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# Theoretical Assessment of the Impact of Climatic Factors in a *Vibrio Cholerae* Model

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**Abstract** A mathematical model for *Vibrio Cholerae* (*V. Cholerae*) in a closed environment is considered, with the aim of investigating the impact of climatic factors which exerts a direct influence on the bacterial metabolism and on the bacterial reservoir capacity. We first propose a *V. Cholerae* mathematical model in a closed environment. A sensitivity analysis using the eFast method was performed to show the most important parameters of the model. After, we extend this *V. cholerae* model by taking account climatic factors that influence the bacterial reservoir capacity. We present the theoretical analysis of the model. More precisely, we compute equilibria and study their stabilities. The stability of equilibria was investigated using the theory of periodic cooperative systems with a concave nonlinearity. Theoretical results are supported by numerical simulations which further suggest the necessity to implement sanitation campaigns of aquatic environments by using suitable products against the bacteria during the periods of growth of aquatic reservoirs.

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**Keywords** Mathematical model · *V. Cholerae* · Climatic factors · Cooperative systems · Stability

## 1 Introduction

*Vibrio Cholerae* (*V. Cholerae*) is a Gram-negative, comma-shaped bacterium that causes cholera in humans. Cholera is an acute intestinal infection caused by the ingestion of contaminated foods and water with *V. Cholerae* bacterium. There are 200 serogroups of *V. Cholerae* but only the *V. Cholerae* O1 and O139 are responsible of epidemics (WHO 2017). The etiological agent passes through and survives the gastric acid barrier of the stomach and then penetrates the mucus lining that coats the intestinal epithelial (Reidl and Karl Klose 2002). Once they colonise the intestinal gut, they produce enterotoxin (which stimulates water and electrolyte secretion by the endothelial cells of the small intestine) that leads to watery diarrhea. *Vibrio Cholerae* has been reported to be associated with a variety of living organisms, including animals with an exoskeleton of chitin, aquatic plants, protozoa, bivalves, waterbirds, as well as abiotic substrates (e.g. sediments). Most of these are well-known or putative environmental reservoirs for the bacterium, defined as places where the pathogen lives over time, with the potential to be released and to cause human infection. These bacteria are then associated with these environmental reservoirs forming commensal relationships (Huq et al. 1984). In many mathematical model of cholera (Copasso and Paveri-Fontana 1979; Cláudia 2001; Pascual et al. 2002; Hartley et al. 2005) and recently (Mushayabasa and Bhunu 2012; Mwasia and Tchuente 2011), bacteria dynamic are generally simplified using one linear differential equation with constant parameters. From the mathematical point of view, the constant parameter assumption has the advantage of simplifying the models and analysis, and facilitating the use of some well-known theory in autonomous dynamical systems. But it is important for mathematical cholera studies to incorporate these seasonal factors that modify the metabolism of bacteria, their density and the number of their environmental reservoirs along a year.

The aim of this paper is to improve our understanding on the impact of climatic factors of the dynamics of *V. Cholerae*. We first formulate and analyze a mathematical model of *V. Cholerae* in a closed environment. The sensitivity analysis of the model is carried out to show the most important parameters of the model. After, we extend this model by taking into account the variation of climatic factors. Threshold and equilibria are obtained and global stabilities examined. Numerical simulations are presented to support the theory and to get insight on the role of climatic factors on the dynamics of the population of *V. Cholerae*.

The rest of the paper is organized as follows. In Sect. 2, we introduced and studied a minimalistic *V. Cholerae* model. After, this model was extended by considering the seasonality of climatic factors on the bacterial reservoir capacity in Sect. 3. We present a qualitative analysis of the model. More precisely, we compute equilibria and study their stabilities. Numerical simulations are presented to illustrate and confirm theoretical results. We conclude the paper on Sect. 4.

