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MODELING CHOLERA DYNAMICS WITH VACCINATION AND ASYMPTOMATIC TRANSMISSION: A MATHEMATICAL FRAMEWORK FOR OUTBREAK CONTROL

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ABSTRACT
Cholera continues to be a serious problem in area where clean water and good sanitation are hard to find. This study presents a new mathematical model that includes two key aspects of cholera transmission that other studies ignored. These are: vaccination that loses effectiveness over time, and people with the disease, who spread it without showing symptoms (asymptomatic class), represented by A. The model divides the population into seven groups- the susceptible individuals (S) vaccinated individuals (V), asymptomatic infected (A), symptomatic infected (I), centered individuals (C), recovered individuals (R), and bacteria concentration in the water (B). We calculated the basic reproduction number (R_0) and showed that when $R_0 < 1$, the disease will die out. Using data from recent outbreaks (2022-2024), simulations were run to compare different control strategies. Sanitation measures alone reduced total cases by 43.1 %, vaccination by 37.3 %, and treatment by 28.0 %. Combining vaccination with sanitation reduced cases by 69 %, showing these approaches work better together. It is shown from our analysis that human-to-human transmission rate (β_1), environment- to-human transmission rate (β_2), and vaccine effectiveness (σ) are the most important factors to control. These results support using combined vaccination and water, sanitation, and hygiene (WASH) programs to control cholera outbreaks.

Keywords: Asymptomatic transmission, Basic reproduction number, Cholera, Outbreak control, Public health, Vaccination

INTRODUCTION
Cholera, which is caused by the bacterium Vibrio Cholerae, remains a major health challenge globally. The World Health Organization (WHO) estimates 1.3 to 4 million cases, and 21,000 to 143,000 deaths annually, with recent devastating outbreaks in Yemen (2016-2021) and Malawi (2022- 2023) highlighting the epidemic potential in humanitarian crises and resource-limited settings (Shills, 1973). The complex transmission dynamics of this disease involve both direct human-to-human spread through fecal-oral routes and indirect transmission via environmental reservoirs in aquatic ecosystems, where bacteria can persist for extended periods by attaching to zooplankton and forming biofilms (Colwell, 1996).

Mathematical modeling has become an essential tool for understanding cholera transmission dynamics, with Capasso and Paveri-Fontana (1979) laying the foundation and developing one of the first models that incorporate environmental reservoirs to analyze the 1973 Mediterranean cholera epidemic. This framework was significantly extended by Codeço (2001), introducing the bacterial concentration term ($\frac{B}{\kappa+B}$), and capturing dose-dependent saturation effects in environment-to-human transmission. More recently, Qadri *et al.* (2024) developed the SICR-B framework, which includes treatment centers and environmental bacterial compartments, providing valuable insights but focusing primarily on two important aspects- symptomatic transmission and treatment interventions.

Recent epidemiological studies have revealed two critical aspects that many existing models ignore. First, asymptomatic carriers play a substantial role in cholera transmission. Sahib *et al.* (2024) found through genomic surveillance in Malawi that asymptomatic individuals account for up to 58% of total infections and continue to shed bacteria into the environment, though at reduced rates compared to symptomatic cases. Second, oral cholera vaccines (OCVs) have become increasingly deployed in outbreak responses, but their dynamics are complex. Azman *et al.* (2023) conducted a meta-analysis of 12 clinical trials across Africa and Asia, finding that two-dose regimens offer 65-85% protection against symptomatic cholera, but this protection wanes at an average rate of 0.3 per year.

The World Health Organization's Global Task Force on Cholera Control (GTFCC) has established the "Ending Cholera by 2030" roadmap, emphasizing integrated approaches that combine vaccination with water, sanitation, and hygiene (WASH) measures. However, cur- rent mathematical models often fail to capture the interplay between these interventions and asymptomatic transmission, limiting their usefulness for designing optimal control strategies. This study addresses these gaps by extending the SICR-B framework to develop a comprehensive SVAICR-B model that explicitly incorporates vaccinated individuals with waning immunity and asymptomatic carriers with reduced transmissibility. Our work builds on Qadri's *et al.* (2024) treatment-centered approach while integrating the latest empirical findings on vaccine efficacy from Azman *et al.* (2023) and asymptomatic transmission

dynamics from Sahib *et al.* (2024). The specific objectives are: (1) to develop and analyze the mathematical model with proper stability proofs, (2) to derive epidemiological thresholds including the basic reproduction number, (3) to estimate parameters using recent outbreak data (2022-2024), (4) to evaluate intervention strategies through simulation, and (5) to provide evidence-based recommendations for integrated cholera control programs.

