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MODELING CHOLERA SPREAD THROUGH A ONE HEALTH LENS: THE ROLE OF FISH IN DISEASE TRANSMISSION AND CONTROL

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ABSTRACT

Cholera being a disease caused by the bacterium *Vibrio cholerae*, continues to affect millions worldwide, especially in areas with poor access to clean water and sanitation. Despite repeated efforts to control outbreaks, the disease continues to resurface. In this study, the authors build on the mathematical model developed by Albalawi *et al.* (2023), expanding it to include a key but often overlooked component: the role of fish in the transmission cycle. By applying a One Health approach, which considers the interconnected health of humans, animals, and the environment, the authors explore how fish may contribute to the spread and persistence of cholera. Our model examines how the disease moves between people, fish, and contaminated water sources. The results show that if the basic reproduction number $\Re_0$ is below one, the disease is likely to die out; if it rises above one, outbreaks can occur. This threshold was calculated using the next-generation matrix method. Through sensitivity analysis, the major factors that drive transmission, such as human-to-human contact, exposure to contaminated water, and interactions between fish and infected environments, are identified. Using simulations run in Maplesoft 2025, the various intervention strategies are tested. The results show that combining efforts, such as vaccination, hygiene, and water treatment, can significantly reduce infection rates. Our findings underscore the importance of incorporating environmental and animal-related factors into cholera control strategies. By adopting a more integrated, One Health perspective, it is easier to understand and manage the complex dynamics of this disease.

Keywords: Fish-borne transmission, One Health, Public health strategy, zoonotic transmission, Sensitivity analysis, *Vibrio cholerae*

1.0 INTRODUCTION

Infectious diseases continue to threaten global health, and cholera, an acute diarrheal illness caused by *Vibrio* cholerae, remains especially burdensome in settings marked by poverty, conflict, and inadequate sanitation. Transmitted primarily through contaminated water or food, cholera can cause rapid dehydration and death within hours if untreated (WHO, 2024b). Global estimates indicate 1.3 - 4.0 million cases and 21,000 - 143,000 deaths annually (Ali *et al.*, 2015). Recent trends show resurgence: in 2023, WHO recorded 535,321 cases across 45 countries, and major outbreaks persisted in 2024, including Sudan's conflict-driven epidemic with over 24,000 cases and nearly 700 deaths by November (Mednick, 2024), alongside sustained transmission in Nigeria (Maruf, 2024; Shobayo, 2024). These setbacks challenge the global goal of reducing cholera deaths by 90% by 2030 (Global Task Force on Cholera Control, 2024), particularly as 419 million people still practice open defecation, an ongoing driver of water contamination (OiEau, 2024; United Nations, 2024).

Cholera transmission involves both direct human exposure and indirect spread through contaminated environments. A key epidemiological insight is its zoonotic dimension. Evidence shows aquatic animals, including fish, shellfish, and

waterfowl, serve as active carriers of *V. cholerae* (Almagro-Moreno and Taylor, 2013; Yan *et al.*, 2022). Copepods act as reservoirs; fish become infected by feeding on them; and waterfowl disperse the bacteria over long distances (Laviad-Shitrit *et al.*, 2019). This human - animal - environment interface underscores the need for a One Health framework (Gibbs, 2014).

Mathematical modelling is central to understanding these transmission dynamics and assessing interventions. Classic SIR and SEIR models (Kermack and McKendrick, 1927; Bjornstad *et al.*, 2020). Cholera model extension generate key metrics such as the basic reproduction number, which determines persistence or elimination (Rock *et al.*, 2014). Models have demonstrated the effectiveness of vaccination (Albalawi *et al.*, 2023), improved water and sanitation (Maliki and Chibuike, 2021), treatment (Lemo-Paiao *et al.*, 2020), and health education (Mwasa and Tchuenche, 2011), with combined strategies performing best (Wang and Modnak, 2011; Mustapha *et al.*, 2024). However, most omit explicit zoonotic pathways; even advanced models like Albalawi *et al.* (2023) exclude animal vectors. Recent work incorporating flies and fish (Suma'Inna *et al.*, 2022) shows fish-related contact rates strongly influence transmission, yet full One Health integration remains limited.

This study addresses this gap by developing a deterministic cholera model that explicitly incorporates a fish population as an animal vector, extending the framework of Albalawi *et al.* (2023) in alignment with One Health principles. Objectives include: (i) adding a fish compartment; (ii) analysing positivity and boundedness; (iii) determining disease-free and endemic equilibria; (iv) computing ; (v) proving local and global stability; (vi) performing sensitivity analysis; and (vii) conducting numerical simulations to explore outbreak behaviour and intervention effects. Incorporating this zoonotic component offers a more ecologically realistic tool for guiding integrated cholera control strategies.

2.0 MODEL FORMULATION

The researchers considered three (3) populations of interest in essence the human, aquatic animal (fish), and bacterial populations. The human population is subdivided into five (5) compartments, namely, susceptible human $S_h(t)$, infected human $I_h(t)$, vaccinated human $V_h(t)$, quarantined human $Q_h(t)$ and recovered human $R_h(t)$. The fish population is split into two groups: susceptible $S_a(t)$, and infected $I_a(t)$ fishes.

