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Mathematical Modeling and Integrated Optimal Control of HCV–Typhoid Coinfection in Developing Nations

***[1]Olawuyi, O.M., & [2]Nkuturum, C.**
[1]Department of Mathematics, Alvan Ikoku Federal University of Education, Owerri, Imo State
[2]Department of Mathematics, Rivers State University, Port Harcourt, Rivers State

***Corresponding author email**: mojiseye213@gmail.com

Abstract
Hepatitis C virus (HCV) and Typhoid fever present low-level endemic status in developing countries. Unregulated use of medical practices, inadequate sanitation, and poor health infrastructure aggravate the situation. Past studies have concentrated on the transmission of the diseases and employed basic modelling. However, the need for integrated control strategies has been largely ignored. The present study employs a deterministic mathematical model to examine the impact of integrated control strategies (Typhoid intervention, Typhoid and HCV treatment, sanitation, and HCV screening) on reducing the burden of Typhoid and HCV coinfection in a high-risk population. Optimal control theory is used to model the implementation of the strategies for a period of 400 days. The study findings indicate that simultaneous implementation of the strategies Multiple Health Interventions for HCV and Typhoid treatments, coupled with improved control of environmental sanitation, and Typhoid and HCV screening, would be most effective in addressing the dual burden of Typhoid and HCV coinfection in developing countries such as Nigeria.

Keywords: HCV, Typhoid Fever, Coinfection, Optimal Control, Sanitation

Introduction

Typhoid fever and Hepatitis C Virus infections are major public health concerns around the world and in developing countries. Hepatitis C Virus infections are caused by the exposure to blood-borne viruses. Typhoid enteric fever is caused by Salmonella Typhi and is transmitted through food and water contamination. In Nigeria and many countries, these diseases are the primary causes of morbidity and mortality. Many of these diseases affect the same systems of the body, so one of the main grievances caused by these diseases is the difficulty in diagnosis and treatment. Typhoid enteric fever and Hepatitis C virus co-infection has been researched, and the purpose of this research is to provide a framework of possible public health Integrated Control options to help unravel the complex systems of combined risks and burdens to develop safe and simple public health Integrated Control options. Many countries and regions of the world have high incidences of both Typhoid Enteric Fever and Hepatitis C Virus infections. Public Health control options for these regions are often limited to blood-borne virus control and water and sanitation. The impact among co-infected individuals is the complex burden of reduced immunity, delayed healing, and increased risks of complications. The integration of control measures for both Typhoid fever and Hepatitis C virus is urgently needed. Mathematical modelling can be used in conjunction with optimal control theory to offer a different perspective on how different interventions relate to one another and how to efficiently allocate limited resources. Thus far, few models have been developed that combine and integrate the HCV and Typhoid control strategy approaches. For policy makers, this article attempts to develop a composite model that spans many control measures to contribute to the understanding of how to reduce the co-infection burden with a limited amount of resources. The implementation of optimal control strategies to reduce the burden of HCV and Typhoid co-infection in Low-and Middle-Income (LMIC) settings, and, more specifically, the impact of control strategies on reducing co-infection, as well as the combined impact of control

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of Typhoid, screening, and treatment of HCV, and environmental sanitation. Although research has been conducted on Typhoid fever and Hepatitis C in isolation, few research studies have assessed the combined impacts on populations, particularly within and among the majority of the developing countries. Recent research shows concerning trends in Hepatitis C prevalence reporting that 58 million people in the world currently living with HCV. HCV is a major contributor to the burden of chronic liver disease and hepatocellular carcinoma. The development of direct-acting antivirals that provide a very high cure rate, but are demanding of the country's healthcare system.

In most regions of sub-Saharan Africa, including Nigeria, typhoid fever is one of the major contributors to illness and death due to the presence of inadequate sanitation and the availability of contaminated water. According to WHO (2023), about 5.4 billion people are estimated to be at risk of typhoid, with over 200,000 people dying from the illness each year. Vaccination, treatment, and sanitation are examples of optimal control models (with positive control) that significantly reduce the incidence and prevalence of infectious diseases. There has been previous work on optimal control theory and the modeling of typhoid and HCV infections separately and jointly. Lawal et al. (2024) highlighted the importance of environmental sanitation, treatment, and personal hygiene to incomplete control of typhoid fever. There is almost no literature that combines HCV and typhoid fever (TF) modelling, particularly for the developing (or low resource) part of the world. This creates the possibility to holistically use the optimal control theory to model comprehensive intervention approaches.

Methodology

This study extends a deterministic compartmental model on co-infection of HCV and Typhoid fever. The population consists of multiple compartments that include susceptible, exposed, acute infected, recovered, and environmental bacteria.

Model development

The population is classified into eight compartments: the susceptible represented by $S(t)$, **exposed Typhoid** $E_T(t)$, acute typhoid $A_T(t)$, **exposed HCV** $E_H(t)$, acute HCV $A_H(t)$, acute typhoid-HCV co-infection $A_C(t)$, those who had recovered from infected population $R(t)$ and bacteria population $B(t)$ all in time t. The entire human population size $N(t)$ can be described as:

$$N(t) = S(t) + E_T(t) + A_T(t) + E_H(t) + A_H(t) + A_C(t) + R(t) \quad (1)$$

People start as *susceptible* if they are healthy and have no immunity to either disease. They enter the susceptible group through births (Λ) newborns are assumed to be susceptible. Susceptible individuals acquire Typhoid through contact with the bacterial by a force of infection λ_T and HCV λ_H expressed as $\lambda_T = \beta_T\left(\frac{\eta_1 A_T + \eta_2 A_C + B}{N}\right)$ and $\lambda_H = \beta_H\left(\frac{A_H + A_C}{N}\right)$ respectively. In these expressions, β_T and β_H denotes the effective contact rate for Typhoid fever and HCV infections. The parameters η_1 and η_2 accounts for the relative transmissibility potential of Typhoid fever from A_T and A_C respectively. The susceptible decreases also by natural death denoted by μ. The resulting ode for the susceptible population is given as:

$$\frac{dS}{dt} = \Lambda - (\lambda_T + \lambda_H + \mu)S \quad (2)$$

Exposed population to typhoid (E_T)

The exposed population gives the typhoid infection pathway. Once a susceptible person is infected with Typhoid bacteria; they **first enter the exposed Typhoid group**(E_T), meaning they are infected but not yet infectious. They leave E_T by: becoming acutely infectious (σ_1) progressing acute typhoid fever A_T and dying naturally at the rate of μ. The ode for the exposed population is given as:

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$$\frac{dE_T}{dt} = \lambda_T S - (\sigma_1 + \mu)E_T \quad (3)$$

Acute typhoid (A_T)

These individuals have symptoms and can infect others through direct contact or contaminating the environment. The population progress to acquire HCV-Typhoid co-infection through contact with HCV-Typhoid at the rate $\lambda_H A_T$ which becomes infected with HCV and re-infected with A_T at the rate of θ_1. The population decreases by death due to Typhoid at δ_1, natural death by μ or recovers by γ_1. The dynamics is represented by:

$$\frac{dA_T}{dt} = \sigma_1 E_T + \theta_1 A_C - \lambda_H A_T - (\gamma_1 + \delta_1 + \mu)A_T \quad (4)$$

Exposed population to HCV (E_H)

The HCV exposed population acts as the HCV infection pathway in this case. If a susceptible person gets HCV that occurs at $\lambda_H S$, they **enter the exposed HCV group**(E_H) infected but not infectious. They leave E_H by progressing to acute HCV (σ_2) and die at the rate of μ. The system is described as:

$$\frac{dE_H}{dt} = \lambda_H S - (\sigma_2 + \mu)E_H \quad (5)$$

Acute HCV (A_H)

This individual shows symptom and can infect others through direct contact. The population progress A_C by acquiring typhoid to become HCV-Typhoid co-infected through $\lambda_T A_H$ and re-infected with HCV at the rate of θ_2. They leave the population through death due to HCV at δ_2, by natural death at the rate of μ or recovers by γ_2. The system is represented by:

$$\frac{dA_H}{dt} = \sigma_2 E_H + \theta_2 A_C - \lambda_T A_H - (\gamma_2 + \delta_2 + \mu)A_T \quad (6)$$

HCV-Typhoid co-infection (A_C).

Both the acute Typhoid patients A_T and acute HCV A_H who get infected by HCV and Typhoid move into co-infection at $\lambda_H A_T$ and $\lambda_T A_H$ respectively. Outflow from A_C when they recover from both diseases and transition into the recovered class R at the rate of $\gamma_3 A_C$. Partial regression to the acute Typhoid compartment A_T (e.g., if HCV symptoms become dormant/controlled but Typhoid persists) occurs at the rate of $\theta_1 A_C$. Similarly, partial regression to the acute HCV compartment A_H (e.g., if Typhoid is treated but HCV persists) occurs at the rate of $\theta_2 A_C$. Also, the population reduces by the death due to the disease and natural death at the rate of $\delta_3 A_C$ and μA_C respectively. The dynamics for this description are given as:

$$\frac{dA_C}{dt} = \lambda_H A_T + \lambda_T A_H - (\gamma_3 + \theta_1 + \theta_2 + \delta_3 + \mu)A_C \quad (7)$$

Recovered population (R)

The two diseases acute Typhoid infected individuals (A_T) and acute HCV (A_H) infected individuals recover at rate γ_1 and γ_2 respectively to R. Similarly, co-infected individuals (A_C) recover from both infections simultaneously recover at rate γ_3 and move into the recovered class R. The outflow from the recovered class is **natural death at the rate of** μ and the ordinary differential equation is given as:

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$$\frac{dR}{dt} = \gamma_1 A_T + \gamma_2 A_H + \gamma_3 A_C - \mu R \quad (8)$$

Environmental bacteria (B)

Typhoid bacteria survive in the environment (water, food, etc.) and contribute to new infections. Bacteria are shed into the environment by acute typhoid $(\epsilon_1 A_T)$, acute HCV- Typhoid $(\epsilon_2 A_C)$, and the bacteria naturally die or decay at rate τ. This gives the following ode:

$$\frac{dB}{dt} = \epsilon_1 A_T + \epsilon_2 A_C - \tau B \quad (9)$$

Therefore, the dynamical system associated with the schematic diagram in Figure 1 is;

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - (\lambda_T + \lambda_H + \mu)S \\
\frac{dE_T}{dt} &= \lambda_T S - (\sigma_1 + \mu)E_T \\
\frac{dA_T}{dt} &= \sigma_1 E_T + \theta_1 A_c - \lambda_H A_T - (\gamma_1 + \delta_1 + \mu)A_T \\
\frac{dE_H}{dt} &= \lambda_H S - (\sigma_2 + \mu)E_H \\
\frac{dA_H}{dt} &= \sigma_2 E_H + \theta_2 A_c - \lambda_T A_H - (\gamma_2 + \delta_2 + \mu)A_H \quad (10) \\
\frac{dA_C}{dt} &= \lambda_H A_T + \lambda_T A_H - (\gamma_3 + \theta_1 + \theta_2 + \delta_3 + \mu)A_C \\
\frac{dR}{dt} &= \gamma_1 A_T + \gamma_2 A_H + \gamma_3 A_C - \mu R \\
\frac{dB}{dt} &= \epsilon_1 A_T + \epsilon_2 A_C - \tau B
\end{aligned}$$

where $\lambda_T = \beta_T \left(\frac{\eta_1 A_T + \eta_2 A_C}{N} + \frac{B}{K+B}\right)$ and $\lambda_H = \beta_H \left(\frac{A_H + A_C}{N}\right)$

with initial conditions

$S(0) = S_0 > 0, E_T(0) = E_{T0} \geq 0, A_T(0) = A_{T0} \geq 0, E_H(0) = E_{H0} \geq 0, A_H(0) = A_{H0} \geq 0,$

$A_C(0) = A_{C0} \geq 0, B(0) = B_0 \geq 0, R(0) = R_0 \geq 0$ (Olawuyi, et al., 2026).

The model incorporates four key interventions: Typhoid prevention (vaccination), treatment of Typhoid, HCV screening and treatment, and environmental sanitation.

Optimal-control extension of HCV–Typhoid co-infection model

An introduction of four public-health relevant control measures, written as the controlled ODE system, give a reasonable objective functional, state the Pontryagin necessary conditions (Hamiltonian, adjoints, and the optimal control characterizations), and finish with practical implementation notes (numerical method, parameter suggestions, and public-health interpretation).

Control measures (public-health relevant) were chosen as follows. By introducing four time-dependent controls $u_i(t) \epsilon [0,1]$;

$u_1(t)$: **Vaccination** against typhoid for susceptible (moves some S to R). (HCV: no vaccine, so vaccination targets typhoid only.)

$u_2(t)$: **Treatment for typhoid infectives** (increases recovery of A_T and helps coinfected A_T).

