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Original article

Local Stability and Threshold Analysis of an SEITR Model with Standard Incidence for Hepatitis B Transmission

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Abstract
This study analyses the SEITR model of hepatitis B virus (HBV) transmission with a standard incidence rate of $\beta SI/N$ in a population of variable size. The model is reduced to an autonomous system expressed in population proportions. The disease-free and endemic equilibria and their Jacobian matrices are derived, and the basic reproduction number is obtained using the next generation matrix method. The Routh-Hurwitz criterion establishes that the disease-free equilibrium is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. For disease-induced mortality $\mu_2 > 0$, shows that the endemic equilibrium is unique and is locally asymptotically stable. For $\mu_2 = 0$, the endemic equilibrium exists for $R_0 > 1$ and is locally asymptotically stable, with a forward bifurcation occurring at $R_0 = 1$. Numerical simulations calibrated to 2022 surveillance data from Ambon City, Indonesia, confirm the analytical results: the critical transmission rate is $\beta^* \approx 0.432$, and the critical vaccination coverage is $\sigma^* \approx 0.622$. The baseline value of $R_0 \approx 0.926$ lies just below the threshold, resulting in very slow extinction of the infection over hundreds of years. Normalised sensitivity analysis indicates that R_0 is most sensitive to vaccination coverage, followed by the transmission rate and the treatment rate.
Keywords. Hepatitis B Transmission, SEITR Model, Standard Incidence Rate, Basic Reproduction Number, Routh Hurwitz Criterion, Sensitivity Analysis.

Introduction

Mathematical modelling is an important tool for understanding the mechanisms of infectious diseases and developing public health strategies and prevention [1]. The SEITR model is an adaptation of traditional epidemic models such as SIR and SEIR. It includes an additional compartment for Treated individuals (T) alongside the standard compartments (Susceptible (S), Exposed (E), Infectious (I), and Recovered (R)) [2,3]. This five-compartment model can be used to model Hepatitis B due to including a latent period and long-term treatment. This can allow accurate assessment of treatment.

Regarding hepatitis B virus (HBV) in particular, deterministic models have been used to understand the transmission dynamics within high-prevalence communities [4,5], and develop effective intervention strategies [6]. Many recent studies have extended the model to include passive immunity and various intervention strategies [7]; calibrated models using real data to predict up to 2030 have been proposed [8]; spatial diffusion models to include the chronic stage have been introduced [9]; coinfection models with HIV have shown that backward bifurcation in HBV dynamics can occur [10], and fractional order models including memory effects have been analysed [11].

Most epidemic models use a bilinear incidence rate βSI (mass action), which is appropriate only when the total population size is constant. In populations of varying size, it implies that contacts per individual increase with population size, which is not realistic for most human infections [12]. The standard incidence rate ($\beta SI/N$) can be used to overcome this limitation. This means the force of infection depends on the proportion of infectious individuals. This allows the model to be expressed in terms of proportions of the population, simplifying the analysis of equilibria [13]. Recent HBV models generally have threshold analysis by finding the basic reproduction number R_0 assessing local stability using linearisation and the Routh-Hurwitz criteria, and performing sensitivity analysis on the value of R_0. Such analyses also show that the direction of bifurcation should be assessed at $R_0 = 1$. It has been reported that backward bifurcation can occur in HBV models [14,15]. Backward bifurcation occurs when there is a stable endemic equilibrium along with a disease-free equilibrium when $R_0 < 1$. This has also been reported in models with standard incidence with treatment [16]. Therefore, the direction of bifurcation at $R_0 = 1$ should be found.

A SEITR model has been recently developed to model Hepatitis B using a standard incidence rate ($\beta SI/N$). This model was solved numerically using the fourth-order Runge-Kutta method [17]. This study focuses on the local stability and threshold analysis of this model. The model was simplified to be expressed in terms of the proportion of the population. The disease-free and endemic equilibria are derived, and R_0 is obtained using the next generation matrix approach [18,19]. The local stability of both equilibria was analysed using linearisation [20] and the Routh-Hurwitz criteria. These analytical results are supported by numerical simulations based on data from Ambon City [21], a normalised sensitivity analysis of R_0 [22], and analysis of the bifurcation occurring at $R_0 = 1$ [23].

Mathematical Model

The $SEITR$ model is formulated under the following assumptions [17]:

The total population $N(t) = S(t) + E(t) + I(t) + T(t) + R(t)$ is variable in size; natural mortality acts at rate μ_1 on all subpopulations, while the infected subpopulation an additional disease induced mortality μ_2.

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Unvaccinated and vaccinated newborns enter S and R at rates$(1-\sigma)\kappa N$, and $\sigma\kappa N$ respectively, and transmission follows the standard incidence $\beta SI/N$.
Progression occurs sequentially: S → E at rate $\beta SI/N$, E → I at rate α, I → T at rate γ, and T → R at rate δ; recovered individuals acquire durable immunity and do not return to S.
Vaccination is fully effective (vaccinated newborns enter R directly) and T is noninfectious.
Based on *these assumptions* and the flow diagram in (Figure 1), the governing system is*:*

$$\frac{dS}{dt} = (1-\sigma)\kappa N - \frac{\beta SI}{N} - \mu_1 S \quad (1)$$

$$\frac{dE}{dt} = \beta SI/N - (\alpha + \mu_1)E \quad (2)$$

$$\frac{dI}{dt} = \alpha E - (\gamma + \mu_1 + \mu_2)I \quad (3)$$

$$\frac{dT}{dt} = \gamma I - (\mu_1 + \delta)T \quad (4)$$

$$\frac{dR}{dt} = \sigma\kappa N + \delta T - \mu_1 R \quad (5)$$

Where:
κ is the natural birth rate
σ is the vaccination coverage rate.
β is the transmission rate.
α is the rate of progression from exposure to infection.
γ is the rate of those receiving treatment
δ is the recovery rate after treatment.
μ_1 is the natural mortality rate
μ_2 is the mortality rate due to the disease.

The terms $(1-\sigma)\kappa N$ and $\sigma\kappa N$ distribute newborns between the Susceptible and Recovered subpopulations according to the vaccination coverage ratio σ. The remaining terms represent: natural mortality in each subpopulation $(\mu_1 S, \mu_1 E, \mu_1 I, \mu_1 T, \mu_1 R)$; disease-induced mortality $(\mu_2 I)$; progression from exposure to infection (αE); entry into Treatment (γI); and Recovery after Treatment (δT).