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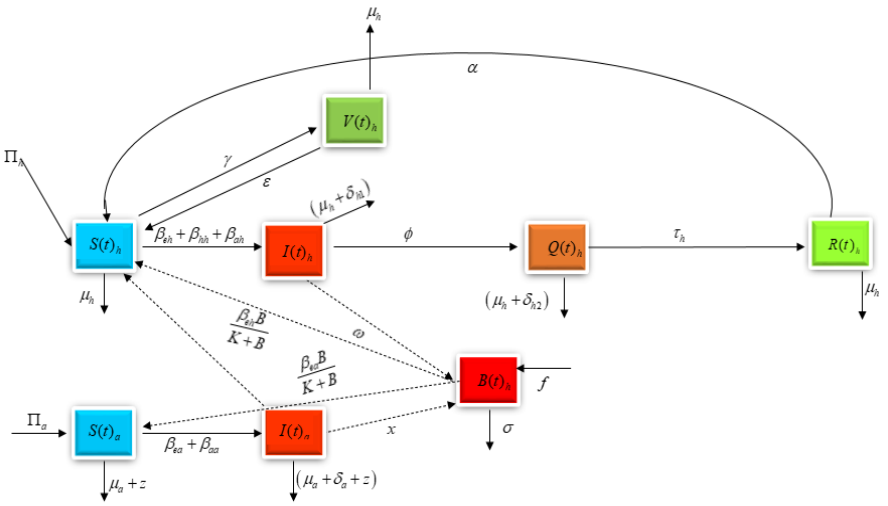

Figure 1: Schematic Diagram of Cholera Disease Dynamics Model.

Table 1: Variables of Cholera Disease Model

Variables	Description
$S_h(t)$	Susceptible human population at time t,
$I_h(t)$	Infected human population at time t,
$V_h(t)$	Vaccinated human population at time t,
$Q_h(t)$	Quarantined human population at time t,
$R_h(t)$	Recovered human population at time t,
$S_a(t)$	Susceptible fish population at time t,
$I_a(t)$	Infected fish population at time t,
$B(t)$	Bacteria *(V. cholerae)* population at time t,

Table 2: Parameters of Cholera Disease Model

Parameters	Description	Baseline values	Sources
Π_h	Recruitment rate due to birth and immigration in humans	0.00913	Hartley *et al.* (2006)
Π_a	Recruitment rate due to birth and immigration in fish	0.2881394595	Suma'inna *et al.*, 2022
β_{eh}	Rate of exposure of susceptible humans to contaminated water by bacteria (*V. cholera*)	0.0040963	Albalawi *et al.*, 2023
β_{hh}	The rate of contact susceptible human and with infected human	0.2143	Hartley *et al.* (2006)
β_{ea}	Rate of exposure of fish to contaminated water by bacteria (*V. cholera*)	0.9433743078	Suma'inna *et al.*, 2022

β_{aa}	The rate of contact between infected fish with susceptible fish	0.9433743078	Suma'inna *et al.*, 2022
β_{ah}	The rate of contact of susceptible humans with infected fish	0.00714285	Suma'inna *et al.*, 2022
τ_h	Recovery rate of the quarantined class due to treatment	0.02	Okolo *et al.* (2020)
τ_a	Recovery rate of infected fish due to treatment	0.2	Assumed
δ_{h1}	The rate of Infection-induced death in infected humans	0.013	(Wang *et al.* 2011)
δ_{h2}	Rate of infection-induced death in the quarantined human	0.008	WHO 2010
δ_a	Rate of Infection-induced death in fish	0.2881394595	Suma'inna *et al.*, 2022
K	Bacteria load (concentration)	10^6	Sanches *et al.* (2011)
μ_h	Natural death rate in the human population	0.025	(Falaye *et al.*, 2018)
μ_a	Natural death rate in the fish population	0.2881394595	Suma'inna *et al.*, 2022
α	Rate of loss of temporally acquired immunity due to treatment from the recovered compartment	0.001096	Neilan *et al.* (2010)
ω	Contribution rate of each infected human to the bacteria (*V. cholera)* population	10	Wang and Modnak (2011)
x	Contribution rate of each infected fish to the bacteria (*V. cholera)* population	10	Assumed
ε	Rate of loss of vaccine-induced immunity	0.00091	Levine *et al.*, (2021)
γ	The rate of vaccination of the susceptible population	0.04	Okolo *et al.* (2020)
η	Hygiene practices	0.4	Maliki and Chibuike (2021)
C_w	Rate of compliance with water hygiene	0.01	Okolo *et al.* (2020)
σ	The death rate of bacteria	0.33	(Misra *et al.*, 2011).
ϕ	The rate of quarantine of infected humans	0.00331428	Albalawi *et al.*, (2023)
f	Growth rate of the bacteria	0.028	Falaye *et al.* (2018)
z	Fishing rate	0.02	Assumed

2.1 DESCRIPTION OF THE MODEL

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The population of susceptible individuals increases through constant recruitment at a rate Π_h and by the inflow of treated individuals who have lost temporary immunity and return to susceptibility at a rate α. The susceptible human population decreases due to infection acquired through the ingestion of contaminated or improperly cooked food, consumption of infected aquatic animals, or contact with infected humans or aquatic animals. This occurs at the force of infection rate $\frac{\beta_{eh}(1-\eta)B}{K+B}+\beta_{hh}(1-\eta)I+\beta_{ah}(1-\eta)I_a$, where β_{eh} denotes the transmission rate through exposure to contaminated water containing *V. cholerae*, β_{hh} represents human-to-human transmission, and β_{ah} represents transmission from aquatic animals to humans. Additionally, susceptible individuals die naturally at a rate μ_h. The population of infected individuals increases due to exposure and infection at a rate $\frac{\beta_{eh}(1-\eta)B}{K+B}+\beta_{hh}(1-\eta)I+\beta_{ah}(1-\eta)I_a$ and decreases through disease-induced death at a rate δ_{h1}, quarantine at a rate ϕ, and natural death at a rate μ_h. The quarantined population increases at a rate ϕ (the quarantine rate) and decreases due to treatment at a rate τ_h, disease-induced death at a rate δ_h, and natural death at a rate μ_h. The recovered population increases due to treatment at a rate τ_h and decreases through loss of temporary immunity at rate α, and natural death at rate μ_h. The vaccinated population increases through vaccination of susceptible rate γ, respectively, and decreases due to waning vaccine protection at the rate ε (assuming vaccine efficacy lasts approximately three years) and natural death at the rate μ_h.

The bacterial population of *V. cholerae* in the environment increases through shedding from infected humans and aquatic animals at rates a and x, respectively. The bacterial growth is also enhanced by binary fission at the rate f, and the population decreases due to bacterial death at the rate σ, influenced by environmental sanitation and hygiene conditions.