**Table 1** Numerical values for the parameters of model (1)

Definition	Symbol	Estimated	Source
Growth rate of <i>V. Cholerae</i>	$r$	$1/7 \text{ day}^{-1}$	Rosa et al. (2003)
Daily death-rate of HI bacteria	$\mu_H$	$1/60 \text{ day}^{-1}$	Kaper and Morris (1995)
Daily death-rate of Non-HI bacteria	$\mu_L$	$1/30 \text{ day}^{-1}$	Tudor and Strati (1977)
Transition rate from HI to Non-HI state	$\nu$	$1/30 \text{ day}^{-1}$	Yibeltal (2009)
<i>V. Cholerae</i> capacity of the environment	$K$	$10^6 \text{ cell/ml}$	Assumed

## 2 A Minimalistic *V. Cholerae* Model

### 2.1 The Model

We consider two distinct population of bacteria according to their infectiousness status: Hyperinfectious bacteria (HI) and latent or Non-Hyperinfectious bacteria (Non-HI) denoted by  $B_H$  and  $B_L$ , respectively. The carrying capacity of *V. Cholerae* in environment is denoted by  $K$ . For the population of hyperinfectious bacteria, we assume that the growth rate  $rB_L$  could be limited due to a plausible limitation of shared resources in the medium where they live  $1 - B_H/K$ , that is, the growth rate in the HI compartment is modeled by the logistic term  $rB_L(1 - B_H/K)$ . Hyperinfectious bacteria become Non-Hyperinfectious bacteria at constant rate  $\nu$ . Hyperinfectious and Non-Hyperinfectious bacteria die at constant rates  $\mu_H$  and  $\mu_L$ , respectively. Then, the dynamics of the population of *V. Cholerae* in a closed environment can be modeled by the following system of differential equations:

$$\begin{cases} \dot{B}_H = rB_L \left(1 - \frac{B_H}{K}\right) - (\mu_H + \nu)B_H, \\ \dot{B}_L = \nu B_H - \mu_L B_L. \end{cases} \quad (1)$$

Biological significance and values of parameters of model (1) are given in Table 1.

### 2.2 Theoretical Analysis

We first study the basic properties of the model's solutions (1), which are essential in the proofs of stability results. The right-hand side of model (1) is a continuously differentiable map ( $C^1$ ). Model system (1) is biologically well posed: if the initial data are in  $\mathbb{R}_+^2$ , then the solution stays in  $\mathbb{R}_+^2$ ; indeed, it is straightforward to show that the compact:

$$\Omega = \left\{ (B_H, B_L) \in \mathbb{R}_+^2 : B_H \leq K, B_L \leq \frac{\nu}{\mu_L} K \right\}, \quad (2)$$

is a global attractor and positively invariant set for model (1). Then, by the Cauchy Lipschitz theorem, model (1) provides a unique maximal solution for every initial condition given.

To derive some qualitative results from our model (1), we compute equilibria and study their stabilities. Let  $\mathcal{F}$  be a threshold that represents the mean number of Non-HI bacteria produced by one Non-HI bacteria over its lifespan:

$$\mathcal{F} = \frac{rv}{(\mu_H + v)\mu_L}. \quad (3)$$

The threshold (3) is sometimes called the basic offspring number. We have the following result.

**Lemma 1** : *Model system (1) has two possible equilibria:*

- (i) a trivial equilibrium  $E_0 = (0, 0)$ ;
- (ii) a positive equilibrium  $E^* = (B_H^*, B_L^*)$  when  $\mathcal{F} > 1$  defined as follows:

$$B_H^* = \left(1 - \frac{1}{\mathcal{F}}\right)K \quad \text{and} \quad B_L^* = \frac{v}{\mu_L} \left(1 - \frac{1}{\mathcal{F}}\right)K. \quad (4)$$

The stability of equilibria of model (1) is summarized in Proposition 1.

