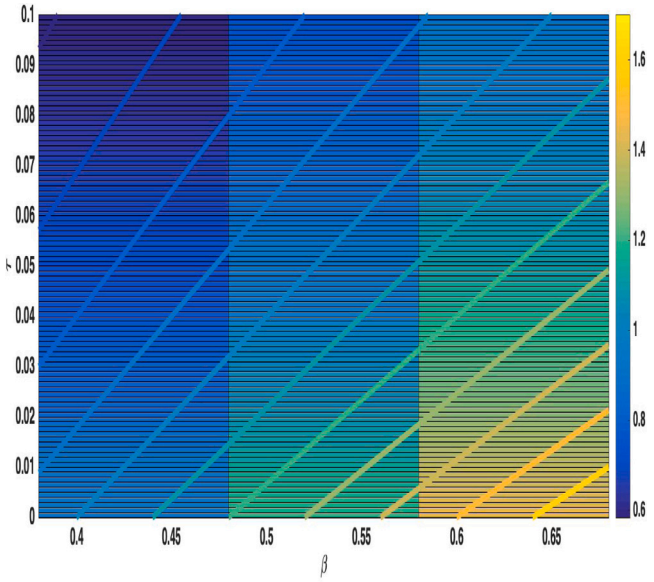

Fig. 3. Impact of the disease transmission and treatment rates $\langle\beta\rangle$ and τ in the $R_0(\langle\beta\rangle, \tau)$-plane, $(0.6 < R_0 < 1.8)$. (For interpretation of the references to colorin this figure legend, the reader is referred to the web version of this article).

Figs. 3 depicts the impact of the disease transmission and treatment rates $\langle\beta\rangle$ and τ, represented by the color gradient from dark blue (high reduction) to yellowish (low reduction), with isoclines (straight lines) represented by a contour plot.

Incorporating functional forms for $\beta(t)$

The transmission dynamics is crucial to understanding and designing implementation of control measures during epidemics (e.g., influenza) or pandemics (e.g., COVID-19). We consider three cases with different functional forms of the contact/transmission rate β. As noted above, the time-dependent contact rate $\beta(t)$ does not explicitly reflect the media effect (the latter is indirectly reflected), as media coverage of deaths directly impacts on the treatment rate. That is, as noted above, $\beta(t)$ herein does not explicitly reflect the media effect which has been considered by some authors [8]. While this is a limitation, in a future study, we shall consider the explicit dependence on I and D of the transmission parameter $\beta(I, D)$, because media coverage of deaths would not only motivate infected individuals to seek treatment, but it could also influence susceptibles to avoid infection (by limiting or minimizing contacts).

Case 1: $\beta(t) = \beta_0\left[1 + \sin\left(\frac{2\pi}{365}t + q\right)\right]$ is a periodic function. In practice, the impact of the intervention is not instantaneous, therefore it is realistic to assume that between the time of the onset of the intervention to the time of full compliance, the transmission rate is periodic (the intervention can also be seasonal [25,27,28,30].

Case 2: $\beta(t)$ is a sigmoid function of the form

$$\beta(t) = \beta_0 + \frac{\beta_1 - \beta_0}{1 + e^{-qt}.}$$

Case 3: $\beta(t)$ is a monotonically decreasing function of the form

$$\beta(t) = \beta_0\left(1 + e^{-qt}\right).$$

A part from the seasonal periodic form, the other two functional forms (cases 2 and 3) are transient between some initial β_i and β_j on a timescale $\frac{1}{q}$. Even though we are concerned with timescales long enough that recruitment should be considered, all these functional forms of the contact rate are used to investigate whether they impact the expression of the basic reproduction number.

Using theory of dynamical systems, we show below that these functional forms do not affect the expression of the basic reproduction number, and consequently will not have any major effect of the disease control problem. This assertion agrees with our definition of the basic reproduction number in terms of the average, where the average of case 1 and 3 is β_0 while the average of case 2 is β_1. Thus, the reproduction number is insensitive to the functional form because it is averaged out.

Case 1: Suppose $\beta(t)$ is a periodic function of period w. In practice, the impact of the intervention is not instantaneous, therefore it is realistic to assume that between the time of the onset of the intervention to the time of full compliance, the transmission rate is periodic of period w.

Let $v(t) = I(t) - I_0$, then the linearized perturbed equation of the I compartment around the disease-free equilibrium is

$$\begin{cases} \dfrac{dv(t)}{dt} &= \beta(t)S^0(1+\varepsilon)v(t) - (\tau+\alpha+\mu)v(t), \quad \varepsilon \in (0\ ,\ 1), \\ v(0) &= 0 \in \mathbb{R}, \end{cases} \tag{4}$$

where ε is an infinitesimal parameter, and $I_0 = 0$ at the DFE. The solution to Eq. (4) is given by

$$v_\varepsilon(t) = U_\varepsilon(t,t_0)v(t_0), \quad \forall\ t \geqslant t_0 \geqslant 0\ ,$$

where

$$U_\varepsilon(t,t_0) = e^{\int_{t_0}^{t}\left(\beta(s)S_\varepsilon^0 - (\tau+\alpha+\mu)\right)ds}, \quad \forall\ t,\ t_0 \geqslant 0, \quad \text{and} \quad S_\varepsilon^0 = S^0(1+\varepsilon).$$

Note that the evolution family $\left\{U_\varepsilon(t,t_0)\right\}_{t,t_0\in\mathbb{R}_+}$ is periodic in the sense that

$$U_\varepsilon(t+w,t_0+w) = U_\varepsilon(t,t_0), \quad \forall\ t,\ t_0 \geqslant 0.$$

Therefore, setting

$$U_\varepsilon(w) = \frac{\ln\left(U_\varepsilon(w,0)\right)}{w} = \frac{1}{w}\int_0^w \left(\beta(s)S_\varepsilon^0 - (\tau+\alpha+\mu)\right)ds,$$

then, for each $\eta < -U_\varepsilon(w)$, there exists $M := M(\eta) \geqslant 1$ such that

$$U_\varepsilon(w) \leqslant Me^{-\eta w} \quad \forall\ w \geqslant 0.$$

Thus, following the approach in [31], $U_0(w,0)$ is a constant and $\frac{d}{dw}U_0(w,0) = 0$, that is $\frac{d}{dw}\int_0^w \left(\beta(s)S^0 - (\tau+\alpha+\mu)\right)ds = 0$, and the basic reproduction number is given by (2).