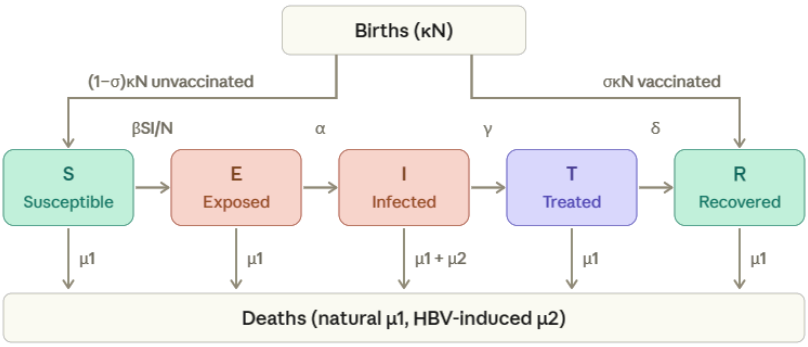


Figure 1. SEITR model flowchart for hepatitis B disease Transmission.

By summing Eqs. (1-5), the total population function will be as follows:

$$dN/dt = (\kappa - \mu_1)N - \mu_2 I \quad (6)$$

In order to define a proper equilibrium analysis for a population of variable size, the following population ratios are studied in accordance with the standard demographic epidemiological approach [12]:

$$s = \frac{S}{N} \; ; \; e = \frac{E}{N} \; ; \; i = \frac{I}{N} \; ; \; \tau = \frac{T}{N} \; ; \; r = \frac{R}{N} \quad (7)$$

where $s + e + i + \tau + r = 1$ for all $t \geq 0$.

By Applying the quotient rule $d(X/N)/dt = X'/N - (X/N)(N'/N)$ where $N'/N = \kappa - \mu_1 - \mu_2 i$ and substituting Eq. (7) in Eqs. (1-5) will get the reduced system as follows:

$$\frac{ds}{dt} = \kappa(1-\sigma-s) - \beta si + \mu_2 si \quad (8)$$

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$$\frac{de}{dt} = \beta si - (\alpha + \kappa)e + \mu_2 ei \quad (9)$$

$$\frac{di}{dt} = \alpha e - (\gamma + \kappa)i - \mu_2 i(1 - i) \quad (10)$$

$$\frac{d\tau}{dt} = \gamma i - (\delta + \kappa)\tau + \mu_2 \tau i \quad (11)$$

$$\frac{dr}{dt} = \kappa(\sigma - r) + \delta\tau + \mu_2 ri \quad (12)$$

Equilibrium Points

There are two equilibrium points: the disease-free equilibrium point (DFE) and the endemic equilibrium point. The equilibrium point is determined by the condition $\frac{ds}{dt} = \frac{de}{dt} = \frac{di}{dt} = \frac{d\tau}{dt} = \frac{dr}{dt} = 0$. To examine the stability of these equilibrium points, the Jacobian matrix corresponding to Eqs. (8 -12) can be constructed as follows [20]:

$$J = \begin{pmatrix} \frac{\partial s(t)}{\partial s} & \frac{\partial s(t)}{\partial e} & \frac{\partial s(t)}{\partial i} & \frac{\partial s(t)}{\partial \tau} & \frac{\partial s(t)}{\partial r} \\ \frac{\partial e(t)}{\partial s} & \frac{\partial e(t)}{\partial e} & \frac{\partial e(t)}{\partial i} & \frac{\partial e(t)}{\partial \tau} & \frac{\partial e(t)}{\partial r} \\ \frac{\partial i(t)}{\partial s} & \frac{\partial i(t)}{\partial e} & \frac{\partial i(t)}{\partial i} & \frac{\partial i(t)}{\partial \tau} & \frac{\partial i(t)}{\partial r} \\ \frac{\partial \tau(t)}{\partial s} & \frac{\partial \tau(t)}{\partial e} & \frac{\partial \tau(t)}{\partial i} & \frac{\partial \tau(t)}{\partial \tau} & \frac{\partial \tau(t)}{\partial r} \\ \frac{\partial r(t)}{\partial s} & \frac{\partial r(t)}{\partial e} & \frac{\partial r(t)}{\partial i} & \frac{\partial r(t)}{\partial \tau} & \frac{\partial r(t)}{\partial r} \end{pmatrix}$$

$$J = \begin{pmatrix} -(\kappa + \beta i - \mu_2 i) & 0 & s(\mu_2 - \beta) & 0 & 0 \\ \beta i & -(\alpha + \kappa) + \mu_2 i & \beta s + \mu_2 e & 0 & 0 \\ 0 & \alpha & -(\gamma + \kappa) - \mu_2(1 - 2i) & 0 & 0 \\ 0 & 0 & \gamma + \mu_2 \tau & -(\delta + \kappa) + \mu_2 i & 0 \\ 0 & 0 & \mu_2 r & \delta & -\kappa + \mu_2 i \end{pmatrix}$$

Disease-Free Equilibrium Point

In the case of the disease-free equilibrium, the proportion of the population infected with Hepatitis B virus is zero so that $i = 0$. Accordingly, the disease-free equilibrium point of the Hepatitis B epidemic model is given by:

$$E_0 = (s(t), e(t), i(t), \tau(t), r(t)) = (1 - \sigma, 0, 0, 0, \sigma)$$

Endemic Equilibrium Point

The endemic equilibrium point reflects the presence of the infected subpopulation. The proportion infected with the Hepatitis B virus is nonzero i.e. $i \neq 0$, and is obtained as:

$$E_1 = (s(t), e(t), i(t), \tau(t), r(t)) = (s^*(t), e^*(t), i^*(t), \tau^*(t), r^*(t))$$

Where $i^* \neq 0$ ($i^* \in (0,1)$ it is obtained by:

From Eqs. (9) and (10) get:

$$s^* = \frac{[(\alpha + \kappa) - \mu_2 i^*][(\gamma + \kappa) + \mu_2(1 - i^*)]}{\alpha\beta} \quad (13)$$

From Eq. (8) get

$$s^* = \frac{\kappa(1 - \sigma)}{\kappa + (\beta - \mu_2)i^*} \quad (14)$$

By equating Eqs. (13) and (14) we obtain the equation that determines the value of i^*:

$$\frac{(A - \mu_2 i^*)(B - \mu_2 i^*)}{\alpha\beta} = \frac{\kappa(1 - \sigma)}{\kappa + (\beta - \mu_2)i^*}$$

Where

$$A = (\alpha + \kappa); \qquad B = \gamma + \kappa + \mu_2$$

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Then yields a cubic polynomial equation of the form

$$c_3 i^{*3} + c_2 i^{*2} + c_1 i^* + c_0 = 0 \quad (15)$$

With coefficients

$$c_3 = {\mu_2}^2(\beta - \mu_2)$$
$$c_2 = \kappa{\mu_2}^2 - \mu_2(\beta - \mu_2)(A + B)$$
$$c_1 = AB(\beta - \mu_2) - \kappa\mu_2(A + B)$$
$$c_0 = AB\kappa - \alpha\beta\kappa(1 - \sigma)$$