$u_3(t)$: **Treatment for HCV infectives** (increases recovery of A_H and helps coinfected A_C).

$u_4(t)$: **Environmental / sanitation & prevention** (reduces transmission: lowers β_T, β_H and increases bacterial clearanceτ; represents improved water, sanitation, hygiene, and risk-reduction campaigns).

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The controlled model (modifications introduced highlighted). Write N as total human population (unchanged). Let constants p_1, p_2, p_{c1}, p_{c2} be effectiveness factors of treatment/vaccination on recovery/transmission. Let ϕ be the gain in bacterial clearance due to sanitation. For simplicity we model transmission reduction multiplicatively by $(1 - u_4)$.

Controlled forces of infection:

$$\lambda_T = \beta_T(1 - u_4)\left(\frac{\eta_1 A_T + \eta_2 A_C}{N} + \frac{B}{K + B}\right) \quad (11)$$

$$\lambda_H = \beta_H(1 - u_4)\left(\frac{A_H + A_C}{N}\right) \quad (12)$$

Controlled ODEs for HCV – typhoid co-infection in a given population is:

$$\frac{dS}{dt} = \Lambda - (\lambda_T(u_4) + \lambda_H(u_4) + \mu)S - u_1 S \quad (3.143)$$

$$\frac{dE_T}{dt} = \lambda_T(u_4)S - (\sigma_1 + \mu)E_T \quad (13)$$

$$\frac{dA_T}{dt} = \sigma_1 E_T + \theta_1 A_c - \lambda_H A_T - (\gamma_1 + p_1 u_2 + \delta_1 + \mu)A_T \quad (14)$$

$$\frac{dE_H}{dt} = \lambda_H(u_4)S - (\sigma_2 + \mu)E_H \quad (15)$$

$$\frac{dA_H}{dt} = \sigma_2 E_H + \theta_2 A_c - \lambda_T A_H - (\gamma_2 + p_2 u_3 + \delta_2 + \mu)A_H \quad (16)$$

$$\frac{dA_C}{dt} = \lambda_H(u_4)A_T + \lambda_T(u_4)A_H - (\gamma_3 + p_{c1}u_2 + p_{c2}u_3 + \theta_1 + \theta_2 + \delta_3 + \mu)A_C \quad (17)$$

$$\frac{dR}{dt} = \gamma_1 A_T + \gamma_2 A_H + \gamma_3 A_C + u_1 S + (p_{c1}u_2 + p_{c2}u_3)A_C - \mu R \quad (18)$$

$$\frac{dB}{dt} = \epsilon_1 A_T + \epsilon_2 A_C - (\tau + \phi u_4)B \quad (19)$$

Vaccination $u_1 S$ moves directly to R (temporarily immune/protected). Treatment terms $p_1 u_2, p_2 u_3$increase effective recovery rates. $p_{c1}u_2, p_{c2}u_3$ Represent how treatment of single infections affects confected recovery. Sanitation u_4 both reduces transmission $\beta_T, \beta_H . 1 - u_4$
And speeds bacterial clearance $\tau + \phi u_4$

These interventions are modeled using control functions that vary over time to minimize disease prevalence while balancing costs.

Optimal Control Analysis

Typical objective: reduce burden (infectious classes + bacterial exposure) and keep control costs moderate: Therefore, we proceed to compute the optimal control problem as performed in Omowumi, Tunde and Afeez (2024).
The objective functional for the fixed T is given as

$$J(u_1, u_2, u_3, u_4) = \int_0^T \left(w_1 A_T + w_2 A_H + w_3 A_C + w_4 B + \frac{1}{2}(c_1 u_1^2 + c_2 u_2^2 + c_3 u_3^2 + c_4 u_4^2)\right) dt \quad (20)$$

where w_i weight epidemiologic importance and $C_i > 0$ are cost weights (quadratic cost penalizes high control effort). The terminal time T is fixed (e.g., 1–5 years). To minimize J subject to the controlled ODEs and control bounds $0 \leq u_i(t) \leq 1$. The Hamiltonian $\mathcal{H}$ function for both the linear and quadratic form of the control measures in J and the objective function enhances the Hamiltomian corrected with the optimal control problem to be maximized. Therefore, we intend to find the optimal control $(u_1^*, u_2^*, u_3^*, u_4^*)\epsilon U$ which satisfied equation J above by applying the Pontryagin maximum Principle — Hamiltonian & adjoint system

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$$J(u_1^*, u_2^*, u_3^*, u_4^*) = min\{J(u_1, u_2, u_3, u_4) | (u_1, u_2, u_3, u_4) \in U\} \quad (21)$$

Therefore, the associated Pseudo-Hamiltonian is:

$$\begin{aligned}\mathcal{H} = w_1 A_T + w_2 A_H + w_3 A_C + w_4 B + \tfrac{1}{2}\textstyle\sum_{i=1}^{4} C_i u_i^2 \\ +\pi_1[\Lambda - (u_4\lambda_T + u_4\lambda_H + \mu)S - u_1 S] \\ +\pi_2[u_4\lambda_T S - (\sigma_1 + \mu)E_T] \\ +\pi_3[\sigma_1 E_T + \theta_1 A_C - (\gamma_1 + p_1 u_2 + \delta_1 + \mu)A_T] \\ +\pi_4[u_4\lambda_H S - (\sigma_2 + \mu)E_H] \\ +\pi_5[\sigma_2 E_H + \theta_2 A_C - (\gamma_2 + p_2 u_3 + \delta_2 + \mu)A_H] \\ +\pi_6[u_4\lambda_H A_T + u_4\lambda_T A_H - (\gamma_3 + p_{c1}u_2 + p_{c2}u_3 + \theta_1 + \theta_2 + \delta_3 + \mu)A_C] \\ +\pi_7[\gamma_1 A_T + \gamma_2 A_H + \gamma_3 A_C + u_1 S + p_1 u_2 A_T + p_2 u_3 A_H + (p_{c1}u_2 + p_{c2}u_3)A_C - \mu R] \\ +\pi_8[\epsilon_1 A_T + \epsilon_2 A_C - (\tau + \phi u_4)B]\end{aligned}$$

where $\pi_i, i = 1, \dots, 8$ are the adjoint variables satisfying:

$\frac{d\pi_1}{dt} = -\frac{d\mathcal{H}}{dS}$, $\frac{d\pi_2}{dt} = -\frac{d\mathcal{H}}{dE_T}$, $\frac{d\pi_3}{dt} = -\frac{d\mathcal{H}}{dA_T}$, $\frac{d\pi_4}{dt} = -\frac{d\mathcal{H}}{dE_H}$, $\frac{d\pi_5}{dt} = -\frac{d\mathcal{H}}{dA_H}$, $\frac{d\pi_6}{dt} = -\frac{d\mathcal{H}}{dA_C}$, $\frac{d\pi_7}{dt} = -\frac{d\mathcal{H}}{dR}$, $\frac{d\pi_8}{dt} = -\frac{d\mathcal{H}}{dB}$. Next, we obtained the adjoint variables in the form of a differential equations $\mathcal{H}$ as

$$\frac{d\mathcal{H}}{dS} = \pi_1[-u_4\lambda_T - u_4\lambda_H - \mu - u_1] + \pi_2 u_4\lambda_T + \pi_4 u_4\lambda_H + \pi_7 u_1$$

$$\Rightarrow \frac{d\pi_1}{dt} = -\frac{d\mathcal{H}}{dS} = u_4\beta_T(\pi_2 - \pi_4)(1 - u_4)\left(\frac{\eta_1 A_T + \eta_2 A_C}{N}\right) + u_4\beta_H(\pi_1 - \pi_4)\left(\frac{A_H + A_C}{N}\right) + u_1(\pi_7 - \pi_1)$$

$$\frac{d\mathcal{H}}{dE_T} = -\pi_2(\sigma_1 + \mu) + \pi_3\sigma_1$$

$$\Rightarrow \frac{d\pi_2}{dt} = -\frac{d\mathcal{H}}{dE_T} = \sigma_1(\pi_2 - \pi_3) + \pi_2\mu$$

$$\frac{d\mathcal{H}}{dA_T} = w_1 - \pi_3(\gamma_1 + p_1 u_2 + \delta_1 + \mu) + \pi_6 u_4\lambda_H + \pi_7\gamma_1 + \pi_7 p_1 u_2 + \pi_8\epsilon_1$$

$$\Rightarrow \frac{d\pi_3}{dt} = -\frac{d\mathcal{H}}{dA_T} = (\pi_7 - \pi_3)(p_1 u_2 - \gamma_1) + \pi_3(\delta_1 + \mu) - \pi_6 u_4\beta_H\left(\frac{A_H + A_C}{N}\right) - \pi_8\epsilon_1$$

$$\frac{d\mathcal{H}}{dE_H} = -\pi_4(\sigma_2 + \mu) + \pi_5\sigma_2$$

$$\Rightarrow \frac{d\pi_4}{dt} = -\frac{d\mathcal{H}}{dE_H} = \sigma_2(\pi_4 - \pi_5) + \pi_4\mu$$

$$\frac{d\mathcal{H}}{dA_H} = w_2 - \pi_5(\gamma_2 + p_2 u_3 + \delta_2 + \mu) + \pi_6 u_4\lambda_T + \pi_7\gamma_2 + \pi_7 p_2 u_3$$

$$\Rightarrow \frac{d\pi_5}{dt} = -\frac{dH}{dA_H} = (\pi_5 - \pi_7)(p_2 u_3 + \gamma_2) + \pi_5(\delta_2 + \mu) - \pi_6 u_4\beta_T\left(\frac{\eta_1 A_T + \eta_2 A_C}{N}\right) - w_2$$

$$\frac{d\mathcal{H}}{dA_C} = w_3 - \pi_6(\gamma_3 + p_{c1}u_2 + p_{c2}u_3 + \theta_1 + \theta_2 + \delta_3 + \mu) + \pi_7\gamma_3 + \pi_8\epsilon_2 + \pi_3\theta_1 + \pi_5\theta_2$$

$$\Rightarrow \frac{d\pi_6}{dt} = -\frac{d\mathcal{H}}{dA_C} = \pi_6(p_{c1}u_2 + p_{c2}u_3 + \delta_3 + \mu) + \theta_1(\pi_6 - \pi_3) + \theta_2(\pi_6 - \pi_5) + \gamma_3(\pi_6 - \pi_7) - \pi_8\epsilon_2 - w_3$$

$$\frac{d\mathcal{H}}{dR} = -\mu\pi_7$$

$$\Rightarrow \frac{d\pi_7}{dt} = -\frac{d\mathcal{H}}{dR} = \mu\pi_7$$

$$\frac{d\mathcal{H}}{dB} = -\pi_8(\tau + \phi u_4) \Rightarrow \frac{d\pi_8}{dt} = -\frac{d\mathcal{H}}{dB} = \pi_8(\tau + \phi u_4)$$

The necessary and sufficient optimality conditions are

$$\frac{\partial\mathcal{H}}{\partial u_1^*} = 0, \frac{\partial\mathcal{H}}{\partial u_2^*} = 0, \frac{\partial\mathcal{H}}{\partial u_3^*} = 0, \frac{\partial\mathcal{H}}{\partial u_4^*} = 0 \quad (22)$$

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These give the optimal controls from the associated Pseudo-Hamiltonian given above. Hence,

$$\frac{\partial \mathcal{H}}{\partial u_1^*} = c_1 u_1^* - \pi_1 S + \pi_7 S = 0$$

$$\Rightarrow u_1^* = \frac{(\pi_1 - \pi_7)S}{c_1} \tag{23}$$

$$\frac{\partial \mathcal{H}}{\partial u_2^*} = c_2 u_2^* - \pi_3 p_1 A_T - \pi_6 p_{c1} A_C + \pi_7 p_1 A_T + \pi_7 p_{c1} A_C = 0$$

$$\Rightarrow u_2^* = \frac{(\pi_7 - \pi_3) p_1 A_T + (\pi_6 - \pi_7) p_{c1} A_C}{c_2} \tag{24}$$

$$\frac{\partial \mathcal{H}}{\partial u_3^*} = c_3 u_3^* - \pi_5 p_2 A_H - \pi_6 p_{c2} A_C + \pi_7 p_2 A_H + \pi_7 p_{c2} A_C = 0$$

$$\Rightarrow u_3^* = \frac{(\pi_5 - \pi_7) p_2 A_H + (\pi_6 - \pi_7) p_{c2} A_C}{c_3} \tag{25}$$

$$\frac{\partial \mathcal{H}}{\partial u_4^*} = c_2 u_4^* - \pi_1 \lambda_T S - \pi_1 \lambda_H S + \pi_2 \lambda_T S + \pi_4 \lambda_H S + \pi_6 \lambda_H A_T + \pi_6 \lambda_T A_H - \tau \phi B = 0$$

$$\Rightarrow u_4^* = \frac{(\pi_1 - \pi_2)\lambda_T S + (\pi_1 - \pi_4)\lambda_H S - \pi_6 \lambda_H A_T - \pi_6 \lambda_T A_H + \tau \phi B}{c_4} \tag{26}$$

The mathematical formulation follows the standard process of constructing a system of ordinary differential equations (ODEs) to describe the dynamics of disease transmission. Pontryagin's Maximum Principle is applied to derive the optimal control conditions, and numerical simulations are used to evaluate the model's performance under various control scenarios.