Lemma 2. *Assume that $\mathcal{R}_0 < 1$. Then, there exists $\varepsilon_0 > 0$ such that $\mathcal{R}_\varepsilon < 1$ for all $\varepsilon \in (0,\varepsilon_0)$.*
Let $\eta \in (0, -\frac{ln\mathcal{R}_\varepsilon}{w})$ be given and fixed. Then, there exists $M := M(\eta) \geqslant 1$ such that

$$U_\varepsilon(t,t_0) \leqslant Me^{-\eta(t-t_0)} \quad \forall\ t,\ t_0 \geqslant 0.$$

Proof. Since $\lim_{\varepsilon\to 0}\mathcal{R}_\varepsilon = \mathcal{R}_0 < 1$, there exists $\varepsilon_0 > 0$ such that $\mathcal{R}_\varepsilon < 1$ for all $\varepsilon \in (0,\varepsilon_0)$. Thus,

$$\mathcal{R}_\varepsilon < 1 \Leftrightarrow \frac{\ln U_\varepsilon(w,0)}{w} = \frac{\ln \mathcal{R}_\varepsilon}{w} < 1.$$

The proof of the second part of the Lemma follows from the above discussion (under Eq. 1). The following result stated without proof will be used subsequently. □

Lemma 3. *Let $(S_0, I_0, T_0, R_0, D_0) \in \mathbb{R}_+^5$ be given. Then, for each $\varepsilon > 0$ there exists $t_0 := t_0(\varepsilon, S_0)$ such that*

$$0 \leqslant S^0 \leqslant (1+\varepsilon)S^0, \quad \forall\ t, \leqslant t_0.$$

Lemma 4. *Assume that $\mathcal{R}_0 < 1$, and let $\varepsilon \in (0,\varepsilon_0)$ be fixed, there exists $t_0 := t_0(\varepsilon, S_0)$ such that*

$$0 \leqslant I(t) \leqslant Me^{-\eta(t-t_0)}I(t_0), \quad \forall\ t \leqslant t_0,$$

with η and M the constants provided in Lemma 2.

Proof. From Lemma 3, there exists $t_0 := t_0(\varepsilon, S_0) > 0$ such that

$$0 \leqslant S^0 \leqslant (1+\varepsilon)S^0, \quad \forall\ t \leqslant t_0.$$

From the second equation (infective component) of the system (1), we have $\forall\ t \leqslant t_0$

$$\frac{dI}{dt} \leqslant \beta(t)S_\varepsilon^0 I - (\tau+\alpha+\mu)I.$$

So that

$$I(t) \leqslant U_\varepsilon(t,t_0)I_{t_0},$$

and the result follows from Lemma 2. As a consequence of Lemma 4, we have

$$\lim_{t\to+\infty} I(t) = 0 \quad \text{if} \quad \mathcal{R}_0 < 1.$$

Moreover, we have

$$0 \leqslant I(t) \leqslant M e^{-\eta(t-t_0)} I(t_0), \quad \forall \ t \leqslant t_0.$$

Since the system is uniformly persistent [30], set $T^\infty = \lim_{t\to+\infty} \sup T(t)$ and $T_\infty = \lim_{t\to+\infty} \inf T(t)$, we note that if $T^\infty = T_\infty$, then $\lim_{t\to+\infty} T(t)$ exists and

$$\lim_{t\to+\infty} T(t) = T^\infty = T_\infty.$$

Similarly, the same holds for $S(t), R(t)$ and $D(t)$. To prove this, we add an additional expression ζD to the death class where ζ accounts for the safe disposal of the deaths [32]. Because $\lim_{t\to+\infty} I(t) = 0$, we have $I^\infty = I_\infty = 0$. Thus, since $t \longmapsto T(t)$, $t \longmapsto S(t)$, $t \longmapsto R(t)$ and $t \longmapsto D(t)$, are bounded with bounded derivative, then

$$\begin{cases} 0 = -(\mu+\kappa)T^\infty - (1-\lambda)\alpha T^\infty, \\ 0 = \kappa T^\infty - (\mu+\delta)R^\infty, \\ 0 = \alpha I^\infty + (1-\lambda)\alpha T^\infty - \zeta D^\infty, \\ 0 = \Lambda + \delta R^\infty - \mu S^\infty. \end{cases} \tag{5}$$

The first equation of system (5) implies $T^\infty = 0$. Therefore, from the second equation, we have

$$0 - (\mu+\delta)R^\infty = 0, \quad i.e., \ R^\infty = 0.$$

Also, $D^\infty = 0$, and from the last equation, $S^\infty = \dfrac{\Lambda}{\mu} S^0$

We can use the same arguments to prove that

$$T_\infty = 0, \quad R_\infty = 0, \quad D_\infty = 0, \quad \text{and} \quad S_\infty = \frac{\Lambda}{\mu} S^0.$$

Thus,

$$\lim_{t\to+\infty} T(t) = 0, \quad \lim_{t\to+\infty} R(t) = 0, \quad \lim_{t\to+\infty} D(t) = 0 \quad \text{and} \quad \lim_{t\to+\infty} S(t) = \frac{\Lambda}{\mu} S^0. \quad \square$$

Case 2: Suppose $\beta(t)$ is a sigmoid function (that is, a general mathematical function that has an S-shaped curve, or sigmoid curve, which is bounded, differentiable, real, and has a non-negative derivative at each point and exactly one inflection point) of the form

$$\beta(t) = \beta_0 + \frac{\beta_1 - \beta_0}{1 + e^{-qt}}.$$

Then, $\forall \ t, \ t_0 \ \geqslant \ 0$, we have

$$\begin{aligned} U(t,t_0) &= e^{\int_{t_0}^{t} \left(\beta(s)S^0 - (\tau+\alpha+\mu)\right) ds}, \quad \text{and} \\ \ln U(t,t_0) &= \int_{t_0}^{t} \left(\beta(s)S^0 - (\tau+\alpha+\mu)\right) ds, \\ &= S^0 \int_{t_0}^{t} \beta(s) ds - (\tau+\alpha+\mu)(t-t_0), \\ \ln U(t+t_0,t_0) &= S^0 \int_{t_0}^{t+t_0} \beta(s) ds - (\tau+\alpha+\mu)t. \end{aligned}$$

Since

$$\int_{t_0}^{t+t_0} \beta(s) ds = \beta_0 t + (\beta_1 - \beta_0) \int_{t_0}^{t+t_0} \frac{1}{1+e^{-qs}} ds,$$

then

$$\begin{aligned} \frac{\ln(U(t+t_0,t_0))}{t} &= \beta_0 S^0 - (\tau+\alpha+\mu) + (\beta_1-\beta_0)S^0 \frac{1}{t} \int_{t_0}^{t+t_0} \frac{1}{1+e^{-qs}} ds, \\ &= \beta_0 S^0 - (\tau+\alpha+\mu) + (\beta_1-\beta_0)S^0 \frac{1}{t} \int_{t_0}^{t+t_0} \frac{e^{qs}}{1+e^{qs}} ds, \\ &= \beta_0 S^0 - (\tau+\alpha+\mu) + (\beta_1-\beta_0)S^0 \frac{1}{t} \frac{1}{q} \ln\Big(\frac{1+e^{q(t+t_0)}}{1+e^{t_0}}\Big). \end{aligned}$$