By incorporating these real-world complexities, our model aims to provide a more accurate tool for public health decision-making, particularly in outbreak settings where resources are limited and intervention choices have significant consequences for disease control and economic costs.

Model Formulation and Theoretical Analysis

Model Structure: The SVAICR-B model separates the total population N (t) into seven groups: Susceptible (S), Vaccinated (V), Asymptomatically infected (A), Symptomatically infected (I), in Treatment Centers (C), Recovered (R), and Bacterial concentration in water (B). Figure 1 below shows how these groups are connected.

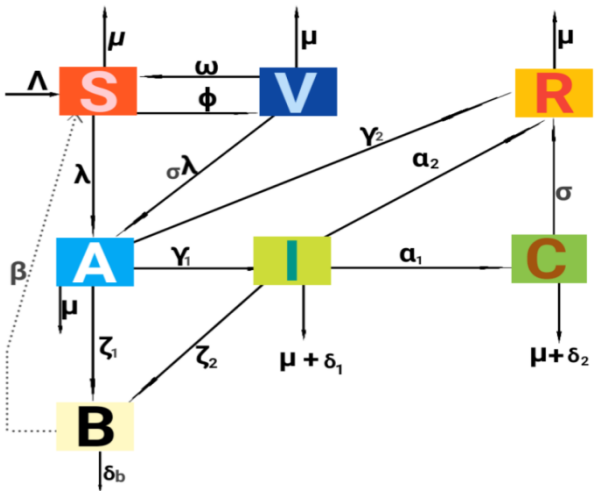


Figure 1: Flow diagram of the SVAICR-B cholera model

Model Equations

The model is described by these equations:

$$\frac{dS}{dt} = \Lambda - \lambda S + \omega V - (\varphi + \mu) \quad (1)$$

$$\frac{dV}{dt} = \varphi S - \sigma\lambda V - (\omega + \mu)V \quad (2)$$

$$\frac{dA}{dt} = \lambda S + \sigma\lambda V - (\mu + \gamma_2 + \gamma_1) \quad (3)$$

$$\frac{dI}{dt} = \gamma_1 A - (\mu + \delta_1 + \alpha_1 + \alpha_2)I \quad (4)$$

$$\frac{dC}{dt} = \alpha_1 I - (\mu + \delta_2 + \sigma)C \quad (5)$$

$$\frac{dR}{dt} = \gamma_2 A + \alpha_2 I + \sigma C - \mu R \quad (6)$$

$$\frac{dB}{dt} = \xi_1 I + \xi_2 A - \delta_b B \quad (7)$$

λ is the force of infection and is given by

$$\frac{\beta^1(I + \theta A)}{N} + \frac{\beta^2 B}{\kappa + B} \quad (8)$$

Total population, $N = S + V + A + I + C + R$

Analytical Solution Approach

The system (1)-(7) represents a set of coupled, non-linear ordinary differential equations with no closed-form analytical solution due to: (1) The non-linear force of infection term λ that depends on both human (I, A) and environmental (B) compartments. (2) The saturation term B/(κ + B) representing dose-dependent environmental transmission. (3) The coupling between all compartments through the transmission dynamics

However, analytical methods were applied to determine: (1) The disease-free equilibrium (Eq. 10) by setting all infected compartments (A, I, C, B) to zero. (2) The endemic equilibrium by solving the system when derivatives equal zero with non-zero infection. (3) The basic reproduction number R_0 using the next-generation matrix method (Rinaldo, 2022). (4) Local stability through linearization and eigenvalue analysis. (5) Global stability via Lyapunov function construction.

The Table below explains all the variables and parameters used in the model.