Necessary conditions for optimal control and control profiles

The Pontryagin's maximum principle was applied to derive the necessary conditions for optimal control and the control profiles that minimise the infection levels and intervention cost are determined in section 3.5

Optimal control Analysis

A simulation of the four main plots were considered, using the same parameter values and control profiles that were used in the previous simulation.

Results

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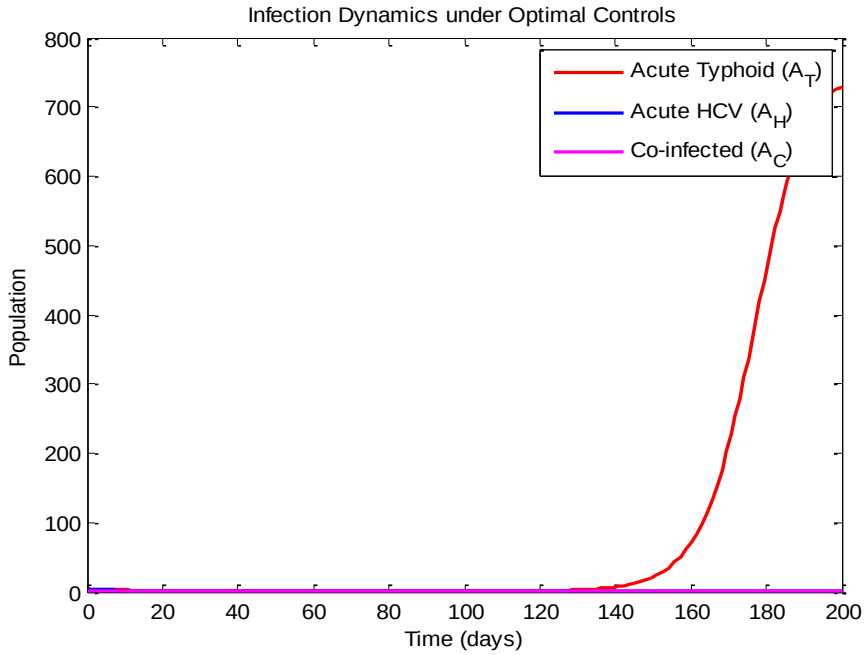

Figure 1: The behaviour of the disease dynamics under optimal control

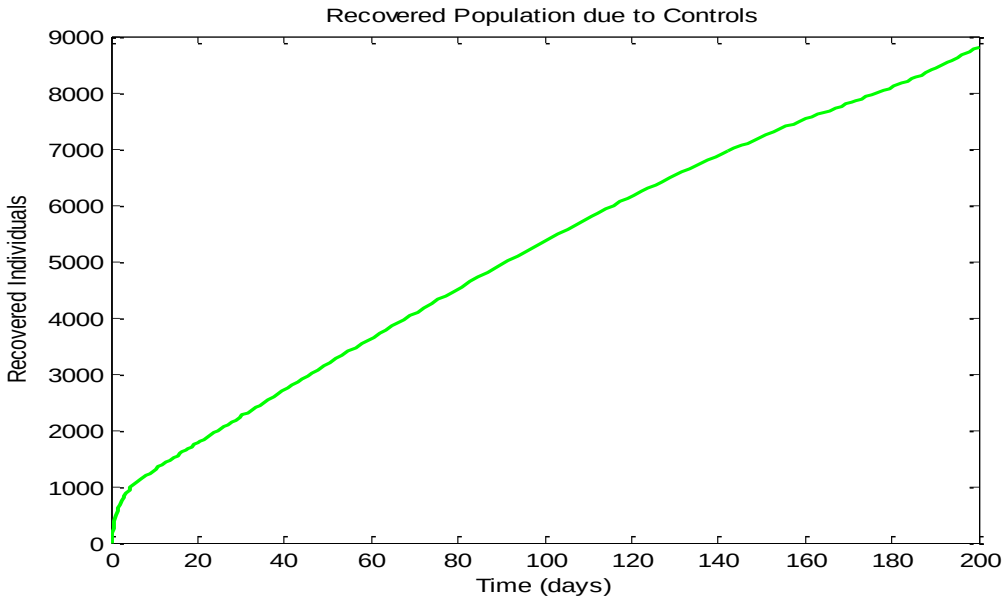

Figure 2: Recovered Population Curve due to optimal control measures

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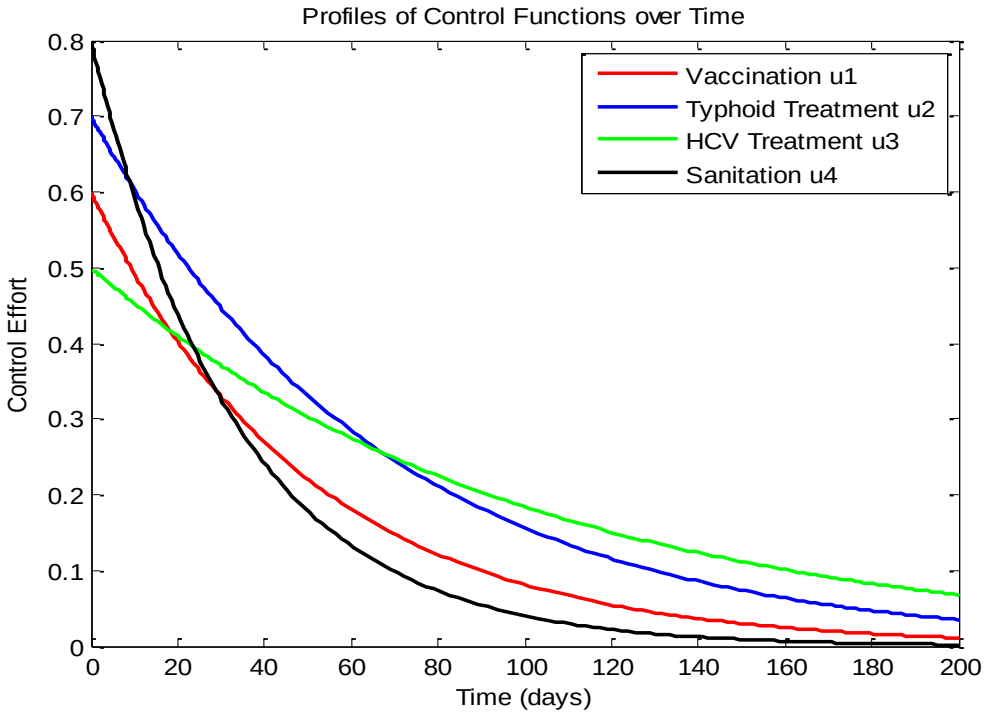

Figure 3: The effect of control measures on A_T, A_H, A_C and B.