Therefore, when numerical values of the parameters $(\alpha, \beta, \gamma, \kappa, \mu_2, \sigma)$ are available Eq. (15) can be solved directly by Cardano's method or numerically. Then the remaining equilibrium components follow immediately from the expressions derived in terms of it as follows:

$$s^* = \frac{\kappa(1 - \sigma)}{\kappa + (\beta - \mu_2)i^*} \quad (16)$$

$$e^* = \frac{i^*[(\gamma + \kappa) + \mu_2(1 - i^*)]}{\alpha} \quad (17)$$

$$\tau^* = \frac{\gamma i^*}{(\delta + \kappa) - \mu_2 i^*} \quad (18)$$

$$r^* = \frac{\kappa\sigma + \delta\tau^*}{\kappa - \mu_2 i^*} \quad (19)$$

For $\mu_2 = 0, c_3 = c_2 = 0$ and $c_1 = AB\beta$, thus Eq. (15) is linear and obtain

$$i^* = \frac{\kappa[\alpha\beta(1 - \sigma) - (\alpha + \kappa)(\gamma + \kappa)]}{\beta(\alpha + \kappa)(\gamma + \kappa)}$$

Note that i^* is positive if and only if $\alpha\beta(1 - \sigma) > (\alpha + \kappa)(\gamma + \kappa)$, then $i^* < \kappa/(\gamma + \kappa) < 1$.

Basic Reproduction Number

The basic reproduction number (R_0) is the average number of secondary infections generated by a single infected individual within an entirely susceptible population. The disease is expected to die out over time when $R_0 < 1$ whereas $R_0 > 1$ indicates that the infection will spread and lead to an outbreak. The disease-free equilibrium point was employed to derive R_0, using the next generation matrix method applied to the exposed and infected subpopulations, e, i and τ, governed by Eqs. (9) and (10). Let φ be a vector for new infections and ψ be the vector for subpopulation movements:

$$\varphi = \begin{pmatrix} \beta si \\ 0 \\ 0 \end{pmatrix} \text{ and } \psi = \begin{pmatrix} (\alpha + \kappa)e - \mu_2 ei \\ (\gamma + \kappa)i + \mu_2 i(1 - i) - \alpha e \\ (\delta + \kappa)\tau - \mu_2 \tau i - \gamma i \end{pmatrix}$$

Let F and V respectively denote the Jacobians with respect to the state variables to e, i and τ thus we get:

$$F = \begin{pmatrix} 0 & \beta s & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \; ; \; V = \begin{pmatrix} \mu_2 i - (\alpha + \kappa) & -\mu_2 e & 0 \\ -\alpha & (\gamma + \kappa) + \mu_2(1 - 2i) & 0 \\ 0 & -\mu_2\tau - \gamma & (\delta + \kappa) - \mu_2 i \end{pmatrix}$$

Since F and V represent to new infection rate matrix and subpopulation movement rate matrix respectively. By substituting the disease-free equilibrium point in F and V, we obtain

$$F = \begin{pmatrix} 0 & \beta(1 - \sigma) & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \; ; \; V = \begin{pmatrix} \alpha + \kappa & 0 & 0 \\ -\alpha & \gamma + \kappa + \mu_2 & 0 \\ 0 & -\gamma & \delta + \kappa \end{pmatrix}$$

The next generation matrix (G) is obtained as follow:

$$G = FV^{-1} = \begin{pmatrix} \frac{\alpha\beta(1 - \sigma)}{(\alpha + \kappa)(\gamma + \kappa + \mu_2)} & \frac{\beta(1 - \sigma)}{(\gamma + \kappa + \mu_2)} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

Since G as a basic matrix (the second row being zero), the basic reproduction number R_0 (the radius of the spectrum) is:

$$R_0 = \frac{\alpha\beta(1 - \sigma)}{(\alpha + \kappa)(\gamma + \kappa + \mu_2)} \quad (20)$$

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Local Stability at the Disease-Free Equilibrium

The Jacobian matrix of the system evaluated at the disease-free equilibrium is given by

$$J(E_0) = \begin{pmatrix} -\kappa & 0 & -(1-\sigma)(\beta-\mu_2) & 0 & 0 \\ 0 & -(\alpha+\kappa) & \beta(1-\sigma) & 0 & 0 \\ 0 & \alpha & -(\gamma+\kappa+\mu_2) & 0 & 0 \\ 0 & 0 & \gamma & -(\delta+\kappa) & 0 \\ 0 & 0 & \mu_2\sigma & \delta & -\kappa \end{pmatrix}$$

By forming $|\lambda I - J(E_0)| = 0$, where λ denotes the eigenvalues to be determined and I is the identity matrix, then obtained:

$$\left| \begin{pmatrix} \lambda & 0 & 0 & 0 & 0 \\ 0 & \lambda & 0 & 0 & 0 \\ 0 & 0 & \lambda & 0 & 0 \\ 0 & 0 & 0 & \lambda & 0 \\ 0 & 0 & 0 & 0 & \lambda \end{pmatrix} - \begin{pmatrix} -\kappa & 0 & -(1-\sigma)(\beta-\mu_2) & 0 & 0 \\ 0 & -(\alpha+\kappa) & \beta(1-\sigma) & 0 & 0 \\ 0 & \alpha & -(\gamma+\kappa+\mu_2) & 0 & 0 \\ 0 & 0 & \gamma & -(\delta+\kappa) & 0 \\ 0 & 0 & \mu_2\sigma & \delta & -\kappa \end{pmatrix} \right| = 0$$

Which simplifies to:

$$\left| \begin{pmatrix} \lambda+\kappa & 0 & (1-\sigma)(\beta-\mu_2) & 0 & 0 \\ 0 & \lambda+(\alpha+\kappa) & -\beta(1-\sigma) & 0 & 0 \\ 0 & -\alpha & \lambda+(\gamma+\kappa+\mu_2) & 0 & 0 \\ 0 & 0 & -\gamma & \lambda+(\delta+\kappa) & 0 \\ 0 & 0 & -\mu_2\sigma & -\delta & \lambda+\kappa \end{pmatrix} \right| = 0$$

$$(\lambda+\kappa)\big(\lambda+(\delta+\kappa)\big)(\lambda+\kappa) \left| \begin{pmatrix} \lambda+(\alpha+\kappa) & -\beta(1-\sigma) \\ -\alpha & \lambda+(\gamma+\kappa+\mu_2) \end{pmatrix} \right| = 0$$

Note that the fourth and fifth columns yield three negative eigenvalues directly:

$$\lambda_1 = -\kappa; \quad \lambda_2 = -(\delta+\kappa)\,;\ \lambda_3 = -\kappa$$

Since κ, δ represent the natural birth rate and the recovery rate, respectively, the eigenvalues $\lambda_1, \lambda_2, \lambda_3$ are negative and, unlike the remaining two, they do not depend on μ_2 at all. From the determinant of the remaining submatrix (of order 2×2), we obtain:

$$\lambda^2 + \lambda[(\alpha+\kappa)+(\gamma+\kappa+\mu_2)] + [(\alpha+\kappa)(\gamma+\kappa+\mu_2) - \alpha\beta(1-\sigma)] = 0$$

According to the Routh Hurwitz stability criterion, the remaining roots λ_4 and λ_5 are negative or possess negative real parts if and only if all coefficients of the characteristic equation are strictly positive. Since all parameters are biologically strictly positive ($\alpha, \kappa, \gamma, \mu_2 \geq 0$), $[(\alpha+\kappa)+(\gamma+\kappa+\mu_2)] > 0$ is always satisfied.

