



# Dynamics of Cholera Pathogen Carriers and Effect of Hygiene Consciousness in Cholera Outbreaks

S.F. Abubakar\*, , M.O. Ibrahim 

## Abstract

We derive a deterministic mathematical model that scrutinizes the dynamics of cholera pathogen carriers and the hygiene consciousness of individuals, before the illness, during its prevalence, and after the disease's outbreaks. The dynamics can effectively help in curtailing the disease, but its effects had less coverage in the literature. Boundedness of the solution of the model, its existence, and uniqueness are ascertained. Effects of cholera pathogen carriers and hygiene consciousness of individuals in controlling the disease or allowing its further spread are analyzed. The differential transformation method is used to obtain series solutions of the differential equations that make the system of the model. Simulations of

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the series solutions of the model are carried out and displayed in graphs. The dynamics of the concerned state variables and parameters in the model are interpreted via the obtained graphs. It is observed that higher hygiene consciousness of individuals can drastically reduce catching cholera disease at onset and further spread of its infections in the population, this in turn, shortens the period of cholera epidemic.

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## 1 Introduction

Mathematical modeling can be defined as the process of selecting and utilizing relevant mathematical ideas to scrutinize and portray situations in a better way to understand and apply safer decisions about physical phenomena, and the entire real-life problems. Models are believed to aid in understanding various ways phenomena and real-life problems operate. This includes epidemic processes, and it helps in designing effective control strategies [1].

The world has witnessed a remarkable improvement in hygiene, sanitary measures, and effective treatment of infectious diseases. This has drastically reduced the threat of diseases like cholera in many countries. In addition to this, many studies, experiments, and theories that were carried out and developed now and in the past have aided in reducing cholera mobility [8]. A report from “acap” stated that “*there have been 93,932 suspected cholera cases, that caused 3,293 deaths from these cases in Nigeria and that 5,343 confirmed cholera cases with 156 deaths in Niger Republic as of October 2021*”.

Due to poor sanitation systems, the shortage of good drinking water, and the high density of the population, Africa become a victim of cholera outbreaks, leading to a higher risk of outbreaks, and the spread of the disease. This coupled with shifting priority to COVID-19 resulted in the diversion of most resources and attention to it, leaving other health problems. This in-

turn caused lapses in the entire health sector and affected the process of contact tracing and thorough management of cholera [27].

## 1.1 Cholera

Cholera is an illness caused by gram-negative, nearly comma-shaped bacillus bacteria called *Vibrio cholerae*. The sickness's symptoms are mostly, severe gray color diarrhea, with flecks of mucus, extreme thirst, drop in blood pressure, abdominal pain, and kidney failure [29]. It usually comes with vomiting and can dehydrates the patient in quite short period of time if prompt treatment measures are not taken. Its outbreaks usually comes after natural and artificial catastrophes that disrupt the public strong sanitation service systems. The disease is still a threat and problem to global public health [26], especially in developing countries.

Mishandling faeces and vomit of infected individuals easily trigger spread of the disease. This is so because *Vibrio cholerae* bacteria that pass through the human digestive system are more infective and stubborn to control by human body immunity than others. Among its crucial characteristics is that it can survive even with limited oxygen and iron [17]. It has been estimated that (though, depending on the individual's age, stomach acidity and blood group), an individual has to ingest up to a dose range of  $10^3$  to  $10^8$  *Vibrio cholerae* bacteria cells, before developing cholera disease. The disease's incubation period ranges (that is, from ingestion of *Vibrio cholerae* bacteria to cholera infection) from 12 hours to 5 days [11, 20]. The range is pegged at, from 18 hours to 5 days, in [24]'s study (as cited in [12]). *Vibrio cholerae*'s mechanism of causing cholera was described by [25] that usually start with surviving the stomach acidity, shedding its flagellum, swim deep into intestine mucus. It colonizes and secretes a toxin that stimulates intestine *mucosal* cells to pump body water due to osmosis. This leads to dehydrating the patient.

The onset outbreak of cholera is usually caused by the consumption of contaminated water. The subsequent spread of the disease can be through contaminated food, contact with cholera patients and unhygienic handling of

faeces, and vomit of cholera patients. Most mathematical models on cholera we visited, have not categorically scrutinized cholera pathogens since the pathogens cannot on their own spread without human contribution. It is hence vital to study the dynamics of *Vibrio cholerae* as causative agents or pathogens that cause cholera disease [28].

