A STOCHASTIC POPULATION MODEL OF CHOLERA DISEASE

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Abstract. A cholera population model with stochastic transmission and with stochasticity on the environmental reservoir of the cholera bacteria is presented. It is shown that solutions are well-behaved. In comparison with the underlying deterministic model, the stochastic perturbation is shown to enhance stability of the disease-free equilibrium. The main extinction theorem is formulated in terms of an invariant which is a modification of the basic reproduction number of the underlying deterministic model. As an application, the model is calibrated as for a certain province of Nigeria. In particular, a recent outbreak (2019) in Nigeria is analysed and featured through simulations. Simulations include making forward projections in the form of confidence intervals. Also, the extinction theorem is illustrated through simulations.

1. **Introduction.** Cholera is a diarrheal infection caused by ingestion of food or water contaminated with the bacterium *Vibrio cholerae*, which affects both children and adults. It continues to be a serious burden for countries with limited access to safe drinking water and an inadequate sanitation system. It can be transmitted through direct faecal-oral contamination or via ingestion of contaminated water and/or food [28]. Between 12 hours and 5 days after the initial infection, severe acute watery diarrhoea sets in. Cholera at its most severe form, can lead to death by severe dehydration and kidney failure. It can kill within hours if untreated. The spread of the disease is mainly caused by contaminated water and insufficient sanitation, in generally poor environmental conditions. In particular, peri-urban

slums where basic infrastructure is not available, or camps for internally displaced people or refugees, where minimum requirements of clean water and sanitation are not met, are some of the highly at-risk areas [28]. It is globally estimated that from 3 to 5 million new cholera cases and about 150,000 cholera related deaths occur annually. In 2012, sub-Sahara African countries accounted for 71% of the total reported new cases and 86% of cholera related deaths [4].

The authors in [21] reported Nigeria to be one of the world's three major current cholera foci. Nigeria experienced her first series of cholera outbreaks between 1970 to 1990 and subsequent recurrent outbreaks has since then followed [11]. The northern parts of the country recorded over 40, 000 estimated new cases of cholera in 2010, which resulted in over 1,500 cholera related deaths and with case fatality rate (CFR) of 4.1%, which is well over the WHO acceptable rate of 1% [3, 29]. Cholera outbreaks in Nigeria has remained persistent since the beginning of 2018.

Mathematical modelling of epidemiological diseases is commonly used for making future projections of the prevalence of a disease in a population and to assess the effect of public health interventions. Many studies of this type have been carried out on the epidemiology of cholera disease, for example, [23, 22], and most of these studies are focussed on the deterministic modelling of cholera dynamics. In this paper, we will incorporate stochasticity into the cholera model presented in [23], in order to factor in the possible effects of randomness.

Stochasticity enters into natural systems in many ways. If the stochasticity is accommodated explicitly in a model, then it supports an analysis of its effects, whether positive or negative. In this paper we present a model of cholera disease dynamics in the form of a system of stochastic differential equations (SDEs). The two main results of this article are as follows. Firstly, we prove a theorem on extinction of the disease according to the stochastic model. Secondly, the underlying deterministic model is calibrated for a particular region and then the state of this population immediately subsequent to a recent cholera outbreak is computed. For both of these results we provide illustrating computations. SDE models of cholera dynamics have been studied in [20, 14, 13, 30]. In the SDE model of cholera in [20] there are computations, but no detail on positivity of solutions, or stability of the disease-free equilibrium. The model in [14] differs from ours essentially in that it has only one mode of infection, driven by the cholera reservoir, while excluding infection of susceptibles through their contact with infected humans. It does probe an extinction theorem (inter alia) but their theorem does not cover the main extinction theorem in the current paper. The other papers [13, 30] focus mostly on persistence, and does not have an extinction theorem similar to ours.

We briefly introduce an existing deterministic model, see [23], in Section 2. In Section 3 we present the stochastic model and we show that it has well-behaved solutions. The extinction theorem is proved in Section 4. In Section 5 we study the 2019 outbreak in Nigeria, focusing on the Adamawa State. We explain in detail how we estimate some of the population-specific parameters from the literature. Simulations are shown in Section 6 and concluding remarks appear in Section 7. The main contributions of this work are firstly the extinction result of Theorem 4.4, secondly the detailed parameter estimation in Section 5 and thirdly, the simulations in Section 6.

2. **A deterministic cholera model.** We briefly present the deterministic model (1) which is a special case of the model that was introduced and analysed in [23].

The model (1) will serve as the basis for our stochastic model that will be presented in Section 3. Our model sub-divides the total human population, denoted by N, into sub-populations of susceptible humans (S), infected humans (I) and recovered humans (R) , so that $N = S + I + R$, while the intensity of the bacteria population (or reservoir) in the environment will be denoted by B.

The recruitment rate is denoted by Λ, while the natural mortality rate is μ. We shall write Λ as $\Lambda = \mu P$, where P is the size of the total population when disease-free.

