

Stochastic Cholera Model with Jump Perturbation

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ABSTRACT

We revisit a deterministic $\chi I - F$ model of Codeco that is linked to endemics and epidemics in order to achieve this. A stochastic differential equation SDE for the number of infectious people $I(t)$ that are impacted by the aquatic environment is then produced by extending this model to a stochastic jump diffusion model. The existence and uniqueness of the solution to the SDE was shown. The basic reproduction number R_0 and some qualitative properties (positivity and invariant region) were found for the deterministic model. Also, the stability and the biological meaning of the end points of the endemic and disease-free equilibria were investigated. The stochastic jump diffusion model was numerically simulated by the method of Euler Maruyama to obtain sample paths of $I(t)$. The results showed that the stochastic jump diffusion trajectories fluctuate around the deterministic trajectory of the $\chi I - F$ model and are continuously differentiable, but not continuously differentiable. Moreover, it is theoretically relevant and easy to understand that the proposed stochastic jump diffusion model is applicable.

Keywords: Cholera; Stochastic; Jump Diffusion; Stability Analysis; Reproduction Number R_0 ; Endemic Equilibrium

INTRODUCTION

The battle against cholera is far from victory yet and hence investigations both theoretically and numerically to assess cholera transmission become more and more significant. Research that could be used to enhance the control, prediction or prevention of severe cholera outbreaks is of significant importance especially because mortality is so high in these outbreaks. Mathematical models are useful tools for understanding how diseases are spread and for assessing the effectiveness of interventions in various epidemiological situations.

Cholera is an acute infection of the gut which causes severe diarrhea and vomiting due to the ingestion of the bacterium *Vibrio cholerae* spread by ingesting contaminated water or food. It has a short incubation period (1-5 days). After infection, *Vibrio cholerae* is able to produce an enterotoxin that hastens the dehydration process and renders the organisms incapable of absorbing fluids, ultimately producing death if treatment does not occur within a few hours after infection [1]. Crooks and Atesmachew report that access to potable drinking water and good environment sanitation is still a major problem in many affected areas of Africa and Asia and the incidence of cholera is still on the increase [2].

Cholera is one of the most ancient diseases to impact both children and adults, and, despite its long control, it is still causing epidemic and pandemic outbreaks. Cholera is a disease that has many causes that are environmental. Changes in the hydrological cycle can significantly affect pathogen levels in water bodies as *Vibrio cholerae* persists and proliferates in water. The rainfall pattern and intensity can either increase or decrease the transmission with droughts or floods [3]. These environmental disruptions are usually sudden and erratic and result in unforeseen contamination of water sources, displacement of people, and disruption of sanitation and can lead to unexpected cases of cholera.

After onset of symptoms, the person suffering from cholera has watery diarrhea without pain, profuse vomiting, irregular heart rate and low blood pressure [4]. Many infected people do not experience any symptoms or have only mild symptoms, but continue to excrete the pathogen in their stool for 7-14 days after infection making water sources and susceptible people vulnerable to contamination [5]. About ten percent of those infected get seriously ill, with the potential for dehydration and death to become very rapid without treatment. There are both direct and indirect routes of transmission for cholera [6]. Direct transmission is defined as close contact with infected persons and indirect transmission is through ingestion of food or water contaminated with *Vibrio cholerae* [7]. The environmental reservoir is thus an important factor in the perpetuation of transmission and in the occurrence of recurrent outbreaks.

Cholera epidemics have been a growing health challenge all over the world, and much research effort has been focused on elucidating the epidemiological dynamics of cholera. The interaction of *Vibrio cholerae*, humans, and the aquatic environment have demonstrated that cholera is a much more complex process of transmission than first thought [3]. This fact has led to the development of many mathematical models to study the epidemiology and endemism of cholera. One of the first deterministic models to investigate the 1973 outbreak of cholera in the Mediterranean was developed by Capasso and Paveri Fontana [8]. For this purpose, Codeco developed an extension of this model, explicitly including the aquatic environmental reservoir in the transmission. Hntsa and Kahsay studied a fractional order cholera model, and Opoku and Afriye analyzed the effect of environmental factors and health care interventions towards the control of cholera. The work of Mukandavire was extended by Wang and Modnak to incorporate treatment, vaccination and sanitation interventions in a cholera model. Other contributions are from Mark, Ochoche, Fatima, Togbenon and Moyo, Posny and Wang, Nyabadza, and Lemospaiaao, along with others [3,4,7,9-22].

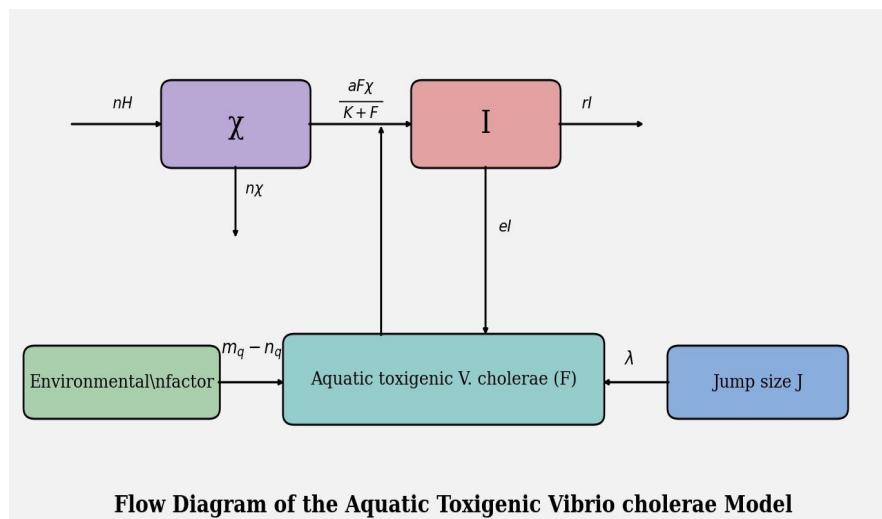
Deterministic models have made a large improvement in the understanding of cholera dynamics, but most of them assume that environmental conditions are continuously varying with time. In fact, there are random fluctuations in the environment many of which affect cholera transmission, and sudden catastrophic events like floods, heavy rainfall, failure of water supply systems, sewage overflow, population displacement and contamination episodes. While jump processes are useful for modelling abrupt and unpredictable events, continuous random fluctuations can be represented by Brownian motion. SDEs with jump diffusion processes integrate these two types of uncertainty, both Wiener process and jumps driven by a Poisson process, into a single equation, hence the name, giving a more realistic representation of an epidemic system with environmental shocks. In a cholera context, a jump diffusion could be a dramatic spike or drop in the density of pathogens, a sudden shift in transmission rates, or a sudden outbreak as a result of unforeseen events like a natural disaster.

Liang, have reported mathematical models for the analysis and prediction of dynamic behavior in the biological systems are successful [23]. Inspired by the necessity to capture the continuous variation in the environment as well as outbreak triggering events, this study extends the deterministic model proposed by Codeco to a stochastic jump-diffusion model of cholera transmission [3]. First the deterministic system is analyzed, with the derivation of the basic reproduction number (R_0), positivity and invariant regions of the solutions and analysis of stability of the disease-free and endemic equilibria. The model is then modified to be a stochastic jump-diffusion model by adding Gaussian white noise and Poisson jump perturbations, so that the dynamics can include sudden events in the environment that influence cholera transmission. A resulting stochastic model is shown to have unique and existing positive solutions. Lastly, numerical simulations are carried out with the Euler-Maruyama scheme and with Poisson jump simulation methods, and implemented in python. The numerical results show the effects of the intensity of the jumps and jump size on the trajectories of the infectious individuals and the concentration of the environmental pathogen; this indicates that the representation of the jumps by the jump-diffusion formulation yields a more realistic representation of cholera outbreaks with sudden disturbances in the environment and irregular surges in the number of infected individuals.