Hence,

$$\sup_{t_0\in\mathbb{R}} \frac{\ln(U(t+t_0,t_0))}{t} = \beta_0 S^0 - (\tau+\alpha+\mu) + (\beta_1-\beta_0)S^0 \frac{1}{t} \frac{1}{q} \sup_{t_0\in\mathbb{R}} \ln\Big(\frac{1+e^{q(t+t_0)}}{1+e^{t_0}}\Big).$$

As the function $t_0 \longmapsto \dfrac{1+e^{q(t+t_0)}}{1+e^{t_0}}$ is increasing, we have

$$\sup_{t_0\in\mathbb{R}} \ln\Big(\frac{1+e^{q(t+t_0)}}{1+e^{t_0}}\Big) = \lim_{t_0\to+\infty} \Big(\frac{1+e^{q(t+t_0)}}{1+e^{t_0}}\Big) = 0.$$

Consequently,

$$\sup_{t_0\in\mathbb{R}} \frac{\ln(U(t+t_0,t_0))}{t} = \beta_0 S^0 - (\tau+\alpha+\mu),$$

and

$$\lim_{t\to+\infty} \sup_{t_0\in\mathbb{R}} \frac{\ln(U(t+t_0,t_0))}{t} = \beta_0 S^0 - (\tau+\alpha+\mu). \tag{6}$$

The right hand side of Eq. (7) implies that for all $\eta < -\beta_0 S^0 + (\tau+\alpha+\mu)$, there exist $M := M(\eta) \geqslant 1$ such that

$$U(t,t_0) \leqslant M e^{-\eta(t-t_0)} \quad \forall\ t,\ t_0 \geqslant 0.$$

As in case 1 above, from [29], the basic reproduction number given by Eq. (2).

Case 3: When $\beta(t)$ is a monotonically decreasing function of the form

$$\beta(t) = \beta_0\left(1+e^{-qt}\right).$$

Then, $\forall\ t,\ t_0 \geqslant 0$, we have

$$\begin{aligned} U(t,t_0) &= e^{\int_{t_0}^{t}\left(\beta(s)S^0-(\tau+\alpha+\mu)\right)ds}, \quad \text{and} \\ \ln U(t,t_0) &= \int_{t_0}^{t}\left(\beta(s)S^0-(\tau+\alpha+\mu)\right)ds, \\ &= S^0\int_{t_0}^{t}\beta(s)ds-(\tau+\alpha+\mu)(t-t_0), \\ \ln U(t+t_0,t_0) &= S^0\int_{t_0}^{t+t_0}\beta(s)ds-(\tau+\alpha+\mu)t. \end{aligned}$$

Since

$$\int_{t_0}^{t+t_0}\beta(s)ds = \beta_0 t + \beta_0\frac{e^{-qt_0}}{q}\left(\frac{e^{qt}-1}{e^{qt}}\right),$$

then,

$$\frac{\ln(U(t+t_0,t_0))}{t} = \beta_0 S^0 - (\tau+\alpha+\mu) + S^0\frac{1}{t} + \beta_0\frac{e^{-qt_0}}{q}\left(\frac{e^{qt}-1}{e^{qt}}\right).$$

Consequently,

$$\lim_{t\to+\infty} \sup_{t_0\in\mathbb{R}} \frac{\ln(U(t+t_0,t_0))}{t} = \beta_0 S^0 - (\tau+\alpha+\mu). \tag{7}$$

The right-hand side of Eq. (7) implies that for all $\eta < -\beta_0 S^0 + (\tau+\alpha+\mu)$, there exists $M := M(\eta) \geqslant 1$ such that

$$U(t,t_0) \leqslant M e^{-\eta(t-t_0)} \quad \forall\ t,\ t_0 \geqslant 0.$$

From Eq. (7), the basic reproduction number is given by Eq. (2).

The optimal control problem

Understanding how to control and eliminate/eradicate infectious diseases is one of the main goals of mathematical epidemiology [25] as well public health. We investigate how to optimally implement two therapeutic measures, vaccination (with a 100% effective vaccine) and treatment to mitigate the spread of the disease. To this effect, we introduce into the system (8) a set of time-dependent control variables $(u_1(t), u_2(t))$ where

(a) $u_1(t)$ represents the vaccination of susceptible individuals, and
(b) $u_2(t)$ represents the treatment of infectives.

The model (8) with optimal control $(u_1(t), u_2(t))$ consists of the following non-autonomous system of non-linear ordinary differential equations.

$$\begin{aligned} \frac{dS}{dt} &= \Lambda - \beta_0 SI + \delta R - [\mu + u_1(t)]S, \\ \frac{dI}{dt} &= \beta_0 SI - u_2(t)\left(1+\frac{\gamma D}{m_D+D}\right)I - (\mu+\alpha)I, \\ \frac{dT}{dt} &= u_2(t)\left(1+\frac{\gamma D}{m_D+D}\right)I - (\mu+\kappa)T - (1-\lambda)\alpha T, \\ \frac{dR}{dt} &= u_1(t)S(t) + \kappa T - (\mu+\delta)R, \\ \frac{dD}{dt} &= \alpha I + (1-\lambda)\alpha T, \end{aligned} \tag{8}$$

subject to the non-negative initial conditions.

Our control problem is to minimize the number of symptomatic individuals as well as minimizing the cost of treatment via minimization of the following cost functional

$$J(u_1,u_2)=\int_0^{t_f}\left[AI+B_1u_1(t)S(t)+B_2u_1^2(t)+C_1u_2(t)\left(1+\frac{\gamma D}{m_D+D}\right)I(t)+C_2u_2^2(t)\right]dt, \tag{9}$$

subject to the differential equations (8), where t_f is the final time, and the coefficients, A, B_1, B_2, C_1, C_2 are balancing cost factors. This performance specification involves the number of symptomatic individuals over time, as well as the cost for implementing vaccination and treatment controls (u_1,u_2) respectively in susceptible and symptomatic individuals. The choice of a quadratic control function is because the positive balancing cost factors transfer the integral into monetary quantity over a finite period of time [33], as this has to do with the logistics of delivering arbitrarily large amounts of control (drugs or vaccines), while the non-linearity of the control efforts is chosen for technical reason. Thus, one expects the cost of the control actions to be a nonlinear function of the two controls, a reason for the choice of the linear terms with coefficients B_1 and C_1 for the part of costs of the actions depending on the number of individuals (like S and I respectively), with the quadratic terms representing the non-linearity. The total cost includes not only the consumption of drugs for every symptomatic individual, but also the cost of hospitalization, etc. Hence, the cost function is nonlinear. Herein, we used a quadratic function to measure the control cost. This objective functional is not new as numerous example could be found in the literature on epidemic control [34–42]. Thus, we seek to find an optimal control, u_1^*, u_2^*, such that the optimal control function

$$J(u_1^*,u_2^*)=\inf_{\mathcal{U}}\{J(u_1,u_2)\}, \tag{10}$$

where $\mathcal{U}=\{(u_1(t),u_2(t))\in(L^2(0,t_f))^2 \mid a\le u_1(t)\le b,\quad c\le u_2(t)\le d \quad , t\in[0,t_f]\}$ is the control set, and a,b,c,d, are fixed positive constants.

