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"Mathematical Optimization of Cholera Vaccination: Applying Pontryagin's Maximum Principle to SIR-B Modeling in South Sudan's 2006-2007"

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Abstract

This study aims to develop optimal vaccination strategies to control cholera outbreaks in resource-limited settings such as South Sudan, where conventional models struggle to balance epidemiologic efficacy and economic feasibility. By applying optimal control techniques (Pontryagin's maximum principle) to the extended SIR-B model and using advanced numerical algorithms to analyze the 2006-2007 outbreak data, the study aims to achieve three main objectives: First, to design effective vaccination strategies capable of significantly reducing the number of infected individuals; second, to minimize operational costs while maintaining efficiency; and third, to optimize the allocation of limited resources. The results showed that this methodology is capable of radically modifying epidemiological behavior, as the numerical simulation led to a significant reduction in prevalence rates and an improvement in public health indicators, confirming the applicability of this model as an effective tool to support health decisions in the face of epidemics, with the possibility of generalizing it to other diseases in the future.

Keywords*: South Sudan, Cholera model, Pontryagin's Maximum Principle, Basic reproduction.*

المستخلص :

تهدف هذه الدراسة إلى تطوير استراتيجيات تطعيم مثلى لمكافحة تفشي الكوليرا في المناطق محدودة الموارد مثل جنوب السودان، حيث تواجه النماذج التقليدية صعوبات في تحقيق التوازن بين الفعالية الوبائية والجدوى الاقتصادية. من خلال تطبيق تقنيات التحكم الأمثل (مبدأ بونترياغين الأقصى) على نموذجSIR-B المعزز، واستخدام خوارزميات عددية متقدمة لتحليل بيانات تفشي 2006-2007، تسعى الدراسة إلى تحقيق ثلاثة أهداف رئيسية: أولاً، تصميم استراتيجيات تطعيم فعالة قادرة على خفض عدد المصابين بشكل ملحوظ؛ ثانياً، تقليل التكاليف التشغيلية مع الحفاظ على الكفاءة؛ ثالثا ، تحسين توزيع الموارد المحدودة. أظهرت النتائج أن هذه المنهجية قادرة على تعديل السلوك الوبائي بشكل جذري، حيث أدت المحاكاة العددية إلى انخفاض ملموس في معدلات الانتشار وتحسين مؤشرات الصحة العامة، مما يؤكد إمكانية تطبيق هذا النموذج كأداة فعالة لدعم القرارات الصحية في مواجهة الأوبئة، مع إمكانية تعميمه على أمراض أخرى في المستقبل.

كلمات مفتاحية: *جنوب السودان ، نموذج الكوليرا ، مبداء بونترجين ، الرقم الأساسي للإنتاج*