Model Formulation

The cholera model in adopts a classic SIR framework to explain the relationship between the human population and the aquatic environment by including the concentration of *Vibrio cholerae* bacteria in water F . In this paper, the random environmental fluctuations are simulated with the help of the Brownian motion (Wiener process) which leads to the stochastic extension of the deterministic model [20]. The model is composed of three compartments: bacterial concentration in water F , susceptible individuals χ and infected individuals I . The recruitment of susceptible people is at rate n . The contact of polluted water causes infection at rate $\frac{aF}{K+F}$.

The bacteria in the aquatic reservoir grow at rate b , infected individuals shed bacteria to the environment at rate e and recover at rate r .



Deterministic Model

The Deterministic Model Equations. In this model, the deterministic compartmental human population and a Vibrio cholera bacteria population. The total human population is divided into three classes which are the susceptible population χ , the cholera infectious population I and a Vibrio cholerae bacteria population F . Susceptible are recruited with the rate of n through birth, susceptible can be infected at the rate of $\frac{aF}{K+F}$ the contact rate with untreated water and $\frac{aF}{K+F}$ is the probability of an individual to catch cholera.

Infected individuals also contribute to the enhancement of the Vibrio cholera bacteria population through excretion at the rate of e . The infected individual may recover at the rate of r . In the aquatic environment, the bacteria population also grows at a rate determined by environmental factors, and b is the size of bacteria in the aquatic environment. A susceptible individual dies naturally at a to extend the deterministic model in to a stochastic jump diffusion, we reformulate the basic model as follows. The above assumptions of the model lead to the system of ordinary differential equations shown below as in

Variable	Definition
χ	Number of susceptible individuals.
I	Number of infected individuals.
F	Toxigenic concentration of Vibrio cholerae in aquatic

$$\left. \begin{aligned} \frac{d\chi}{dt} &= n(H - \chi) - \frac{aF\chi}{K + F} \\ \frac{dI}{dt} &= \frac{aF\chi}{K + F} - rI \\ \frac{dF}{dt} &= F(n_q - m_q) + eI \end{aligned} \right\} \dots\dots (1)$$

Parameters	Definition
H	Human population
a	Exposure rate of individuals to contaminated water per day
n	Birth and death rates of humans per day
r	Recovery rate per day
K	Vibrio cholerae concentration in water reservoir per day Growth and death difference rate
$n_q - m_q$	Infected rate of individuals contributing to the bacteria population in water
e	

Table 1: Model parameters and their definitions

Theorem 1

Positivity:

In order to get realistic model, our solution must be non-negative for all t . Therefore if the initial values of the parameters $\chi(0) \geq 0$, $I(0) \geq 0$, $F(0) \geq 0$, then the solution set $\{\chi(t), I(t), F(t)\}$ is non-negative for all $t \geq 0$.

Proof

Let $t_* = \sup\{t > 0 : \chi(t) > 0, I(t) > 0, F(t) > 0\}$. Since $\chi(t)$, $I(t)$ and $F(t)$ are continuous, then we $t_* > 0$. If $t_* = +\infty$, the positivity holds however if $0 < t_* < +\infty$, $\chi(t) = 0$ or $I(t) = 0$ or $F(t) = 0$.

Consider the model equation

$$\frac{d\chi(t)}{dt} = nH - \frac{aF(t)\chi(t)}{K+F(t)} - n\chi(t)$$

We can write the above model equation as

$$\frac{d\chi(t)}{dt} + (n + \lambda(t))\chi(t) = nH$$

Where $\lambda(t) = \frac{aF(t)\chi(t)}{K+F(t)}$

$$\frac{d\chi(t)}{dt} + (n + \lambda(t))\chi(t) = nH.$$

The integrating factor is

$$IF = e^{\int (n+\lambda(t)) dt}$$

$$\implies = e^{nt + \int \lambda(t) dt}$$

Multiplying the differential equation by the integrating factor,

$$e^{nt + \int \lambda(t) dt} \frac{d\chi}{dt} + (n + \lambda(t))\chi(t)e^{nt + \int \lambda(t) dt} = nHe^{nt + \int \lambda(t) dt}$$

Hence,

$$\frac{d\chi(t)}{dt} (\chi(t)e^{nt + \int \lambda(t) dt}) = nHe^{nt + \int \lambda(t) dt}$$

Integrating both sides from 0 to t_* ,

$$\int_0^{t_*} \left(\frac{d}{dt} [\chi(t)e^{nt + \int \lambda(t) dt}] \right) dt = \int_0^{t_*} (nHe^{nt + \int \lambda(t) dt}) dt$$

Therefore,

$$\chi(t)e^{nt + \int \lambda(t) dt} \Big|_0^{t_*} = \int_0^{t_*} (nHe^{nt + \int \lambda(t) dt}) dt$$

That is,

$$\chi(t_*)e^{nt_* + \int \lambda(t) dt} - \chi(0) = \int_0^{t_*} (nHe^{nt + \int \lambda(t) dt}) dt$$

$$\chi(t_*)e^{nt_* + \int \lambda(t) dt} = \chi(0) + \int_0^{t_*} (nHe^{nt + \int \lambda(t) dt}) dt$$

$$\chi(t_*) = \chi(0)e^{-(nt + \int \lambda(t)dt)} + e^{-(nt + \int \lambda(t)dt)} \int_0^{t_*} (nHe^{nt + \int \lambda(t)dt}) dt$$

$$\chi(t_*) = T_1\chi(0) + T_1 \int_0^{t_*} (nHe^{nt + \int \lambda(t)dt}) dt$$

where $T_1 = e^{-\left(nt + \int_0^t \lambda(t)dt\right)} > 0$

$$\chi(0) > 0. \text{ Hence, the solution } \chi(t_*) > 0$$

Implies that $\chi(t_*) \neq 0$.

Also,

$$\frac{dI}{dt} = \frac{aF(t)\chi(t)}{K+F(t)} - rI(t).$$

$$\Rightarrow \frac{dI}{dt} = \lambda\chi(t) - rI(t).$$

where $\lambda = \frac{aF(t)}{K+F(t)}$

$$\frac{dI}{dt} + rI(t) = \lambda\chi(t).$$

$$IF = e^{\int r dt}.$$

$$\Rightarrow IF = e^{rt}.$$

Multiplying both sides of the above equation by the integrating factor,

$$e^{rt} \frac{dI}{dt} + rI(t)e^{rt} = \lambda\chi(t)e^{rt}.$$

$$\frac{dI}{dt} (e^{rt}I(t)) = \lambda\chi(t)e^{rt}.$$

$$\int_0^{t_*} \left(e^{rt} \frac{dI}{dt} \right) = \int_0^{t_*} (\lambda\chi(t)e^{rt}) dt$$

$$Ie^{rt} \Big|_0^{t_*} = \int_0^{t_*} (\lambda\chi(t)e^{rt}) dt$$

$$I(t_*)e^{rt} - I(0) = \int_0^{t_*} (\lambda\chi(t)e^{rt}) dt$$

$$I(t_*)e^{rt} = I(0) + \int_0^{t_*} (\lambda\chi(t)e^{rt}) dt$$

$$I(t_*) = I(0)e^{-rt} + e^{-rt} \int_0^{t_*} (\lambda\chi(t)e^{rt}) dt$$

$$I(t_*) = I(0)T_1 + T_1 \int_0^{t_*} (\lambda\chi(t)e^{rt}) dt$$

Where $T_1 = e^{-rt} > 0$

$I(0) > 0$, implies that the solution $I(t_*) > 0$, hence $I(t_*) \neq 0$ The model equation,

$$\begin{aligned}\frac{dF}{dt} &= F(t)(m_q - n_q) + eI(t) \\ \Rightarrow \frac{dF}{dt} + F(t)(m_q - n_q) &= eI(t)\end{aligned}$$