The Parameter sensitivity analysis on the co-infection dynamic

This was explored in Figure 4.11 to 4.15. The parameters sensitivity of HCV-typhoid co-infection model were using four plots. Each plot shows how changing a key parameter (by ±30%) affects the total infected populations $A_T + A_H + A_C$, which gives the overall infection burden in the community.

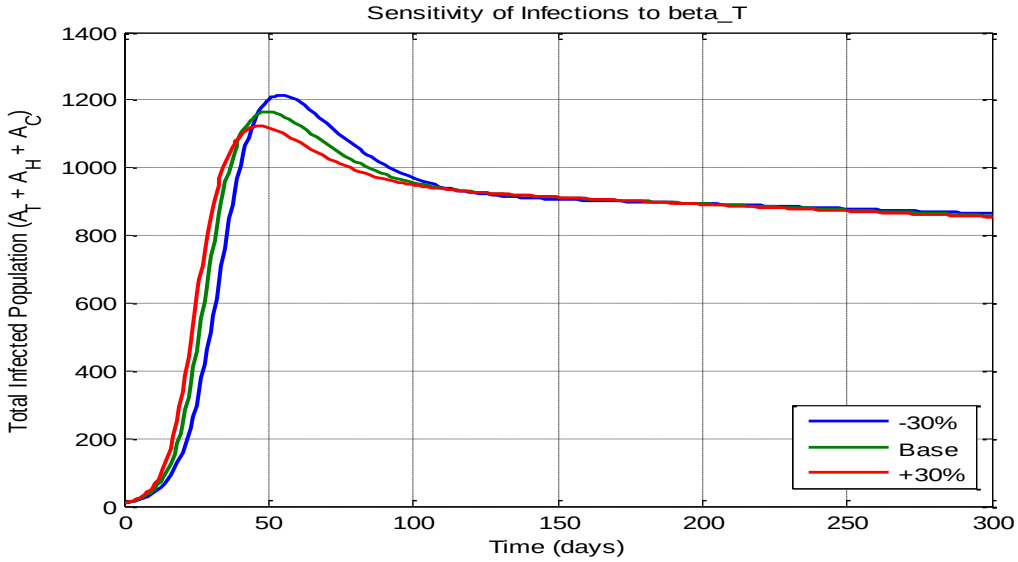

Figure 4: Sensitivity to β_T (Typhoid transmission rate), parameter varied $\beta_T = 0.5 \times (0.7,1.0,1.3)$

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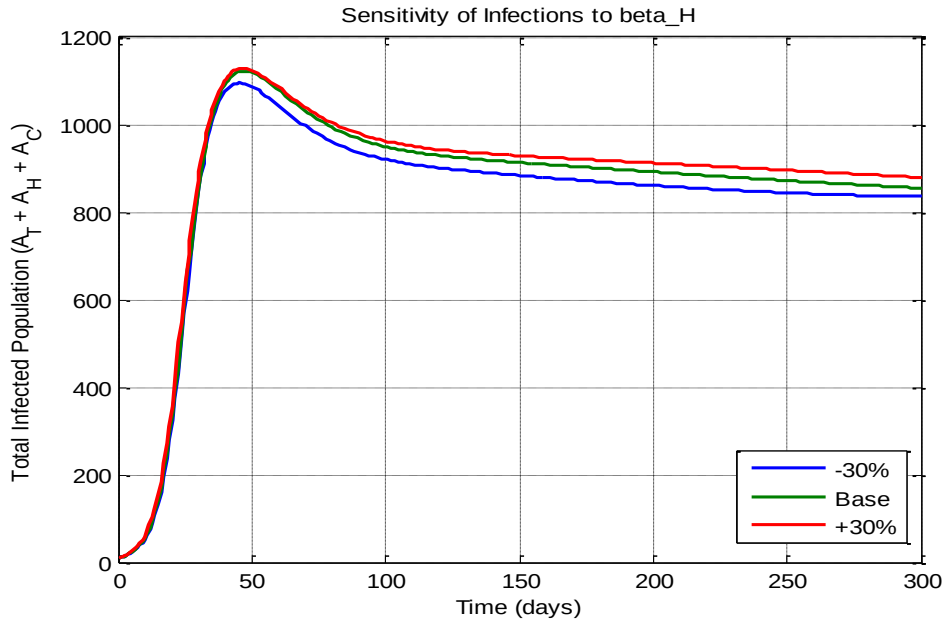

Figure 5: Sensitivity to β_H (HCV transmission rate), parameter varied $\beta_H = 0.6 \times (0.7,1.0,1.3)$

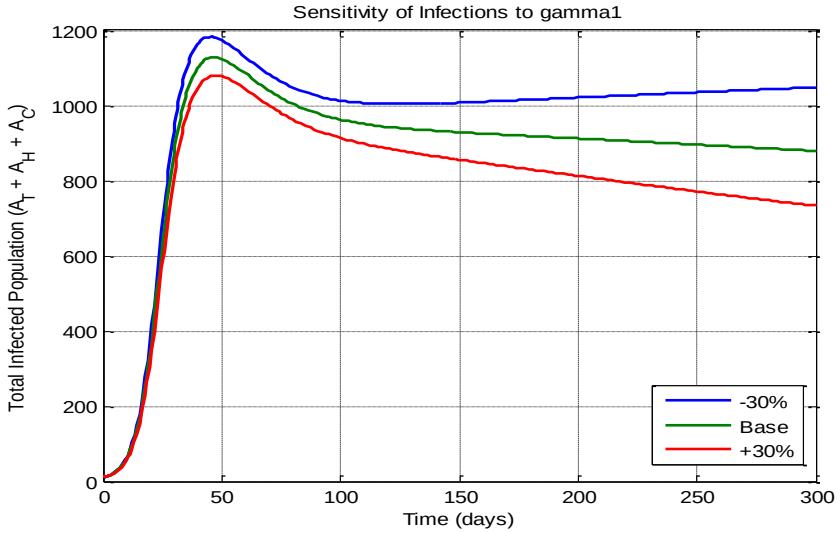

Figure 6: Sensitivity to γ_1 (Typhoid recovery rate), parameter varied $\gamma_1 = 0.05 \times (0.7,1.0,1.3)$