**Proposition 1** *Assume that the initial condition  $(B_H(0), B_L(0)) \in \Omega$ .*

- 1 *When  $\mathcal{F} < 1$ , the trivial equilibrium  $E_0$  is globally asymptotically stable (GAS), which implies that the population of *V. Cholerae* will go until extinction, whatever the initial population.*
- 2 *When  $\mathcal{F} > 1$ , the trivial equilibrium is unstable and the positive equilibrium  $E^*$  is globally asymptotically stable, that is the population of *V. cholerae* persists in the environment.*

*Proof* To prove Proposition 1, we will use some results related to cooperative systems (Smith 2008). These kind of systems are very important and well-known in Biology and Ecology, and have been studied extensively, see Smith (2008). For reader's convenience, we recall the following general definition. Consider a  $n$ -dimensional autonomous differential system:

$$\dot{x} = f(x), \quad x(0) = x_0, \quad (5)$$

where  $f$  is a given vector function, i.e.  $f = (f)_i$ , with  $f_i : \mathbb{R}^n \rightarrow \mathbb{R}$ . Then, we recall the following

**Definition 1** (Smith 2008) System (5) is called *cooperative* if for every  $i, j \in \{1, 2, 3, \dots, n\}$  such that  $i \neq j$  the function  $f_i(x_1, \dots, x_n)$  is monotone increasing with respect to  $x_j$ .

It is easy to verify that model (1) is a cooperative system in  $\Omega$ . In the following, we consider the basins of attraction represented as  $n$ -dimensional intervals: given  $a, b \in \mathbb{R}^n$  with  $a \leq b$  then  $[a, b] = \{x \in \mathbb{R}^n : a \leq x \leq b\}$ . To show GAS, we will use the following theorem:

**Theorem 1** (Anguelov et al. 2012) Let  $a, b \in \Omega$  be such that  $a < b, ]a; b[ \subset \Omega$  and  $f(b) \leq 0 \leq f(a)$ . Then, system (5) defines a (positive) dynamical system on  $[a, b]$ . Moreover, if  $[a, b]$  contains a unique equilibrium  $p$  then  $p$  is globally asymptotically stable on  $[a, b]$ .

We are now able to prove Proposition 1.

- When  $\mathcal{T} \leq 1$ , model (1) has only the trivial equilibrium  $E_0$ . Thus, taking  $a = E_0$  and  $b = \left(K, \frac{\nu}{\mu_L}K\right)$  we have that  $f(a) = 0$  and  $f(b) \leq 0$ . It then follows from Theorem 6 (Anguelov et al. 2012), that the trivial  $E_0$  is globally asymptotically stable on  $[a, b]$ , hence on  $\Omega$ , when  $\mathcal{T} < 1$
- When  $\mathcal{T} > 1$ , there exists  $\epsilon > 0$  such that  $\mathcal{T} > 1 + \epsilon$ . Let  $H_\epsilon$  sufficiently small such that:  $H_\epsilon < \epsilon$  and  $L_\epsilon = \frac{\nu(1+\epsilon)}{\mu_L \mathcal{T}} H_\epsilon < \epsilon$ . Let  $a_\epsilon = (H_\epsilon, L_\epsilon)^T$ . Then, from the right-hand side of model (1) and the fact that  $\mathcal{T} > 1 + \epsilon$  and  $K \gg 1$ , we can deduce that

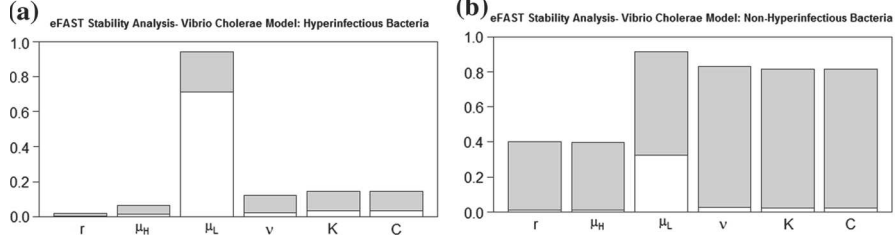
$$f(a_\epsilon) \geq \begin{pmatrix} (\nu + \mu_L)\epsilon \left(1 - \frac{1+\epsilon}{\mathcal{T}}\right) H_\epsilon \\ \nu \left(1 - \frac{1+\epsilon}{\mathcal{T}}\right) H_\epsilon \end{pmatrix} \geq 0. \quad (6)$$