Introduction

Cholera is a disease caused by the bacterium Vibrio cholera, which primarily resides in two reservoirs: humans and aquatic environments, particularly saltwater habitats like oysters. The disease is transmitted directly person-to-person contact or environmental exposure (Onyuma et al., 2017; Sciarra et al., 2018; Fitria, 2019; Shuai et al., 2011; Hartley et al., 2005; Wang et al., 2011; Ujjiga et al., 2015; Bayleyegn et al., 2009; He et al., 2018; Kwasi et al., 2020; Wang, 2017). Once ingested, Vibrio cholerae bacteria penetrate the acidic barrier of the stomach and colonize the intestines, where they release enterotoxins. These toxins result in symptoms such as severe fluid and electrolyte loss, nausea, muscle cramps, vomiting, and leg cramps (Wang, 2017). If left untreated, dehydration and circulatory failure can lead to death within 12 to 24 hours (Bayleyegn et al., 2009; Misra et al., 2012; Sciarra et al., 2018; Ogunmiloro et al., 2019). The incubation period for cholera varies from a few hours to five days (Misra et al., 2012). Effective treatment primarily involves oral rehydration solutions, composed of water, salt, and sugar, which have been instrumental in saving millions of lives. While antibiotics are also used, their efficacy is increasingly challenged by the rise of antimicrobial resistance (Yang et al., 2019). Despite being a preventable disease identified over two centuries ago, cholera continues to present significant public health challenges (Fitria, 2019; Misra et al., 2012; Onyuma et al., 2017). Cholera remains most prevalent in developing regions, including Africa, parts of Asia, and South and Central America, where inadequate sanitation systems and limited access to clean water are widespread (Wang et al., 2011). Several significant cholera outbreaks occurred between 2007 and 2018 in countries like Angola, Haiti, Zimbabwe, Yemen, India (2007), Congo (2008), Iraq (2008), Zimbabwe (20082009), Vietnam (2009), Nigeria (2010), Kenya (2010), Haiti (2010), Cameroon (20102011), and Yemen (20162018) (Lemos et al., 2018; Shuai et al., 2011; Bayleyegn et al., 2009; Al et al., 2013; Tian et al., 2010; Hartley et al., 2005; He et al., 2018; Yang et al., 2019; Wang et al., 2011; Muhseen et al., 2016). In 2012, 94,553 cholera cases and 1,834 deaths were reported across 25 African nations. For instance, Kenya recorded 14,878 cases and 234 deaths between December 2014 and May 2016. According to WHO data in 2018, cholera affected an estimated 1.3 to 4 million people globally, resulting in 21,000 to 143,000 deaths annually (Ogunmiloro et al., 2019; Onyuma et al., 2017). In resource-limited regions such as South Sudan, traditional strategies for controlling cholera outbreaks face significant challenges in striking an effective balance between epidemiological efficiency and economic viability. Current epidemiological models are inadequate to represent this balance. Therefore, this study aims to explore the application of optimal control techniques to design effective vaccination strategies within the SIR-B model, focusing on the 2006-2007 cholera outbreak in South Sudan, with the aim of minimizing the number of infections and associated costs. The research problem can be summarized in the following questions

How can optimal control techniques be used to develop effective vaccination strategies to control cholera outbreaks?

What are the most effective vaccination strategies to reduce the number of infections and minimize costs?

What is the impact of optimal control strategies on the behavior and spread of the cholera epidemic in South Sudan during 2006-2007?

This study attempts to apply optimal control techniques based on Pontryagin's principle to design effective vaccination strategies within the SIR-B model, focusing on the case of a cholera outbreak in South Sudan in 2006-2007. The work aims to achieve a significant reduction in the number of infected people while minimizing operational costs and ensuring optimal use of available resources.

Theoretically, the study contributes to the development of an integrated mathematical framework linking disease dynamics and control mechanisms, while in practice it provides an effective tool for public health decision makers to predict the course of an outbreak, determine the best timing of intervention, and optimize resource allocation. The study also opens prospects for applying the presented methodology to other epidemic diseases, contributing to the development of more efficient systems to support management decisions in the face of epidemics.

The structure of this paper is organized as follows: Section 2 covers the materials and methods, with Subsection 2.1 focusing on the development of the mathematical model and Subsection 2.2 providing a detailed analysis of the epidemic model. Subsections 2.3 and 2.4 delve into the optimal control analysis, while Subsection 2.5 presents the numerical simulations. Section 3 highlights the results and their interpretations, and finally, Section 4 concludes the paper with a summary of the findings and key takeaways.

Materials and Method

Model Formulation

Cholera transmission occurs through multiple pathways. Direct human-to-human transmission is represented mathematically by the mass action term αSI, where α is the constant infection rate. Alternatively, indirect transmission from the environment to humans is modeled using a Holling type-III functional response, expressed as $\frac{\eta S B_2}{k+B_2}$, where η represents the constant infection rate. To mitigate the spread of infection, a control function, u(t), is introduced as a vaccination strategy. This approach aims to reduce the infection rate while simultaneously increasing the number of both susceptible and recovered individuals. Importantly, 2 this strategy seeks to achieve these outcomes while maintaining low vaccination costs. Inspired by the work of Magdoleen and Eihab (Bakheet et al., 2023), the deterministic cholera model has been formulated as a system of ordinary differential equations, providing a robust framework for analyzing the dynamics of the disease and the effectiveness of control measures.