To find the integrating factor of the above differential equation,

$$\text{IF} = e^{\int (m_q - n_q) dt}$$

Multiplying through by the integrating factor,

$$\begin{aligned}e^{\int (m_q - n_q) dt} \frac{dF}{dt} + F(t)(m_q - n_q)e^{\int (m_q - n_q) dt} &= eI(t)e^{\int (m_q - n_q) dt} \\ \Rightarrow \frac{d}{dt} [F(t)e^{\int (m_q - n_q) dt}] &= eI(t)e^{\int (m_q - n_q) dt}\end{aligned}$$

Integrating both sides from 0 to t ,

$$F(t)e^{\int_0^t (m_q - n_q) d\chi} - F(0) = \int_0^t eI(\chi)e^{\int_0^\chi (m_q - n_q) dt} d\chi + \int_0^t e^{\int_0^\chi (m_q - n_q) dt} d\chi$$

Hence,

$$F(t)e^{\int_0^t (m_q - n_q) d\chi} = F(0) + \int_0^t (eI(\chi)e^{\int_0^\chi (m_q - n_q) dt}) d\chi + \int_0^t (\chi e^{\int_0^\chi (m_q - n_q) dt}) d\chi$$

Multiplying by $e^{-\int_0^t (m_q - n_q) d\chi}$

The equation becomes

$$F(t_*) = F(0)e^{-\int_0^t (m_q - n_q) d\chi} + \int_0^t eI(t)$$

Let $k_1 = e^{-\int_0^t (m_q - n_q) d\chi}$

$$F(t) = F(0)e^{-\int_0^t (m_q - n_q) dt} + e^{-\int_0^t (m_q - n_q) dt} \int_0^t e^{\int_0^\chi (m_q - n_q) dt} (eI(t)) dt.$$

Let $e^{-\int_0^t (m_q - n_q) dt} = k_1$

Then $F(t) = F(0)k_1 + k_1 \int_0^t e^{\int_0^\chi (m_q - n_q) dt} eI(t) dt > 0$

$$k_1 = \frac{1}{e^{\int_0^t (m_q - n_q) dt}} > 0$$

$\Rightarrow m_q - n_q > 0$ and $F(0) > 0$

Therefore

$$\begin{aligned}
k_1 F(0) + k_1 \int_0^{t_*} e^{\int (m_q - n_q) dt} eI(t) dt &> 0 \\
k_1 F(0) + k_1 \int_0^{t_*} (eI(t)) e^{\int (m_q - n_q) dt} dt &> 0 \\
F(t) = k_1 F(0) + k_1 \int_0^{t_*} eI(t) e^{\int (m_q - n_q) dt} dt &> 0 \\
F(0) > 0, \quad I(t) > 0, &\implies F(t) > 0 \\
F(t) &\neq 0
\end{aligned}$$

By definition t_* is not finite, which implies that $t_* = +\infty$. Therefore the model solution of the system (1) is always positive

Theorem 2

Invariant Region for the Deterministic Model :

In this section, we obtain the invariant region of the model Equation (1). The model so

$$\text{lution are in the region } \Omega = \{(\chi, I, F) : \chi \geq 0, I \geq 0, 0 \leq \chi + I \leq \frac{\Delta}{n}, 0 \leq F \leq \frac{e\Delta}{nq}\} \quad \forall \quad t_* \geq 0$$

Consider the total human population

$$\begin{aligned}
H(t) &= \chi(t) + I(t) \\
\implies \frac{dH(t)}{dt} &= \frac{d\chi(t)}{dt} + \frac{dI(t)}{dt} \\
\frac{dH(t)}{dt} &= nH(t) - \frac{qF(t)\chi(t)}{K + F(t)} - nS(t) + \frac{qF(t)\chi(t)}{K + F(t)} - rI(t).
\end{aligned}$$

$$\text{Let } nH = \Delta, \quad \chi = H - I$$

Therefore we have

$$\begin{aligned}
\frac{dH(t)}{dt} &= \Delta - n(H - I) - rI(t) \\
\frac{dH(t)}{dt} &= \Delta - nH(t) + nI(t) - rI(t) \\
\frac{dH(t)}{dt} &\leq \Delta - nH(t) \\
\implies H(t) &\leq \frac{\Delta}{n} + Ke^{-nt} \\
\text{At } t = 0, \quad H(0) - \frac{\Delta}{n} &\leq K \\
\text{Therefore } H(t) &\leq \frac{\Delta}{n} + \left(H(0) - \frac{\Delta}{n}\right) e^{-nt} \\
\implies H(t) &\leq \frac{\Delta}{n} \quad \text{And } 0 \leq H(t) \leq \frac{\Delta}{n} \\
\text{Similarly } \frac{dF(t)}{dt} &= eI(t) - F(m_q - n_q)
\end{aligned}$$

$$\frac{dF(t)}{dt} = eI(t) - qF(m_q - n_q) \quad \text{where } q = (m_q - n_q)$$

$$\frac{dF(t)}{dt} + qF = eI(t)$$

To find the integrating factor of the above first order DE

$$IF = e^{\int q dt}$$

$$\int \frac{d}{dt} (F e^{\int q dt}) = \int eI(t) e^{\int q dt} dt$$

$$F e^{\int q dt} = \frac{eI(t)}{q} e^{\int q dt} + K$$

$$F(t) = \frac{eI}{q} + K e^{-qt}$$

$$\text{At } t=0 \quad F(0) - \frac{e\Delta}{nq} \leq K$$

Hence the feasible solution set of the system of equation (1) is in the region $\Omega = \{(\chi, I, F) : \chi \geq 0, I \geq 0, 0 \leq \chi + I \leq \frac{\Delta}{n}, 0 \leq F \leq \frac{e\Delta}{nq}\}$. Therefore the model is well posed and have biological sense.

Existence and Stability of disease-Free equilibrium :

The model's disease free equilibrium is obtained by equating the susceptible χ , infected I and the disease compartment F

$$\frac{d\chi}{dt} = \frac{dI}{dt} = \frac{dF}{dt} = 0$$

This implies that ,

$$\frac{d\chi}{dt} = n(H - \chi) - \frac{aF\chi}{K+F} = 0$$

$$\frac{dI}{dt} = \frac{aF\chi}{K+F} - rI = 0$$

$$\frac{dF}{dt} = eI + F(n_q - m_q) = 0$$

Hence, the equilibrium equations are

$$n(H - \chi) - \frac{aF\chi}{K+F} = 0 \quad \dots \quad (2)$$

$$\frac{aF\chi}{K+F} - rI = 0 \quad \dots \dots \dots (3)$$

$$eI + F(n_q - m_q) = 0 \dots (4)$$

This implies that there exists no infection at the disease-free equilibrium; Thus, $I = 0$

By substituting $I = 0$ into equation (4).

we have, $F(n_q - m_q) = 0$ it follows that $F = 0$

Provided that $(n_q - m_q) \neq 0$

Also, substituting $F = 0$ into equation (2), we obtain $n(H - \chi) = 0$ Since $n \neq 0$, $\chi = H$

Therefore, the disease-free equilibrium is $E_0 = (H, 0, 0)$

$$F(t) \leq \frac{e\Delta}{nq} + \left(F(0) - \frac{e\Delta}{nq}\right) e^{-qt}$$

Taking the limit as $t \rightarrow \infty$

$$\text{Therefore } \lim_{t \rightarrow \infty} F(t) \leq \frac{e\Delta}{nq}$$

$$\text{Which implies that } 0 \leq F(t) \leq \frac{e\Delta}{nq}$$

Therefore, the feasibility of the solution set of the system of equations (1) is in the region

$$\Omega = \left\{ (\chi, I, F) : \chi \geq 0, I \geq 0, 0 \leq \chi + I \leq \frac{\Delta}{n}, 0 \leq F \leq \frac{e\Delta}{nq} \right\}.$$

Hence, the model is well-posed and biologically meaningful, as the jump size alters F .