Existence of an optimal control

The existence of solution of the system (8) for a finite time interval, given control in the admissible control set $\mathcal{U}$ can be established using results from Lukes [43]. Hence, the following result holds.

Theorem 1. *Given any control $(u_1,u_2)\in\mathcal{U}$, there exists a bounded solution to system (8).*

Since the state variables and the controls are uniformly bounded, existence of an optimal control follows boundedness of solutions and their derivatives of the system (8) for a finite time interval [43]. The boundedness property and the convexity of the objective functional give enough compactness for the existence of an optimal control [44,45]. Thus, with the objective functional J in Eq. (9) subject to the control set $\mathcal{U}$, there exists an optimal control $(u_1^*,u_2^*)\in\mathcal{U}$ such that

$$J(u_1^*,u_2^*)=\min_{u_1,u_2\in\mathcal{U}}J(u_1,u_2).$$

Characterization of optimal controls

The necessary conditions that an optimal control must satisfy come from the Pontryagin's Maximum Principle [46]. This principle converts (8) and (9) into a problem of minimizing pointwise a Hamiltonian H, with respect to (u_1,u_2). First, we formulate the Hamiltonian from the cost functional (9), and the governing dynamics (8) to obtain the optimality conditions.

$$\begin{aligned}H(\beta(t)) &= AI+B_1u_1(t)S+B_2u_1^2(t)+C_1I\left(1+\frac{\gamma D}{m_D+D}\right)u_2(t)+C_2u_2^2\\ &+ \lambda_S\left\{\Lambda-\beta(t)SI+\delta R-[u_1(t)+\mu]S\right\}\\ &+ \lambda_I\left\{\beta(t)SI-u_2\left(1+\frac{\gamma D}{m_D+D}\right)I-(\mu+\alpha)I\right\}\\ &+ \lambda_T\left[u_2\left(1+\frac{\gamma D}{m_D+D}\right)I-(\mu+\kappa)T-(1-\lambda)\alpha T\right]\\ &+ \lambda_R\left[u_1(t)S+\kappa T-(\mu+\delta)R\right]\\ &+ \lambda_D\left[\alpha I+(1-\lambda)\alpha T\right],\end{aligned} \tag{11}$$

where the $\lambda_S,\ \lambda_I,\ \lambda_T,\ \lambda_R,\ \lambda_D$ are the associated adjoints for the state variables S, I, T, R, D. The system of equations is found by taking the appropriate partial derivatives of the Hamiltonian (11) with respect to the associated state variable.

Theorem 2. *Given the optimal control (u_1^*, u_2^*) and the corresponding state solutions S^*, I^*, T^*, R^*, D^* of system (8) that minimizes $J(u_1, u_2)$ over $\mathcal{U}$, there exists $\lambda_S,\ \lambda_I,\ \lambda_T,\ \lambda_R,\ \lambda_D$ satisfying the adjoint system*

$$\begin{aligned}
\frac{d\lambda_S}{dt} &= -B_1u_1^* + \beta(t)I(\lambda_S - \lambda_I) + u_1^*(\lambda_S - \lambda_R) + \mu\lambda_S,\\
\frac{d\lambda_I}{dt} &= -A - C_1\left(1 + \frac{\gamma D}{m_D + D}\right)u_2^* + \beta(t)S(\lambda_S - \lambda_I) +\\
&\quad u_2^*\left(1 + \frac{\gamma D}{m_D + D}\right)(\lambda_I - \lambda_T) + \alpha(\lambda_I - \lambda_D) + \mu\lambda_I,\\
\frac{d\lambda_T}{dt} &= \alpha(1-\lambda)(\lambda_T - \lambda_D) + \kappa(\lambda_T - \lambda_R) + \mu\lambda_T,\\
\frac{d\lambda_R}{dt} &= \delta(\lambda_R - \lambda_S) + \mu\lambda_R,\\
\frac{d\lambda_D}{dt} &= -C_1\frac{\gamma m_D}{(m_D + D)^2}Iu_2^* + \frac{\gamma D}{(m_D + D)^2}Iu_2^*(\lambda_T - \lambda_I),
\end{aligned} \tag{12}$$

with transversality conditions

$$\lambda_S(t_f) = 0,\ \lambda_I(t_f) = 0,\ \lambda_T(t_f) = 0,\ \lambda_R(t_f) = 0,\ \lambda_D(t_f) = 0, \tag{13}$$

Furthermore, we have the optimal control characterizations

$$\begin{aligned}
u_1^* &= min\left\{b, max\left[a,\ \frac{-B_1S + S(\lambda_S - \lambda_R)}{2B_2}\right]\right\},\\
u_2^* &= min\left\{d, max\left[c,\ \left(\frac{I(\lambda_I - \lambda_T - C_1)}{2C_2}\right)\left(1 + \frac{\gamma D}{m_D + D}\right)\right]\right\}.
\end{aligned} \tag{14}$$

Proof. Given the existence of an optimal control pair, the differential equations governing the adjoint variables are obtained by differentiating the Hamiltonian function, given as

$$\begin{aligned}
\frac{d\lambda_S}{dt} &= -\frac{\partial H}{\partial S} = -B_1u_1 + \beta_0 I(\lambda_S - \lambda_I) + u_1(\lambda_S - \lambda_R) + \mu\lambda_S,\\
\frac{d\lambda_I}{dt} &= -\frac{\partial H}{\partial I} = -A - C_1\left(1 + \frac{\gamma D}{m_D + D}\right)u_2 + \beta_0 S(\lambda_S - \lambda_I)\\
&\quad + u_2\left(1 + \frac{\gamma D}{m_D + D}\right)(\lambda_I - \lambda_T) + \alpha(\lambda_I - \lambda_D) + \mu\lambda_I,\\
\frac{d\lambda_T}{dt} &= -\frac{\partial H}{\partial T} = \alpha(1-\lambda)(\lambda_T - \lambda_D) + \kappa(\lambda_T - \lambda_R) + \mu\lambda_T,\\
\frac{d\lambda_R}{dt} &= -\frac{\partial H}{\partial R} = \delta(\lambda_R - \lambda_S) + \mu\lambda_R,\\
\frac{d\lambda_D}{dt} &= -\frac{\partial H}{\partial D} = -C_1\frac{\gamma m_D}{(m_D + D)^2}Iu_2^* + \frac{\gamma D}{(m_D + D)^2}Iu_2^*(\lambda_T - \lambda_I).
\end{aligned}$$