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \left(\eta \frac{B^2 S}{k+B^2}\right) - \alpha I S + \rho R - \mu S - u(t) S \\
\frac{dI}{dt} &= \left(\eta \frac{B^2 S}{k+B^2}\right) + \alpha I S - (\gamma + \mu + d) I \\
\frac{dR}{dt} &= \gamma I - (\mu + \rho) R + u(t) S \\
\frac{dB}{dt} &= \zeta_1 I - \zeta B
\end{aligned} \tag{2.1}$$

where the condition

$$S(0) > 0, \quad I(0) \geq 0, \quad R(0) \geq 0, \quad B(0) \geq 0 \tag{2.2}$$

are satisfied, and control u is a functions of time t when t ∈ [0, T] T > 0, it is assumed to be bounded with

$$0 \leq u \leq u_{max}, \quad u \in [0, 1] \tag{2.3}$$

The total human population N is divided into three compartments depending on the epidemiological status of individuals. These compartments include: Susceptible S(t) symptomatically infected, I(t) and Recovered R(t). The concentration of the vibrios in the environment (that is contaminated water) is denoted by B(t). Furthermore, the susceptible population increases due to the incoming of immigrants and recovered individuals at the rates Λ and $\gamma > 0$ respectively . Any recovered individual can lose the immunity after some while and the recovered individuals become susceptible again at rate $\rho \geq 0$. The parameter $d \geq 0$ is the death rate associated with the disease. Each infected individual has contribution to the bacteria concentration at rate ξ . The natural decay rate of V. cholera is $\delta > 0$ whereas $k > 0$ is the concentration of vibrios in contaminated water in the environment (concentration of V. cholera in water that yields 50%. It is assumed that natural immunity and immunity acquired from vaccination are diminished at the same rate ρ(Miller et al., 2009).

Mathematical analysis of the epidemic model

In particular, when no control measures are applied, the system described above (2.1) simplifies to the original model proposed by Magdoleen and Eihab (Bakheet, 2023). Based on their work, the following results are derived:

Proposition 2.1 The basic reproduction number of original model is given

$$R_0 = \rho\left(F_0 V_0^{-1}\right) = \frac{\alpha\,\Lambda}{\mu\,(\gamma + \mu + d)} \tag{2.4}$$

TheR_0describes how human transmit cholera to other human at the rate (α). The stability of the equilibrium points is investigated using the basic reproduction number

Theorem 1 The disease-free equilibrium (DFE) of the model (2.1) $E_0 = (\frac{\Lambda}{\mu}, 0, 0, 0)$ is locally and globally asymptotically stable if$R_0 < 1$ and unstable if $R_0 > 1$.

Theorem 2 The endemic equilibrium of the model (2.1) $E_1 = (S^*, I^*, R^*, B^*)$ is locally and globally asymptotically stable if $R_0 > 1$.

Optimal control analysis

In this section, aims to minimize the objective functional, which is designed to reduce the number of infected individuals, increase the number of recovered individuals, and lower the costs of implementing control measures. This approach is based on the methods described by (Lenhart and Workman (2007)) and (Laarabi et al. (2015)). The objective functional to be optimized is

$$J\left(u_1^*\right) = \int_{t_0}^{T} \left[AI + \frac{Cu^2}{2}\right] dt \tag{2.5}$$

We seek to find our optimal control condition, u^* such that

$$J\left(u^*\right) = \min\left\{J(u) : u \in [0,1]\right\} \tag{2.6}$$

Where the coefficients ($A > 0$) represent the cost associated with minimizing infections, and ($C > 0$) represents the absolute cost generated by implementing the control measures (such as the cost of a vaccination program at time (t)) over a finite time (T) (the duration of the control campaign). The term (A I(t)) represents the cost of infection. The squared expression of the control is included to account for nonlinear costs, which are modeled as a convex function and are likely to emerge at high intervention levels (Miller, 2009).