Disease-Free Equilibrium Stability :

To analyze the disease-free equilibrium stability, let us consider taking the Jacobian of the model equations (2) and (4) to prove that they are locally asymptotically stable around the equilibrium point as in [3]. When it comes to disease spread, local asymptotic stability means that if a small change or perturbation is introduced into the system, the system will still return to the disease-free equilibrium.

Basic Reproduction Number for the Deterministic Model:

The basic reproduction number R_0 is calculated from the disease compartment as follows:

- F_i : rate of appearance of new infections,
- V_i^- : transfer rate out of the disease compartment,
- V_i^+ : transfer rate into the disease compartment by other means.

$$V_i^- = \begin{pmatrix} rI \\ 0 \end{pmatrix}, \quad V_i^+ = \begin{pmatrix} 0 \\ (m_q - n_q) + eI \end{pmatrix}.$$

Therefore,

$$V_i = V_i^- - V_i^+ = \begin{pmatrix} rI - 0 \\ -(m_q - n_q) - eI \end{pmatrix}$$

Hence,

$$V_i = \begin{pmatrix} rI \\ (m_q - n_q) - eI \end{pmatrix}$$

$\frac{\partial F(E_0)}{\partial \chi_i}$, the Jacobian of F with respect to the disease compartment (χ_i) evaluated at the disease-free equilibrium.

The model disease-free equilibrium point is obtained by setting

$$\frac{d\chi(t)}{dt} = \frac{dI(t)}{dt} = dF(t)dt = 0$$

$$nH(t) - \frac{aF(t)\chi(t)}{K+F(t)} - n\chi(t) = 0$$

$$\frac{aF(t)\chi(t)}{K+F(t)} - rI(t) = 0$$

$$(n_q - m_q)F(t) + eI(t) = 0$$

$$nH(t) - \left(\frac{aF(t)}{K+F(t)} + n \right) \chi(t) = 0$$

Therefore

$$\begin{aligned}\chi^* &= \frac{nH}{\frac{aF^*}{K+F^*} + n} \\ I^* &= \frac{aF^*S^*}{r(K+F^*)} \\ F^* &= \frac{eI^*}{(n_q - m_q)}\end{aligned}$$

From the equation above, $I^* = 0$ and $(n_q - m_q) = 0$

provided that $n_q - m_q \neq 0$

By putting $F^* = 0$ in to

$$\chi^* = \frac{nH}{\frac{aF^*}{K+F^*} + n}$$

we have $\chi^* = H$

Therefore,

$$E_0 = (\chi(t), I(t), F(t)) = (H, 0, 0)$$

The Jacobian matrix of is

$$F = \begin{pmatrix} \frac{\partial m_1}{\partial I} & \frac{\partial m_1}{\partial F} \\ \frac{\partial m_2}{\partial I} & \frac{\partial m_2}{\partial F} \end{pmatrix}.$$

where $m_1 = \frac{aF(t)\chi(t)}{K+F(t)} - rI(t)$ and $m_2 = (n_q - m_q) + eI(t)$

Thus,

$$F = \begin{pmatrix} 0 & \frac{a\chi F}{(K+F)^2} \\ 0 & 0 \end{pmatrix}.$$

At the disease-free equilibrium $\chi = H$ $F = 0$

Hence

$$F = \begin{pmatrix} 0 & \frac{aH}{K} \\ 0 & 0 \end{pmatrix}.$$

Also, $V = \frac{\partial V_i(E_0)}{\partial \chi_i}$

which is the Jacobian matrix of V_i with respect to the disease compartments χ_i , evaluated at the disease-free equilibrium (D.F.E).

Therefore,

$$\begin{aligned}
 V &= \begin{pmatrix} r & 0 \\ -e & (m_q - n_q) \end{pmatrix} \\
 V^{-1} &= \frac{1}{r(m_q - n_q)} \begin{pmatrix} (m_q - n_q) & 0 \\ e & r \end{pmatrix} \\
 V^{-1} &= \begin{pmatrix} \frac{1}{r} & 0 \\ \frac{e}{r(m_q - n_q)} & \frac{1}{(m_q - n_q)} \end{pmatrix} \\
 FV^{-1} &= \begin{pmatrix} 0 & \frac{qH}{K} \\ 0 & 0 \end{pmatrix} \begin{pmatrix} \frac{1}{r} & 0 \\ \frac{e}{r(m_q - n_q)} & \frac{1}{(m_q - n_q)} \end{pmatrix} \\
 &= \begin{pmatrix} 0 & \frac{qH}{K} \\ 0 & 0 \end{pmatrix} \begin{pmatrix} \frac{1}{r} & 0 \\ \frac{e}{r(m_q - n_q)} & \frac{1}{(m_q - n_q)} \end{pmatrix} \\
 &= \begin{pmatrix} \frac{aeH}{Kr(m_q - n_q)} & \frac{qH}{K(m_q - n_q)} \\ 0 & 0 \end{pmatrix}
 \end{aligned}$$

The eigenvalues satisfy

$$\begin{vmatrix} \frac{qHe}{Kr(m_q - n_q)} - \lambda & \frac{qH}{K(m_q - n_q)} \\ 0 & -\lambda \end{vmatrix} = 0.$$

Therefore,

$$\left(\frac{qHe}{Kr(m_q - n_q)} - \lambda \right) (-\lambda) = 0.$$

$$\text{Hence } \frac{qHe}{Kr(m_q - n_q)} - \lambda = 0 \quad \text{or} \quad \lambda = 0$$

$$\text{Thus } \lambda_1 = \frac{qHe}{Kr(m_q - n_q)}, \quad \lambda_2 = 0$$

Clearly λ_1 is the dominant eigenvalue and therefore becomes the basic reproduction number R_0 of the model:

$$R_0 = \frac{aeH}{Kr(m_q - n_q)}$$

Theorem 3

The disease-free equilibrium point of the system is asymptotically stable if and only if $R_0 < 1$

proof

$$J = \begin{pmatrix} -n - \frac{aF}{K+F} & 0 & -\frac{a\chi K}{(K+F)^2} \\ \frac{aF}{K+F} & -r & \frac{a\chi K}{(K+F)^2} \\ 0 & e & (n_q - m_q) \end{pmatrix}$$

Also, at the disease-free equilibrium point,

$$E_0 = (H, 0, 0) = (\chi_0, I_0, F_0)$$

We have

$$J(E_0) = \begin{pmatrix} -n & 0 & -\frac{aH}{K} \\ 0 & -r & \frac{aH}{K} \\ 0 & e & (n_q - m_q) \end{pmatrix}$$

The equilibrium point is asymptotically stable if the following condition (Routh-Hurwitz) holds for the characteristic polynomial and its determinant

$$|J(\chi_0, I_0, F_0) - \lambda I| = \begin{vmatrix} -n - \lambda & 0 & -\frac{aH}{K} \\ 0 & -r - \lambda & \frac{aH}{K} \\ 0 & e & (n_q - m_q) - \lambda \end{vmatrix}$$

$$= \lambda^3 + (n+r)\lambda^2 + ((n_q - m_q))\lambda + (n_q - m_q)(n+r) - rn + \frac{aHe}{K} = 0$$