Considering the optimality conditions, the Hamiltonian function is differentiated with respect to the control variables resulting in

$$\begin{aligned}
0 = \frac{\partial H}{\partial u_1} &= 2B_2u_1^* + B_1S - S(\lambda_S - \lambda_R),\\
0 = \frac{\partial H}{\partial u_2} &= 2C_2u_2^* - I(\lambda_I - \lambda_T - C_1)\left(1 + \frac{\gamma D}{m_D + D}\right),
\end{aligned} \tag{15}$$

on the interior of the control set $\mathcal{U}$. Then, solving for u_2^* (on the interior of the control set) gives

$$\begin{aligned}
u_1^* &= \frac{-B_1S + S(\lambda_S - \lambda_R)}{2B_2},\\
u_2^* &= \left[\frac{I(\lambda_I - \lambda_T - C_1)}{2C_2}\right]\left(1 + \frac{\gamma D}{m_D + D}\right).
\end{aligned} \tag{16}$$

Using the bounds on the controls, we obtain the characterization given in (14) and we conclude that

$$\begin{aligned}
u_1^* &= min\left\{b, max\left[a,\ \frac{-B_1S + S(\lambda_S - \lambda_R)}{2B_2}\right]\right\},\\
u_2^* &= min\left\{d, max\left[c,\ \left(\frac{I(\lambda_I - \lambda_T - C_1)}{2C_2}\right)\left(1 + \frac{\gamma D}{m_D + D}\right)\right]\right\},
\end{aligned}$$

□ □

To support the analytical results, graphical representations are illustrated in the next section using the hypothetical model parameter values in Table 1. Because for $u_1(t) = 0$, there is no vaccination at all, we set the lower bound of the controls to 0 and the upper bound to 1, that is, $a = c = 0$, $b = d = 1$. Thus, $0 \le u_1(t), u_2(t) \le 1$ [39].

Table 1
Description of the model parameters.

Parameter	Description	Baseline value
κ	Recovery rate	0.65
λ	Effectiveness of the drug as a reduction factor in disease-induced death ($0 < \lambda \leq 1$)	0.85
β_0, β_1	Disease transmission rate	0.00025, 0.00023
Λ	Recruitment rate of individuals into the population	0.8
μ	Natural death (or emigration) rate which is assumed to be the same for all sub-populations	$1/(59 \times 365)$
τ	treated at a constant rate	0.65
α	Disease induced death rate	0.04
δ	Rate of lost of immunity	0.56
m_D	Half saturation constant	0.50
γ	Half regulation constant	0.08333
q	Transition rate from β_0 to β_1	0.5

Connecting $\beta(t)$ functional forms to the control problem

Here, we graphically connect the analytical results in Section "Analysis of the model" to the control problem in Figs. 4(a)–5(b) where the contact rate is a constant for each of the aforementioned cases 1,2 and 3. It is important to note that the shape of the figures are almost unchanged irrespective of which functional form of the contact rate is used.

Numerical simulations with and without optimal control

Numerical solutions to the optimality system comprising of the state equations (8) and adjoint equations (12) are carried out using parameter values in Table 1 (except otherwise stated), and the following weight factors with the initial conditions:

$$A = 0.725, B_1 = 0.115, B_2 = 0.125, C_1 = 0.315, C_2 = 0.125, S(0) = 1000, I(0) = 120,$$

$$T(0) = 20, R(0) = 30, D(0) = 0, a = c = 0, b = d = 1$$

The numerical solutions were obtained using the forward–backward scheme, starting with an initial guess for the optimal controls u_1 and u_2; the state variables are solved forward in time from the dynamics (8) using a Runge–Kutta method of the fourth order. Then, those state variables and initial guess for u_1, u_2 are used to solve the adjoint equations (12) backward in time with given final conditions (14) again employing a fourth order Runge–Kutta method. The controls u_1 and u_2 are updated and used to solve the state and then the adjoint system. This iterative process terminates when current state, adjoint, and control values fall below a predefined threshold [42].

We start with the fixed transmission rate $\beta(t) = \beta_0$; the numerical simulations for the different functional forms for $\beta(t)$ are given below. For each graphical representation, the term "with control" refers to the application of both controls $u_1(t)$ for vaccination of susceptibles and $u_2(t)$ for treatment of infectives. The control problem allows for 2 controls and an optimal solution with one of those controls being 0 is possible.

Figs. 4(a) and 4(b) depict the dynamics of the infectives and treated without and with controls (vaccination of susceptible individuals $u_1(t)$, and treatment of infectives $u_2(t)$). Concurrently applying both control interventions clearly indicates that this could help to mitigate the spread of the disease. Media coverage of disease outbreaks can help elicit behavioral change, thereby lowering the number of infections as depicted in Figs. 2(a) and 2(b), a combination of media coverage and concurrent implementation of treatment of infectives and prevention measures such as vaccination would potentially have a larger impact on disease dynamics as depicted in Fig. 4 where we observed fewer number of infected and deaths in the presence of the control unlike when controls are absent. We also observed from the figures that there are more treated and recovered individuals when both controls are used.

The control profiles $u_1(t)$ and $u_2(t)$ are shown graphically in Figs. 5(a) and 5(b). Both treatment and vaccination start at their respective upper bounds, almost at the same time with $u_2(t)$ starting a little later from the lower bound. However, the vaccination level drops off after a short period of time, and is maintained at a lower bound close to the end of the implementation period, which is about 1,500 days. On the other hand, treatment remains at its upper bound for a while before gradually dropping to the lower bound about half way (750 days) into the implementation period.

In the sequel, the graph without control is in green, while blue represents Case 1, red for Case 2, and black for Case 3.

Case 1: $\beta(t) = \beta_0\left[1 + sin\left(\frac{2\pi}{365}t + q\right)\right]$

Figs. 6(a)–6(d) (blue color) depict the numerical simulations and the time dependent controls when $\beta(t)$ is a periodic function. We observe in Figs. 6(a) and 6(d) a higher number of infected and deaths when both controls ($u_1(t)$ for vaccination and $u_2(t)$ for treatment) are used. Also, Figs. 6(b) and 6(c) show more treated and recovered individuals when the controls are implemented concurrently. The peak of the trajectory in Fig. 6(a) is higher than the peak in Fig. 4(a), indicating that more individuals are infected with this functional form; however, the deaths are fewer unlike the number observed