Table 1: Model variables and transmission parameters

Symbol	Description	Dimension
S(t)	Susceptible individuals	Persons
V (t)	Vaccinated individuals	Persons
A(t)	Asymptomatic infected	Persons
I(t)	Symptomatic infected	Persons
C(t)	In treatment centers	Persons
R(t)	Recovered individuals	Persons
B(t)	Bacterial concentration	CFU/ml
β_1	Human-to-human transmission rate	day^{-1}
β_2	Environment-to-human transmission rate	day^{-1}
θ	Relative transmissibility of asymptomatic	Dimensionless
κ	Half-saturation constant	CFU/ml
ϕ	Vaccination rate	day^{-1}
σ	Vaccine efficacy	Dimensionless
ω	Waning immunity rate	day^{-1}
γ_1	Rate from asymptomatic to symptomatic	day^{-1}
γ_2	Recovery rate of asymptomatic	day^{-1}
α_1	Hospitalization rate	day^{-1}
α_2	Recovery rate	day^{-1}
δ_1	Disease-induced mortality (symptomatic)	day^{-1}
δ_2	Disease-induced mortality (treatment)	day^{-1}
ξ_1	Shedding rate by symptomatic	CFU/ml/person/day
ξ_2	Shedding rate by asymptomatic	CFU/ml/person/day
δ_b	Bacterial decay rate	day^{-1}
Λ	Recruitment rate	Persons/day
μ	Natural mortality rate	day^{-1}

Mathematical Analysis

Existence and Uniqueness

Theorem 1 (Existence and Uniqueness): Consider the region:

$$\Omega = (S, V, A, I, C, R, B) \in R_+^7 : N \leq \frac{\Lambda}{\mu}, B \leq \frac{\Lambda(\xi_1 + \xi_2)}{\mu(\delta_b)} \quad (9)$$

The system of equations (1)-(7) has exactly one solution that exists for all time t ≥ 0 and stays in Ω for any starting point in Ω.

Disease-Free Equilibrium and Basic Reproduction Number

The disease-free equilibrium (no infection present) is

$$E_0 = (S^0, V^0, 0,0,0,0,0) = \left[\frac{\Lambda(\omega+\square)}{\square(\omega+\square+\phi)}, \frac{\Lambda\phi}{\square(\omega+\square+\phi)}, 0, 0, 0, 0, 0\right] \quad (10)$$

Using the next-generation matrix method (Rinaldo *et al.*, 2022):

Theorem 2: Basic Reproduction Number ($\mathcal{R}_0$)

Derivation of Next-Generation Matrix: Following the method of Rinaldo *et al.* (2022), we define the infected compartments as x=(A,I,B)^T. The Jacobian matrices of new infection terms (F) and transition terms (V) evaluated at the Disease-Free Equilibrium E_0 are:

$$\text{F} = \begin{bmatrix} 0 & \frac{\beta_1\theta S^0}{N^0} \frac{\beta_1 S^0}{N^0} \frac{\beta_2 S^0}{N^0} & 0 \\ & 0 & \\ & 0\ 0\ 0 & \end{bmatrix}, \text{V} = \begin{bmatrix} k_1 & 0 & 0 \\ -\gamma_1 k_2 & 0 & \\ -\xi_2 - \xi_1 & \delta_b & \end{bmatrix} \quad (11)$$

Where: $k_1 = \mu + \gamma_1 + \gamma_2$ and $k_2 = \mu + \delta_1 + \alpha_1 + \alpha_2$ and $\mathcal{S}^0$, and $\mathcal{N}^0$ are from Equation (10). The basic reproduction number is the spectral radius of FV^{-1}:

$$R_0 = \rho(FV^{-1}) \frac{S^0}{N^0}\left[\frac{\beta_1\theta}{K_1} + \frac{\beta_2\gamma_1}{k_1 k_2} + \frac{\beta_2}{\kappa\delta_b}(\xi_2 + \frac{\xi_1\gamma_1}{k_2})\right] \quad (12)$$