It is observed that the Jacobian matrix characteristic equation has three roots. Now we write the equation of characteristic as

$$P(\lambda) = \lambda^3 + A\lambda^2 + \lambda + C = 0.$$

where

$$A = n + r + (m_q - n_q)$$

$$\begin{aligned} F &= nr + (n+r)(m_q - n_q) - \frac{aHe}{K} \times \frac{r(m_q - n_q)}{r - (m_q - n_q)} \\ &= nr + (n+r)(m_q - n_q) - \frac{aHe}{K} \\ &= nr + (n+r)(m_q - n_q) - \frac{aHe}{K} \times \frac{r(m_q - n_q)}{Kr(m_q - n_q)}. \end{aligned}$$

Hence

$$F = nr + (m_q - n_q + r) + (m_q - n_q)(1 - R_0).$$

$$C = nr(m_q - n_q) - \frac{aHen}{K} = nr(m_q - n_q)(1 - R_0).$$

Therefore,

$$AB - C = \left((m_q - n_q)\delta_3 + n + r\right) \left(n(m_q - n_q) + r\right) - (mq - n_q)(1 - R_0).$$

Hence the eigenvalues that correspond to the equilibrium E_0 are

$$\lambda_1 = -n$$

$$\text{From the quadratic formula, } \lambda_{2,3} = \frac{-b \pm \sqrt{b^2 - 4ac}}{2a}$$

thus,

$$\lambda_{2,3} = \frac{-(m_q - n_q + r) \pm \sqrt{((m_q - n_q + r))^2 - 4(m_q - n_q)(1 - R_0)}}{2}.$$

We have $A > 0$, $B > 0$, $C > 0$, $AB - C > 0$

and according to the Routh-Hurwitz criterion, all the characteristic equation roots have negative real part when $R_0 < 1$ and $(m_q - n_q) > 0$. Hence, the disease-free equilibrium is stable, given that $R_0 < 1$.

Existence and stability of endemic equilibrium:

In the endemic equilibrium state $I \neq 0$, we use equations: 2, 3, 4 to find the endemic equilibrium state

$$n(H - \chi) - \frac{aF\chi}{K + F} = 0, \quad (2)$$

$$\frac{qF\chi}{K + F} - rI = 0, \quad (3)$$

$$F(m_q - n_q) + eI = 0, \quad (4)$$

This implies,

$$F = \frac{eI}{(m_q - n_q)}. \quad (5)$$

$$\chi = \frac{(K + F)rI}{aF}. \quad (6)$$

$$nH - n\chi + \frac{aF\chi}{K + F} = 0. \quad (7)$$

$$nH - \frac{(K + F)rI}{aF} \left(n + \frac{aF}{K + F} \right) = 0,$$

$$nH - \left(\frac{K + F}{qF} rI \right) n - \left(\frac{K + F}{aF} rI \right) \left(\frac{aF}{K + F} \right) = 0,$$

Substituting the value of χ in equation (7)

$$nH + \left(\frac{K + F}{aF} rI \right) n - rI = 0,$$

$$nH - rI \left(n \left(\frac{K + F}{aF} \right) + 1 \right) = 0,$$

$$nH - rI \left(\frac{n(K + F) + aF}{aF} \right) = 0.$$

Since $F = \frac{eI}{(n_q - m_q)}$

We obtain $I = \frac{nHa - rn(n_q - m_q)K}{e(a + n)}$

Or equivalently,

$$I = \frac{nHa - rn(n_q - m_q)K}{er(a + n)}$$

Suppose $I > 0$

Then $nHa - rn(n_q - m_q)K > 0$

which implies $R_0 > 1$

$$\text{since } R_0 = \frac{aHe}{rK(n_q - m_q)} > 1$$

We observed that from the analysis, if $R_0 > 0$ and $(n_q - m_q) > 0$, there exist a positive endemic equilibrium state

Theorem 4

The endemic equilibrium state E^* is locally asymptotically stable if $R_0 > 1$.

Proof

Let $E^* = (\chi^*, I^*, F^*)$.

The Jacobian matrix evaluated at the endemic equilibrium is

$$J(E^*) = \begin{pmatrix} -n - u & 0 & -U \\ u & -r & U \\ 0 & e & -A \end{pmatrix}.$$

Therefore,

$$|J(E^*) - \lambda I| = \begin{vmatrix} -n - u - \lambda & 0 & -U \\ u & -r - \lambda & U \\ 0 & e & -A - \lambda \end{vmatrix}.$$

$$= -\lambda^3 - \lambda^2(n + u + r + A) - \lambda(nr + nA + ur + uA + rA - ue) - nrA - urA + nue + uAe = 0.$$

Multiplying through by -1 gives

$$\lambda^3 + \lambda^2(n + u + r + A) + \lambda(nr + nA + ur + uA + rA - ue) + nrA + urA - nue - uAe = 0.$$

Hence

$$\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0$$

Where

$$a_1 = n + u + r + A$$

$$a_2 = nr + nA + ur + uA + rA - ue$$

$$a_3 = nrA + urA - nue - uAe$$

From the Routh–Hurwitz criterion, the equilibrium is locally asymptotically stable provided

$$a_0 > 0, \quad a_1 > 0, \quad a_2 > 0, \quad a_3 > 0,$$

$$A = \begin{pmatrix} a_0 & a_1 \\ a_2 & a_3 \end{pmatrix}$$

The symmetric difference of A is

$$a_1a_2 - a_0a_3 > 0$$

Which implies that the endemic equilibrium state is locally asymptotically stable. That is all the roots of the characteristic polynomial have negative real parts.

Clearly,

$$a_0 > 0, \quad a_1 > 0$$

since $n, u > 0$ and $R_0 > 1$.

Similarly,

$$a_2 > 0, \quad a_3 > 0$$

since $n, e, r, q > 0$ and $(nl - mwI) > 0$

And $R_0 > 1$ if and only if

$$nr + nA + ur + rA > ue \text{ and } nrA + urA > nue$$

Now, we need to show that

$$a_1a_2 - a_0a_3 > 0$$

Hence,

$$\begin{aligned} & (n + u + r + A)(nr + nA + ur + uA + rA - ue) - (nrA + urA - nue) \\ &= n^2r + n^2A + nur + nuA + nr^2 + nrA - nue + unr + unA + u^2r + u^2A + urA - u^2e \\ &+ nr^2 + nrA + r^2u + urA + Ar^2 - rue + nAr + nA^2 + urA + uA^2 + rA^2 - Aue - nrA - urA + nue > 0. \end{aligned}$$

Therefore, for $R_0 > 1$, the model system (1) has a unique endemic equilibrium E^* and it is stable if

$$n^2r + n^2v + nwr + nuA + wnr + wuA + w^2r + w^2A + wrA + nr^2 + nrA + wr^2 + wrA + Ar^2 + nAr + nA^2 + wrA + wuA + rA^2 > (wue + rue + Aue).$$

And $(mA - nA) > 0$.

Transition Probabilities

Consider the stochastic differential equation with jumps

$$dX(t) = f(X(t), t) dt + g(X(t), t) dW(t) + h(X(t), t) dN(t) \text{ where}$$

$f(X, t)$ is the deterministic (drift) part

$g(X(t), t)$ is the diffusion part and

$h(X(t), t)$ is the jump-size part.