Therefore, the form $[(\alpha+\kappa)(\gamma+\kappa+\mu_2) - \alpha\beta(1-\sigma)]$ must be positive so that:

$$[(\alpha+\kappa)(\gamma+\kappa+\mu_2) - \alpha\beta(1-\sigma)] > 0$$

$$(\alpha+\kappa)(\gamma+\kappa+\mu_2)\left(1 - \frac{\alpha\beta(1-\sigma)}{(\alpha+\kappa)(\gamma+\kappa+\mu_2)}\right) > 0$$

which can be written compactly as:

$$(\alpha+\kappa)(\gamma+\kappa+\mu_2)(1-R_0) > 0$$

$$(1-R_0) > 0$$

$$R_0 < 1$$

Local Stability at the Endemic Equilibrium

An endemic equilibrium is a point of Ω at which the right-hand sides of Eqs. (8-12) vanish and $i^* > 0$. Only the equilibria that lie in Ω have an epidemiological meaning which can be defined as:

$$\Omega = \{(s, e, i, \tau, r) \in \mathbb{R}^5:\ s, e, i, \tau, r \geq 0, \Sigma = s + e + i + \tau + r = 1\}.$$

Lemma 1 Let $E_1 = (s^*, e^*, i^*, \tau^*, r^*) \in \Omega$ be an endemic equilibrium and $u = \mu_2 i^*$ then

(i) $0 < u < \kappa$ and $0 < i^* < 1$; in particular $u < \mu_2$ and $\delta + \kappa - u > 0$;

(ii) $P > 0$, $Q > \alpha > 0$, $H > \gamma + \mu_2 > 0$ and $R > \gamma > 0$ where P, Q, H, and R defined as:

$$P = \kappa + (\beta - \mu_2)i^*;\ \ Q = A - u;\ \ H = B - u;\ R = B - 2u = H - u;\ \ \text{since}\ \ A = (\alpha+\kappa);\ \ B = \gamma + \kappa + \mu_2\,.$$

Proof. Since $i^* > 0$ and $\tau^* \geq 0$, and $(\delta + \kappa - u)\tau^* = \gamma i^* > 0$ from Eq. (18); hence $\tau^* > 0$ and $\delta + \kappa - u > 0$. From Eq. (19) $(\kappa - u)r^* = \kappa\sigma + \delta\tau^* > 0$ with $r^* \geq 0$, thus $r^* > 0$ and $\kappa - u > 0$. Since $\kappa - u > 0$ so that $\mu_2 i^* < \kappa$ then get $i^* < (\kappa/\mu_2)$. The Eq. (16) gives $Ps^* = \kappa(1-\sigma) > 0$ with $s^* \geq 0$, so $s^* > 0$ and $P > 0$. From Eqs. (13) and (17) obtains $Qe^* = \beta s^* i^* > 0$ hence $e^* > 0$ and $Q > 0$, and the Eq. (17) gives $Hi^* = \alpha e^* > 0$, hence $H > 0$. Because $s^*, e^*, \tau^*, r^* > 0$ and $\Sigma = 1$, we get $i^* < 1$, and therefore $u = \mu_2 i^* < \mu_2$. Finally, since $\mu_2,\ \gamma, \alpha,$ and κ are positive parameters then we get $Q = \alpha + (\kappa - u) > \alpha > 0$, $H = \gamma + \mu_2 + (\kappa - u) > \gamma + \mu_2 > 0$ and $R = \gamma + (\kappa - u) + (\mu_2 - u) > \gamma > 0$. ■

Lemma 2. Let $\mu_2 > 0$. A point $E_1 \in \Omega$ with $i^* > 0$ is an endemic equilibrium if and only if $i^* \in (0, \kappa/\mu_2)$ is a root of $\phi(i) = c$.

Proof. The first will define the three linear functions $p(i) = \kappa + (\beta - \mu_2)i$, $q(i) = A - \mu_2 i$, $w(i) = B - \mu_2 i$, the cubic polynomial $\phi(i) = p(i)\ q(i)\ w(i)$ and the constant $c = \alpha\beta\kappa(1-\sigma)$. Therefore, can be rewritten Eq. (15) as $\phi(i^*) = c$. Note that $P = p(i^*)$, $Q = q(i^*)$, $H = w(i^*)$ and $\phi(0) = \kappa AB$, so that $c/\phi(0) = R_0$.By Lemma 1, every endemic equilibrium in Ω has $i^* \in (0, \kappa/\mu_2)$, Eq (13) gives $\alpha\beta s^* = QH$ with $s^* = \kappa(1-\sigma)/P$ is $PQH = c$, i.e. $\Phi(i^*) = c$. Conversely, let $i^* \in (0, \kappa/\mu_2)$

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satisfy $\phi(i^*) = c$. $p > 0$ (it is linear with $p(0) = \kappa > 0$ and $p(\kappa/\mu_2) = \kappa\beta/\mu_2 > 0$), $q \geq A - \kappa = \alpha > 0$, $w \geq B - \kappa = \gamma + \mu_2 > 0$, and $\kappa - \mu_2 i > 0$ for $i < \kappa/\mu_2$. Therefore Eqs. (16-19) are positive. Now by adding Eqs. (8-12) and will get $d\Sigma/dt = (\kappa - u)(1 - \Sigma)$ then $(\kappa - u)(1 - \Sigma) = 0$ with $\kappa - u > 0$, so $\Sigma = 1$ and the point belongs to Ω. So that the endemic equilibria in Ω correspond one-to-one to roots of $\phi = c$ in $(0, \kappa/\mu_2)$. ■

Based the endemic equilibrium point $E_1 = (s^*, e^*, i^*, \tau^*, r^*)$, the following Jacobian matrix is formed:

$$J(E_1) = \begin{pmatrix} a_{11} & 0 & a_{13} & 0 & 0 \\ a_{21} & a_{22} & a_{23} & 0 & 0 \\ 0 & a_{32} & a_{33} & 0 & 0 \\ 0 & 0 & a_{43} & a_{44} & 0 \\ 0 & 0 & a_{53} & a_{54} & a_{55} \end{pmatrix}$$