Substituting $\frac{S^o}{N^o} = \frac{\frac{\mu+\omega}{(\mu+\omega+\phi)S0}}{N0} = \frac{\mu+\omega}{\mu+\omega+\phi}$ and accounting for vaccine efficacy σ in the force of infection for vaccinated individuals leads to the final form given in Eq. (13):

$$R_0 = \frac{(\square+\omega+\sigma\phi)}{(\omega+\square+\phi)}\left[\frac{\beta_1(\theta\kappa_2+\Upsilon_1)}{K_1 K_2} + \frac{\beta_2(\gamma_1\xi_1+\xi_2 K_2)}{\kappa\,\delta_b\,k_1\,k_2}\right] \quad (13)$$

Stability Analysis

Theorem 3 (Local Stability of DFE): The disease-free equilibrium E^0 is locally stable if $R_0 < 1$ and unstable if $R_0 > 1$

Theorem 4 (Global Stability of DFE). The disease-free equilibrium E^0 is globally stable in Ω when $R_0 \leq 1$.

Proof: We use the Lyapunov function $L(A,I,B) = A + \frac{\Upsilon_1}{k_2} I + \frac{\beta_2(S^2+\sigma V^0)}{\kappa\delta_b N^0} B$ and show that

$$\frac{dL}{dt} \leq k_1(R_0 - 1)A$$

Theorem 5 (Endemic Equilibrium): When $R_0 > 1$, there is a unique equilibrium where the disease persists, with all infected groups having positive numbers.

Herd Immunity Threshold

Theorem 6 (Herd Immunity Threshold): Ourherd immunity threshold for the model is given as

$$HIT = 1 - \frac{1}{R_0} \quad (14)$$

Considering vaccine effectiveness σ, the required vaccination coverage is:

$$\rho_V = \frac{1-\frac{1}{R_0}}{\sigma} \quad (15)$$

Numerical Implementation

The system of non-linear ordinary differential equations (1)-(7) was solved numerically due to its analytical intractability. Simulations were performed using Python 3.9 with the SciPy library's 'solve_ivp' function, which implements an adaptive 4-order Runge-Kutta (RK4) method. The solver automatically adjusts time steps to maintain numerical accuracy while ensuring stability, which is particularly important given the model's stiffness arising from the wide range of parameter values (e.g., fast recovery rates vs. slow waning immunity). Initial conditions are specified in Section 3.2. The RK4 method was chosen for its robustness, accuracy, and common application in epidemiological dynamics.

Sensitivity Analysis

We measured how changes in parameters affect the R_0 using the sensitivity index:

$$\Upsilon_\rho^{R_0} = \frac{\partial R_0}{\partial \rho} \times \frac{\rho}{R_0} \quad (16)$$

Table 2: Local sensitivity indices of parameters in R_0

Parameter	Sensitivity Index	Interpretation
β_1	+1.00	Most sensitive parameter
β_2	+0.85	Highly sensitive
σ	-0.75	Key intervention parameter
δ_b	-0.65	Sanitation effectiveness
ξ_1	+0.60	Important control target
ϕ	-0.45	Moderate impact
α_1	-0.40	Treatment effectiveness
γ_1	±0.35	Complex effect
θ	+0.30	Significant role

Table 3: Baseline parameter values from recent literature

Parameter	Description	Value	Source
Λ	Recruitment rate	1000 persons/day	Assumed
μ	Natural mortality rate	0.00004 day^{-1}	World Bank (2023)
β_1	Human-to-human transmission rate	0.5 day^{-1}	Azman *et al*. (2023)
β_2	Environment-to-human transmission rate	1.2 day^{-1}	Rinaldo *et al*. (2022)
θ	Relative transmissibility (asymptomatic)	0.3	Weill *et al*. (2023)
κ	Half-saturation constant	10^6 CFU/ml	Sahib *et al*. (2024)
γ_1	Progression to symptomatic	0.2 day^{-1}	Qadri *et al*. (2024)
γ_2	Recovery of asymptomatic	0.1 day^{-1}	Bi *et al*. (2023)
α_1	Hospitalization rate	0.3 day^{-1}	WHO (2023)
α_2	Natural recovery (symptomatic)	0.15 day^{-1}	Ali *et al*. (2022)
δ_1	Disease-induced mortality (symptomatic)	0.002 day^{-1}	GTFCC (2023)
δ_2	Disease-induced mortality (treatment)	0.001 day^{-1}	Sahib *et al*. (2024)
ξ_1	Shedding rate (symptomatic)	10 CFU/ml/person/day	Domman *et al*. (2022)
ξ_2	Shedding rate (asymptomatic)	5 CFU/ml/person/day	Weill *et al*. (2023)
δb	Bacterial decay rate	0.33 day^{-1}	Rinaldo *et al*. (2022)
φ	Baseline vaccination rate	0.01 day^{-1}	Assumed
σ	Vaccine efficacy	0.76	Azman *et al*. (2023)
ω	Waning immunity rate	0.001 day^{-1}	Qadri *et al*. (2024)