For the aquatic animal population, the susceptible fish population increases through recruitment at the rate Π_a and recovery from infection at a rate τ_a. It decreases due to infection from ingesting infected copepods or aquatic organisms at the rate β_{aa}, from environmental exposure at the rate β_{ea}, natural death at the rate μ_a, and fishing at the rate $_z$. The infected fish population increases through infection at rates β_{aa} and β_{ea}, and decreases due to disease-induced death at the rate δ_a, natural death at the rate μ_a, recovery at the rate τ_a, and fishing at the rate $_z$.

Based on these assumptions and transitions, the cholera transmission model is formulated as a system of ordinary differential equations (ODEs), presented in Equation (1).

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$$\left.\begin{aligned}
\frac{dS_h(t)}{dt} &= \Pi_h + \alpha R_h + \varepsilon V_h - \left[\lambda_h + \gamma + \mu_h\right] S_h \\
\frac{dI_h(t)}{dt} &= \lambda_h S_h - \left(\phi + \mu_h + \delta_{h1}\right) I_h \\
\frac{dV_h(t)}{dt} &= \gamma S_h - \left(\varepsilon + \mu_h\right) V_h \\
\frac{dQ_h(t)}{dt} &= \phi I_h - \left(\mu_h + \delta_{h2} + \tau_h\right) Q_h \\
\frac{dR_h(t)}{dt} &= \tau_h Q_h - \left(\alpha + \mu_h\right) R_h \\
\frac{dB(t)}{dt} &= \omega\left(1-\eta\right) I_h + x I_a + \left(f - \sigma\right) B \\
\frac{dS_a(t)}{dt} &= \Pi_a + \tau_a I_a - \left(\lambda_a + \mu_a + z\right) S_a \\
\frac{dI_a(t)}{dt} &= \lambda_a S_a - \left(\tau_a + \mu_a + \delta_a + z\right) I_a
\end{aligned}\right\} \quad (1)$$

Where λ is the force of infection;

$$\left.\begin{aligned}
\lambda_h &= \left(1-\eta\right)\left(\frac{\beta_{eh} B}{K+B} + \beta_{hh} I_h + \beta_{ah} I_a\right) \\
\lambda_a &= \left(1-C_w\right)\left(\frac{\beta_{ea} B}{K+B}\left(1-\eta\right) + \beta_{aa} I_a\right)
\end{aligned}\right\} \quad (2)$$

With initial conditions $\left(S_h(0), I_h(0), V_h(0), Q_h(0), R_h(0), B(0), S_a(0), I_a(0)\right) \geq 0$ (3)

3.0 MODEL ANALYSIS

The mathematical model(1) of cholera disease dynamics considers both human and fish populations, all its state variables and parameters are non-negative for all time t. Hence, the following result holds for all the variables in the model equations(1).

3.1 POSITIVITY

Theorem 1: The solutions of the Cholera disease model(1) $\left\{S_h, I_h, V_h, Q_h, R_h, B, S_a, I_a\right\}$ with non-negative initial conditions $S_h(0), I_h(0), V_h(0), Q_h(0), R_h(0), B(0), S_a(0), I_a(0)$ remain non-negative for all time $t > 0$.

Proof:

Using the method of proof from Wangari *et al.* (2023), when $S_h(t) = 0$ before all other state variables $I_h, V_h, Q_h, R_h, B, S_a, I_a$. Then, from the first equation of the model equation (1) we have

$$\left.\frac{S_h(t)}{dt}\right|_{S_h=0} = \Pi_h + \alpha R_h + \varepsilon V_h > 0 \quad (4)$$

Following a similar process in(4), we can deduce that for all $t > 0$ $\left\{I_h(t), V_h(t), Q_h(t), R_h(t), B(t), S_a(t), I_a(t)\right\} \geq 0$, hence, the non-negativity of the cholera model(1) is established.

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3.2 INVARIANT REGION

Theorem 2: The biologically feasible region.

$$\Gamma=\left\{\left(S_h,I_h,V_h,Q_h,R_h,B,S_a,I_a\right)\in\mathbb{R}_+^8 : N_h(t)\le\frac{\Pi_h}{\mu_h}, N_a(t)\le\frac{\Pi_a}{\left(\mu_a+z\right)}, B(t)\le\frac{\theta}{\sigma-f}\right\} \quad (5)$$

Implies that the region Γ is positively invariant with respect to the model (1)

Proof:

We first consider the human population in the absence of disease-induced mortality $\delta_{h1}=\delta_{h2}=0$, is given by; $\frac{dN_h}{dt}=\frac{dS_h}{dt}+\frac{dI_h}{dt}+\frac{dV_h}{dt}+\frac{dQ_h}{dt}+\frac{dR_h}{dt}$, will yield;

$$\frac{dN_h}{dt}+\mu_h N_h(t)\le\Pi_h \quad (6)$$

Using the integrating factor method, integrate the equation (6), we obtained;

$$N_h(t)\le\frac{\Pi_h}{\mu_h}, \text{ as } t\to\infty \quad (7)$$

Applying the same approach to the fish population, we obtain

$$N_a(t)\le\frac{\Pi_a}{\left(\mu_a+z\right)} \text{ as } t\to\infty \quad (8)$$

Similarly, to the bacteria population in the contaminated environment:

$$\frac{dB(t)}{dt}=\omega\left(1-\eta\right)I_h+xI_a+\left(f-\sigma\right)B \quad (9)$$

Equation (9) becomes: $\frac{dB(t)}{dt}\le\frac{\omega\left(1-\eta\right)\Pi_h}{\mu_h}+\frac{x\Pi_a}{\left(\mu_a+z\right)}-\left(\sigma-f\right)B$

Since $I_h(t)\le N_h(t)\le\frac{\Pi_h}{\mu_h}$, and $I_a(t)\le N_a(t)\le\frac{\Pi_a}{\left(\mu_a+z\right)}$

then $\frac{dB(t)}{dt}\le\theta-\left(\sigma-f\right)B$ (10)

where; $\theta=\frac{\omega\left(1-\eta\right)\Pi_h}{\mu_h}+\frac{x\Pi_a}{\left(\mu_a+z\right)}$

$$\frac{dB(t)}{dt}+\left(\sigma-f\right)B\le\theta \quad (11)$$

Using the integrating factor for $f<\sigma$ (that is, bacteria die faster than they reproduce):

$$\Rightarrow B(t)\le\frac{\theta}{\sigma-f}, \text{ as } t\to\infty \quad (12)$$

Hence, all the solutions of the model (1) is well posed, mathematically meaningful (Hethcote,2000; Bjornstand *et al.,* 2020).