It is assumed that there are two possible ways in which a susceptible human can get infected. This can be either through infection as a result of interaction with an infectious person, or infection caused by the aquatic population B of $V.\ cholerae$. The parameter β_h is the effective contact rate between a given susceptible human and an infective human. Similarly, β_c is the (average) level of effective contact rate between a given susceptibe and the cholera reservoir. The parameter K is a constant which is the value of B at which the reservoir produce one new infection per week. The force of infection arising directly from interaction of a susceptible human with infected humans is $\beta_h I$. The parameter σ denotes the average rate of the contribution of each cholera infected individual to the aquatic population B of $V.\ cholerae$. The parameter α is the recovery rate, while ϵ is the cholera induced mortality rate. It is assumed that recovered humans have permanent immunity or at least, have immunity for a very long time. Here, ω is the rate at which $V.\ cholerae$ leaves the environment. The model is described by the following system of ordinary differential equations.

$$
\begin{aligned}
dS &= \left[\Lambda - \beta_c S \frac{B}{K + B} - \beta_h SI - \mu S\right] dt, \\
dI &= \left[\beta_c S \frac{B}{K + B} + \beta_h SI - (\alpha + \mu + \epsilon)I\right] dt, \\
dR &= \left[\alpha I - \mu R\right] dt, \\
dB &= \left[\sigma I - \omega B\right] dt.
\end{aligned}
\tag{1}
$$

An extensive analysis of this model can be found in the paper [23]. For our purposes, which focus on eradication of cholera in a given population, it is important to note that the basic reproduction number of this model is

$$
R_0 = \frac{\Lambda[\beta_c \sigma + K \omega \beta_h]}{\mu K \omega (\alpha + \mu + \epsilon)}.
$$

3. **A stochastic cholera model.** Randomness is present in a variety of real life situations and phenomena. It is therefore helpful when building models, such as disease models, to allow for the effect of stochasticity, [9]. When parametrizing a model, it is possible that there could be errors, even if very small. The inclusion of stochastic perturbations on an ODE model, informs the users of the model of how the dynamics of the system in point can possibly deviate from the expected outcome, as also mentioned in [6]. In this model we apply stochastic perturbations on the rate of transmission of cholera from infected humans to susceptible humans, in a complementary couple, and also we perturb the rate of removal of cholera from the reservoir.

We assume a pair of mutually independent standard Brownian motions $\{W(t)\}_{t>0}$ and $\{Y(t)\}_{t>0}$, defined on a complete filtered probability space, satisfying the usual conditions. The model is described by the following system of SDEs. This system can be seen to be a modification of the model (1), by introduction of stochastic perturbations. The constant, ξ, is the intensity of the perturbation on β_h, and ζ is the intensity of the pertubation on ω.

$$
\begin{aligned}
dS &= \left[\Lambda - \beta_c S \frac{B}{K+B} - \beta_h SI - \mu S\right] dt - \xi SI dW, \\
dI &= \left[\beta_c S \frac{B}{K+B} + \beta_h SI - (\alpha + \mu + \epsilon)I\right] dt + \xi SI dW, \\
dR &= \left[\alpha I - \mu R\right] dt, \\
dB &= \left[\sigma I - \omega B\right] dt + \zeta B dY.
\end{aligned}
\tag{2}
$$

Note, we shall often use the notation:

$$
\mu_1 = \mu + \alpha + \epsilon.
\tag{3}
$$

We prove below, that this system has solutions that exist globally and are positive almost surely (a.s.). The method that we use is commonly applied for such purposes, see for instance [6, 10, 25].
Let
   $\Delta = \{x \in \mathbb{R}^4 : x_i > 0 \;\; \forall\, i, \text{ and } x_1 + x_2 + x_3 < \frac{\Lambda}{\mu}\}$.
Similarly as for instance in [24], we prove the following proposition.

Proposition 3.1. *Suppose that for some T, there is a local solution*

$$
X(t,\omega) = (S(t,\omega), I(t,\omega), R(t,\omega), B(t,\omega)) \;\; \text{on} \;\; t \in [0, T)
$$

for the system, with $X(t,\omega) \in \mathbb{R}_+^4$ for each $t \in [0,T)$, $\omega \in \Omega$. If $N(0,\omega) \leq \Lambda/\mu$, then $N(t,\omega) \leq \Lambda/\mu$ for each $t \in [0,T)$.

Proof. Given any such local solution $X(t,\omega)$, then

$$
\frac{d(N(t,\omega) - \Lambda/\mu)}{dt} = \Lambda - \mu S(t,\omega) - (\mu + \epsilon)I(t,\omega) = -\mu(N(t,\omega) - \Lambda/\mu) - \epsilon I(t,\omega).
$$

Consequently,

$$
\frac{d(N(t,\omega) - \frac{\Lambda}{\mu})}{dt} + \mu(N(t,\omega) - \frac{\Lambda}{\mu}) = -\epsilon I(t,\omega) \leq 0.
$$

Thus we have an ordinary differential equation which is linear and of first order. For this equation we have that if $N(0,\omega) < K$, then $N(t,\omega) < K$ for all $t \in [0,T)$. $\square$

Theorem 3.2. *Given any point $x_0 \in \Delta$, then there is a solution $X(t)$ of the system (2) with $X(0) = x_0$ such that $X(t)$ is global (a.s.) and $X(t) \in \Delta$ for all $t > 0$ (a.s.).*

Proof. The so-called Lyapunov method of proof by contradiction is popularly used for results such as the current theorem, see for instance [25]. We follow the same

methodology, without giving the complete argument. The coefficients of the system (2) are locally Lipschitz continuous. Consequently there exists a unique local solution $X(t)$, with $X(0) = x_0$. Since $x_0 \in \Delta$, at least for some $t_1 > 0$, we have $\{X(t)|\ 0 \le t < t_1\} \subset \Delta$. Let τ_1 be the explosion time of the local solution $X(t)$. For such a solution $X(t)$ that starts in Δ, we define the function

$$J(t) = \ln \frac{\Lambda}{\mu S(t)} + \ln \frac{\Lambda}{\mu I(t)} + \ln \frac{\Lambda}{\mu R(t)} + \Big[B(t) - \ln B(t)\Big].$$