Hence, it follows from Theorem 6 (Anguelov et al. 2012) that the nontrivial equilibrium  $E^* = (B_H^*, B_L^*)$  is globally asymptotically stable on  $[a_\epsilon, b]$ . Since  $a_\epsilon$  can be selected to be smaller than any  $x > 0$ , we have that  $E^*$  is asymptotically stable on  $\Omega$  with basin of attraction  $\Omega' = \Omega \setminus \{E_0\}$  which implies that  $E_0$  is unstable. This concludes the proof. □

### 2.3 Sensitivity Analysis

Let us recall that the basic offspring number is defined as follows:

$$\mathcal{T} = \frac{r\nu}{(\mu_H + \nu)\mu_L}. \quad (7)$$



**Fig. 1** eFast sensitivity analysis for the **a** Hyperinfectious and **b** Non-Hyperinfectious bacteria. White bar: first-order effects; sum of white and grey bars: total effect

The relevance of the basic offspring number is due to the Proposition 1. In fact, it is the key parameter for the existence of a non-trivial equilibrium. It is important to know the relative impact of different parameters of the model (1) on  $\mathcal{T}$ . To do this, we estimate the sensitivity index of  $\mathcal{T}$  with respect to a parameter  $p$  is  $\frac{\partial \mathcal{T}}{\partial p}$ . Another

measure is the elasticity index  $p$  (Chitnis et al. 2008) that measures the relative change of  $\mathcal{T}$  with respect to  $p$ , defined as follows:

$$\Upsilon_p^{\mathcal{T}} = \frac{\partial \mathcal{T}}{\partial p} \cdot \frac{p}{\mathcal{T}}. \quad (8)$$

In fact, straightforward computations lead to the following result:

$$\Upsilon_r^{\mathcal{T}} = 1, \quad \Upsilon_{\mu_L}^{\mathcal{T}} = -1, \quad \Upsilon_v^{\mathcal{T}} = \frac{\mu_H}{\mu_H + v} \quad \text{and} \quad \Upsilon_{\mu_H}^{\mathcal{T}} = -\frac{\mu_H}{\mu_H + v}. \quad (9)$$

Clearly  $r$  and  $\mu_L$  have the strongest impact on  $\mathcal{T}$ . However, it is slightly different when we focus on the variables  $B_H$  and  $B_L$ . Now, we perform the sensitivity analysis in order to detect the most sensitive parameters, that is the parameters that most influence the output variable of the model. This can help predict the effect of each parameter on the model results and classify them according to their degree of sensitivity. To do so, we use the eFast<sup>1</sup> sensitivity method which is described in (Marino et al. 2008). Figure 1 presents the sensitivity analysis obtained with the eFast method that highlights first-order effects (main effects) and total effects (main and all interaction effects) of the parameters on the Model Outputs,  $B_H$  and  $B_L$ . According to the result obtained in Fig. 1, the parameter  $\mu_L$  has the strongest impact both on  $B_H$  and  $B_L$ . This means that elimination of Latent Bacteria (Non-HI) have the most impact bacteria density. These kind of bacteria are usually found in wetlands, rivers, sewage

<sup>1</sup> Is a variance decomposition method (analogous to ANOVA): input parameters are varied, causing variation in model output. This variation is quantified using the statistical notion of variance  $s^2 = \sum_{i=1}^N (y_i - \bar{y})^2 / (N - 1)$ , where  $N$  = sample size (or equivalently, total number of model runs),  $y_i$   $i$ th model output, and  $\bar{y}$  sample mean. The algorithm then partitions the output variance, determining what fraction of the variance can be explained by variation in each input parameter (i.e. partial variance).