3.3 CHOLERA-FREE EQUILIBRIUM

In the absence of cholera infection, that is when, $I_h=Q_h=B=I_a=0,$, then the system(1) has a point referred to be Cholera-Free Equilibrium (CFE), which is obtained by setting the right-hand side of the equation(1) to zero and solving simultaneously. The CFE obtained is;

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$$E_0=\left(S_{h0},I_{h0},V_{h0},Q_{h0},R_{h0},B_0,S_{a0},I_{a0}\right)=\left(\frac{\left(\mu_h+\varepsilon\right)\Pi_h}{\mu_h\left(\varepsilon+\gamma+\mu_h\right)},0,\frac{\gamma\Pi_h}{\mu_h\left(\varepsilon+\gamma+\mu_h\right)},0,0,0,\frac{\Pi_a}{\left(\mu_a+z\right)},0\right) \quad (13)$$

3.4. CHOLERA PRESENT EQUILIBRIUM

The state where disease is prevalent in the population; that is infectiousness is not zero. Let the endemic equilibrium point be denoted by E_0^{**} such that $E_0^{**}=\left(S_h^{**},V_h^{**},I_h^{**},Q_h^{**},R_h^{**},B^{**},S_a^{**},I_a^{**}\right)$ and set $S_h\neq 0, V_h\neq 0, I_h\neq 0, Q_h\neq 0, R_h\neq 0, B_h\neq 0, S_a\neq 0, I_a\neq 0$.

The endemic equilibrium points of the system (1), is obtained as;

$$\left.\begin{aligned}
S_h^{**}&=\frac{\Pi_h l_7 l_4 l_1 l_6}{\left(\lambda_h^{**}+l_5\right)l_7 l_4 l_1 l_6-\alpha\tau\phi\lambda_h^{**}-\varepsilon\gamma l_7 l_4 l_1}\\
I_h^{**}&=\frac{\lambda_h^{**}\Pi_h l_7 l_4 l_6}{\left(\lambda_h^{**}+l_5\right)l_7 l_4 l_1 l_6-\alpha\tau\phi\lambda_h^{**}-\varepsilon\gamma l_7 l_4 l_1}\\
V_h^{**}&=\frac{\gamma\Pi_h l_7 l_4 l_1}{\left(\lambda_h^{**}+l_5\right)l_7 l_4 l_1 l_6-\alpha\tau\phi\lambda_h^{**}-\varepsilon\gamma l_7 l_4 l_1}\\
Q_h^{**}&=\frac{\phi\lambda_h^{**}\Pi_h l_7 l_6}{\left(\lambda_h^{**}+l_5\right)l_7 l_4 l_1 l_6-\alpha\tau\phi\lambda_h^{**}-\varepsilon\gamma l_7 l_4 l_1}\\
R_h^{**}&=\frac{\tau_h\phi\lambda_h^{**}\Pi_h l_6}{\left(\lambda_h^{**}+l_5\right)l_7 l_4 l_1 l_6-\alpha\tau\phi\lambda_h^{**}-\varepsilon\gamma l_7 l_4 l_1}\\
B^{**}&=\frac{\omega\left(\lambda_h^{**}\Pi_h l_7 l_4 l_6\right)}{\left(\left(\lambda_h^{**}+l_5\right)l_7 l_4 l_1 l_6-\alpha\tau\phi\lambda_h^{**}-\varepsilon\gamma l_7 l_4 l_1\right)l_3}+\frac{x\lambda_h^{**}\Pi_a}{\left(l_8 l_4-\tau_a\lambda_a^{**}\right)l_3}\\
S_a^{**}&=\frac{\Pi_a l_4}{l_8 l_4-\tau_a\lambda_a^{**}}\\
I_a^{**}&=\frac{\lambda_a^{**}\Pi_a}{l_8 l_4-\tau_a\lambda_a^{**}}
\end{aligned}\right\} \quad (14)$$

3.5 BASIC REPRODUCTION NUMBER

The Basic Reproduction Number $\Re_0$, represents the average number of new infections that a single infected individual is expected to cause within a susceptible population (Albalawi *et al.*, 2023). To obtain the basic reproduction number, the next generation matrix method is used (Driessche and Watmough 2002). The basic reproduction number is the dominant eigenvalue, which is given by;

$$\Re_0=\frac{\left(\Re_{0h}+\Re_{0a}\right)\pm\sqrt{\left(\Re_{0h}+\Re_{0a}\right)^2-4\left(\Re_{0h}\Re_{0a}-A_{41}A_{14}\right)}}{2}$$

Where;

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$$\Re_{0h}=\frac{\beta_{hh}\eta_e(\mu_h+\varepsilon)\Pi_h}{l_1\mu_h(\varepsilon+\gamma+\mu_h)}+\frac{\beta_{eh}\omega\eta_e^{\,2}(\mu_h+\varepsilon)\Pi_h}{l_1l_3\mu_h(\varepsilon+\gamma+\mu_h)},\Re_{0a}=\frac{x\beta_{ea}\eta_e\upsilon_w\Pi_a}{l_3l_4K(\mu_a+z)}+\frac{\beta_{aa}\upsilon_w\Pi_a}{l_4(\mu_a+z)}$$

$$A_{14}=\frac{x\beta_{eh}\eta_e(\mu_h+\varepsilon)\Pi_h}{l_3l_4K\mu_h(\varepsilon+\gamma+\mu_h)}+\frac{\beta_{ah}\eta_e(\mu_h+\varepsilon)\Pi_h}{l_4\mu_h(\varepsilon+\gamma+\mu_h)},A_{41}=\frac{\beta_{ea}\omega\eta_e^2\upsilon_w\Pi_a}{l_1l_3K(\mu_a+z)},$$

$$\eta_e=1-\eta,\upsilon_w=1-C_w,l_4=(\tau_a+\mu_a+\delta_a+z),l_2=(\mu_h+\delta_{h2}+\tau_h),l_3=(f-\sigma),l_1=(\phi+\mu_h+\delta_{h1})\}$$