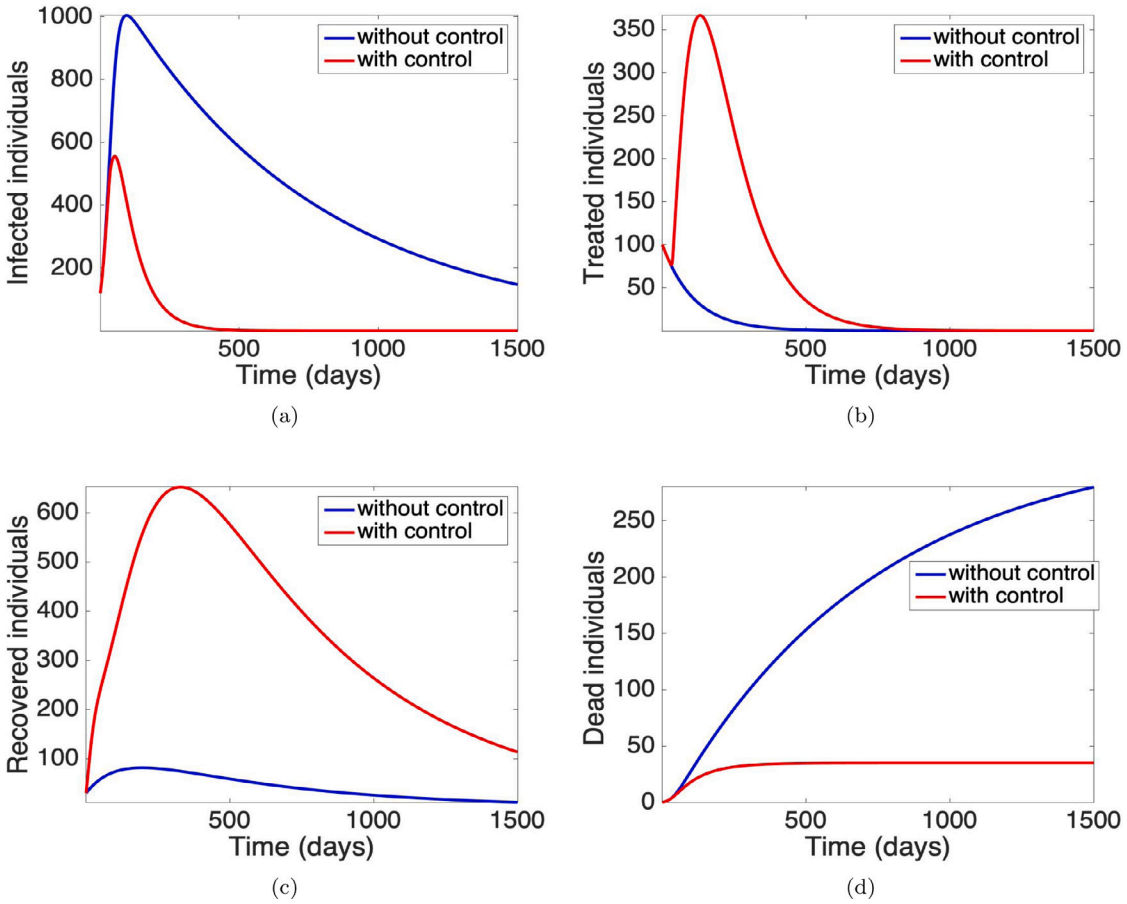

Fig. 4. Numerical simulation when $\beta(t) = \beta_0$ a constant function. (a) Time series of $I(t)$; (b) Time series of $T(t)$; (c) Time series of $R(t)$; and (d) Time series of $D(t)$.

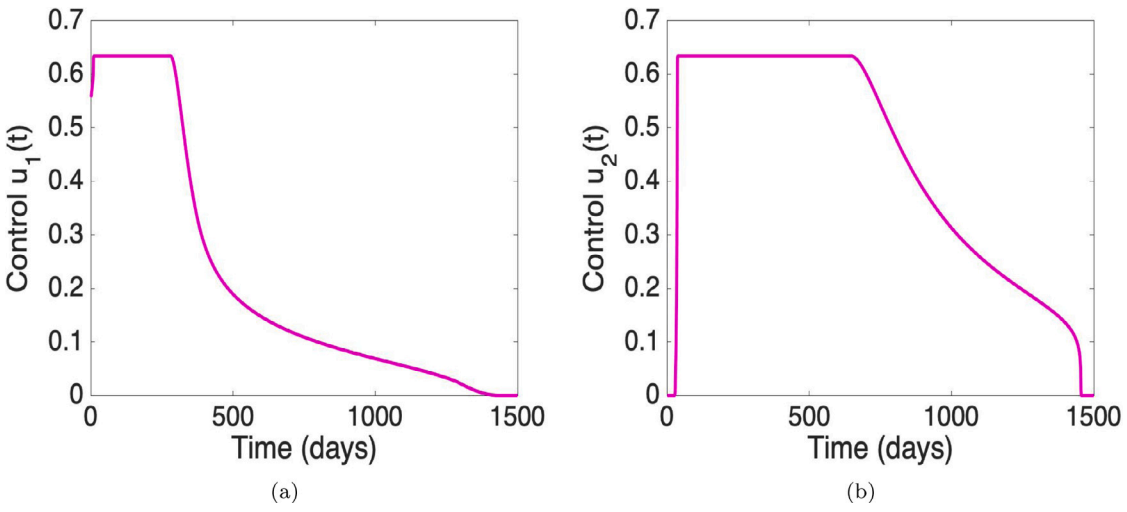

Fig. 5. Time dependent controls for $\beta(t) = \beta_0$. (a) Optimal control profile of $u_1(t)$; and (b) Optimal control profile of $u_2(t)$.

in Fig. 4(d). More treated and recovered are obtained with this functional form compared to the treated and recovered obtained for the constant contact rate $\beta(t) = \beta_0$.

The optimal control profiles $u_1(t)$ and $u_2(t)$ for this periodic functional form (case 1; blue color) are shown in Figs. 7(a) and 7(b). The treatment control $u_1(t)$ starts and stays at the upper bound for more than half of the simulation/implementation period before declining to the lower bound unlike what we observe in Fig. 5(a). The optimal control $u_2(t)$ on the other hand, starts a little earlier from the lower bound from about 30 days but quickly jumps to the upper bound where it mostly remains before dropping to the lower bound close to the end of the implementation period (at about day 460). The trajectory of control $u_2(t)$ differs from the trajectory of the control $u_2(t)$ in Fig. 5(b) for $\beta(t) = \beta_0$.

Case 2: $\beta(t) = \beta_0 + \frac{\beta_1 - \beta_0}{1 + e^{-qt}}$

Figs. 6(a)–6(d) (red color) depict the time series of the infected, treated, recovered, dead when $\beta(t)$ is the sigmoid function given above. We observe in Figs. 6(a) and 6(d) (red color) fewer number of infected individuals and deaths in the presence of both controls $u_1(t)$ and $u_2(t)$ compared to Case 1. Similarly, in Figs. 6(b) and 6(c), there are fewer treated and recovered individuals when both controls are used. Notice that the peak of the trajectory in Fig. 6(a) is lower than the peak in Figs. 4(a) and 6(a), indicating that fewer individuals are infected with this functional form; the number of deaths are also fewer unlike the number observed in Figs. 4(d) and 6(d) compared to the baseline case without control (when $\beta(t) = \beta_0$) and Case 1.

The optimal control profiles $u_1(t)$ and $u_2(t)$ (red color) for this functional form are shown in Figs. 7(a) and 7(b). Like the control profile depicted for Case 1 (blue color), the vaccination control $u_1(t)$ starts and stays at the upper bound for more than half of the simulation period before gradually declining to the lower bound by the end of the simulation period. The decline starts a little bit earlier, and its slope is steeper than the one in Case 1. On the other hand, the treatment control $u_2(t)$ started a bit later from the lower bound (from about 50 days) than the control in Case 1. The trajectory of the optimal control $u_2(t)$ quickly jumps to the upper bound where it mostly remains before dropping to the lower bound close to the end of the implementation period.