and water sources contaminated by human excrements (Xu 1982). Therefore, sensitivity analysis results suggest that effective control strategy to reduce bacteria density would be the implementation of a serious sanitation campaigns of aquatic environments (wetlands, rivers, sewage and water sources contaminated by human excrements) by using suitable products during the periods of growth of aquatic reservoirs and also during cholera epidemic periods. These sanitation actions could be initiated and organized by the public health agents. In Haïti for example, the new solution implemented by United Nation Organization to eradicate Cholera concerns improving access to drinking water and sanitation (Guillaume et al. 2012).

### 3 A *V. Cholerae* Model in a Periodic Environment

#### 3.1 The Model

Climatic factors such as water temperature may explain the seasonality of the disease either by exerting a direct influence on the bacterial reservoir capacity (the value of  $K$ ) or even on parameters affecting their growing ( $r$ ), their survival ( $\mu_H$  and  $\mu_L$ ) and their metabolism ( $v$ ) (Colwell 1996; Islam et al. 1999). But the bacterial reservoir capacity ( $K$ ) have an important role on persistence and resurgences of cholera disease. In fact bacteria are strongly associated with both phytoplanktonous and zooplanktonous organisms forming commensal or symbiotic relationships. When *V. Cholerae* bacteria are associated with such reservoirs, they generally have an advanced survival (Xu 1982). Their environmental adaptation will lead to metabolic and phenotypic changes that will condition their survival; This can be compared to a phenomenon of dormancy. Cells are considered “viable but non-culturable” (VNC) because the main effect of this change is the loss of ability live on bacteriological culture media (Roszak and Colwell 1987). Changing for a cultivable state is possible particularly when the factors that cause stress become favorable to the development and growth of the bacterial population. The variation of these climatic factors are such all the parameters excepted  $C$  must be time-dependent and strictly bounded by positive values (Colwell 1996; Islam et al. 1999; Xu 1982; Roszak and Colwell 1987; Huq et al. 1984). The model (1) becomes :

$$\begin{cases} \dot{B}_H = r(t)B_L\left(1 - \frac{B_H}{K(t) + C}\right) - (\mu_H(t) + v(t))B_H, \\ \dot{B}_L = v(t)B_H - \mu_L(t)B_L, \end{cases} \quad (10)$$

where  $C$  is a minimal constant bacterial capacity value. Notice that the model in (10) belongs to the family of periodic cooperative systems with a concave nonlinearity (Smith 2008; Jifa 1993). Let us define a periodic cooperative system of  $n$  differential equations:

$$\dot{x} = f(t, x), \quad (11)$$

where  $f : \mathbb{R}_+ \times \mathbb{R}^n \rightarrow \mathbb{R}^n$ , with concave non-linearities. Let  $A(t)$  be a  $n \times n$  continuous matrix in  $\mathbb{R}$ ,  $\tau$ -periodic in  $t$ , we denote:

$$\bar{a}_{i,j} = \max_{0 \leq t \leq \tau} a_{i,j}(t) \quad \text{and} \quad \underline{a}_{i,j} = \min_{0 \leq t \leq \tau} a_{i,j}(t), \quad (12)$$

and set

$$\bar{A} = (\bar{a}_{i,j}) \quad \text{and} \quad \underline{A} = (\underline{a}_{i,j}). \quad (13)$$

Then,

$$\underline{A} \leq A(t) \leq \bar{A} \quad \text{for} \quad 0 \leq t \leq \tau. \quad (14)$$

Set  $P_0$  and  $Q_0$  in  $\mathbb{R}_+$ . For reader's convenience, let us recall Theorem 5.5, p. 230 of (Smith 2008).