3.5 LOCAL STABILITY OF THE CHOLERA-FREE EQUILIBRIUM

Lemma 1: The cholera-free equilibrium of the model (1) is locally asymptotically stable (LAS) if $R_0<1$ and unstable otherwise(Akinyemi *et al.*, 2018).

Proof

From the obtained negative eigenvalues

$$\lambda_1=-l_8,\lambda_2=-l_7,\lambda_3=-l_2 \quad (15)$$

$$\lambda_4=-\frac{1}{2}\left((l_5+l_6)-\left(\sqrt{4\gamma\varepsilon+l_5^2-2l_5l_6+l_6^2}\right)\right) \quad (16)$$

Whenever; $(l_5+l_6)>\sqrt{4\gamma\varepsilon+l_5^2-2l_5l_6+l_6^2}$

$$\lambda_5=-\frac{1}{2}\left((l_5+l_6)+\left(\sqrt{4\gamma\varepsilon+l_5^2-2l_5l_6+l_6^2}\right)\right) \quad (17)$$

$$\lambda_6=N-l_4 \quad (18)$$

Whenever; $N<l_4$

$$\lambda_7=-\frac{1}{2}\left((D+l_1-l_3)-\sqrt{4E\eta_e\omega+D^2+2Dl_1+2Dl_3+l_1^2+2l_1l_3+l_3^2}\right) \quad (19)$$

Whenever; $(D+l_1-l_3)>\sqrt{4E\eta_e\omega+D^2+2Dl_1+2Dl_3+l_1^2+2l_1l_3+l_3^2}$

$$\lambda_8=-\frac{1}{2}(D+l_1-l_3)-\frac{1}{2}\sqrt{4E\eta_e\omega+D^2+2Dl_1+2Dl_3+l_1^2+2l_1l_3+l_3^2} \quad (20)$$

$$\lambda_8=-\frac{1}{2}\left((D+l_1-l_3)+\frac{1}{2}\sqrt{4E\eta_e\omega+D^2+2Dl_1+2Dl_3+l_1^2+2l_1l_3+l_3^2}\right)$$

Where;

$$\left.\begin{aligned}D=\frac{\beta_{hh}\eta_e(\mu_h+\varepsilon)\Pi_h}{\mu_h(\varepsilon+\gamma+\mu_h)},E=\frac{\beta_{eh}\eta_e(\mu_h+\varepsilon)\Pi_h}{K\mu_h(\varepsilon+\gamma+\mu_h)},G=\frac{\beta_{ah}\eta_e(\mu_h+\varepsilon)\Pi_h}{\mu_h(\varepsilon+\gamma+\mu_h)},\\ H=\frac{\beta_{ea}\upsilon_w\eta_e\Pi_a}{Kl_8},N=\frac{\beta_{aa}\upsilon_w\Pi_a}{l_8},l_5=\gamma+\mu_h,l_6=\varepsilon+\mu_h,l_7=\alpha+\mu_h \text{ and } l_8=\mu_a+z\end{aligned}\right\}$$

the disease-free equilibrium is locally asymptotically stable.

3.6 GLOBAL STABILITY OF THE CHOLERA-FREE EQUILIBRIUM

Theorem 3: The Cholera-free equilibrium of the system(1) is globally asymptotically stable if the $\Re_0<1$. Using the method of Castillo-Chavez *et al.* (2002);

Proof:

To prove Theorem 3, the model(1) can be restructured in the form

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$$\left.\begin{aligned}\frac{dX_1}{dt} &= F\left(X_1,\ X_2\right)\\ \frac{dX_2}{dt} &= G\left(X_1,\ X_2\right),\ G\left(X_1,\ 0\right)=0\end{aligned}\right\} \tag{21}$$

Where $X_1=\left(S_h,V_h,R_h,S_a\right)$ and $X_2=\left(I_h,Q_h,B,I_a\right)$

Let the CFE of the model(1) be given by $E_0=\left(X_1^*,0\right)$. Then, the following properties must be satisfied to establish the global asymptotic stability of the model (1) (Brauer *et al.,* 2016).

$H_1:\frac{dX_1}{dt}=F\left(X_1^*,0\right),X_1^*$ is globally asymptotically stable

$H_2:G\left(X_1,X_2\right)=AX_2-\hat{G}\left(X_1,X_2\right),\hat{G}\left(X_1,X_2\right)\geq 0$ for $\left(X_1,X_2\right)\in\Gamma$

$A=\frac{\partial G}{\partial X_2}$ Is a matrix evaluated at $\left(X_1^*,0\right)$,

where, the off-diagonal elements are non-negative.