The drift coefficient is given by

$$f(X(t), t) = E \left[\frac{\Delta X}{\Delta t} \right]$$

The diffusion coefficient is

$$g(X(t), t) = \sqrt{V} = \sqrt{E \left[\frac{\Delta X (\Delta X)^T}{\Delta t} \right]}$$

The jump-size coefficient is $h(X(t), t) = \lambda J(t)$

Since time is a continuous variable, the state variables $\chi(t)$ and $I(t)$ are continuous random variables.

Let $\Delta X = X(t + \Delta t) - X(t)$ and

$$\Delta I = I(t + \Delta t) - I(t)$$

These increments are approximately normally distributed,

$\Delta X(t) \sim N(\mu(X)\Delta t, \sigma^2(X)\Delta t)$ and
 $\Delta I(t) \sim N(\mu(I)\Delta t, \sigma^2(I)\Delta t)$
 For small time intervals Δt ,
 Assume $X(t) = (X_1, X_2)^T$

where X_1 and X_2 correspond to the number of individuals $\chi(t)$ and $I(t)$, respectively, with $\chi(t), I(t) \in (0, H)$ and $F(t)$ denotes the concentration of *Vibrio cholerae* bacteria.

$$\text{Therefore } E(\Delta X) = \sum_{i=1}^4 P_i \Delta X_i + \lambda J(t).$$

According to Allen [?], $g = \sqrt{V}$.

Hence,

$$g = V^{\frac{1}{2}} = \frac{1}{\eta} \begin{pmatrix} \gamma + \Theta & \rho \\ \rho & + \Theta \end{pmatrix}$$

Where $\Theta = \sqrt{\gamma\psi - \rho^2}$, $\eta = \sqrt{\gamma + \rho + 2\Theta}$ and

$$\gamma = n(H + \chi) + \frac{aF\chi}{K+B}, \quad \rho = -\frac{aF\chi}{K+F} \quad \text{and} \quad \psi = \frac{aF\chi}{K+F} + rI.$$

Substituting the expressions for γ, ρ, ψ

$$\Theta = \sqrt{\left(n(H + S) + \frac{aF\chi}{K+F}\right) \left(\frac{aF\chi}{K+F} + rI\right) - \left(-\frac{aF\chi}{K+F}\right)^2}.$$

Expanding,

$$\Theta = \sqrt{n(H + \chi) \frac{aF\chi}{K+F} + \frac{aF\chi}{K+F} rI + n(H + \chi) rI}.$$

NUMERICAL RESULTS AND DISCUSSION

Numerical simulations of the suggested stochastic cholera model with jump perturbation were considered for the dynamic behavior of the model. The susceptible human population $S(t)$, the infected human population $I(t)$, and the concentration of toxic *Vibrio cholerae* bacteria $F(t)$ in the aquatic environment are the three interacting state variables that make up the model. The stochastic part of the model incorporates diffusion in situations of environmental uncertainty and abrupt environmental perturbations (Poisson jump processes), whereas the deterministic part of the model predicts the spread of cholera through polluted water. This means that the model can incorporate both large scale contamination events and gradual environmental changes that may impact the transmission of cholera.

The following parameter values were chosen for the numerical experiments: $n = 0.0001$ per day, $a = 0.5$ per day, $K = 106$ cell/ml, $r = 0.2$ per day, $m_q - n_q = 0.33$ per day, $e = 10$ cell/ml per day per person and $\lambda = 0.05$ per day. In this instance n is the recruitment rate for susceptible individuals, n is the recruitment rate for contaminated water, K is the half-saturation constant related to the concentration of the bacteria, r is the recovery rate, $m_q - n_q$ is the net removal rate of bacteria, e is the infected individual that contributes bacteria to the aquatic environment, and λ is the intensity of the Poisson jump process.

The estimated jump diffusion reproduction number R_{jp} shows that in addition to the deterministic reproduction number R_0 , ongoing environmental changes and abrupt epidemic disruptions also have an impact on the dynamics of typhoid disease transmission. In particular, the contribution by the diffusion component includes a negative correction factor $-\eta I^2/2R$

which implies that environmental stochasticity can reduce the potential for the infection to be effective in reproduction. On the contrary, if the size of the jump is positive J_I , then the contribution is positive, The term $\frac{\lambda_I \ln(1+J_I)}{r}$

indicates that if there are significant, or frequent spikes in the number of infections, then disease may be spread. Thus, $R_{jp} < 1$ represents a tendency to extinction of the disease, and $R_{jp} > 1$ represents a potential for disease persistence. The jump-diffusion formulation accounts for the random fluctuations in the infected population, along with dramatic changes in that population, and as such provides a more realistic epidemic threshold.

Poisson jump simulation and the Euler-Maruyama method for the stochastic component were used in the numerical computations. This numerical approach is suitable for the stochastic differential formulation as it allows the discontinuous nature of the disturbances generated by the Poisson process to be incorporated into the numerical trajectories as well as the continuous random fluctuations generated by the Wiener process. The consequence is that the stochastic formulation can give several possible epidemic paths, rather than a single deterministic path. The development of the model in particular aims to represent fast environmental events which can lead to abrupt changes in the concentration of pathogens and thus influence the spread of cholera.

Figure 28 shows the difference between the deterministic trajectory of the infected population $I(t)$ and the stochastic jump-diffusion trajectory of $I(t)$. The deterministic trajectory has a very small number of infected individuals at the start and a sharp increase following this. In the first portion of the simulation, the amount of the affected population starts to rise rapidly, indicating the beginning of an epidemic outbreak. Vulnerable individuals are susceptible to the disease when they come in contact with water contaminated with *Vibrio cholerae* bacteria, so the quick rise is associated with the environmental pathway of transmission.

The number of people infected is at its peak between Days 22 and 25, at about 345–350 infected people. This point is the maximum intensity of the simulated outbreak, given the baseline parameter values. The timing of the peak implies that the number of new infections that are occurring during this time is high enough to surpass the number of people who are dying from the infection or recovering. This leads to the number of affected people increasing until the peak of the epidemic.