**Theorem 2** *Let  $F(t, x)$  be continuous in  $\mathbb{R} \times [0, P_0] \times [0, Q_0]$ ,  $\tau$ -periodic in  $t$  for a fixed  $x$  and assume  $D_x F(t, x)$  exists and is continuous in  $\mathbb{R} \times [0, P_0] \times [0, Q_0]$ . Assume also that all solutions are bounded in  $[0, P_0] \times [0, Q_0]$  and  $F(t, 0) = 0$ . Assume*

- (a)  $\frac{\partial F_i}{\partial x_j} \geq 0$ ,  $(t, x) \in \mathbb{R} \times [0, P_0] \times [0, Q_0]$ ,
- (b)  $A(t) = D_x F(t, 0)$  is irreducible for any  $t \in \mathbb{R}$ ,
- (c) if  $0 < x < y$ , then  $D_x F(t, x) > D_x F(t, y)$ .

Then

1. If all principal minors of  $-\bar{A}$  are non-negative, then  $\lim_{x \rightarrow \infty} x(t) = 0$  for every solution of system (11) in  $[0, P_0] \times [0, Q_0]$
2. If  $-\underline{A}$  has at least one negative principal minor, then system (11) possesses a unique positive  $\tau$ -periodic solution which attracts all initial conditions in  $[0, P_0] \times [0, Q_0]$ .

In this case, we can take  $P_0 = Q_0 = \bar{K} + C$ . Let  $x = (B_H, B_L) \in [0, P_0] \times [0, Q_0]$ . Without losing to the generality we can suppose  $B_H \leq K + C$  and  $B_L \leq K + C$ . One has

$$F(t, x) = \begin{pmatrix} r(t)B_L \left(1 - \frac{B_H}{K(t) + C}\right) - (\mu_H(t) + v(t))B_H \\ v(t)B_H - \mu_L(t)B_L \end{pmatrix}.$$

Clearly,  $F$  is continuous and  $\tau$ -periodic. We also have  $F(t, 0) \equiv 0$ . Moreover,

$$D_x F(t, x) = \begin{pmatrix} -(\mu_H(t) + \nu(t)) - \frac{r(t)B_L}{K(t) + C} & r(t) \left(1 - \frac{B_H}{K(t) + C}\right) \\ \nu(t) & -\mu_L(t) \end{pmatrix}, \quad (15)$$

which implies that  $D_x F(t, x)$  is continuous and  $\frac{\partial F_i}{\partial x_j} \geq 0$  for all  $(B_H, B_L) \in \mathbb{R}_+^2$ . When  $0 < x < y$ , we have obviously  $D_x F(t, y) > D_x F(t, x)$ . Finally, we have

$$A = D_x F(t, 0) = \begin{pmatrix} -(\mu_H(t) + \nu(t)) & r(t) \\ \nu(t) & -\mu_L(t) \end{pmatrix}. \quad (16)$$

Clearly,  $A$  is irreducible for all  $t$ . One notes  $p_{\min} \leq p(t) \leq p_{\max}$  for all  $t \geq 0$  and for  $p = r, \nu, \mu_H, \mu_L, K$ . Using the fact that all time-dependent parameters have positive lower and upper bounds, we can deduce that

$$-\underline{A} = \begin{pmatrix} \mu_{H\max} + \nu_{\max} & -r_{\min} \\ -\nu_{\min} & \mu_{L\max} \end{pmatrix} \quad \text{and} \quad -\bar{A} = \begin{pmatrix} \mu_{H\min} + \nu_{\min} & -r_{\max} \\ -\nu_{\max} & \mu_{L\min} \end{pmatrix}. \quad (17)$$

First, according to Theorem 2, we need to study all principal minors of  $-\bar{A}$ . Diagonal terms are positive, and a straightforward computation shows that  $\det(-\bar{A}) \geq 0$  if the following inequality is verified

$$\frac{r_{\max} \nu_{\max}}{(\mu_{H\min} + \nu_{\min}) \mu_{L\min}} \leq 1.$$

From Theorem 2, since all diagonal terms of  $-\underline{A}$  are positive, it remains to verify that  $\det(-\underline{A}) < 0$ . A simple computation gives