We note that as a consequence of Proposition 3.1, if each of the first three terms on the right hand side are positive while $X(t) \in \Delta$. Also,

$$\lim_{x \to 0^+} \frac{\Lambda}{\mu x} = \infty.$$

We furthermore note that $x - \ln x > 0$ for $x > 0$ and that

$$\lim_{x \to 0^+} (x - \ln x) = \infty \quad \text{and} \quad \lim_{x \to \infty} (x - \ln x) = \infty\ .$$

Therefore the function $J(t)$ is a good candidate to furnish a proof using the Lyapunov method. Let τ_1 be the explosion time of $J(t)$. Then

$$0 < \tau_1 \le \tau_0.$$

In order to complete this proof it suffices to prove that $\tau_1 = \infty$, and this is what we shall prove.

We calculate the differential of $J(t)$.

$$dJ(t) = \mathcal{L}J(t)dt + \Big[-\frac{1}{S(t)}\Big(-\xi S(t)I(t)dW\Big)\Big]$$
$$-\frac{1}{I(t)}\Big[\xi S(t)I(t)dW\Big] + \Big(1 - \frac{1}{B(t)}\Big)\Big[\zeta B dY\Big].$$

By the martingale property it follows that

$$\mathbb{E}\Big[\int_0^t \Big(\frac{1}{S(z)} - \frac{1}{I(z)}\Big)\xi S(z)I(t)dW(z)\Big] = 0 \text{ (a.s.)},$$

and

$$\mathbb{E}\Big[\int_0^t \Big(1 - \frac{1}{B(z)}\Big)\zeta B(z)dY(z)\Big] = 0 \text{ (a.s.)}.$$

Therefore

$$\mathbb{E}\Big[J(t)\Big] = \mathbb{E}\Big[\int_0^t \mathcal{L}J(z)dz\Big] \text{ (a.s.)}.$$

Now

$$\mathcal{L}J(t) = -\frac{1}{S(t)}\Big[\Lambda - \beta_c S(t)\frac{B}{K+B} - \beta_h S(t)I(t) - \mu S(t) + \eta R(t)\Big]$$
$$-\frac{1}{I(t)}\Big[(\beta_c S(t)\frac{B}{K+B} + \beta_h S(t)I(t) - (\alpha + \mu + \epsilon)I(t)\Big]$$
$$-\frac{1}{R(t)}\Big[\alpha I(t) - (\eta + \mu)R(t)\Big] + \Big(1 - \frac{1}{B(t)}\Big)\Big[\sigma I(t) - \omega B(t)\Big]$$
$$+\frac{1}{2S^2(t)}\Big[(\xi S(t)I(t))^2\Big] + \frac{1}{2I^2(t)}\Big[(\xi S(t)I(t))^2\Big] + \frac{1}{2B^2(t)}\Big[(\zeta B(t))^2\Big]$$

and so,

$$
\begin{aligned}
\mathcal{L}J \;\leq\; & \frac{1}{S}\left[\frac{\beta_c BS}{K+B}+\beta_h SI\right]+\frac{1}{I}(\mu_1 I)+\frac{1}{R}\left[(\eta+\mu)R\right]+\sigma I+\frac{1}{B}\left[\omega B\right] \\
& +\frac{\xi^2}{2}(I^2+S^2)+\frac{\zeta^2}{2} \\
\leq\; & \beta_c+\beta_h I-\mu_1+(\eta+\mu)+\sigma I+\omega+\frac{\xi^2}{2}\left(I^2+S^2\right)+\zeta^2/2 \\
\leq\; & M,
\end{aligned}
$$

where $M=\beta_c+\beta_h\frac{\Lambda}{\mu}+\mu_1+(\eta+\mu)+\sigma\frac{\Lambda}{\mu}+\omega+\xi^2\left(\frac{\Lambda^2}{\mu^2}\right)+\zeta^2/2$. Therefore the Lyapunov argument will produce a proof of Theorem 3.2. $\square$

Remark 3.3. In the analysis we shall encounter the following function (given constants $g>0$ and non-negative ζ,ξ) for a variable x.

$$
h(S,x)=\xi^2 S^2 x+\frac{\zeta^2}{g^2}\frac{(1-x)^2}{x},\quad 0<x\leq 1.
$$

Then we take the partial derivative of h with respect to x and note that it vanishes when

$$
x=\frac{\zeta}{g}\left[\left(\frac{\zeta}{g}\right)^2+S^2\xi^2\right]^{-\frac{1}{2}}=:x^*.
$$

Hence we conclude that $h(S,x)\geq h(S,x^*)$ for all $0<x\leq 1$. From this we can derive the inequality:

$$
h(S,x)\geq S^2 k\quad\text{with}\quad k=\xi^2\frac{\zeta}{g}\left[\left(\frac{\zeta}{g}\right)^2+\left(\frac{\Lambda\xi}{\mu}\right)^2\right]^{-\frac{1}{2}}. \tag{4}
$$