Where the entries are expressed in terms of the equilibrium values and the model parameters as follows:

$$a_{11} = -[\kappa + i^*(\beta - \mu_2)] = -P, \quad a_{13} = -s^*(\beta - \mu_2);$$

$a_{21} = \beta i^*$, $a_{22} = -(\alpha + \kappa) + \mu_2 i^* = -Q$, $a_{23} = \beta s^* + \mu_2 e^*$;

$a_{32} = \alpha$, $a_{33} = -(\gamma + \kappa + \mu^2) + 2\mu_2 i^* = -R$;

$a_{43} = \gamma + \mu_2 \tau^*$, $a_{44} = -(\delta + \kappa) + \mu_2 i^* = -(\delta + \kappa - u)$;

$a_{53} = \mu_2 r^*$, $a_{54} = \delta$, $a_{55} = -\kappa + \mu_2 i^* = -(\kappa - u)$

By forming $|\lambda I - J(E_1)| = 0$, we obtain:

$$(\lambda - a_{55})(\lambda - a_{44})(-\lambda^3 + A_1\lambda^2 + A_2\lambda + A_3) = 0$$

with coefficients:

$$A_1 = a_{11} + a_{22} + a_{33}$$
$$A_2 = -(a_{11}a_{22} + a_{11}a_{33} + a_{22}a_{33} - a_{23}a_{32})$$
$$A_3 = a_{11}(a_{22}a_{33} - a_{23}a_{32}) + a_{13}a_{21}a_{32}$$

Therefore, we obtain

$$\lambda_1 = a_{55}; \quad \lambda_2 = a_{44}$$

By Lemma 1 $a_{44} < 0$ and $a_{55} < 0$. Since $\kappa < \delta + \kappa$ for every $\delta > 0$, they are negative if and only if:

$$i^* < \min\left(\frac{\delta + \kappa}{\mu_2}, \frac{\kappa}{\mu_2}\right) \tag{21}$$

For cubic characteristic equation by multiplying −1 recasts this in the standard monic form required by the Routh Hurwitz criterion as follow:

$$\lambda^3 + B_1\lambda^2 + B_2\lambda + B_3 = 0,$$

where $B_1 = -A_1 = P + Q + R$, $B_2 = -A_2 = PQ + PR + QR - \alpha a_{23}$,

$$B_3 = -A_3 = P(QR - \alpha a_{23}) + \alpha\beta i^* s^*(\beta - \mu_2)$$

By the Routh Hurwitz criterion, all three roots of this cubic have negative real part if and only if

$$B_1 > 0, \quad B_3 > 0, \quad B_1 B_2 > B_3$$

Lemma 3. At E_1: $\alpha\beta s^* = QH$, $\alpha(\beta s^* + \mu_2 e^*) = AH$, and the Routh Hurwitz coefficients of the (s, e, i) in $J(E_1)$ are $B_1 = P + Q + R$; $B_2 = P(Q + R) - u(Q + H)$; $B_3 = P[QH - u(Q + H)] - \kappa QH = i^*\phi'(i^*)$.

Proof. Multiply $Hi^* = \alpha e^*$ by Q and use $Qe^* = \beta s^* i^*$; also $\alpha\mu_2 e^* = uH$. Then $QR - AH = -u(Q + H)$, and substituting in the entries of $J(E_1)$ gives B_2, B_3 with $(\beta - \mu_2)i^* = P - \kappa$. ■

Lemma 4. At E_1: $\kappa\rho - i^* QH > 0$, $P = \kappa\rho\,\zeta/(\kappa\rho - i^* QH)$ and $P > \kappa$ where $\zeta = \kappa - u$ and $\rho = \alpha(1 - \sigma)$.

Proof. $PQH = \kappa\rho\beta$ and $P = \kappa + (\beta - \mu_2)i^*$ give $\beta(\kappa\rho - i^* QH) = QH\zeta > 0$, hence the first two claims. $P > \kappa$ is equivalent to $QH > \rho\mu_2$, true since $Q > \alpha \geq \rho$ and $H > \mu_2$. ■

Lemma 5. At E_1: $B_3 > 0$ and $B_1 B_2 > B_3$.

Proof. B_3: inserting P from Lemma 4 gives $1/\kappa\,(\kappa\rho - i^* QH)\,B_3 = i^*\{Q^2H^2 - \rho\mu_2[QH + \zeta(Q + H)]\}$.

With $X = \alpha\mu_2 \geq \rho\mu_2$, $Q = \alpha + \zeta$ and $H \geq \mu_2 + \zeta$, we get $Q^2H^2 - X[QH + a(Q + H)] \geq a\,[\alpha Q(H - \mu_2) + \mu_2 H(Q - \alpha)] > 0$. So $B_3 > 0$.

$B_1 B_2 > B_3$: expanding with

$H = R + u$, $B_1 B_2 - B_3 = P[P(Q + R) + Q^2 - Qu + QR + R^2] + \kappa QH - u(Q + R)(Q + H)$.

Using $P > \kappa > u$ (Lemmas 4, 1), the bracket exceeds $W = \rho Q + \kappa R + Q^2 + QR + R^2 > 0$, hence $B_1 B_2 - B_3 > \kappa(W + QH) - u(Q + R)(Q + H) \geq u[W + QH - (Q + R)(Q + H)] = u\zeta\,(Q + R) > 0$. ■

Theorem. Let $\mu_2 > 0$.

Every endemic equilibrium in Ω is locally asymptotically stable.

If $R_0 > 1$ there is exactly one; if $R_0 \leq 1$ there is none.

Proof.

(i) $J(E_1)$ is block lower-triangular with eigenvalues $-(\delta + \kappa - u) < 0$, $-a < 0$ and those of the (s, e, i) block. By Lemmas 1 and 4, $B_1, B_3 > 0$ and $B_1 B_2 > B_3$, so the Routh Hurwitz criterion.

Let $i_c = \kappa/\mu_2$ and $\psi(i) = \phi(i) - \kappa\rho\beta$. Endemic equilibria in Ω correspond one-to-one to roots of ψ in $(0, i_c)$ (Lemma 1; conversely every such root gives positive s^*, e^*, τ^*, r^* and $\Sigma = 1$). By Lemma 3 and Lemma 5, $\psi'(i^*) = B_3/i^* > 0$ at every such root. If $R_0 > 1$: $\psi(0) = \kappa AB(1 - R_0) < 0$ and $\psi(i_c) = \alpha\beta\kappa(\gamma/\mu_2 + \sigma) > 0$, so a root exists. Two consecutive roots are

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impossible: ψ would be positive right of the first and negative left of the second, forcing a root between them. If $R_0 \leq 1$: $\psi > 0$ just right of 0 (for $R_0 = 1$ since $\psi'(0) = AB(\beta - \mu_2) - \kappa\mu_2(A + B) > 0$, which follows from $\beta \geq AB/\alpha$ and $B > \mu_2$), so the smallest root i_1 would have $\psi'(i_1) \leq 0$, a contradiction. ■

Therefore for $\mu_2 > 0$ the preceding Theorem shows that the conditions $i^* < \kappa/\mu_2$ and $B_1 > 0,\ B_3 > 0,\ B_1B_2 > B_3$ hold automatically at every endemic equilibrium so all five eigenvalues of $J(E_1)$ have negative real parts, and E_1 is locally asymptotically stable.