$F(X_1,X_2)$ and $G(X_1,X_2)$ are obtained from (1) as follows

$$F\left(X_1,X_2\right)=\begin{pmatrix}\Pi_h+\alpha R_h+\varepsilon V_h-\left[\frac{\beta_{eh}\left(1-\eta\right)B}{K+B}+\beta_{hh}\left(1-\eta\right)I_h+\beta_{ah}\left(1-\eta\right)I_a+\gamma+\mu_h\right]S_h\\ \gamma S_h-\left(\varepsilon+\mu_h\right)V_h\\ \tau_hQ_h-\left(\alpha+\mu_h\right)R_h\\ \Pi_a+\tau_aI_a-\left(\frac{\beta_{ea}B}{K+B}\left(1-\eta\right)\left(1-C_w\right)+\beta_{aa}\left(1-C_w\right)I_a+\mu_a+z\right)S_a\end{pmatrix} \tag{22}$$

$$G\left(X_1,X_2\right)=\begin{pmatrix}\left(\frac{\beta_{eh}\left(1-\eta\right)B}{K+B}+\beta_{hh}\left(1-\eta\right)I_h+\beta_{ah}\left(1-\eta\right)I_a\right)S_h-\left(\phi+\mu_h+\delta_{h1}\right)I_h\\ \phi I_h-\left(\mu_h+\delta_{h2}+\tau_2\right)Q_h\\ \omega\left(1-\eta\right)I_h+xI_a+\left(f-\sigma\right)B\\ \left(\frac{\beta_{ea}B}{K+B}\left(1-\eta\right)\left(1-C_w\right)+\beta_{aa}\left(1-C_w\right)I_a\right)S_a-\left(\tau_a+\mu_a+\delta_a+z\right)I_a\end{pmatrix} \tag{23}$$

Evaluating (22) at disease-free equilibrium $H_1:\frac{dX_1}{dt}=F\left(X_1\,0\right)$; implies that;

$$\left.\begin{aligned}\frac{dS_h}{dt} &= \Pi_h+\varepsilon V_h-\left(\gamma+\mu_h\right)S_h\\ \frac{dV_h}{dt} &= \gamma S_h-\left(\varepsilon+\mu_h\right)V_h\\ \frac{dR_h}{dt} &= 0\\ \frac{dS_a}{dt} &= \Pi_a-\left(\mu_a+z\right)S_a\end{aligned}\right\} \tag{24}$$

Solving (24) by integrating each equation, yields

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$$\left.\begin{aligned} S_h(t) &= S_h(0)e^{-(\gamma+\mu_h)t} + \frac{(\mu_h+\varepsilon)\Pi_h}{\mu_h(\varepsilon+\gamma+\mu_h)} \\ V_h(t) &= V_h(0)e^{-(\varepsilon+\mu_h)t} + \frac{\gamma\Pi_h}{\mu_h(\varepsilon+\gamma+\mu_h)} \\ R_h(t) &= R_h(0)e^{-(\alpha+\mu_h)t} \\ S_a(t) &= S_a(0)e^{-(\mu_a+z)t} + \frac{\Pi_a}{(\mu_a+z)} \end{aligned}\right\} \quad (25)$$

Hence, $S_h(t), V_h(t), R_h(t), S_a(t) \to \left(\frac{(\mu_h+\varepsilon)\Pi_h}{\mu_h(\varepsilon+\gamma+\mu_h)}, \frac{\gamma\Pi_h}{\mu_h(\varepsilon+\gamma+\mu_h)}, 0, \frac{\Pi_a}{(\mu_a+z)}\right)$

irrespective of the initial value of the variables as $t \to \infty$. Therefore, X_1^* is globally asymptotically stable, satisfying H_1

Furthermore, the matrix A is given as

$$A = \frac{\partial G}{\partial X_2}\left(X_1^* 0\right) = \begin{pmatrix} -l_1+\beta_{hh}\eta_e S_h(0) & 0 & \frac{\beta_{eh}}{K}\eta_e S_h(0) & \beta_{ah}\eta_e S_h(0) \\ \phi & -l_2 & 0 & 0 \\ \omega\eta_e & 0 & (f-\sigma) & x \\ 0 & 0 & \frac{\beta_{ea}\eta_e\upsilon_w S_a(0)}{K} & -l_4+\beta_{aa}\upsilon_w S_a(0) \end{pmatrix} \begin{pmatrix} I_h \\ Q_h \\ B \\ I_a \end{pmatrix} \quad (26)$$

$$\hat{G}(X_1, X_2) = AX_2 - G(X_1, X_2)$$

$$\begin{pmatrix} \left(-l_1+\beta_{hh}\eta_e S_h(0)\right)I_h + \frac{\beta_{eh}}{K}\eta_e S_h(0)B + \beta_{ah}\eta_e S_h(0)I_a \\ \phi I_h - l_2 Q_h \\ \omega\eta_e I_h + (f-\sigma)B + xI_a \\ \frac{\beta_{ea}\eta_e\upsilon_w S_a(0)B}{K} + \left(-l_4+\beta_{aa}\upsilon_w S_a(0)\right)I_a \end{pmatrix} - \begin{pmatrix} \left(\frac{\beta_{eh}\eta_e B}{K+B} + \beta_{hh}\eta_e I_h + \beta_{ah}\eta_e I_a\right)S_h - l_1 I_h \\ \phi I_h - l_2 Q_h \\ \omega\eta_e I_h + xI_a + (f-\sigma)B \\ \left(\frac{\beta_{ea}B}{K+B}\eta_e\upsilon_w + \beta_{aa}\upsilon_w I_a\right)S_a - l_4 I_a \end{pmatrix} \quad (27)$$

$$\hat{G}(X_1 X_2) = \begin{pmatrix} \left(\frac{\beta_{eh}\eta_e B}{K} + \beta_{hh}\eta_e I_h + \beta_{ah}\eta_e I_a\right)(S_h(0) - S_h) \\ 0 \\ 0 \\ \left(\frac{\beta_{ea}\eta_e\upsilon_w B}{K} + \beta_{aa}\upsilon_w I_a\right)(S_a(0) - S_a) \end{pmatrix} \quad (28)$$

Since $0 \le S_h \le \frac{(\mu_h+\varepsilon)\zeta_h}{\mu_h(\varepsilon+\gamma+\mu_h)}$, $0 \le V_h \le \frac{\gamma\Pi_h}{\mu_h(\varepsilon+\gamma+\mu_h)}$ and $0 \le S_a \le \frac{\Pi_a}{(\mu_a+z)}$, it is obvious that $\hat{G}(X_1, X_2) \ge 0$. Thus H_2 holds. Hence, the cholera-free equilibrium of the system (1) is globally asymptotically stable.