4. **Stability of the disease-free equilibrium. 4.1 Notation**. For a stochastic process $\{x(t)\}$ we write

$$
\langle x\rangle_t=\frac{1}{t}\int_0^t x(z)dz.
$$

For a positive number g, we define the stochastic processes $\{u(t)\}$ and $\{v(t)\}$ as follows:

$$
u(t)=I(t)+gB(t),
$$
$$
\text{and when } u(t)>0,\ \text{ we let } v(t)=\ln u(t). \tag{5}
$$

In particular, we are interested in the Lyapunov exponent Γ of u, assuming $u(t)>0$ for all $t>0$, which is defined as

$$
\Gamma=\limsup_{t\to\infty}\frac{1}{t}\ln u(t). \tag{6}
$$

We calculate $\mathcal{L}v(t)$.

$$
\mathcal{L}v=\frac{1}{u}\left[\beta_c S\frac{B}{K+B}+\beta_h SI-(\alpha+\mu+\epsilon)I+g(\sigma I-\omega B)\right]-\frac{1}{2u^2}\left[(\xi SI)^2+g^2\zeta^2 B^2\right]
$$

Then we can write

$$
\mathcal{L}v=A_1\frac{I}{u}+A_2\frac{B}{u}-E, \tag{7}
$$

where

$A_1 = \beta_h S - \mu_1 + g\sigma$ [with $\mu_1 = \mu + \alpha + \epsilon$, recall equation (3)],

$A_2 = \beta_c \frac{S}{K+B} - g\omega$,

and

$E = \frac{1}{2u^2}[(\xi S I)^2 + g^2\zeta^2 B^2]$.

Proposition 4.2. *For u and v as above,*

$$\limsup_{t\to\infty} \left(\frac{1}{t}v(t)\right) = \limsup_{t\to\infty}\langle\mathcal{L}v\rangle_t \quad (a.s.).$$

Proof. By Itô's lemma

$$v(t) = v(0) + \int_0^t \mathcal{L}v(z)dz + \int_0^t \frac{1}{u(z)}\xi S(z)I(z)dW(z) + \int_0^t \frac{g}{u(z)}\zeta B(z)dY(z).$$

Now we note that

$$\left(\frac{\xi S I}{u}\right)^2 \le \left(\frac{\xi S I}{I}\right)^2 = (\xi S)^2 \le \frac{\xi \Lambda^2}{\mu^2} \text{ and } \left(\frac{\zeta g B}{u}\right)^2 \le \zeta^2.$$

Thus by the strong law of large numbers, see [5] for instance, it follows that

$$\limsup_{t\to\infty} \frac{1}{t}\int_0^t \frac{1}{u(z)}\xi S(z)I(z)dW(z) = 0 \quad (a.s.).$$

and

$$\limsup_{t\to\infty} \frac{1}{t}\int_0^t \frac{g}{u(z)}\zeta B(z)dY(z) = 0 \quad (a.s.).$$

Also

$$\lim_{t\to\infty} \frac{v(0)}{t} = 0.$$

This concludes the proof. $\square$

4.3 Notation. Similarly as in [24] for instance, we can find a special sequence (t_n) which is an increasing unbounded sequence of positive numbers such that

$$\Gamma = \lim_{t_n\to\infty} \frac{1}{t_n} \ln u(t_n),$$

and the following limits exist:

$$i := \lim_{n\to\infty} \left\langle\frac{I}{u}\right\rangle_{t_n}; \qquad b := \lim_{n\to\infty} \left\langle\frac{B}{u}\right\rangle_{t_n}.$$

The identity below, follows immediately:

$$i + gb = 1. \tag{8}$$

We introduce the following invariant of the model (2), related to the basic reproduction number of the underlying deterministic model:

$$R_* = R_0 \times \frac{\mu_1}{\mu_1 + \frac{k\Lambda^2}{2\mu^2}} = \frac{\Lambda[\beta_c\sigma + \omega\beta_h K]}{\mu\omega K\left(\mu_1 + \frac{k\Lambda^2}{2\mu^2}\right)}. \tag{9}$$

We note that $R_* \le R_0$. Stochastic stability analysis described in terms of an invariant which is smaller than the basic reproduction number of the disease-free equilibrium can be observed in, for instance, [6, 10, 24, 8]. In our stability theorem we utilize R_*.

Theorem 4.4. *If $R_* < 1$ and $\Lambda/\mu \le \beta_h/k$, then:*

(a) (I, B) *converges exponentially to 0 almost surely,*

(b) $\lim_{t \to \infty} S(t) = \Lambda/\mu$ *almost surely.*

Proof. **(a)** The inequality $R_* < 1$ implies that

$$\Lambda[\beta_c \sigma + \beta_h \omega K] - \mu \omega K \left(\mu_1 + \frac{k\Lambda^2}{2\mu^2} \right) < 0. \tag{10}$$

We can find z such that $0 < z < 1$ and

$$\Lambda[\beta_c \sigma + \omega \beta_h K] - (1 - z)\mu \omega K \left(\mu_1 + \frac{k\Lambda^2}{2\mu^2} \right) < 0. \tag{11}$$

Let us fix the following constant, g:

$$g = \frac{\beta_c \Lambda}{(1 - z)\mu \omega K}.$$

Now we introduce $u = I + gB$ and $v = \ln u$ as from Notation 4.1. By Proposition 4.2 it suffices to prove that

$$\limsup_{t \to \infty} \left[\frac{1}{t} \int_0^t \mathcal{L}v(\tau)d\tau \right] < 0 \quad \text{(a.s.), i.e., } \Gamma < 0 \text{ (a.s.)}.$$