Proposition. For $\mu_2 = 0$, the endemic equilibrium E_1 exists if and only if $R_0 > 1$ and, whenever it exists, it is locally asymptotically stable.

Proof.

For $\mu_2 = 0$, we get $a_{55} = -\kappa < 0$ and $a_{44} = -(\delta + \kappa) < 0$. In the (s, e, i), $P = \kappa + \beta i^*, Q = \alpha + \kappa$ and $R = \gamma + \kappa$, and the equilibrium relation $\beta s^* = (\alpha + \kappa)(\gamma + \kappa)/\alpha$ gives $\alpha\beta s^* = QR$. Then $B_1 = P + Q + R > 0, B_2 = PQ + PR + QR - \alpha\beta s^* = P(Q + R) > 0$ and $B_3 = \alpha\beta^2 s^* i^* > 0$. Moreover, since $P > \beta i^*$ and $(P + Q + R)(Q + R) > (Q + R)^2 \geq 4QR$, we have $B_1B_2 = P(P + Q + R)(Q + R) > 4\beta i^* QR > \beta i^* QR = B_3$. Hence all Routh Hurwitz conditions hold and E_1 is locally asymptotically stable. ■

Numerical simulation

The model was validated numerically using the 2022 demographic and surveillance data set for Ambon City, Indonesia [21], reproduced in Table 1, with initial subpopulations given in (Table 2).

Table 1. Parameters values used in the simulations.

Parameters	Description	Values
κ	Natural birth rate	0.0153
σ	Rate of vaccinated individuals	0.6497
α	Rate of exposed individuals becoming infected	4
γ	Rate of infected individuals on treatment	0.1354
δ	Rate of individuals who recover after treatment	1
μ_1	Natural mortality rate	0.0141
μ_2	Death rate due to Hepatitis B infection	0

Table 2. Initial conditions [21]

Subpopulations	Initial value (individuals)
S(0)	121,663
E(0)	0
I(0)	792
T(0)	124
R(0)	225,646
N(0)	348,225

Since $\kappa = 0.0153 \neq \mu_1 = 0.0141$, the absolute population grows without bound. Eq. (6) with $\mu_2 = 0$ gives the same result. All differential equations were integrated numerically in Python using the implicit high-order Radau method (suitable for the stiff regimes encountered near $R_0 = 1$). Absolute and relative tolerances were both 10^{-9}. That resolution was needed for the slow long-term decay of the infectious proportion. Because $N(t)$ grows exponentially when $\kappa > \mu_1$ and $\mu_2 = 0$, the long horizons used below are mathematical devices for exhibiting the asymptotic behavior of the proportions s, e, i, τ, r, not demographic projections.

The exact transition point between extinction and endemicity (critical threshold) is found by solving $R_0 = 1$ algebraically:

$$\beta^* = \frac{(\alpha + \kappa)(\gamma + \kappa)}{\alpha(1 - \sigma)} \approx 0.432$$

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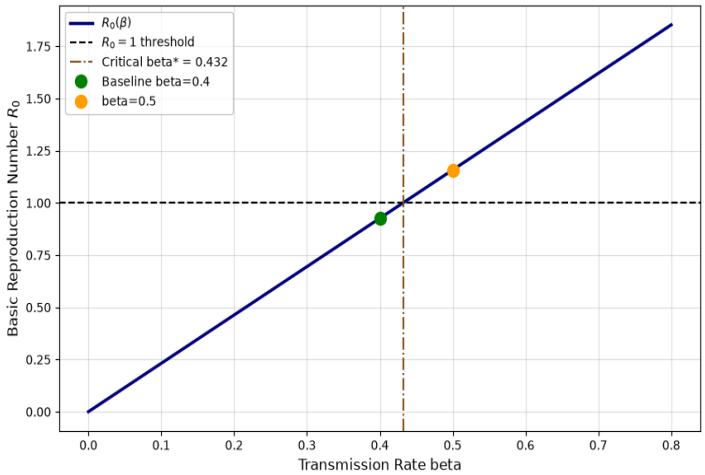


Figure 2. R_0 as a function of the transmission rate β, with critical threshold $\beta^* \approx 0.432$

(Figure 3) presents a long-horizon simulation (3000 years) for both $\beta = 0.4$ and $\beta = 0.5$, tracking $i(t)$ on a logarithmic scale. For $\beta = 0.4$ the curve continues its exponential decline to numerically negligible values ($\approx 10^{-15}\%$) by year 3000 confirming eventual extinction, although the decline is very slow. In contrast, the curve for $\beta = 0.5$ settles at the constant positive level i^* predicted analytically by Eq. (15). For $\mu_2 = 0$, Eq. (15) reduces to $i^* = \kappa(R_0 - 1)/\beta = 0.0153 \times 0.158/0.5 \approx 0.483\%$, in agreement with the simulation.

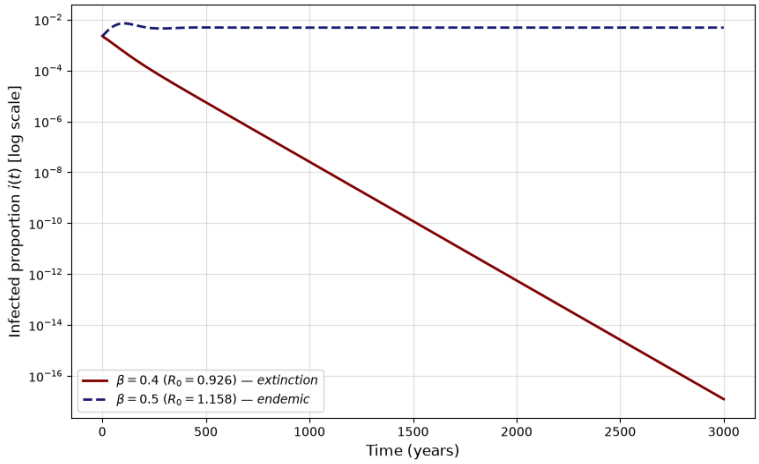


Figure 3. Long-term (3000-year) behaviour of the infectious proportion $i(t)$ on a logarithmic scale