Cholera is an extremely virulent disease that affects all ages and economic classes of a nation. It has been estimated that about two billion people drink water from sources that may be contaminated with faeces and that 1.3 billion people are at risk of cholera disease in cholera endemic areas and regions of the world [29]. 75% of cholera pathogens carriers, manifest no symptoms but spread the pathogen to their immediate environment through faeces for a period of 7–14 days, of ingesting the pathogens by the carriers [13]. People with low gastric acidity are at high risk of catching cholera. Therefore transmission of the disease in such a class of people is rampant [1]. The study intends to apply mathematical modeling to best portray the transmission thresholds of the infections by *Vibrio cholerae* in the host, and the magnitudes it attains so as to get the best ways to eradicate it.

## 1.2 Related literature

Elhia et al. [10] proposed a mathematical model on the global stability of the SIR cholera epidemic model that dealt with two infectious stages and treatment of cholera. They carried out an analysis of the model's global stability, obtained  $R_0$ , and illustrated the analytical results numerically.

A mathematical model on the controls of cholera, including hygiene consciousness as a control strategy and a compartment of the model was formulated in [21].

Mathematical models on the dynamics of individuals who ingested cholera pathogens but have not developed any symptoms of cholera disease are very rare. These categories of individuals unknowingly spread the pathogens of the disease in the environment. This in turn causes the spread of the disease. Therefore, incorporating all possible and necessary compartments in a mathematical model, will surely warrant a chance to determine the essential

dynamics it entails. This will also help in determining the basic mechanisms of transmission of the disease. Its outbreak and ways individuals can be infected by the disease. It can also help in exploring various ways of its spread, methods of controlling it, and what can make it escalate or persist.

These motivate us to formulate a deterministic mathematical model of cholera by marrying [21]'s SICRB cholera model with [10]'s SIUTR cholera model, to come up with SICUTRB deterministic mathematical model of cholera, in order to investigate the dynamics, spread, and control measures of cholera disease, especially through hygiene consciousness. The model has two incidences, linear and nonlinear. The linear incidence represents the human-to-human transmission of cholera, and the nonlinear is the logistic response to the increase in the cholera pathogen  $B$ , and  $\phi$  is represented by  $v1 \frac{B}{K+B}$ .

Differential transform method (DTM) is a numerical-analytical (others call it semi-analytical) technique. It is used in solving linear and nonlinear, ordinary and partial differential equations, usually with known initial values. It is carried out by obtaining the approximate series solutions of the equations. It was first used by [30] in solving linear and nonlinear initial value problems in electrical circuit analysis. Pukhov earlier proposed differential transform by computing the image of a transformed function using differential operations, in his work 'Differential Transformations of Functions and Equations' published in 1980. Zhou pioneered using the method to solve the initial value problem [5].

The method is an iterative procedure for getting analytic Taylor series solutions of differential equations by iterations [4, 31]. In other words, DTM solves an induced recursive equation from a given differential equation by determining coefficients of the Taylor series of the concern function [2]. Its analytical solutions can be constructed in the form of polynomials, whose obtained solutions can be presented in terms of convergence series. This in turn makes it easier to compute especially nonlinear equations. Application of the method reduces the bulk computation and helps arrive at more accurate approximate answers while solving linear and nonlinear differential equations. In recent years, the method has been employed in solving one-dimensional planar Bratu's problem, differential-difference equations, delay differential

equations, differential-algebraic equations, and integro-differential systems [32].

Some basic definitions and properties of DTM as obtained in related literature, such as [2, 3, 14, 18, 19, 23], are as follows: The transformation of the  $b$ th derivative of a function with one variable is

$$G[b] = \frac{1}{b!} \left[ \frac{d^b g(t)}{dt^b} \right]_{t=t_0}. \quad (1)$$

Here  $g(t)$  is the original function, while  $G[b]$  is the transformed function, and the inverse transformation is given as

$$g(t) = \sum_{b=0}^{\infty} G[b](t - t_0)^b. \quad (2)$$

By Taylor series, the inverse transformation of (1) can be obtained as

$$g(t) = \sum_{b=0}^{\infty} t^b G[b]. \quad (3)$$

Substituting (1) into (3) gives

$$g(b) = \sum_{b=0}^{\infty} \left[ \frac{t^b}{b!} \left( \frac{d^b g(t)}{dt^b} \right) \right]_{t=t_0}. \quad (4)$$

Some functions and their respective differential transformations are displayed in Table 1.

## 2 Model formulation

State variables and parameters used in the model are described in Tables 2 and 3, respectively.