We turn to equation (7). Note that $u = I + gB$, and in particular, $\frac{B}{u} = \frac{1}{g}\left(1 - \frac{I}{u}\right)$. So if we write $y = \frac{I}{u}$, then E becomes:

$$E = \frac{1}{2}\left[\xi^2 S^2 y^2 + \frac{\zeta^2}{g^2}(1 - y)^2 \right] = \frac{1}{2}h(S,y)y \geq \frac{k}{2}S^2 \frac{I}{u}.$$

Thus we have

$$\begin{aligned}
\mathcal{L}v &= A_1 \frac{I}{u} + A_2 \frac{B}{u} - E \text{ (a.s.)} \\
&= \left[\beta_h S + g\sigma - \mu_1 \right]\frac{I}{u} + \left[\beta_c \frac{S}{K + B} - g\omega \right]\frac{B}{u} - E \\
&\leq \left[\beta_h S - \frac{1}{2}kS^2 + g\sigma - \mu_1 \right]\frac{I}{u} + \left[\beta_c \frac{S}{K + B} - g\omega \right]\frac{B}{u}.
\end{aligned}$$

Note that the quadratic expression $\beta_h x - \frac{1}{2}kx^2$ represents an increasing function on the interval $0 \leq x \leq \beta/k$. Since $0 \leq S \leq \Lambda/\mu$, and (by assumption) $\Lambda/\mu \leq \beta/k$, the following inequality holds,

$$\beta_h S - \frac{1}{2}kS^2 \leq \beta_h \frac{\Lambda}{\mu} - \frac{1}{2}k\frac{\Lambda^2}{\mu^2}.$$

Now we find that $\mathcal{L}v$ satisfies the inequality

$$\mathcal{L}v \leq D_1 \frac{I}{u} + D_2 \frac{B}{u} \text{ (a.s.)},$$

with

$$D_1 = \beta_h \frac{\Lambda}{\mu} + \frac{\beta_c \Lambda \sigma}{(1 - z)\mu \omega K} - \mu_1 - \frac{k\Lambda^2}{2\mu^2}.$$

and

$$D_2 = \beta_c \frac{S}{K + B} - g\omega.$$

Note that

$$D_1 \leq G_0 G_1,$$

where

$$G_0 = \frac{1}{(1-z)\mu\omega K} \quad \text{and} \quad G_1 = \Lambda[\beta_c\sigma + \omega\beta_h K] - (1-z)\mu\omega K\left(\mu_1 + \frac{k\Lambda^2}{2\mu^2}\right).$$

In view of equation (11) we have $G_1 < 0$ and hence $D_1 < 0$. We further note that,

$$D_2 \leq \beta_c\frac{\Lambda}{\mu K} - g\omega = -\frac{\beta_c\Lambda}{\mu K}\frac{z}{1-z} < 0.$$

Therefore,

$$\Gamma \leq D_1 i + D_2 b.$$

From equation (8) we know that $i + gb = 1$, and therefore we cannot have both of i and b to be zero. Thus we can conclude that $\Gamma < 0$ (a.s.). This concludes the proof of Theorem 4.4(a).

(b) Let us write $S(t) + I(t) = Z(t)$. Then the stochastic process $Z(t,\omega)$ happens to be differentiable and we have:

$$\frac{dZ(t)}{dt} = \Lambda - \mu Z(t) - (\alpha + \epsilon)I(t). \tag{12}$$

We consider two cases, Case (i) and its complement, Case (ii). Case (i) covers the sample paths for which $Z(t)$ is eventually a monotone function, i.e., eventually either non-decreasing or eventually non-increasing.

Case (i): Let us assume that there exists a finite stopping time $\tau > 0$ such that on the interval $[\tau, \infty)$, $Z(t)$ is monotone. Then since $Z(t)$ is bounded above (by Λ/μ) (a.s.) and below by 0 (a.s.), the function $Z(t)$ has a limit Z^*, and $\lim_{t\to\infty} Z'(t) = 0$ (a.s.). Since by Theorem 4.4(a) we have $\lim_{t\to\infty} I(t) = 0$ (a.s.), from equation (12) it follows that $\lim_{t\to\infty} S(t) = \Lambda/\mu$ (a.s.).

Case (ii): This time we consider the complementary case to Case (i). Let $m_0 = \liminf_{t\to\infty} Z(t)$ and let $m_1 = \frac{1}{2}\left(m_0 + \frac{\Lambda}{\mu}\right)$. We note that m_0 is stochastic, and therefore so is m_1. Firstly, (a.s.) there exists an unbounded sequence $(\tau_n)_{n=1}^{\infty}$ of positive numbers τ_n which are such that $I(t) < \frac{1}{n}$ whenever $t \geq \tau_n$. Secondly, there exists a sequence $(t_n)_{n=1}^{\infty}$ of positive numbers such that for every $n \in \mathbb{N}$:

$Z'(t_n) < 0, \ t_n \geq \tau_n$ and $Z(t_n) \leq m_1$ (a.s.).