(Table 3) reports the normalized sensitivity indices of R_0 evaluated numerically at the Ambon baseline ($\beta = 0.4$) by finite differences. These are the elasticities $\Upsilon_p = (\partial R_0/\partial p)(p/R_0)$, which have closed forms as follows:

$$\Upsilon_\sigma = -\frac{\sigma}{1-\sigma}, \quad \Upsilon_\gamma = -\frac{\gamma}{\gamma+\kappa}, \quad \Upsilon_\kappa = -\frac{\kappa}{\alpha+\kappa} - \frac{\kappa}{\gamma+\kappa} \text{ and } \Upsilon_\alpha = \frac{\kappa}{\alpha+\kappa}$$

Table 3. Normalized sensitivity indices of R_0, ranked in descending magnitude

Rank	Parameter	Sensitivity index Y	Biological interpretation
1	σ	−1.855	Most influential; raising vaccination coverage sharply lowers R_0
2	β	+1.000	Directly proportional to R_0 by definition
3	γ	−0.898	Faster entry into treatment lowers R_0 effectively
4	κ	−0.105	Comparatively weak effect over the realistic biological range
5	α	+0.004	Nearly negligible effect

The vaccination coverage σ has the largest normalized index (-1.855). It grows in magnitude as σ approaches 1, while σ itself is bounded above by 1, so the room for improvement is limited (from 0.65 to at most 1). The transmission rate β (index $+1$) and the treatment rate γ (-0.898) follow. The birth rate κ and the incubation rate α have only marginal influence within the realistic Ambon range, so interventions aimed at shortening the incubation period or speeding up diagnosis yield little benefit compared with vaccination and early treatment.

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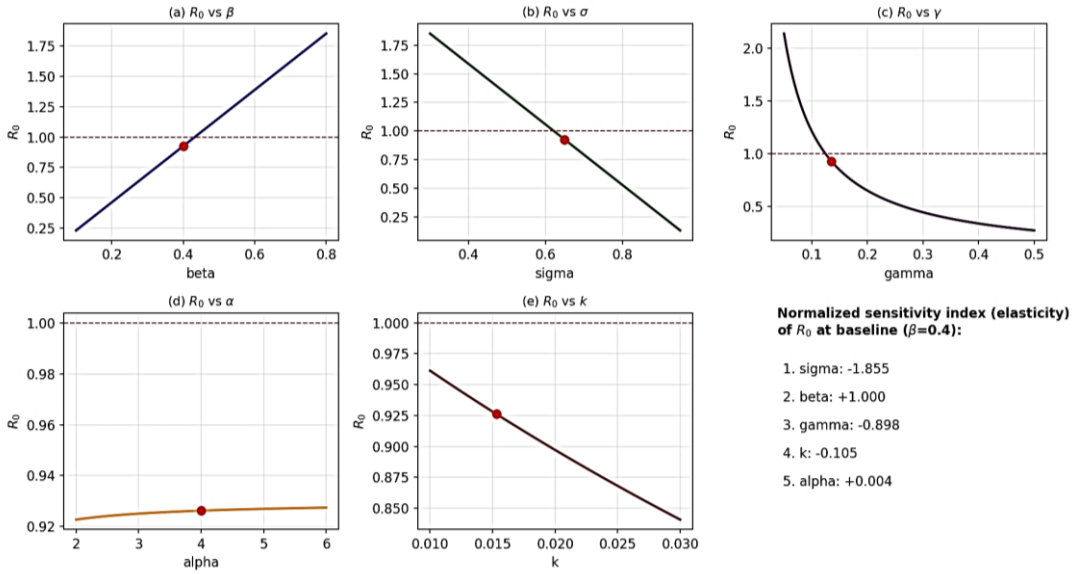


Figure 4. Sensitivity curves of R_0 with respect to each parameter, ranked by sensitivity index

(Figure 4) shows how R_0 responds to each parameter ranked by sensitivity index. The transmission rate β has a proportional effect since R_0 is linear in β (Index +1.000). Vaccination coverage σ has the largest elasticity (−1.855). R_0 is also linear in σ, so each additional percentage point of coverage lowers it by the same absolute amount. What grows with σ is the relative effect, which is about 1.9% per 1% increase in σ at the Ambon level ($\sigma \approx 0.65$), compared with about 0.3% at $\sigma = 0.25$. The treatment rate γ follows (−0.898), then the birth rate κ (−0.105), whereas α has almost no influence (+0.004). These elasticities are local thus, they describe small changes around the baseline. The baseline lies close to the threshold; modest changes in β or σ could move R_0 across 1.

In (Figure 5), three single interventions and their combination were compared at the $\beta = 0.4$ baseline ($R_0 = 0.926$): an increase 50% in vaccination coverage σ, a 50% reduction in the transmission rate β, and a 50% increase in the treatment rate γ. Increasing σ gives the largest reduction ($R_0 \approx 0.067$), followed by reducing ($\beta \approx 0.463$) and increasing ($\gamma \approx 0.639$); the combined strategy gives $R_0 \approx 0.023$. Because R_0 is a product of factors, simultaneous interventions multiply their effects: $0.926 \times 0.0726 \times 0.5 \times 0.690 \approx 0.023$. The combined reduction (0.903) is therefore smaller than the sum of the individual reductions ($0.859 + 0.463 + 0.287 = 1.609$, which even exceeds the baseline value), so no synergistic effect is implied.

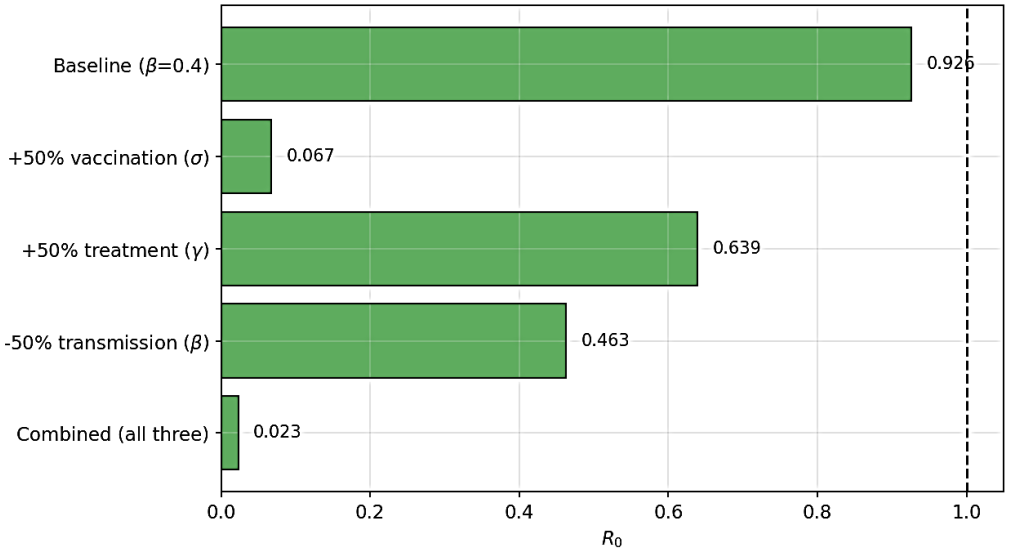


Figure 5. Effect of different control strategies on the basic reproduction number R_0

(Figure 6) shows that i^* emerges continuously from zero at $\beta^* \approx 0.432$ and increases monotonically and concavely with β. For $\mu_2 = 0$ this follows directly from $i^* = \kappa(R_0 - 1)/\beta$ thus the transition at $\beta = \beta^*$ is a forward (transcritical) bifurcation without hysteresis: no stable endemic equilibrium coexists with the disease-free equilibrium when $R_0 < 1$.