Case 3: $\beta(t) = \beta_0 \left(1 + e^{-qt}\right)$

Figs. 6(a)–6(d) (black color) depict the time series of the infected, treated, susceptibles and the controls with $\beta(t)$ the function given above. We observe fewer number of infected and deaths when both controls are used unlike when the controls are absent in Figs. 6(a) and 6(d), and also compared to Case 1. On the other hand, Figs. 6(b) and 6(c) show more treated and recovered individuals when the controls are implemented at the same time. The peak in Fig. 6(a) (Case 3, black color) is lower than the peak in Fig. 6(a) (Cases 1, black color) indicating that fewer individuals are infected with this functional form. Also, more treated and recovered individuals are obtained with this functional form compared to the number of treated and recovered individuals obtained in Case 2 (red color). That is, the graphs of this Case 3 are intermediary between those of Case 1 (higher, blue color) and Case 3 (lower, red color). However,

Figs. 7(a) and 7(b) (black color) show the optimal control profiles $u_1(t)$ and $u_2(t)$ for the functional form $\beta(t) = \beta_0 \left(1 + e^{-qt}\right)$. The treatment control $u_1(t)$ in Fig. 7(a) starts and stays at the upper bound for about half of the simulation period before gradually declining to the lower bound. It is however implemented in a shorter time compared to Cases 1 and 2. The vaccination control $u_2(t)$ on the other hand stayed at the lower bound for about 40 days before rising (in between Cases 1 and 2) to the upper bound where it mostly remains before dropping to the lower bound at about day 460.

When the contact rate $\beta(t) = \beta_0$ is a constant, the timeline of implementing the control measures as depicted in Fig. 5 is about 3 times longer (1,500 days), compared to the other three functional forms in cases 1 to 3, Fig. 7 .

It is important to note that no control measure to mitigate the spread of diseases can be implemented at the optimal level all the time. Though the controls tend to stop at some point in time, this is basically because the implementation of the control measures is for one outbreak of the disease. Since the model is continuous and deterministic, the infection may not truly go to zero except if the disease has been completely eradicated, and thus, with significant recruitment over time, the population presumably grows so that the infection may restart and eventually one would expect an endemic state. The case of the recent Covid-19 outbreak is an example as implementation of the control measures are currently almost non existent/no longer enforced, despite the low resurgence of the disease.

Discussion and conclusion

Highly contagious and viral diseases pose significant threats to the future of human being. In order to better prepare against future epidemic outbreaks, such as Covid-19, avian, swine, and human influenza, it is imperative to theoretically assess the impact of media coverage on a generic disease transmission. Information such as number of deaths and infections reported in mass media such as TV, radio, print media, or social media could potentially impact individuals risk perception and their responses to disease threats. For instance, substantial media reports of the Covid-19 pandemic prompted individuals to adopt protective measures such as social/physical distancing, face mask wearing.

In this study, we formulated an epidemic model incorporating media reports on the number of deaths. The proposed model which accounts for treatment and essential factors such as the media reporting of disease induced deaths is theoretically analyzed. Numerical simulations show the potential impact of media coverage on mitigating the spread of the disease. We extend the basic model by incorporating a pair of time-dependent control variables and using Pontryagin's Maximum Principle, the appropriate conditions for the existence of optimal control and the optimality system are established. Media coverage through reporting of the

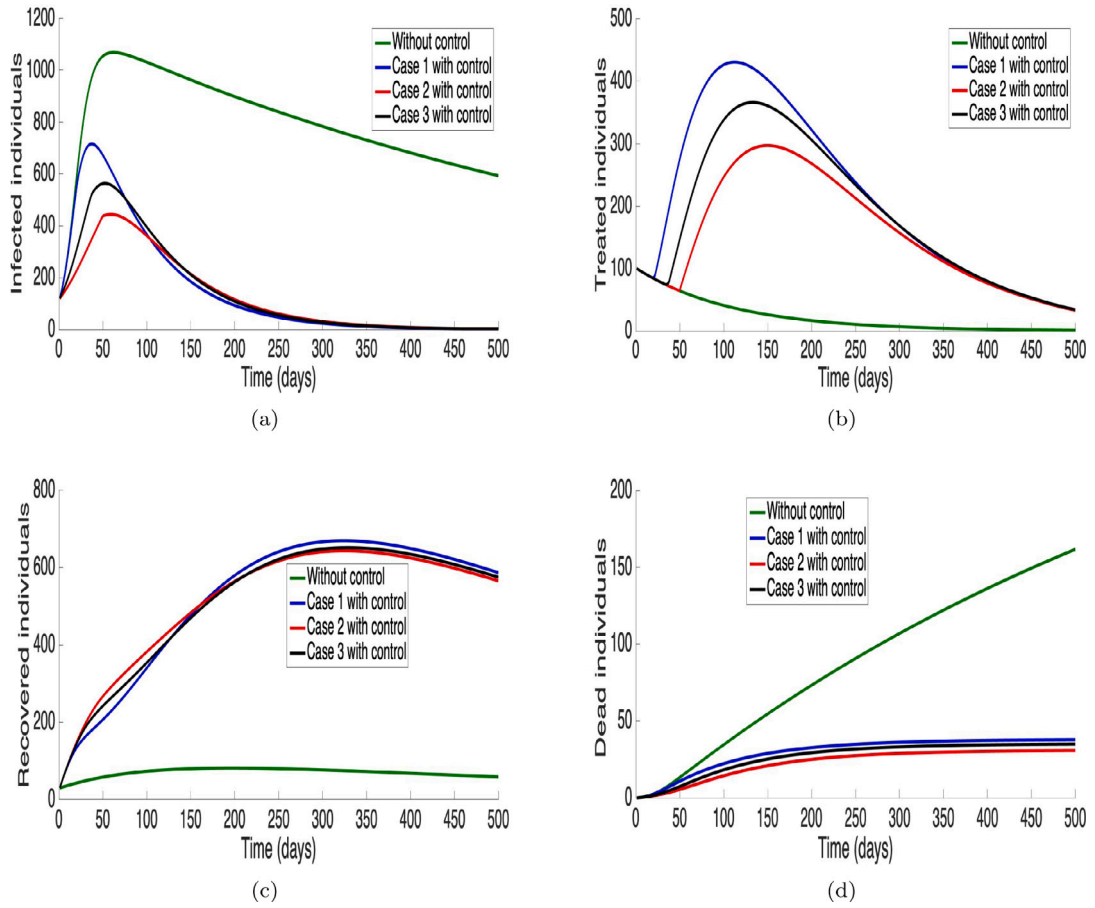

Fig. 6. Numerical simulation for all three cases (a) Time series of $I(t)$; (b) Time series of $T(t)$; (c) Time series of $R(t)$; and (d) Time series of $D(t)$. (For interpretation of the references to colorin this figure legend, the reader is referred to the web version of this article).