Existence and Optimality System

first establish the following theorem on the existence of optimal control:

Theorem 3 There exist an optimal u^* with corresponding states $x^* = (S^*, I^*, R^*, B^*)$ that minimizes the objective functional defined by (2.5)

$$J(u^*) = \min_{u \in U} J(u)$$

where the control set U = [0, 1] ,u∈ U,x = (S, I, R, B) and f(t, x, u) the right-hand side of state system (2.1), given by

$$f(t,x,u) = \begin{pmatrix} \Lambda - \left(\eta \frac{B^2 S}{k+B^2}\right) - \alpha IS + \rho R - \mu S - u(t)S \\ \left(\eta \frac{B^2 S}{k+B^2}\right) + \alpha IS - (\gamma + \mu + d)I \\ \gamma I - (\mu + \rho)R + u(t)S \\ \zeta_1 I - \zeta B \end{pmatrix}$$

To prove the existence of an optimal control, we rely on the results presented by Fleming and Rishel (2012).

1 Convexity and closure of the control set U.

2 Boundedness of the state system by a linear function in the state and control variables.

3 Convexity of the integrand of the objective functional with respect to the control.

4 There exist constants c1, c2 > 0 and c3 > 1 such that the Lagrangian is bounded below by $c_1 (|u|^2)^{\frac{c_3}{2}} - c_2$

After demonstrating the existence of optimal controls (Fleming and Rishel, 2012), to derive the necessary conditions for this optimal control, we do not need to compute the integral in the objective functional (2.5) but rather use the Hamiltonian. The primary tool for studying optimality in system (2.1) is the Pontryagin Maximum Principle (Lenhart and Workman, 2007). Pontryagins maximum principle is applied to the control problem using the Hamiltonian (Lenhart and Workman, 2007; Neilan and Lenhart, 2010; Chachuat, 2007). Pontryagins Maximum Principle introduces adjoint functions that allow us to link the state system of differential equations to the objective functional. The optimal solution can be derived through the Lagrangian and Hamiltonian for the control problem (2.5). The Lagrangian is defined as:

$$L\left(t, x(t, u(t))\right) = AI + \frac{Cu^2}{2}$$

We have to find the minimal value of the Lagrangian for this we define the Hamiltonian H for the control problem as:

$$H\left(t, \vec{x}(t), \vec{u}(t), \vec{\lambda}(t)\right) = f\left(t, \vec{x}(t), \vec{u}(t)\right) + \sum_{i=1}^{n} \lambda_i(t) g_i\left(t, \vec{x}(t), \vec{u}(t)\right)$$

$$\begin{aligned} H(x, U, \lambda) = & \left(AI + \frac{Cu^2}{2}\right) \\ & + \lambda_S(t)\left(\Lambda - \eta \frac{B^2 S}{k+B^2} - \alpha IS + \rho R - \mu S - u(t)S\right) \\ & + \lambda_I(t)\left(\eta \frac{B^2 S}{k+B^2} + \alpha IS - (\gamma + \mu + d)I\right) \\ & + \lambda_R(t)\left(\gamma I - (\mu + \rho)R + u(t)S\right) \\ & + \lambda_B(t)\left(\zeta_1 I - \zeta B\right) \end{aligned}$$

Where $\lambda(t) = \{\lambda_S(t), \lambda_I(t), \lambda_R(t), \lambda_B(t)\}$ is the vector of adjoint variables,which determine the adjoint system, and can be solved by the following system:

$$\begin{aligned}
\frac{d\lambda_S(t)}{dt} &= -\frac{\partial H}{\partial S} = -\lambda_S(t)\left(-\left(\eta\frac{B^2}{k+B^2}\right) - \alpha I - \mu - u(t)\right) - \lambda_I(t)\left(\left(\eta\frac{B^2}{k+B^2}\right) + \alpha I\right) \\
&\quad -\lambda_R(t)u(t) \\
\frac{d\lambda_I(t)}{dt} &= -\frac{\partial H}{\partial I} = -A - \lambda_S(t)(-\alpha S) - \lambda_I(t)(\alpha S - (\gamma + \mu + d)) - \lambda_R(t)(\gamma) - \lambda_B(t)(\zeta_1) \\
\frac{d\lambda_R(t)}{dt} &= -\frac{\partial H}{\partial R} = -\lambda_S(t)\rho + \lambda_R(t)(\rho + \mu) \\
\frac{d\lambda_B(t)}{dt} &= -\frac{\partial H}{\partial B} = -\lambda_S(t)\left(-\frac{2\eta kBS}{(k+B^2)^2}\right) - \lambda_I(t)\left(\left(\frac{2\eta kBS}{(k+B^2)^2}\right)\right) + \lambda_B(t)\zeta
\end{aligned} \tag{2.7}$$