Now since $\frac{dZ}{dt}\big|_{t_n} < 0$ and $\frac{dZ(t)}{dt}$ is continuous, there exist a number $q_n > 0$ such that $\frac{dZ(t)}{dt}$ is negative over the interval $E_n = [t_n, t_n + q_n]$, (a.s.). Next, there exists $t_n^* \in E_n$, such that

$$\int_{E_n} (\Lambda - \mu Z)dt = [\Lambda - \mu Z(t_n^*)]q_n \ \ (\text{a.s.}).$$

In particular then, $Z(t_n^*) \leq m_1$ and consequently

$$\int_{E_n} (\Lambda - \mu Z(t))dt \geq (\Lambda - \mu m_1)q_n \ \ (\text{a.s.}).$$

Now for every $n \in \mathbb{N}$ we have:

$$
\begin{aligned}
0 \quad &> \quad Z(t_n + q_n) - Z(t_n) \\
&= \quad \int_{E_n} Z(t)dt \\
&= \quad \int_{E_n} (\Lambda - \mu Z(t))dt - \int_{E_n} (\alpha + \epsilon)I(t)dt \\
&\geq \quad (\Lambda - \mu m_1)q_n - (\alpha + \epsilon)\frac{1}{n}q_n \quad \text{(a.s.).}
\end{aligned}
$$

Therefore, (a.s.) $\Lambda - \mu m_1 \leq \frac{\alpha + \epsilon}{n}$ for all $n \in \mathbb{N}$. Consequently $\Lambda - \mu m_1 \leq 0$, i.e., $m_1 \geq \frac{\Lambda}{\mu}$ (a.s.). Since $\lim_{n\to\infty} I(t) = 0$ (a.s.), again it follows that $\lim_{n\to\infty} S(t) = \dfrac{\Lambda}{\mu}$ (a.s.). $\square$

The theorem shows that the stochastic perturbation allows for improved stability of the disease-free equilibrium, beyond the range $R_0 < 1$. In particular, the disease-free equilibrium is stable (a.s.) when $R_0 = 1$.

5. **Calibration of the deterministic model.** As an application in this paper, we consider the state of Adamawa in Nigeria, and we focus on the 2019 cholera outbreak. However, where information on Adamawa itself is not readily available, we shall approximate by using the relevant Nigeria information.

Life expectancy in Nigeria during 2015 was 54.49, see Nigeria Life Expectancy 1950-2020 [17]. Then we take μ to be the reciprocal of the life expectancy, and so we have $\mu = 0.0003548$ per week. From [2] we have the population size for Adamawa State in 2019 to be 4 890 000, and this we take as the value of P. We assign to Λ the value $\Lambda = \mu \times P$.

We obtain a value $\epsilon = 0.0149$ per day from the case fatality rate, CFR=836/43996, in the Adamawa cholera outbreak of 2019 [16], while we note that the duration of the infection is taken as 1 week. Duration 1 week implies $\alpha = 1$. From [22] we obtain $\omega = 1.06 - 0.73 = 0.33$ per day. The parameter ω can be taken as $\omega = 0.33$ per day, following [7] (see also [12], [15]).

Remark 5.1. The units in which we measure B, determines the units of K. When the value of K is known, then the units in which B (and consequently also K) is measured, can be changed so as to have the numerical value of K being unity. Thus in our application, with the detail of the units of B not being known and not quite directly relevant, we find it appropriate to omit units and choose $K = 1$. A similar approach has been followed in [27].

We use data on Nigeria cholera statistics to calculate equilibrium values for the human compartments. From [1], we can calculate the average number C of cases per week. On the graph of annual cases, for years 1990 up to 2013 we regard the counts higher than 15000 as major outbreaks, and we do not include them towards calculating the equilibrium number of cases. For the remaining 19 years it gives an annual average of 4395 cases, and thus an average of 84 cases per week over the period 1990 -2013 for Nigeria. We downscale this to Adamawa, and adjust to allow for population growth to get an average number C of weekly infections for Adamawa, at $C = 2$ (per week). Let D be the average number of cholera fatalities per week. At this point we can calculate numerical endemic equilibrium values for

the classes S, I and R by using the equations:

$$D = \epsilon C/\alpha, \quad I^* = D/\epsilon, \quad \mu R^* = \alpha I^* \quad \text{and} \quad S^* + I^* + R^* = P - \epsilon I^*/\mu.$$

In our simulations we do not specify the exact units in which B is measured. The understanding is that a change of units amounts to a (constant) proportionality. The exact value of such a constant is, for our purposes, immaterial. Thus we assume B to be measured in an undisclosed unit. We need to make an assumption about how the reservoir contributes to new infections. Let us assume that when at endemic equilibrium state, then the reservoir produces 50% of the new infections. That is,

$$\beta_c S^* B^* \frac{1}{K + B^*} = C/2.$$

In terms of the units of B, we can obtain a good estimate of β_c as: $\beta_c = 2K/KP = 2/P$. Then B^* can be calculated from the identity $\beta_c S^* \frac{B^*}{K+B^*} = C/2$. We obtain $\beta_c = 4.095 \times 10^{-7}$ per unit of B per week. Since, quite coincidentally, $C/2 = 1$, it follows that $B^* = 1$. Now we can calculate numerical values for the endemic equilibrium state.

$$S^* = 4884200, \ I^* = 2, \ R^* = 5735, \ B^* = 1.$$

Finally we can calculate the remaining parameters. From the equation $\beta_h S^* I^* = C/2$ we obtain $\beta_h = 1.024 \times 10^{-7}$ per week. Also, $\sigma = \omega B^*/I^* = 1.155$. The basic reproduction number is $R_0 = 1.479$.

Table 1. Numerical values of model parameters for Adamawa State, Nigeria, 2019.