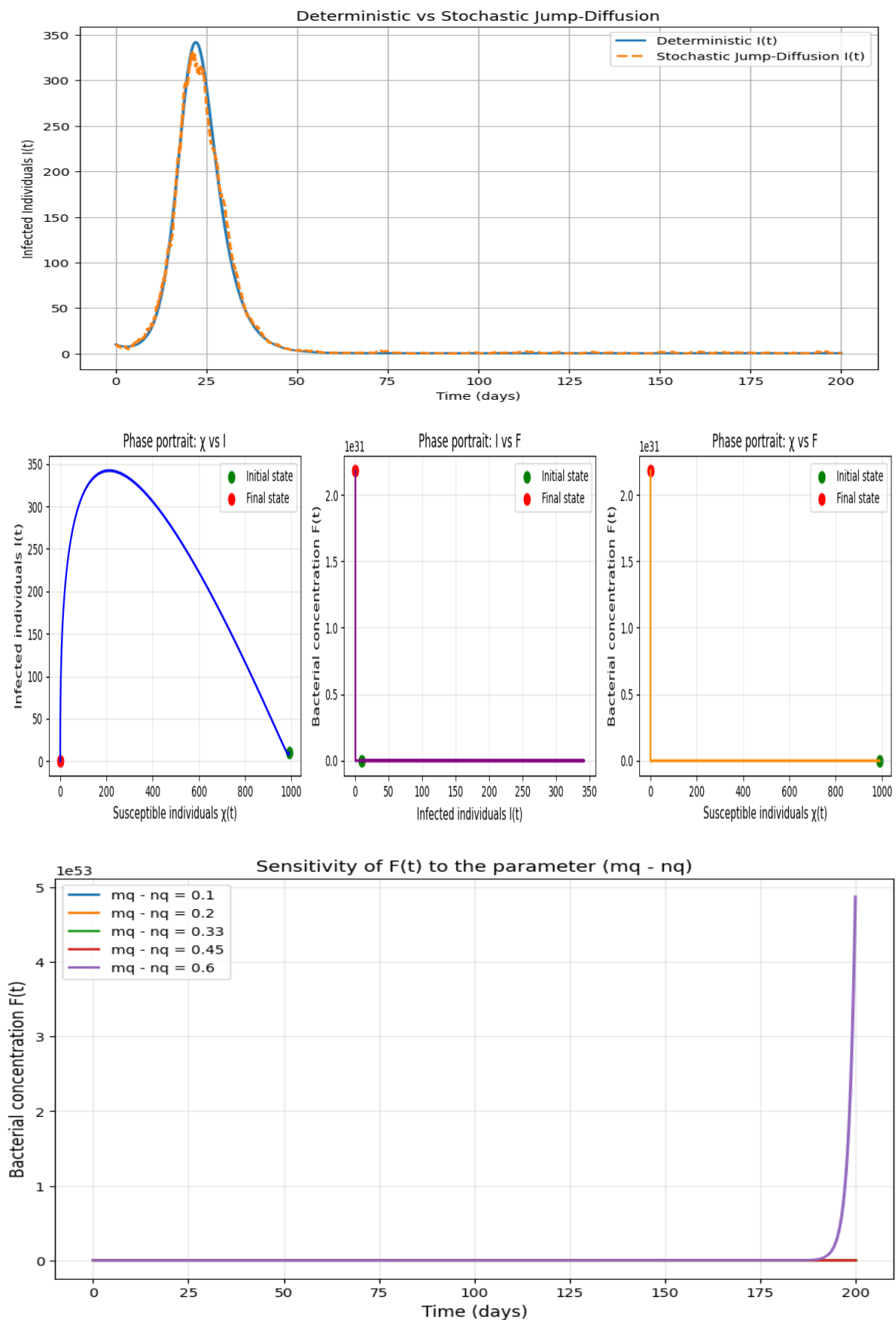
Once infected the population quickly decreases, this decrease is due to the fact that the number of susceptible individuals has decreased significantly in the course of the outbreak, while the infected ones are still recovering at the rate of r . Consequently, there are fewer new cases and epidemics cannot be maintained at high levels of infected persons as occurred during the epidemic. The simulation shows that the infected population drops off rapidly after days 50 and then remains in a low state for the next 200 days of the simulation.

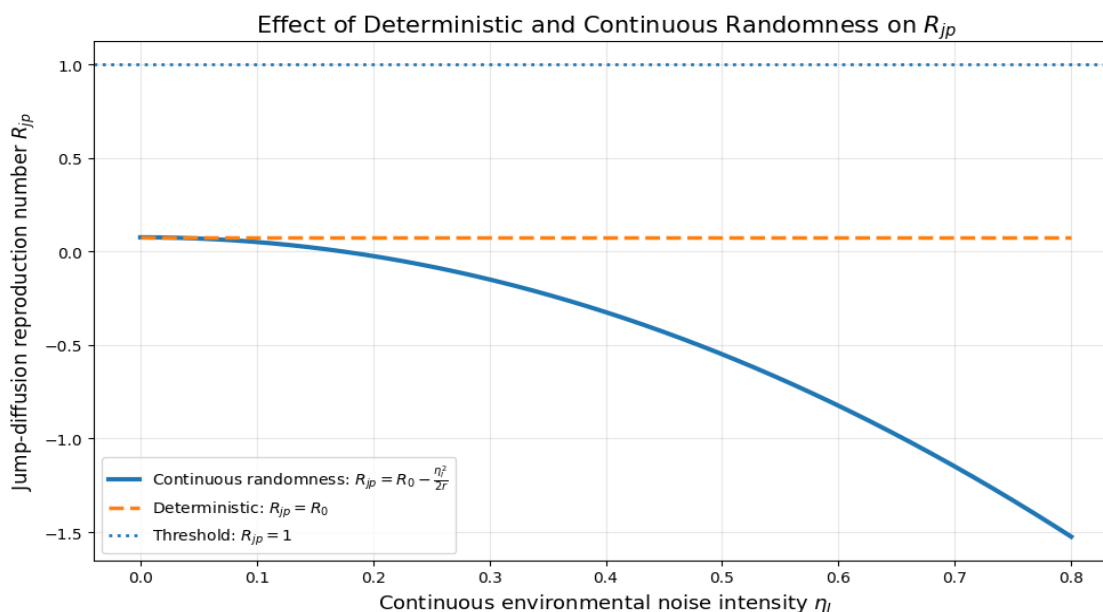
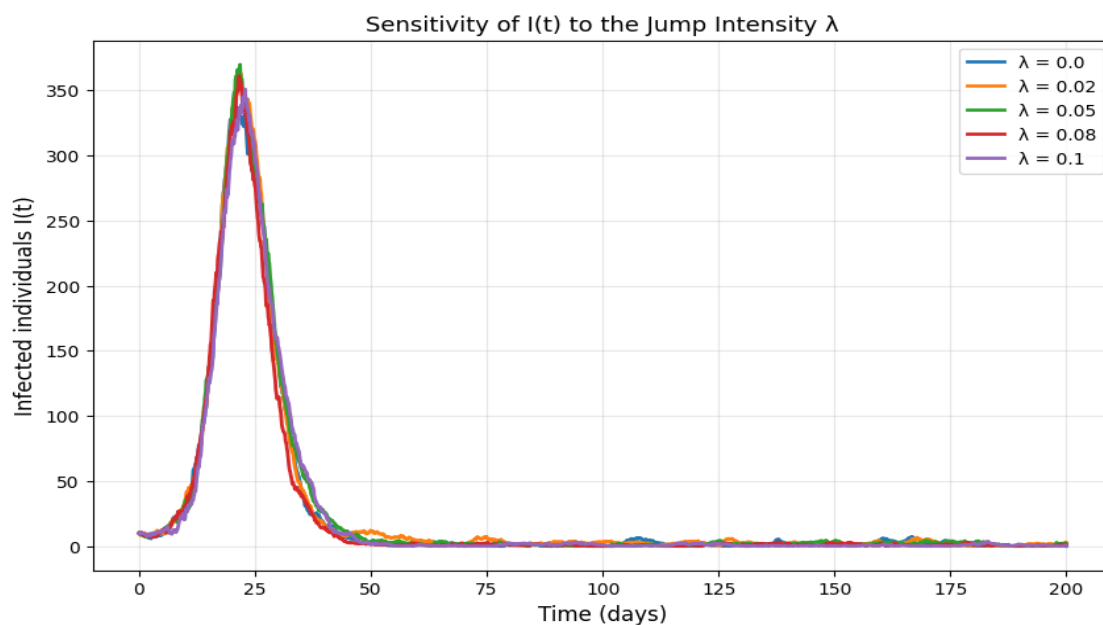
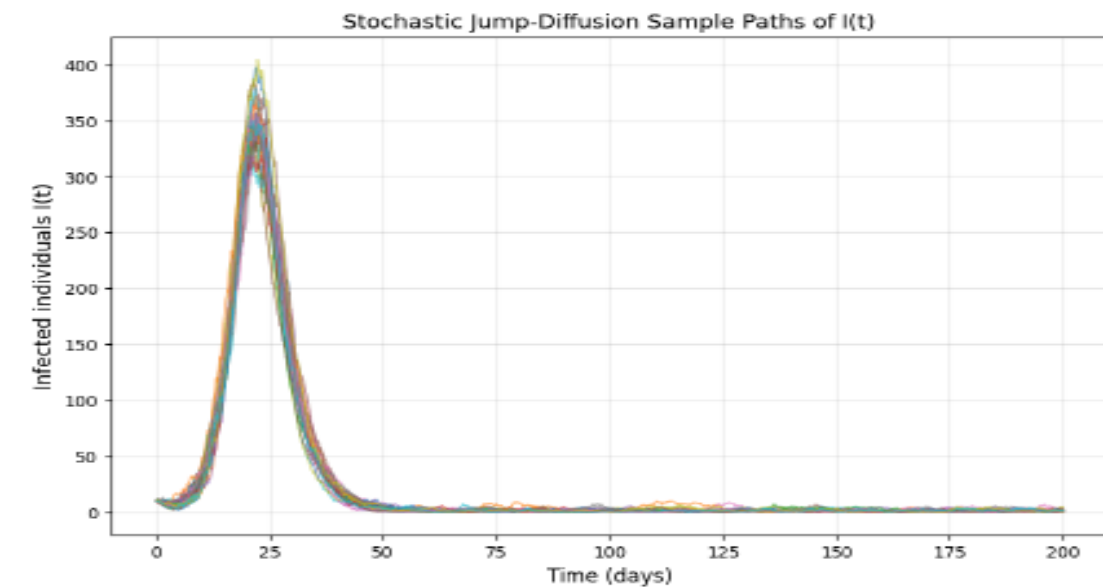
An important characteristic of the model is shown as a reduction in the number of infections following the peak of the epidemic. Susceptible individuals' interactions with a reservoir of environmental bacteria are a major determinant of cholera transmission, which is not only determined by the number of infected individuals. The infection pressure drops as the number of susceptible people is decreased and those who are infected recover. Therefore, the number of infected people can be reduced even though there may be bacteria in the environment.