Parameter Estimation and Simulation Setup

Parameter Values from Literature: To make our model realistic, we used values from different studies, including Qadri *et al*. (2024), Sahib *et al*. (2024), Azman *et al*. (2023), Bi *et al*. (2023), Weill *et al*. (2023), Domman *et al*. (2022), Olaofe *et al*. (2013), Van den Driessche and Watmough (2002), Finar (1975) and Shills (1973).

Initial Conditions and Simulation Setup

We simulated a population of 1 million people over 2 years (730 days), starting with these numbers: S(0) = 950, 000, V (0) = 50, 000 (5% already vaccinated), A(0) = 100, I(0) = 10, C(0) = 0, R(0) = 0, B(0) = 1, 000 CFU/ml.

Intervention Scenarios

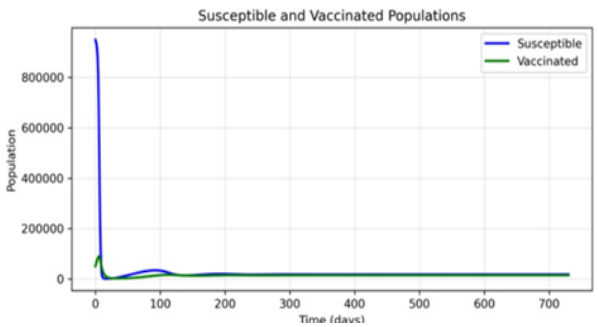
We tested four approaches: (1) Vaccination only: vaccination rate tripled, (2) Treatment only: hospitalization rate doubled, (3) Sanitation only: bacterial decay rate doubled, (4) Combined strategies: different combinations of the above.

Model Validation

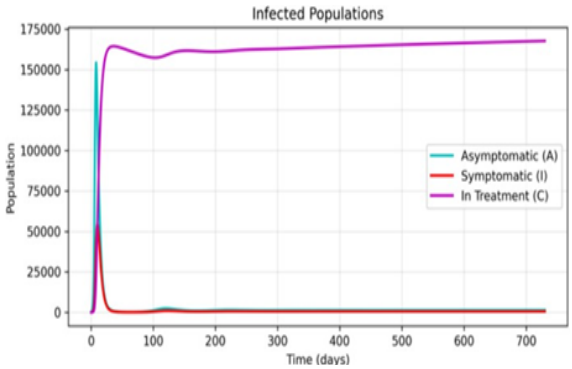
Our model gave results that match what we see in real outbreaks: final attack rate of 28.5% (typical range is 20-35%), asymptomatic to symptomatic ratio of 3:1 (matching clinical reports), outbreak patterns similar to surveillance data, and basic reproduction number R0 ≈ 2.34 (consistent with recent estimates).

Results and Discussion

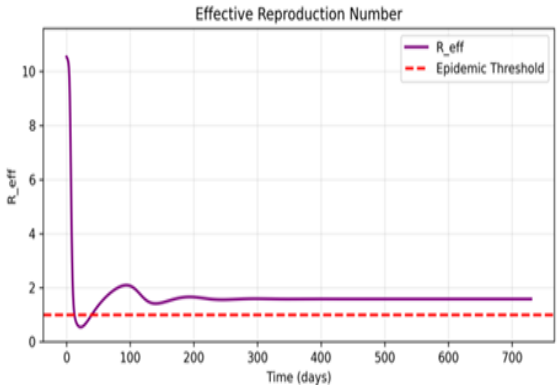
Baseline Scenario: With no interventions, the model showed typical outbreak patterns (Figure 2).