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3.8 SENSITIVITY ANALYSIS OF CHOLERA MODEL

The sensitivity analysis reveals factors responsible for its transmission and prevalence. This reveals the impact of each parameter, which helps in minimising and controlling the spread of the disease. The normalised forward sensitivity index of a variable to a parameter is the ratio of the relative change in the variable to the relative change in the parameter (Chitnis *et al.*, 2008). It is given by;

$$\alpha_{\chi}^{R_0} = \frac{\partial R_0}{\partial \chi} \times \frac{\chi}{R_0} \tag{29}$$

where:

$\Re_0$ Is the basic reproduction number

χ Is the parameter of interest

Solving for the parameters of interest, we obtained the results as presented in

Table 3

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Table 3: Sensitivity index of the basic reproduction number relative to its parameters

Parameters	Sensitivity index	Parameters	Sensitivity index
Π_a	1.000003120	μ_a	-1.296955821
β_{aa}	1.000023881	δ_a	-0.3618585793
β_{ea}	0.00002076099051	γ	$-1.895571959*10^{(-6)}$
x	0.00001986802927	η	–0.00001667133242
σ	0.00002268585050	C_w	-0.6666687452
β_{ah}	$8.929612384*10^{(-7)}$	ϕ	$-2.214486542*10^{(-7)}$
β_{hh}	$2.000057473*10^{(-6)}$	z	-0.09002278430
Π_h	$3.124347491*10^{(-6)}$	τ_a	-0.2511690554
ε	$6.658460541*10^{(-8)}$	δ_{h1}	$-1.002247792*10^{(-6)}$
β_{eh}	$2.293779369*10^{(-7)}$	K	0.00002076099563
ω	$1.122409807*10^{(-6)}$	μ_h	$-2.965686391*10^{(-6)}$
τ_h	0	f	$1.924860043*10^{(-6)}$

The sensitivity index results are presented in

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Table 3 show that those parameters with positive index () increase the $\Re_0$. This implies that the parameters have a great impact on transmitting the disease in the population if they increase in value. Similarly, those parameters with negative sensitivity index () decreases the $\Re_0$, which also implies that the parameters can minimise the disease in the population if their values are increased while others are kept constant.

4.0 CONCLUSION AND RECOMMENDATION

The study highlights the zoonotic transmission of cholera by extending the work of Albalawi *et al.* (2023) by incorporating the aquatic animal (fish) population based on the one health initiative, the model was formulated and analysed (Almagro-Moreno and Taylor, 2013; Gibbs, 2014). The model analysis reveals that the model (1) have two equilibrium points; which are the cholera free and cholera present equilibrium points. The non-negativity and invariant region of solutions was found to be well posed. The basic reproduction number was calculated using the next generation matrix and the global stability of the cholera free equilibrium was obtained using Castillo-Chavez method. Using the normalised forward sensitivity index, we were able to find out the parameter that influence the transmission of cholera, numerical simulation further confirms the findings.

Increasing the values of parameters with negative sensitivity index and reduce the values of parameters with positive sensitivity index in order to curb the transmission of cholera in the human and fish population. The data used in this study were selected from the existing literature on the dynamics of cholera transmission. The study recommends mass vaccination, good hygiene practices, quarantine and water sanitation to effectively control the dynamics of cholera in the population.

Conflict of interest: The authors declare that there is no evidence of any conflicting financial interests or personal relationships influencing any of the works discussed in this paper.

Declaration of AI Generation: During preparation, the authors corrected errors in the manuscript's text using Grammarly. After using these resources to assess and make any necessary edits, the authors assume full responsibility for the publication's content.

REFERENCES

Akinyemi, J. A., Adeniyi, M. O., and Chukwu, A. U. (2018). Stability analysis of infectious diseases model in a dynamic population. *Communication in Mathematical Modelling and Applications,* 3(3), 37-43

Albalawi, W., Nisar, K. S., Aalam, A., Ozair, M., Hussain, T., Shoaib, M., and Zahran, H. Y. (2023). Mathematical modelling approach to cholera transmission with vaccination strategy. *Alexandria Engineering Journal, 75*, 191–207. https://doi.org/10.1016/j.aej.2023.05.053

Ali, M., Nelson, A. R., Lopez, A. L., and Sack, D. A. (2015). Updated global burden of cholera in endemic countries. *PLOS Neglected Tropical Diseases,* 9(6), e0003832. https://doi.org/10.1371/journal.pntd.0003832

Almagro-Moreno, S., and Taylor, R. K. (2013). *Vibrio cholerae* as a zoonotic pathogen: Reservoirs and transmission routes. *Environmental Microbiology Reports, 5* (4), 591–597. . https://doi.org/10.1128/microbiolspec.OH-0003-2012

Bjornstad, O. N., Shea, K., Krzywinski, M., and Altman, N. (2020). Modelling infectious epidemics. *Nature Methods, 17* (5), 455–456. https://doi.org/10.1038/s41592-020-0822-z

Brauer, F. (2017). Mathematical epidemiology: Past, present, and future. *Infectious Disease Modelling, 2* (2), 113–127. https://doi.org/10.1016/j.idm.2017.02.001

Chitnis, N., Hyman, J. M., and Cushing, J. M. (2008). Determining important parameters in the spread of malaria through the sensitivity analysis of a mathematical model. *Bulletin of Mathematical Biology, 70*(5), 1272–1296. https://doi.org/10.1007/s11538-008-9299-0

Codeço, C. T. (2001). Endemic and epidemic dynamics of cholera: The role of the aquatic reservoir. *BMC Infectious Diseases, 1*(1), Article 1. https://doi.org/10.1186/1471-2334-1-1

Diekmann, O., Heesterbeek, J. A. P., and Metz, J. A. P. (1990). On the definition and computation of the basic reproduction ratio in models for infectious diseases in heterogeneous populations. *Journal of Mathematical Biology, 28*(4), 365–382. https://doi.org/10.1007/BF00178324