Param.	Description	Numerical value	Reference/ comment
P	Population size when disease-free	4 890 000	[18],[19]
μ	mortality rate, excluding death directly due to cholera	0.0003548 per week	[2]
Λ	rate of inflow	1.735 per week	$\Lambda = \mu P$
ϵ	rate of human deaths due to cholera	0.0149 per week	[16]
α	Transfer rate from I-class to R-class (recovery rate)	1 per week	[22]
ω	Removal rate of pathogen from the environment	0.33×7 per week	[7] [12] [15]
β_c	a contact rate	4.095×10^{-7} per week	Fitted
β_h	a contact rate	1.024×10^{-7} per week per unit of B	Fitted
K	A threshold value of B	1	Remark 5.1
σ	rate of increase of the levels of the pathogen	1.155 per week	Fitted.

6. **Simulations.** We show the results of three sets of simulations on the model as calibrated for Adamawa Province of Nigeria. Time on the horizontal axis is in weeks. The symbol t_0 will denote the time beginning of the 2019 cholera outbreak, which is the start of the 19th week of the calender year 2019. Note that when

simulating the stochastic model, the intensities of the perturbations will always be taken as

$$\xi = 1/4890000 \ \text{ and } \ \zeta = 1/10 \ .$$

6.1. For the outbreak which is documented in the Situation Report prepared by the Nigeria Centre for Disease Control (NCDC) [16], one can determine the epidemiological state of the population at the end of the outbreak period, by utilising the model in an indirect way. A similar computation was done for a listeriosis outbreak [27]. At the beginning of the outbreak we assume the population to be at the endemic equilibrium state.

We assume that $X(t_0) = X^*$. Let t_2 be a date which is 26 weeks after t_0, So that $[t_0, t_2]$ represents the outbreak interval over Adamawa under consideration. Then we compute the evolution of the system over the time interval $[t_0, t_2]$ by inputting the case data into the model. The relevant parameter values are as in Table 1. The results are shown in Figure 1. The terminal values of the state variables, $X(t_2)$ are as follows

$$S(t_2) = 4889200, \ I(t_2) = 4.8, \ R(t_2) = 6481, \ B(t_2) = 2.9186.$$

From the graph we observe that I reaches a maximal value at time $t_1 = 13$. The state of the system at time t_1 is computed as

$$S(t_1) = 4889500, \ I(t_1) = 69.7, \ R(t_1) = 6168, \ B(t_1) = 3.306.$$

6.2. We show how the system evolves back in the direction of equilibrium. For the stochastic model we ran 999 sample paths. For each t in the interval $[t_1, t_2]$, we calculate the mean value $I_{\mathrm{mn}}(t)$ of the 999 $I(t)$-values and the mean $B_{\mathrm{mn}}(t)$ of the 999 $B(t)$-values. We also determine the 10'th and 90'th percentiles, respectively $I_{\mathrm{ten}}(t)$ and $I_{\mathrm{nty}}(t)$. In this case the $I_{\mathrm{mn}}(t)$ values are very close to the deterministic model's $I(t)$ values and we omit the deterministic graphs. Figure 2 shows $I_{\mathrm{mn}}(t)$ and $B_{\mathrm{mn}}(t)$ over the interval $[13, 43]$. For this simulation the system is assumed to evolve on its own, without external interference. Simulations similar to Figure 2 and Figure 3 appear in the paper [26]. In Figure 3 we have $I_{\mathrm{mn}}(t)$, $I_{\mathrm{ten}}(t)$ and $I_{\mathrm{nty}}(t)$ over an interval of length 20 weeks, starting at the date t_2.

6.3. We illustrate that for the stochastic model, the disease-free equilibrium can be (almost surely exponentially) stable even in cases when the basic reproduction number of the underlying deterministic model exceeds unity. Consider the parameterization as in Table 1 except that we reduce the values of β_c and β_h by a factor 1.45 . This yields $R_0 = 1.020$. Now taking

$$\xi = 1.25/4890000 \text{ and } \zeta = 0.125$$

we obtain

$$R_* = 0.905.$$

This means that for the deterministic model, the disease-free equilibrium will be unstable, but by Theorem 4.4, for the stochastic model the disease-free equilibrium will be almost surely exponentially stable. This is indeed a welcome observation, the fact that the stochasticity enhances extinction of cholera. In order to make this

phenomenon more visible, we run a simulation in which we use parameter values as in Table 1 except that we reduce the values of each of β_c and β_h by a factor 1.45, see Figure 4. Here, we notice that for the deterministic model, the I-values seem to converge to a positive endemic value, while even the 90th percentile (as well as the mean) of the stochastic $I(t)$ over 1000 runs, seem to converge to zero.

7. **Conclusion.** A fairly simple, yet versatile deterministic model of cholera population dynamics was modified by imposing stochastic perturbation on it. The solutions were shown to be positive and suitably bounded. An extinction theorem for the pathogen in the population was proved in terms of an analogue of the basic reproduction number. We presented the 2019 cholera outbreak in the Adamawa Province of Nigeria as a case study. We showed that extinction was easier to obtain in the presence of the stochastic perturbations in comparison with the underlying deterministic model. The model was calibrated through methods that are rather novel (as was also done in [27]). The extinction results and the demonstrated phenomena and processes, such as working through the outbreak period like we did in the computation 6.1, can readily be applied in real life situations. Future investigations in this regard may be devoted to the study of interventions such as vaccination, and optimal control of a stochastic model.