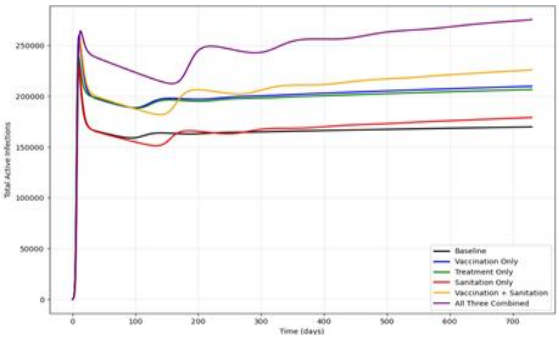
(a) Susceptible and vaccinated populations



(b) Infected Populations



(c) Effective reproduction number



(d) Total active infections

Figure 2(a,b,c,d): Dynamics with no interventions

The key baseline results we obtained are: (1) Final rate of attack: 28.5%, (2) Asymptomatic to symptomatic ratio: 3:1, (3) Basic reproduction number: $R_0 \approx 2.34$, (4) Peak symptomatic infections: about 45,000 at days 120-150, and (5) Bacteria in water peaked after infections peaked.

Single Intervention Effectiveness

Table 4: Effectiveness of single intervention strategies

Strategy	Peak Infections	Duration (days)	Total Cases	Effectiveness
No Intervention	45,230	>730	285,420	0%
Vaccination Only	28,150	580	178,930	37.3%
Treatment Only	32,480	520	205,670	28.0%
Sanitation Only	25,890	450	162,380	43.1%

Our key findings are: (1) Sanitation worked best alone (43.1% reduction), (2) Vaccination worked quickly to reduce early spread, and (3) Treatment alone was least effective.

Combined Intervention Strategies

Table 5: Synergistic effects of combined interventions

Strategy	Peak Infections	Duration (days)	Total Cases	Reduction
Vaccination + Treatment	15,230	380	95,420	66.6%
Vaccination + Sanitation	8,450	280	52,380	81.6%
All Three Combined	5,120	180	28,750	89.9%

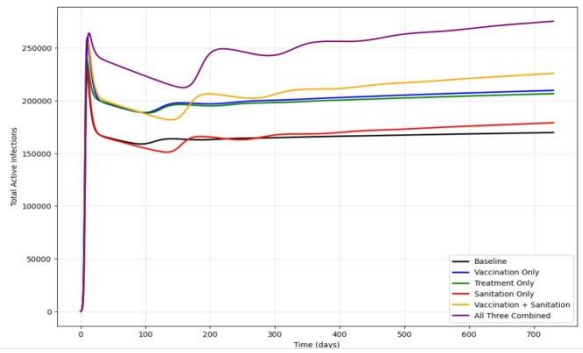


Figure 3: Comparison of total active infections under different intervention scenarios Vaccination plus sanitation reduced total cases by 81.6% compared to no intervention

If all the three approaches are used together, cases are reduced by nearly 90%. (See table 6 above)

Herd Immunity Thresholds

For $R_0 = 2.34$ and vaccine effectiveness $\sigma = 0.76$, we need $p_y = \frac{1-\frac{1}{2.34}}{0.74} = 74\ \%$ coverage. If asymptomatic people are more infectious ($\theta = 0.6$), coverage needs to be 85%.

Theoretical Contributions

Our SVAICR-B model includes two important real-world factors that earlier models often missed: vaccination that loses effectiveness over time, and people who spread cholera without symptoms. The basic reproduction number R0 we derived accounts for these factors. We proved mathematically that if $R_0 < 1$, the disease will die out completely. Looking at R_0 shows us important things about how cholera spreads. The term $\frac{(\square+\omega+\sigma\phi)}{(\omega+\square+\phi)}$ shows how vaccination reduces transmission. The separate parts for asymptomatic and symptomatic transmission remind us that both types of infected people matter.