Giamberardino, P. D., and Iacoviello, D. (2018). Modelling and control of an epidemic disease under possible complication. *In 2018 22nd International Conference on System Theory, Control and Computing (ICSTCC)* (pp. 67–72). IEEE. https://doi.org/10.1109/ICSTCC.2018.8540736

Gibbs, E. P. J. (2014). The evolution of One Health: A decade of progress and challenges for the future. *Veterinary Record, 174*(4), 85–91. https://doi.org/10.1136/vr.g143

Halpern, M., and Izhaki, I. (2017). Fish as hosts of *Vibrio cholerae. Frontiers in Microbiology, 8*, Article 282. https://doi.org/10.3389/fmicb.2017.00282

Hartley, D.M., Morris, J.B., and Smith, D.L. (2006). Hypersinfectivity a critical element in the ability of *V. cholerae* to cause epidemics, *PLoS Medicine. e7*. https://doi.org/10.1371/journal.pmed.0030007

Hethcote, H.W.(2000) The mathematics of infectious diseases, SIAM Rev. 4 (42) 599–653

Hendrix, T. R., (1971). *The Pathophysiology of Medicine*. 47(10): 1169-1180

Kermack, W. O., and McKendrick, A. G. (1927). A contribution to the mathematical theory of epidemics. *Proceedings of the Royal Society of London. Series A, Containing Papers of a Mathematical and Physical Character, 115(772), 700–721. https://doi.org/10.1098/rspa.1927.0118

Laviad-Shitrit, S., Lev-Ari, T., Katz, R., Sharaby, Y., Izhaki, I., and Halpern, M. (2019). Waterfowl as potential vectors of *Vibrio cholerae. Applied and Environmental Microbiology, 85*(19), e00932-19. https://doi.org/10.1111/tmi.13069

Lemos-Paião, A. P., Silva, C. J., Torres, D. F., and Venturino, E. (2020). Optimal control of aquatic diseases: A case study of Yemen's cholera outbreak. *Journal of Optimization Theory and Applications, 185*, 1008–1030. https://doi.org/10.48550/arXiv.2004.07402

Maliki, O. S., and Chibuike, O. C. (2021). A mathematical model for the control of cholera epidemic without natural recovery. *Applied Mathematics, 12*, 655–668. https://doi.org/10.4236/am.2021.128046

Maruf, A. (2024, November 19). Cholera outbreak: Sokoto govt reacts as 25 confirmed dead, 15 hospitalized. *Legit.ng*. https://www.legit.ng/nigeria/1626077-cholera-outbreak-sokoto-govt-reacts-25-confirmed-dead15-hospitalized/

Mednick, S. (2024, November). Hundreds dead, thousands infected in Sudan's months-long cholera outbreak. *Africanews*. https://www.africanews.com/2024/11/18/hundreds-dead-thousands-infected-in-sudans-months-long-cholera-outbreak/

Mustapha, U. T., Qureshi, S., and Yusuf, A. (2024). Assessing vaccines and personal hygiene as control strategies for cholera: A mathematical model. *Journal of Theoretical Biology, 580*, Article 111752. https://doi.org/10.1016/j.jtbi.2024.111752

Mwasa, A. and Tchuenche, J. M. (2011). Mathematical analysis of a cholera model with public health interventions. *Biosystems, 105* (3), 190–200. https://doi.org/10.1016/j.biosystems.2011.05.001

OiEau International Office for Water. (2024, November). World Toilet Day 2024: Effective sanitation, essential to sustainable development. World Toilet Day 2024: effective sanitation, essential to sustainable development

Rock, K., Brand, S., Moir, J., and Keeling, M. J. (2014). Dynamics of infectious diseases. *Reports on Progress in Physics, 77* (2), 026602. https://doi.org/10.1088/0034-4885/77/2/026602

Shobayo, I. (2024, November 19th). Over 60 people hospitalized over cholera outbreak in Plateau communities. *Nigerian Tribune*. https://tribuneonlineng.com/over-60-people-hospitalised-over-cholera-outbreak-in-plateau-communities/amp/

Sule, A., and Abdullahi, M. B. (2022). Unholy stigma and control of leprosy: A mathematical modelling approach. *International Journal of Science for Global Sustainability, 8*(3), https://www.researchgate.net/publication/366182396_Unholy_Stigma_and_Control_of_Leprosy_a_Mathematical_Modeling_Approach

Suma'inna, J. Y., Massora, M., and Sirajuddin, M. (2022). An extended SIWR model for cholera transmission dynamics incorporating fly and fish vectors. *Mathematical Biosciences and Engineering, 19*(9), 8765–8785. https://doi.org.10.1063/5.0082967

United Nations. (2024). World Toilet Day - 19 November. https://www.un.org/en/observances/toilet-day

van den Driessche, P., and Watmough, J. (2002). Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Mathematical Biosciences, 180* (1–2), 29–48. https://doi.org/10.1016/S0025-5564(02)00108-6

Wang, J., and Modnak, C. (2011). Modelling cholera dynamics with controls. *Canadian Applied Mathematics Quarterly, 19*(2), 255–273.

Wangari, I. M., Olaniyi, S., Lebelo, R, S., and Okosun, K. O. (2023). Transmission of COVID-19in the presence of single-dose and double dose vaccines with hesitancy: Mathematical modelling and optimal control analysis. *Frontiers in Applied Mathematics and Statistics, 9.* https://doi.org/10.3389/fams.2023.1292443

World Health Organization. (2023). Cholera vaccines: WHO position paper. *Weekly Epidemiological Record, 96*(48), 561–580.

World Health Organization. (2024). WHO reports surge in cholera cases in

2023, surpassing the numbers recorded in 2022. https://www.choleraalliance.org/en/news/who-reports-surge-cholera-cases-2023-surpassing-numbers-recorded-2022

Yan, M., Li, X., and Li, B. (2022). Aquatic animals as vectors of *Vibrio cholerae*: Implications for zoonotic transmission. *Journal of Water and Health, 20*(3), 409–425. https://doi.org/10.2166/wh.2022.025