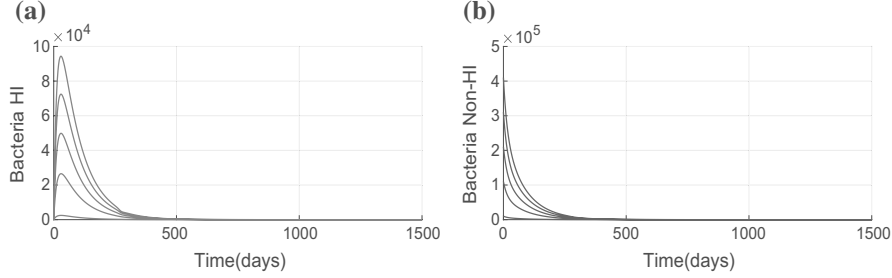
$$\frac{r_{\min} \nu_{\min}}{(\mu_{H\max} + \nu_{\max}) \mu_{L\max}} < 1.$$

Then, applying Theorem 2, we have the following result.

**Theorem 3** *Let*

$$\mathcal{I}_{\min} = \frac{r_{\min} \nu_{\min}}{(\mu_{H\max} + \nu_{\max}) \mu_{L\max}} \quad \text{and} \quad \mathcal{I}_{\max} = \frac{r_{\max} \nu_{\max}}{(\mu_{H\min} + \nu_{\min}) \mu_{L\min}}. \quad (18)$$

*Now, let us assume all parameters to be time-dependent. Then,*



**Fig. 2** Simulation of model (10) using four various initial conditions in  $\mathbb{R}_+^2$  when  $r = 1/40 \text{ day}^{-1}$  (so that  $\mathcal{T} = 0.5$ ). All other parameter values are given in Table 1

- (i) If  $\mathcal{T}_{\min} > 1$ , model (10) has a unique periodic solution which attracts all initial conditions in  $\Omega$ .
- (ii) If  $\mathcal{T}_{\max} \leq 1$ , the solution of model (10) converges to the trivial equilibrium  $E_0$ .

Indeed, the above result is related to the time dependent basic offspring number  $\mathcal{T}(t)$ :

$$\mathcal{T}(t) = \frac{r(t)v(t)}{(\mu_H(t) + v(t))\mu_L(t)}, \quad (19)$$

which satisfies

$$\mathcal{T}_{\min} \leq \mathcal{T}(t) \leq \mathcal{T}_{\max}. \quad (20)$$

Assume now, that all parameters are constant except  $K$ , then  $-\underline{A} = -\bar{A}$  and thus  $\mathcal{T}_{\min} = \mathcal{T}_{\max}$ , then we have the following result.

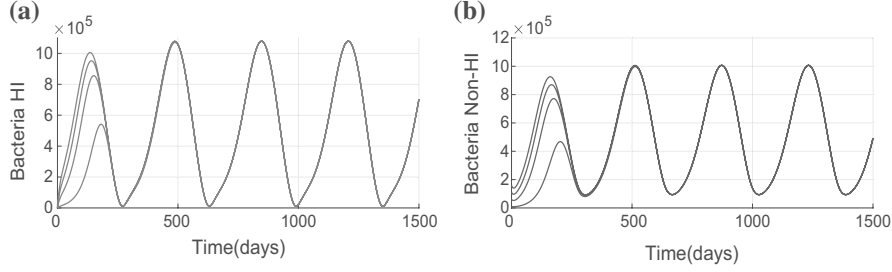
**Theorem 4** Assume that all parameters are constant, except. Then,

- (i) If  $\mathcal{T} > 1$ , model (10) has a unique periodic solution which attracts all initial conditions in  $\Omega$ .
- (ii) if  $\mathcal{T} \leq 1$ , the solution of model (10) converges to the trivial equilibrium  $E_0$ .