hence we obtain system with transversality condition $\lambda_S(t) = \lambda_I(t) = \lambda_R(t) = \lambda_B(t) = 0$ Furthermore, there is optimal control u^* which minimizes J(u) over the region $0 \leq u \leq 1$ characterized by

$$u^* = \frac{(\lambda_S(t) - \lambda_R(t))\,S}{C}$$

In characterizing control, we consider three cases concerning the control bounds.We show this in detail for the characterization of u^* Thus using the bounds of the control its optimal control u(t) is given by

$$u^* = \begin{cases} 0 & \text{if } \frac{(\lambda_S(t)-\lambda_R(t))S}{C} \leq 0 \\ \frac{(\lambda_S(t)-\lambda_R(t))S}{C} & \text{if } 0 < \frac{(\lambda_S(t)-\lambda_R(t))S}{C} < 1 \\ 1 & \text{if } \frac{(\lambda_S(t)-\lambda_R(t))S}{C} \geq 0 \end{cases}$$

Control u(t) can be written in compact form as

$$u^* = \min\{\max\{0, u\}, 1\}, \quad u^* = \min\left\{\max\left\{0, \frac{(\lambda_S(t) - \lambda_R(t))\,S}{C}\right\}, 1\right\}$$

indicating that the optimal control minimize the Hamiltonian. The combination of the ODE system (2.1) and the state system (2.7) is the optimality system, which describes how the system behaves minimize J under the control applications By substituting the value of (u^*) in the control system (5.1) and adjoint system (5.7) we get the following optimality system:

$$
\begin{aligned}
\frac{dS}{dt} &= \Lambda - \left(\eta\frac{B^2S}{k+B^2}\right) - \alpha IS + \rho R - \mu S - u^*(t)S \\
\frac{dI}{dt} &= \left(\eta\frac{B^2S}{k+B^2}\right) + \alpha IS - (\gamma+\mu+d)I \\
\frac{dR}{dt} &= \gamma I - (\mu+\rho)R + u^*(t)S \\
\frac{dB}{dt} &= \zeta_1 I - \zeta B \\
\frac{d\lambda_S(t)}{dt} &= -\lambda_S(t)\left(-\left(\eta\frac{B^2}{k+B^2}\right) - \alpha I - \mu - u^*(t)\right) - \lambda_I(t)\left(\left(\eta\frac{B^2}{k+B^2}\right) + \alpha I\right) \\
&\quad - \lambda_R(t)u^*(t) \\
\frac{d\lambda_I(t)}{dt} &= -A - \lambda_S(t)(-\alpha S) - \lambda_I(t)(\alpha S - (\gamma+\mu+d)) - \lambda_R(t)(\gamma) - \lambda_B(t)(\zeta_1) \\
\frac{d\lambda_R(t)}{dt} &= -\lambda_S(t)\rho + \lambda_R(t)(\rho+\mu) \\
\frac{d\lambda_B(t)}{dt} &= -\lambda_S(t)\left(-\frac{2\eta kBS}{(k+B^2)^2}\right) - \lambda_I(t)\left(\left(\frac{2\eta kBS}{(k+B^2)^2}\right)\right) + \lambda_B(t)\zeta
\end{aligned}
\tag{2.8}
$$

with $S(0) = S_0, I(0) = I_0, R(0) = R_0, B(0) = B_0$ and

$$\lambda_S(T) = \lambda_I(T) = \lambda_R(T) = \lambda_B(T) = 0$$

We note that the optimality system (2.8) consists of the state system of differential equations(2.1) with the initial conditions (2.2), the adjoint equations (2.7) and the final time conditions together with the characterization of optimal control. We cannot solve the optimality system (2.8) directly,Thus we will solve numerically in next section.