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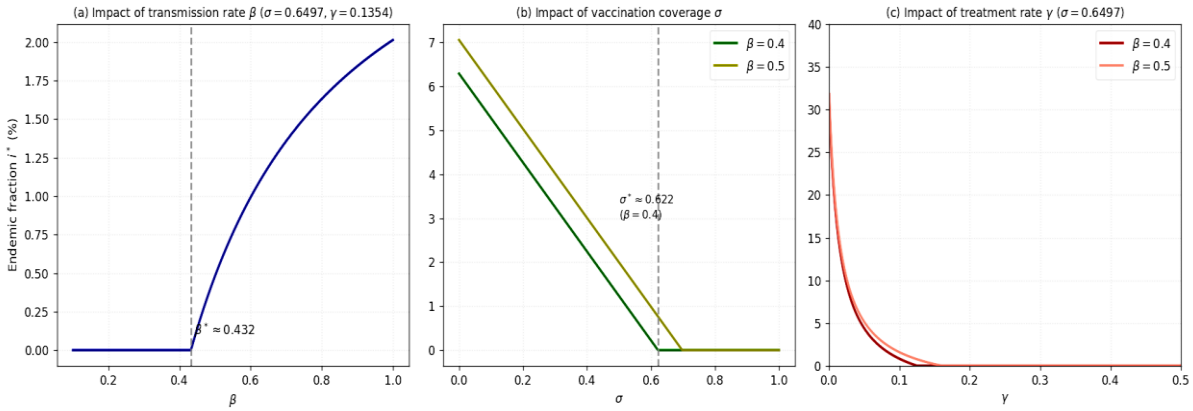


Figure 6. i^* as a function of (a) the transmission rate β (b) the vaccination coverage σ for $\beta = 0.4$ and 0.5, and (c) the treatment rate γ

Similarly, i^* declines monotonically and, for $\mu_2 = 0$, linearly as vaccination coverage σ increases, reaching a critical vaccination threshold $\sigma^* \approx 0.622$, above which the disease cannot persist at $\beta = 0.4$ (Figure 6b). Since Ambon's actual coverage ($\sigma \approx 0.65$) already exceeds σ^*, this quantitatively explains why the baseline R_0 (0.926) falls below unity for the city.

(Figure 7) shows that Ambon's operating point ($\beta \approx 0.4, \sigma \approx 0.65$) lies in the disease-free region but close to the $R_0 = 1$ threshold line, underscoring the sensitivity of the outcome to small parameter changes and the slow decay seen in the long-horizon simulation (Figure 3).

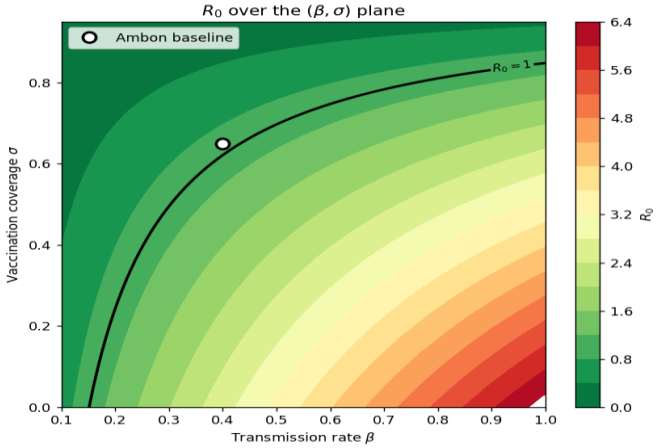


Figure 7. Contour map of R_0 over the (β, σ) plane, with the $R_0 = 1$ threshold line

Discussion

The analysis establishes that the basic reproduction number is the threshold quantity for the local dynamics of the SEITR model under standard incidence: the disease-free equilibrium is locally asymptotically stable when $R_0 < 1$ and unstable when $R_0 > 1$, and for $\mu_2 = 0$ the endemic equilibrium exists and is locally asymptotically stable precisely when $R_0 > 1$. The reduction to population proportions is essential, because with standard incidence and a growing population the absolute variables have no equilibrium, whereas the proportions do. These results are local.

The numerical results for Ambon City illustrate three points of practical significance. First, the baseline operating point ($R_0 \approx 0.926$) is below the threshold, but only narrowly: an increase of the transmission rate from 0.40 to above 0.432, or a fall in vaccination coverage from 0.65 to below 0.622, would make the infection endemic ($i^* \approx 0.5\%$ for $\beta = 0.5$). The control program therefore rests on a narrow margin of safety. Second, even when $R_0 < 1$ the approach to the disease-free state is slow. Third, in relative terms, R_0 responds most strongly to vaccination coverage, followed by the transmission rate and the treatment rate. Because σ cannot exceed 1 and its index is amplified as σ approaches 1, this supports maintaining or modestly increasing coverage and speeding up treatment.

Conclusion

This study analysed a five-compartment SEITR model of Hepatitis B transmission with standard incidence and variable population size. After reducing the model to population proportions, the disease-free and endemic equilibria were derived and the basic reproduction number was obtained by the next generation matrix method. Using the Routh-Hurwitz criterion the disease-free equilibrium was shown to be locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. For $\mu_2 > 0$, a unique endemic equilibrium exists if and only if $R_0 > 1$, and it is locally asymptotically stable. For $\mu_2 = 0$ the

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endemic equilibrium is given explicitly by $i^* = \kappa(R_0 - 1)/\beta$; it exists and is locally asymptotically stable whenever $R_0 > 1$ with a forward bifurcation at $R_0 = 1$. Simulations calibrated to 2022 data from Ambon City gave $R_0 \approx 0.926$ and critical values $\beta^* \approx 0.432$ and $\sigma^* \approx 0.622$, so the city lies just on the disease-free side of the threshold, and the infection would disappear only very slowly. Normalized sensitivity indices ranked vaccination coverage (-1.855) first, followed by the transmission rate $(+1.000)$ and the treatment rate (-0.898). These findings suggest that maintaining or modestly raising vaccination coverage and speeding up treatment are the most effective ways to keep R_0 safely below one.

Conflict of interest. Nil

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