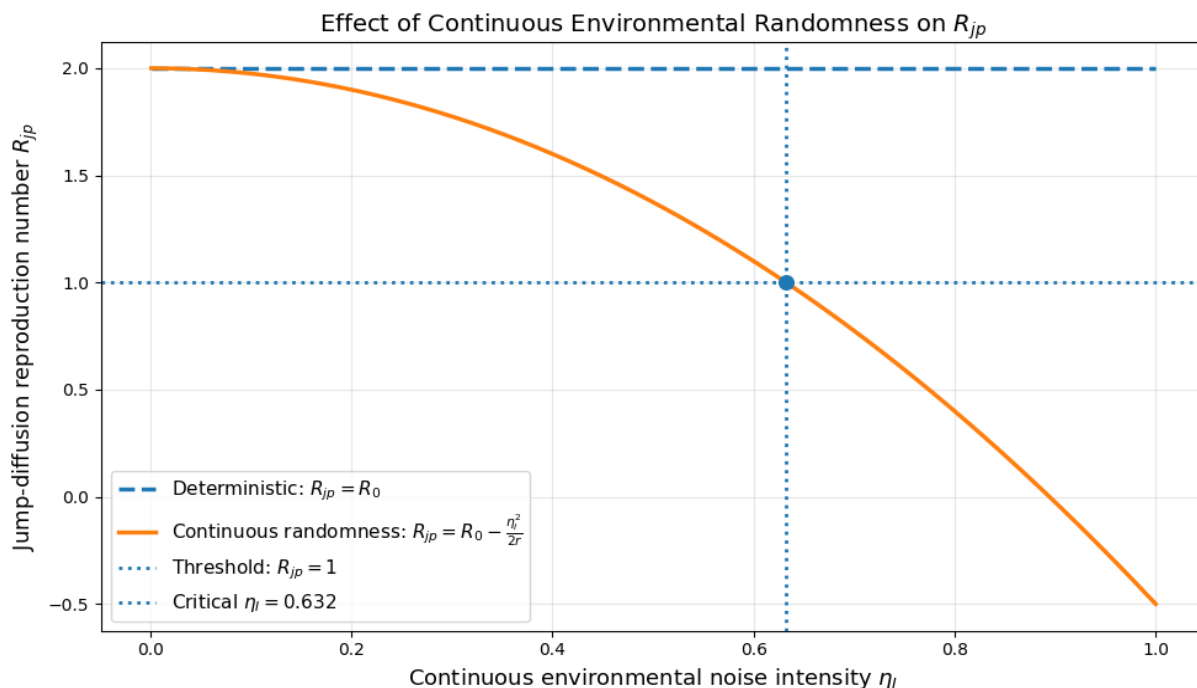
Comparison between Deterministic and Stochastic Solution

The stochastic jump diffusion trajectory shown below does not exactly coincide with the deterministic solution, but it does have the same general shape. On the contrary, the stochastic trajectory is surrounded by the deterministic track. These oscillations are due to environmental unpredictability integrated in the model by stochastic variables. This produces a more diverse epidemic trajectory than the deterministic model, so that the stochastic model produces a greater variety of epidemic trajectories than the deterministic model. One important result is that the basic nature of the epidemic is not completely modified by the inclusion of randomness. Both deterministic and stochastic trajectories show an initial rise in the number of infected individuals to a large epidemic peak followed by a fast decrease. The stochastic model provides more information on uncertainty in the primary epidemic pattern while the deterministic model does. The stochastic simulations show that there can be a range of infected people ranging between about 300 and 400 people on different stochastic sample routes. The peak is also slightly delayed in time. This is important from an epidemiological standpoint because it demonstrates the importance of environmental uncertainty on the size and timing of an outbreak of cholera. 2 outbreaks of equal average conditions can have different epidemic outcomes due to chance environmental variations.

However, environmental uncertainty is not necessarily unimportant for modelling cholera, as seen in the difference between the two paths: deterministic and stochastic. According to the deterministic model, environmental conditions change at predetermined average rates. The stochastic model, on the other hand, recognizes the possibility of unexpected contamination occurrences, and the varying levels of environmental variables. The stochastic model, therefore, does not give a single trajectory for the pandemic, but rather a variety of scenarios.







CONCLUSION

The standard $\chi I - F$ cholera model was extended to a stochastic jump diffusion model, to account for both single-time events of contamination and persistent environmental uncertainty. The deterministic model was shown to be well-posed from a mathematical and a physiological point of view, using positivity and invariant-region analysis. The stability of endemic and disease free equilibria studied by applying Routh-Hurwitz criterion and basic reproduction number R_0 .

The model was stochastic, allowing sudden environmental changes to be modeled by Poisson jump processes, and the continuous environmental changes by Brownian motion. The existence and uniqueness of the stochastic solution were proven under the right circumstances. The Euler Maruyama method for numerical simulation of the stochastic trajectories shows that they fluctuate around the deterministic trajectory, while the impact of the jump events causes short-term deviations without changing the overall shape of the epidemic, compared to the deterministic trajectory.

The numerical results show that the number of infected persons initially increased and was close to 345–350 individuals between Days 22 and 25, followed by a fast decline after Day 50. Differences in the size and timing of the outbreak were due to the different stochastic sample routes, reflecting the influence of environmental uncertainty on cholera transmission. By sensitivity analysis, the bacterial clearance rate $m_q - n_q$ and shedding rate e were determined to be important factors affecting the spread of the disease and outbreak severity. On balance, the proposed stochastic jump diffusion model provides a convenient and statistically tractable model for understanding the transmission of cholera in the context of the environmental uncertainty and sporadic contamination events. The results emphasize how crucial sanitation, water treatment, and strategies that lessen *Vibrio cholerae* persistence and environmental shedding are to containing cholera epidemics.

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