Data availability. No new data was generated during this research. Existing data that was used are freely accessible and clearly indicated in the references.

Conflict of interest. The authors declare that there is no conflict of interest associated with this research paper.
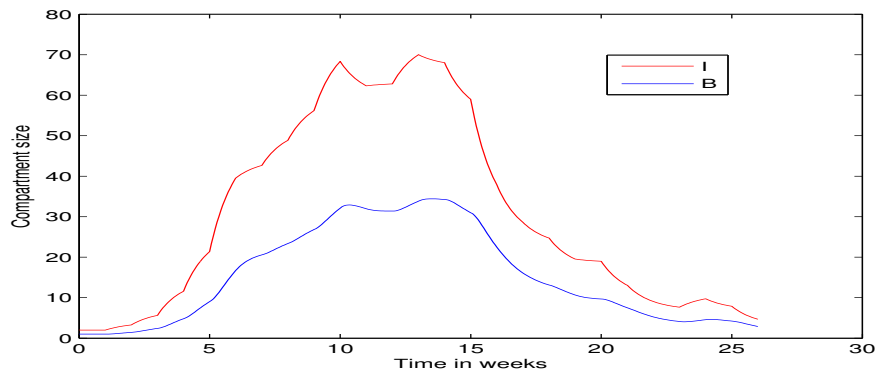
FIGURE 1. The classes $I(t)$ and $B(t)$ in the deterministic model as calculated from the data over the outbreak period $[t_0, t_2]$.
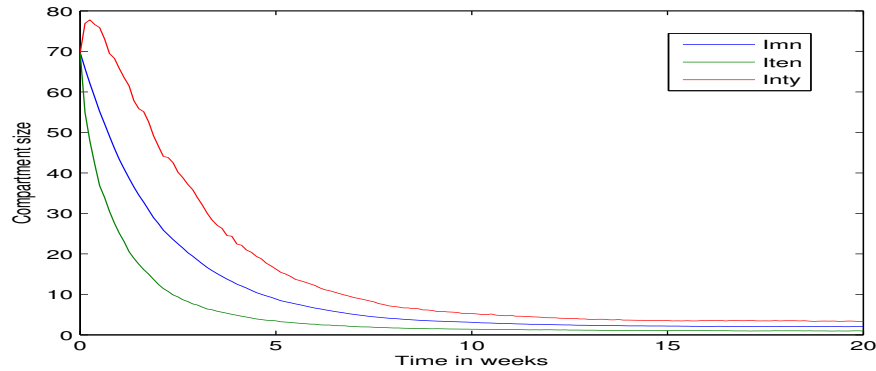
FIGURE 2. Evolution of the $I(t)$ as calculated the stochastic model from the data over the post-outbreak period $[t_1, t_2]$. The date t_1 which is 13 weeks later than t_0, corresponds to $t = 0$.
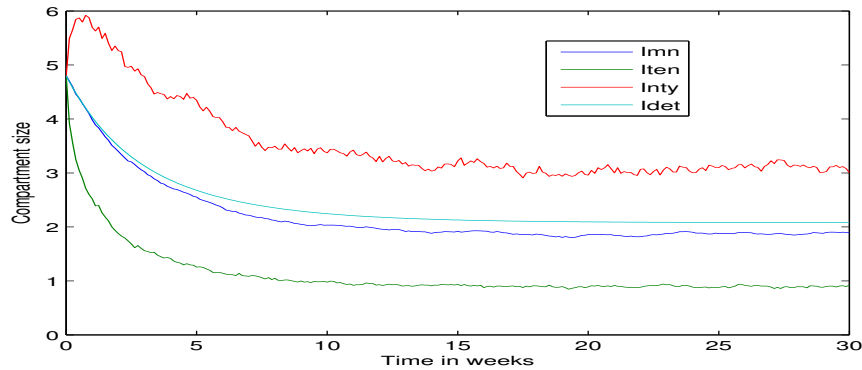

FIGURE 3. Evolution of the $I(t)$ as calculated for the stochastic model from the data over the post-outbreak period $[t_2, t_2 + 30]$.

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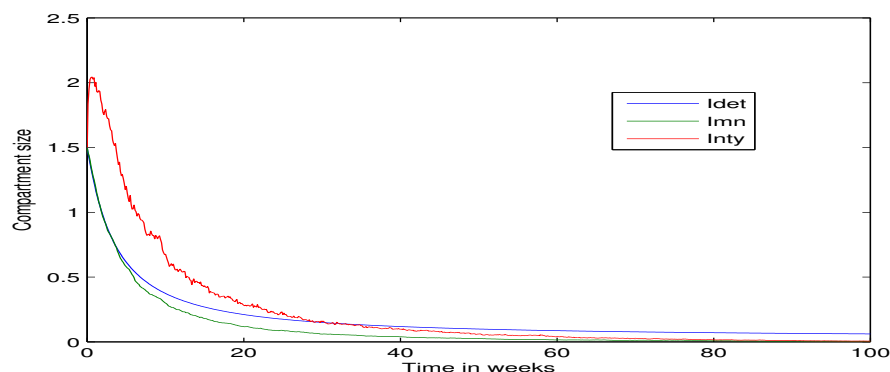
FIGURE 4. Evolution of the model over the post-outbreak period
$[t_2, t_2 + 78]$. We show the deterministic $I(t)$, and for the
stochastic model the mean, tenth and ninetieth percentiles.

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