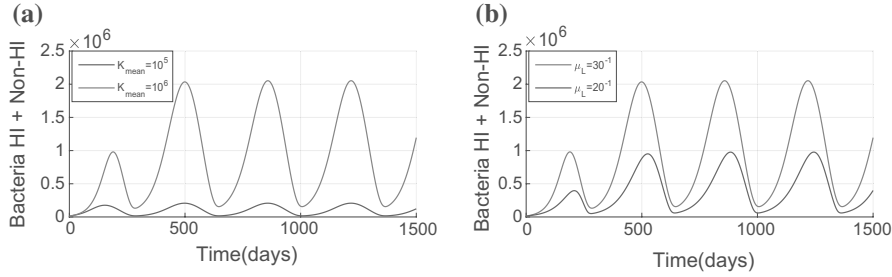
We stress that this result is similar to the one with constant parameters, except that the nontrivial equilibrium is now periodic.

### 3.2 Numerical Simulation

Herein, numerical simulations are presented to illustrate Theorem 4. We used the parameter values given in Table 1. The carrying capacity  $K$  of *V. Cholerae* is



**Fig. 3** Simulation of model (10) using four various initial conditions in  $\mathbb{R}_+^2$  when  $r = 1/7 \text{ day}^{-1}$  (so that  $\mathcal{T} = 2.8571$ ). All other parameter values are given in Table 1



**Fig. 4** Simulation of model (10) for two different values of  $K_{mean}$  and  $\mu_L$ . All other parameter values are given in Table 1

periodic, while all other parameter constant. Several type of functions for  $K$  could be chosen. Among them, we choose one of the simplest:

$$K(t) = K_{mean} \left( 1 + \tilde{k} \sin\left(\frac{2\pi}{T} t\right) \right). \quad (21)$$

The function  $K(t)$  is a periodic function with a period  $T = 365$  days,  $K_{mean} = 10^6$  cell/ml and  $\tilde{k} = 0.99$ . The Fig. 2 illustrates the model (10) dynamics when  $\mathcal{T} < 1$ . As expected, the population decays until extinction. However, when  $\mathcal{T} > 1$  bacteria are maintained in the environment with a periodic density as shown in Fig. 3.

Another simulation (see Fig. 4) illustrates how dynamics depend on climatic factors that affect a direct influence on either the bacteria reservoir capacities ( $K_{mean}$ ) or the bacteria mortality ( $\mu_L$ ).

Figure 4 shows density variations of bacteria when  $K$  is considered periodic and when  $K_{mean}$  and  $\mu_L$  have two different values. The Fig. 4a shows clearly how reservoirs can play important role on persistence of bacteria in environment.

## 4 Conclusion

In this work, we proposed a model of *V. Cholerae* populations growing in a closed environment and qualitatively analyzed their dynamics. The aim was to evaluate the impact of climatic factors on the growth of *V. Cholerae* populations. We first considered a *V. Cholerae* model in a closed environment with all parameters constant. The theoretical analysis of the model has been presented. The sensitivity analysis of the model has been also investigated. We found that the best control action would be preventive sanitation. So it is recommended to use suitable products against *V. Cholerae* during the periods of growth of aquatic reservoirs and also during cholera epidemic periods. After, the climatic variations have been incorporated in this *V. cholera* model by using periodic functions. Critical periods generally correspond to periods where climatic and environmental factors are favorable to the growth and to the propagation of aquatic reservoirs. We presented a qualitative analysis of the model. More precisely, existence, positivity and boundedness of solutions are presented. Threshold and equilibria are obtained and global stabilities examined. We shown that the model can present one or two biologically meaningful equilibria when the basic offspring number  $\mathcal{T}$  is lower or greater than 1 respectively. Equilibrium stabilities were examined. Numerical simulations have been presented to illustrate and confirm theoretical results. Through numerical simulations, we found that the aquatic reservoirs are playing a significant role among the factors explaining the causes of endemicity of these disease.

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