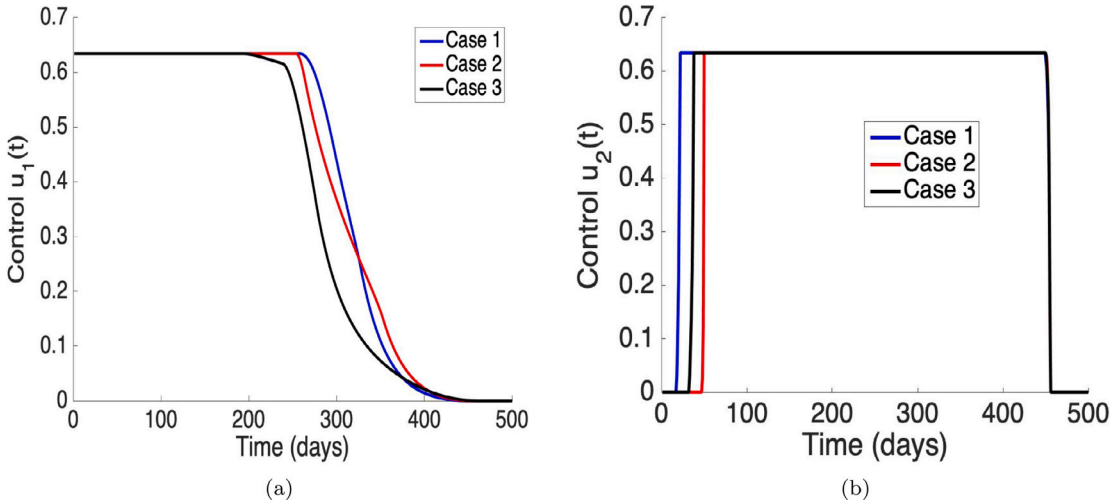

Fig. 7. Profile of the time-dependent controls for the three cases (a) Optimal control profile of $u_1(t)$ and (b) the optimal control profile of $u_2(t)$. (For interpretation of the references to colorin this figure legend, the reader is referred to the web version of this article).

number of case fatalities could have a short-term positive population-level impact on mitigating the spread of the disease. Overall, the numerical simulations show that concurrent implementation of both vaccination and treatment strategies would be more impactful in mitigating the spread of the disease. However, the number of infected, treated, recovered, and dead depends on the form of the incidence function used. When $\beta(t)$ is periodic, there is a higher estimate of the number of individuals in the various classes (infected, treated, recovered, and dead), but lower than when the contact rate is assumed constant. The sigmoid functional form in Case 2 could underestimate the numbers of individuals in the above classes. The functional form in Case 3 is somehow intermediary between Cases 1 and 2.

The theoretical results indicate that depending on the incidence function $\beta(t)$ used, one can obtain either the extinction of the disease or the persistence of the disease according to whether their respective basic reproduction number R_0 is less or greater than unity, but there is also a relationship between the size of the epidemic and media coverage. The chosen media functions contribute to mitigating the spread of the epidemic. However, with the complexity of choosing the best media function, we study the optimal control using three different contact rates and a media function that takes deaths as an argument. The results of the control analysis indicate that the nature of the controls does not significantly vary as the incidence functions are changed. This is important since finding an incidence function for an epidemic remains critical and must be done with great caution. If the results obtained with the considered incidence functions remains true with any incidence function, one could say the nature of the incidence function does not significantly influence the proposed optimal control problem. It would be important in the future to critically investigate why the choice of the incidence function seems to have no impact on the optimal control problem.

Our results show that if the implementation of therapeutic measures (like vaccination and treatment) starts at the same time after a disease outbreak, vaccination will be at its upper bound for a short time while treatment will as expected be implemented on a longer time scale. We summarize the results from this study as follows:

(i) Media reporting to inform the public about the number of disease induced deaths could have a short term benefit in mitigating the spread of infectious disease.
(ii) Vaccination and treatment strategies should be concurrently implemented in order to have impactful results in slowing the spread of the disease.
(iii) Treatment of infectives should be implemented for a longer time compared to vaccination. This is to be expected because the increased treatment of infected is modeled as a Holling type II functional response.

The proposed model has some limitations, for instance, it includes a single control for both treatment and media coverage. Future extension of this work could include two control strategies, namely $u_1(t)$ and $u_2(t)$ (which are bounded, Lebesgue integrable functions), with $u_1(t)$ being the time-dependent optimal strategy associated with treating symptomatic individuals, while $u_2(t)$ is the time-dependent optimal strategy associated with treating symptomatic individuals when deaths are reported (it may represent the use of alternative preventive measures). In this case,

$$\tau\left(1+\frac{D}{m_D+D}\right)$$

may be replaced by

$$\left(\tau_1(t)+\tau_2(t)\frac{D}{m_D+D}\right).$$

This will enable one to assess the impact of concurrently controlling treatment and media coverage as awareness campaign [14], or one in the absence of the other (we note however that, using time-dependent optimal control $u_1(t)$ will only give a reduced number of infected than when time dependent optimal control $u_2(t)$ only is used). Also, media control here does not mean controlling what the media will report or how what is reported is perceived by the public (triggering panic or overconfidence), but that deaths which are more news-worthy are reported, at least from the mathematical standpoint. Also, the proposed model considers a highly fatal disease where recovery is only possible through treatment which may not be applicable if applying this to a specific disease. In this case one could assume that some people may recover without treatment. While media influences only treatment herein, a previous study in which media instead influences the contact structure was carried out in [8], and a future study of a specific disease will consider combining both media influencing contact and treatment rates.

While news coverage may have a significant impact on individuals' behavior, the latter is not instantaneous. Future studies could incorporate time delay or a Markovian process to capture these behavioral responses. Another limitation of this study is its reliance on a deterministic model, which may not fully capture the stochastic (random) nature of real-world epidemics. Such models might oversimplify complex interactions and behavioral dynamics in actual epidemic scenarios. Consequently, a stochastic version of this model to account for random variation in one or more parameters over time is viable. Finally, given that simplified understandings of disease epidemiology propagated through media reporting could impact the disease spread [8], investigating the possibility of the model undergoing a backward/sub-critical bifurcation (co-existence of a stable disease-free equilibrium with a stable endemic equilibrium) is important from a public health stand-point, because in this case, having the basic reproduction number less than unity is not a sufficient condition for disease elimination [33].

CRediT authorship contribution statement

Mamadou L. Diagne: Methodology, Formal analysis, Writing – review & editing. **Folashade B. Agusto:** Conceptualization, Methodology, Formal analysis, Writing – review & editing. **Herieth Rwezaura:** Methodology, Formal analysis, Writing – review & editing. **Jean M. Tchuenche:** Conceptualization, Methodology, Formal analysis, Writing – review & editing. **Suzanne Lenhart:** Conceptualization, Methodology, Formal analysis, Writing – review & editing.

Declaration of competing interest

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

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