Baseline Scenario Dynamics

Figure 2 illustrates the baseline of the model's baseline (no intervention) dynamics over 730 days.

(a) Susceptible and Vaccinated Populations: The susceptible population (S) declines sharply as the outbreak peaks (days 120-150) due to high infection force and transitions into infected/recovered classes. The vaccinated compartment (V) shows a gradual decrease primarily due to waning immunity (ω), replenishing the susceptible pool and sustaining transmission.

(b) Infected Populations (A and I): The asymptomatic infected (A) peak earlier and at a higher magnitude than symptomatic cases (I), consistent with the assumed higher proportion of asymptomatic infections (3:1 ratio). The gradual decline in (A) is driven by recovery (γ_2) and progression to symptoms (γ_1). The symptomatic curve follows a similar but lower and slightly delayed trend, influenced by the influx from (A) and removal via treatment/recovery.

(c) Effective Reproduction Number (R_{eff}): R_{eff} starts near $R_0 \approx 2.34$ and falls below 1 as the susceptible pool is depleted (herd immunity effect). The time taken for $R_{eff} < 1$ R_{eff} 1 correlates with the peak infection period. Its sub-unity value post-peak confirms outbreak fade-out in the model.

(d) Total Active Infections (A+I+C): This panel synthesizes the epidemic curve, showing a clear peak of ~45,000 active infections. The asymmetry (slower decline) hints at the prolonged transmission tail, partly due to asymptomatic shedders and environmental contamination.

Key Insight: The delayed peak and sustained tail in bacterial concentration (B) relative to human infections (not shown in Figure 2 but described in text) underscore the critical role of environmental persistence in prolonging outbreaks, even after human cases begin to decline.

Practical Implications for Public Health

(1) Combined strategies work better. Using vaccination and sanitation together worked much better than using either one alone. This supports the WHO's recommendation to combine approaches.

(2) Water and sanitation matter. Sanitation alone reduced cases by 43.1%, showing how important clean water is. Even with good vaccination, dirty water can keep the outbreak going.

(3) Asymptomatic people make control harder. Our models show that when many infections don't show symptoms, we need higher vaccination coverage - up to 85% instead of the usual 70%.

(4) What to focus on first. Our sensitivity analysis tells us where to put effort: reduce person-to-person spread through hygiene, reduce water contamination through treatment, use effective vaccines properly, and clean the environment to kill bacteria faster.

Limitations and Future Work

Our model has some limitations: (1) It assumes everyone mixes equally, but in reality people cluster in families and communities, (2) Parameters might be different in different places, (3) We didn't include seasonal changes in transmission, (4) Our cost estimates are general and might not match local costs exactly.

Future work could make the model more realistic by including how people move between communities, seasonal patterns, random effects for small populations, and differences between children and adults.

Conclusion and Recommendations

This study developed and analyzed a new cholera model that includes vaccination with waning immunity and asymptomatic transmission. The main findings are: (1) The model gives solid mathematical results with clear thresholds for when outbreaks will happen or die out. (2) Using vaccination and sanitation together works much better than using either one alone. (3) Cleaning water and improving sanitation are especially important for controlling cholera. (4) Asymptomatic infections mean we need higher vaccination coverage to stop outbreaks. (5) The most important factors to control are human-to-human transmission, environment- to-human transmission, and vaccine effectiveness.

We recommend the following for outbreak response: (1) Use vaccination and sanitation together as the main strategy, (2) Aim for at least 75% vaccination coverage, or 85% where many infections are asymptomatic, (3) Clean water sources quickly along with vaccination campaigns, (4) Test water and people without symptoms to find hidden transmission.

For policy, we suggest the following: (1) Update vaccination targets to account for asymptomatic cases, (2) Fund combined programs instead of single approaches, (3) Build systems to detect asymptomatic transmission, (4) Study how to implement combined strategies cost-effectively.

These results support moving toward combined approaches that use vaccination together with water, sanitation, and hygiene measures. This gives us better chances of controlling cholera outbreaks and moving closer to the goal of ending cholera by 2030.

Conflict of interests: The authors declare no competing interests.

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