Numerical Solution and Simulation

To address the optimal control problem presented in this study, we applied the forward-backward sweep method, utilizing a fourth-order Runge-Kutta algorithm. This method allowed for iterative updates of the control function u(t) by solving the state and adjoint equations until convergence was reached. The parameters and initial conditions used for the simulation are provided in Table 2. The numerical simulations conducted underscore the effectiveness of the optimal vaccination strategy in reducing the spread of infection. By administering vaccines at the maximum rate ($u_{max} = 1$) during the initial week, there was a significant drop in the infection rate. Following this, the vaccination rate decreased gradually over the 23-week period, corresponding to the reduced number of susceptible individuals. Our results clearly indicate that early and aggressive vaccination is crucial in controlling the spread of infection.

Result and discussion

The implementation of the optimal control strategy showed a marked reduction in the number of infected individuals and a corresponding increase in the recovered population. The numerical simulations highlight the importance of timely intervention in controlling infectious diseases. Specifically, our findings for South Sudan emphasize that initiating vaccination efforts within the first few weeks after disease detection can have a substantial impact on public health outcomes. The study further demonstrates the utility of optimal control theory in designing and evaluating public health strategies. The forward-backward sweep method proved to be an effective numerical approach for solving the optimal control problem. Future research could explore the application of this method to other infectious diseases, considering additional factors such as varying vaccine efficacy and resource constraints.

Table 1: resumes the parameters in the SIR-B model and recalls their meaning, together with method of evaluation required.

Parameters	Description	Evaluation
Λ	Constant human recruitment rate	Literature
μ	Natural human mortality rate	Literature
ρ	immune period	Literature
k	Half saturation constant	Literature
η	indirect transmission rate for humans and contaminated water	Calibration
α	direct transmission rate between S and I	Calibration
d	Disease induced death rate	Literature
γ	mean infectious period	Literature
ζ_1	Bacteria shed rate into the water supply by infected human	Literature
ζ	Decay rate of vibrio	Literature
A	Cost coefficient (Vaccine)	Literature
C	Quadratic cost coefficient (Vaccine)	Literature

Source: (Naficy, et al. (1998) ;Miller,E. (2009); Hartley, et al . (2006) ; Carr, et al. (2001);Neilan, et al. (2010) ; Codeo, C.T. (2001);Wang, et al .(2011);Rahaman, et al . (1976);Levine, (1988);Levine, M. (1981)) ; (Che, et al.(2021);Bani,S.(2012);Woodward, et al. (1971) ;Kabir, A.(2005) ;Click Here;ClickHere;Click Here)

Table 2: parameter values (week).

Parameter	Value	Units	Source
Λ	680.9052	week	Click Here
μ	0.0002055769	week	Click Here
ρ	0.006410256	weeks	(Woodward, et al. (1971) ,Kabir, A. (2005).) (Levine, et al.(1981) ,Che, et al.(2021)) (Bani, S. (2012))
k	10^6	cells/mL	(Levine,et al.(1988) ,Codeo, C.T. (2001)) (Neilan, et al. (2010))
η	2.69×10^{-8}	week	estimated
α	4.22×10^{-7}	week	estimated
d	9.1111	week	Click Here
γ	0.7143	week	(Rahaman, et l . (1976) ,Levine, (1988)) (Hartley, et al . (2006) ,Wang, et al .(2011).)
ζ_1	70	cells ml week per person	(Neilan, et al. (2010) , Codeo, C.T. (2001))
ζ	2.31	week	(Hartley, et al . (2006) , Carr, et al. (2001).)
A	6	dollars per human/week	(Naficy, et al (1998) ,Miller, E. (2009))
C	10	dollars per effort^2	(Miller, E. (2009).)

Source: (Naficy, et al. (1998) ; Miller,E. (2009); Hartley, et al . (2006) ; Carr, et al. (2001); Neilan, et al. (2010) ; Codeo, C.T. (2001); Wang, et al .(2011); Rahaman, et al . (1976); Levine, (1988);

Levine, M. (1981)) ; (Che, et al.(2021);Bani,S.(2012); Woodward, et al. (1971) ; Kabir, A.(2005) ; Click Here; Click Here; Click Here)