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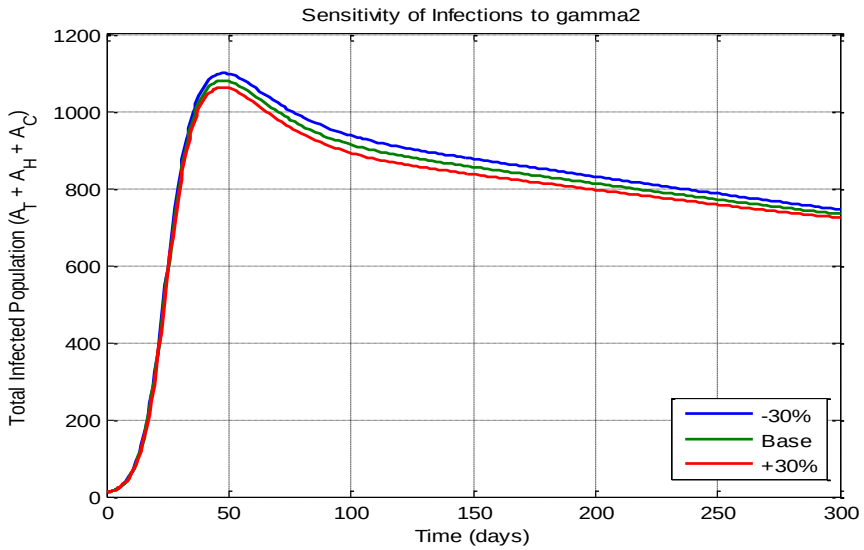

Figure 7: Sensitivity to γ_2 (HCV recovery rate), parameter varied $\gamma_2 = 0.04 \times (0.7,1.0,1.3)$

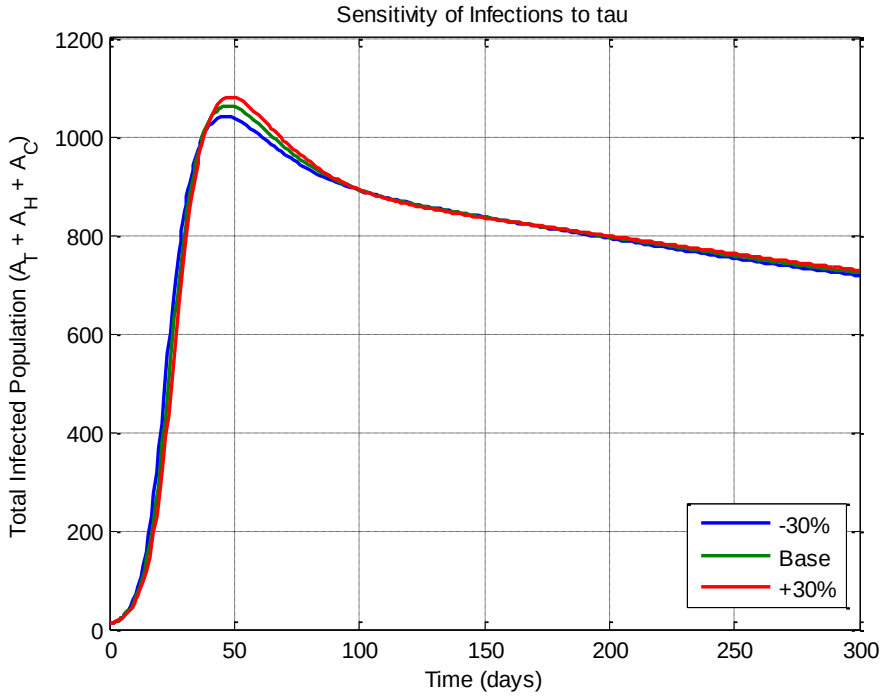

Figure 8: Sensitivity to τ (Environmental clearance rate), parameter varied $\tau = 0.5 \times (0.7,1.0,1.3)$

The effect of basic reproduction number on the model

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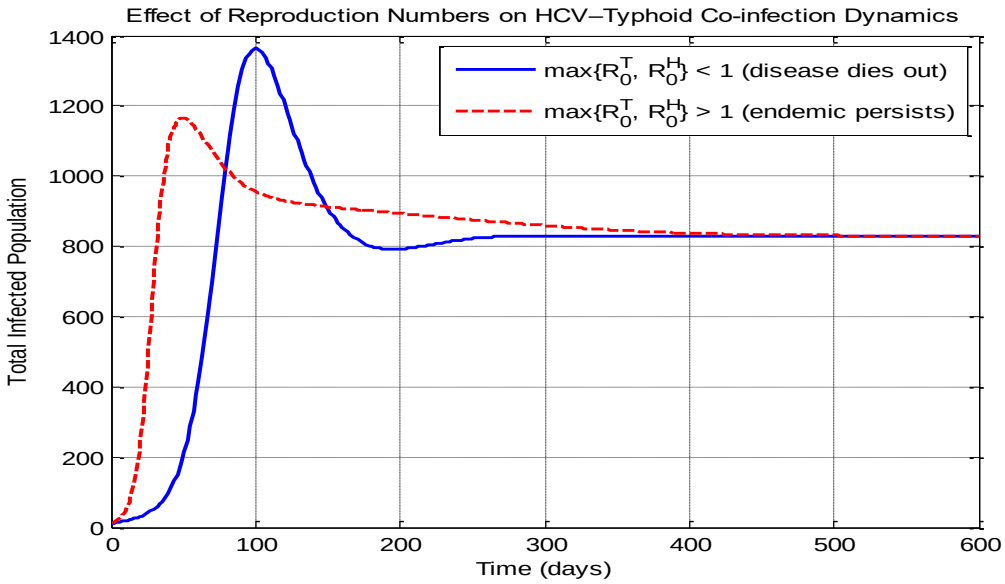

Figure 9: The plot shows both curves on the same axes where blue line indicate infection fades (below threshold) and red dashed line represent infection persists (above threshold)

In Figure 9, when transmission rates (β_T and β_H) are small, the effective reproduction numbers are less than unity, meaning each infectious individual cause fewer than one new case on average. The epidemic cannot sustain itself and quickly vanishes. However, when the transmission coefficients are increased, R_0^T and R_0^H exceed 1, and the infection persists indefinitely at endemic equilibrium — the red dashed curve stabilizes at a nonzero level, confirming the theoretical threshold property of infectious disease dynamics.