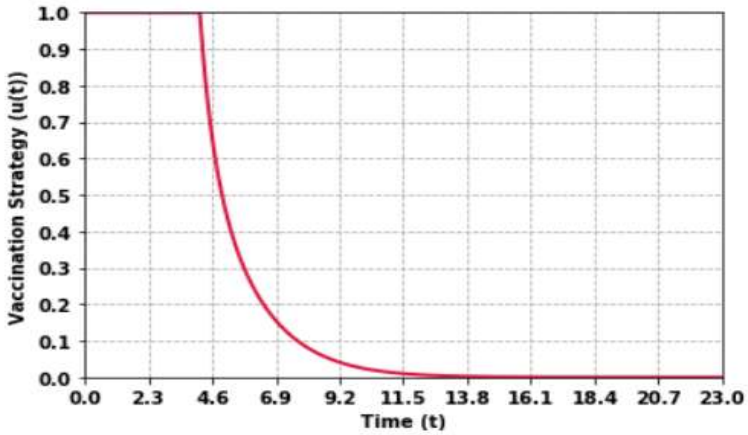

Figure 1: The plot represents the control variable u(t).optimal vaccination rate

Source: Researcher preparation Using Prameter values in Table 2 ,python program

Source: Researcher preparation Using Prameter values in Table 2 ,python program

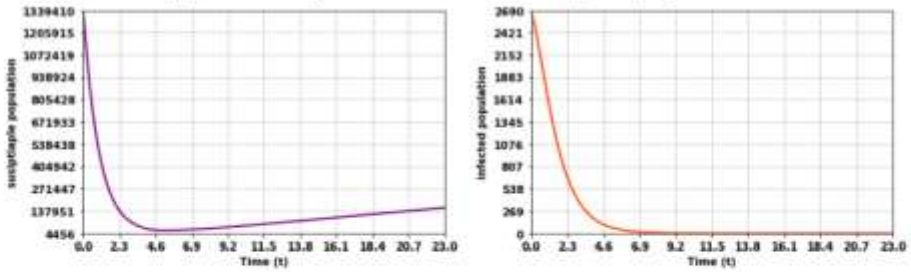

Figure 2: a) The plot shows the population of susceptible individuals with control, the vaccinatione is effective in decreasing susiptiable population. The numerical results show that the number of susceptible individuals increase after the optimal control vaccination and small number of individuals are infected from cholera.
b) The plot represents the population of infected individuals with control, the vaccinatione is effective in reducing the total infections population.
Source: Researcher preparation Using Prameter values in Table 2 ,python program

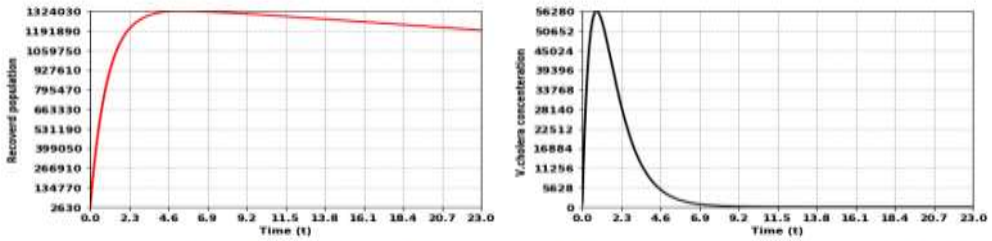

Figure 3: c) The plot shows the population of recovered individuals with control, the vaccinatione is effective in increasing the recovery people.
d)The vaccinatione is effective in reducing the concentration of V.Cholera in equitic enviroment
Source: Researcher preparation Using Prameter values in Table 2 ,python program

Conclusions

In conclusion, this study highlighted the effectiveness of an optimal control approach in reducing the spread of infection through timely vaccination. The numerical simulations demonstrate that early and aggressive vaccination can significantly reduce both the spread of infection and the total number of infected individuals. Findings underscore the critical importance of implementing vaccination strategies at the maximum rate during the initial stages of disease detection. The results from South Sudan serve as a valuable case study, emphasizing the need for swift and decisive public health interventions. Overall, this study provides valuable insights into the practical application of optimal control theory in public health. The proposed model and numerical solutions can serve as a foundation for further research and policy-making aimed at improving disease management and prevention. Future investigations could be build on this work by considering additional variables and constraints to develop more comprehensive and effective control strategies.

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