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Comparison of the Dynamics on Infections

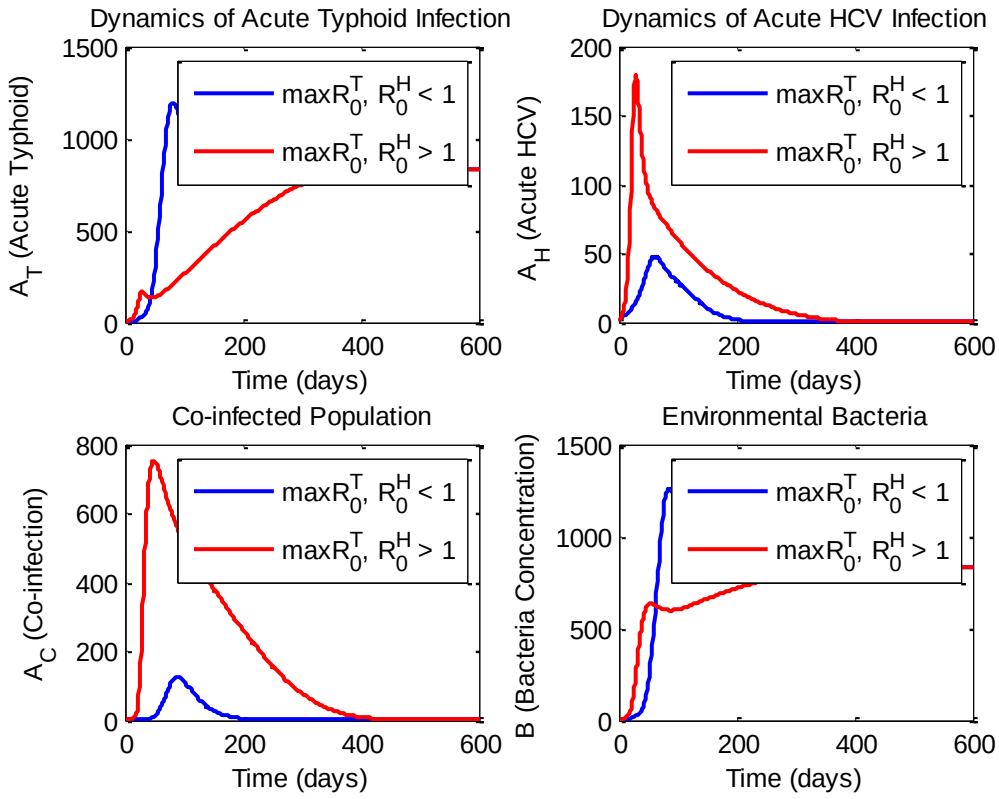

Figure 10: The plots compare R_0^T, R_0^H scenarios on acute typhoid, acute HCV, co-infection and bacterial with low $(\beta_T = \beta_H = 0.2)$ and high $(\beta_T = 0.8, \beta_H = 0.9)$ transimissions

Figure 10 displaced two scenarios where $\max\{R_0^T, R_0^T\} < 1$ and $\max\{R_0^T, R_0^T\} > 1$ on varying the values of transmissions of the two diseases on A_T, A_H, A_C and B respectively. In case 1, the acute and chronic infection curves (A_T, A_H, A_C) rise slightly and then decay to zero over time. This shows that the infection cannot sustain itself new infections decline because the effective contact rates $(\beta_T, \beta_H,)$ are low relative to recovery and natural death. The bacterial concentration (B) also decays, indicating environmental recovery. The system moves toward a disease-free equilibrium (DFE)**.** In the other case, $\max\{R_0^T, R_0^T\} > 1$, the infection populations rise sharply and then stabilize at positive endemic levels (never return to zero). This reflects a self-sustaining epidemic each infection reproduces itself faster than recovery or death can remove it. The **co**-infected population **(A_C)** increases more gradually but stabilizes at a significant level due to mutual reinforcement of both diseases. The environmental bacteria (B) also stabilizes at a high level, feeding back into Typhoid transmission. Thus, the simulation shows the threshold phenomenon the transition from eradication to persistence.

Discussion

The results of the simulations indicated that the combination of the control strategies for both HCV and the environmental sanitation, when used along with treatment for both diseases, and the typhoid vaccination, led to a considerable reduction in both HCV and Typhoid co-infection. Several findings were reached from the simulations in the following areas:

1. Decreasing Infection Rates: Using the treatment in combination with the rest of the interventions led to a more effective decrease in the infection rate.

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2. Recovery Improvement: Recovery of co-infected individuals, among whom the sanitation and treatment extended, was at an elevated level.

3. Environmental Control: Sanitation treatment effectively shrank the Typhoid bacterial reservoir, and as a result, new typhoid infections were lower.

The findings from the optimal control model underscore the importance of integrated disease management. Typhoid fever and HCV co-infection are diseases that deserve control, at both the upper levels and operative levels. Integrating HCV control is essential. Typhoid disease control simulating healthcare systems are more efficient when used at the same operational and control levels. Using sanitation aids to lift the HCV integration load and endurance along with co-infection disease control at the lowest operational level, is at the same time removing the long-term disease. Optimized control schemes signify when to execute long-term interventions for the sake of efficiency, and when to implement both for the sake of balance in health concern.

Conclusion

This study demonstrates the importance of utilizing integrated optimal control methods in the management of Typhoid fever and HCV co-infection. The model also captures the synergistic impact of several combined interventions such as Typhoid vaccine delivery, HCV screening, treatment of both diseases, and environmental sanitation, among others. Evidence points state that combined interventions impact the disease burden more significantly than independent control approaches.

Recommendations

1. Public Health Authorities should develop integrated HCV and Typhoid screening and treatment programs, particularly in areas with poor sanitation and high risk of coinfection.
2. Sanitation should be a central component of Typhoid and HCV control programs, with a focus on improving water quality and waste management.
3. HCV treatment access should be expanded, particularly in regions with high coinfection rates and limited healthcare access.

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