Table 1: Some functions and their respective differential transforms

Original Function	Transformed Function
$g(t) = f(t) \pm h(t)$	$G[b] = F[b] \pm H[b]$
$g(t) = mf(t)$	$G[b] = mF[b]$ $m$ is a constant
$g(t) = 1$	$G[b] = \delta[b]$
$g(t) = t$	$G[b] = \delta[b - 1]$
$g(t) = f(t)h(t)$	$G[b] = \sum_{a=0}^b F[a]H[b - a]$
$g(t) = e^{\lambda t}$	$G[b] = \frac{\lambda^b}{b!}$
$g(t) = e^t$	$G[b] = \frac{1}{b!}$
$g(t) = \frac{df(t)}{dt}$	$G[b] = (b + 1)G[b + 1]$
$g(t) = \frac{d^2f(t)}{dt^2}$	$G[b] = (b + 1)(b + 2)G[b + 2]$
$g(t) = t^a$	$G[b] = \delta(b - a) = \begin{cases} 1, & \text{if } b = a \\ 0, & \text{if } b \neq a \end{cases}$
	where $\delta$ is the Kronecker delta
$g(t) = (1 + t)^a$	$G[b] = \frac{a(a - 1) \cdots (a - b + 1)}{b!}$
$g(t) = \sin(\theta t + \alpha)$	$G[b] = \frac{\theta^b}{b!} \sin\left(\frac{b\pi}{2} + \alpha\right)$
$g(t) = \cos(\theta t + \alpha)$	$G[b] = \frac{\theta^b}{b!} \cos\left(\frac{b\pi}{2} + \alpha\right)$

Table 2: State variables description

Variable	Description
$S$	Susceptible Individuals
$I$	Infected Individuals
$C$	Hygiene Conscious Individuals
$U$	Untreated <i>Vibrio cholerae</i> Carriers
$T$	Infected Individuals Under Treatment
$R$	Recovered Individuals
$B$	Concentration of <i>Vibrio cholerae</i> Bacteria in Food and Water Consume by the Human Population

Table 3: Parameters description

State Variables & Parameter	Description	Value	Reference
$S$	Susceptible Individuals	900	Estimated
$I$	Infected Individuals	50	Calculated
$C$	Hygiene Conscious Individuals	500	Estimated
$U$	Untreated <i>Vibrio cholerae</i> Carriers	25	Calculated
$T$	Infected Individuals Under Treatment	22	Estimated
$R$	Recovered Individuals	3	Calculated
$B$	Concentration of <i>Vibrio cholerae</i> Bacteria in Food	600	Estimated
$q$	Recruitment rate of human population	0.104	Estimated
$\delta$	Per capital natural human death rate	0.0101	[10]
$v_1$	Rate of ingesting <i>Vibrio Cholerae</i> bacteria from contaminated food and water consume by the human population	0.5	[21]
$v_2$	Rate of getting <i>Vibrio cholerae</i> bacteria through human to human interaction	0.03	[21]
$\theta$	Recovery rate of hygiene individuals	0.38	[21]
$h$	Hygiene consciousness of individuals	0.6	Estimated
$\beta$	Rate of <i>Vibrio cholerae</i> decrease	0.78	Estimated
$\eta$	Rate at which infected individuals increase <i>Vibrio cholerae</i> bacteria in the environment	10	[6]
$f$	Rate of freedom from <i>Vibrio cholerae</i> by the hygiene conscious individuals	0.7	Estimated
$k$	Concentration of cholera pathogen that yields 50% chance of individual developing cholera disease	$10^6$	[6]
$g_1$	Progression of recovery of treated individuals to untreated <i>Vibrio cholerae</i> carriers	0.08	Estimated
$g_2$	Rate of interruption of treatment	0.2	[10]
$r_1$	Recovery rate of untreated <i>Vibrio Cholerae</i> carriers	0.8	Estimated
$r_2$	Recovery of treated cholera patients	0.9	Estimated
$\epsilon$	Disease induced death rate for individuals in $I$ class	0.105	Estimated
$\mu$	Disease induced death rate for individuals in $U$ class	0.1	Estimated
$\alpha$	Disease induced death rate for individuals in $T$ class	0.08	Estimated
$m$	Rate of movement of the infected class	0.4	[10]



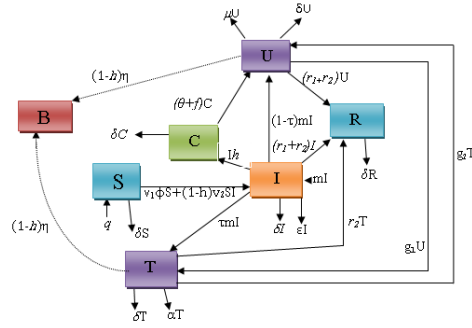


Figure 1: Schematic diagram of the model

### 2.1 Basic assumptions of the model

The following basic assumptions are used in formulating the model. They serve as conditions under which the model will be meaningful and work efficiently.

1. A homogeneous human population is used.
2. Infected individuals are classified into under-treatment and untreated healthy *Vibrio cholerae* carriers.
3. State variables and parameters of the model are positive and remain positive.
4. Only few cholera pathogen carriers develop severe cholera disease (about 75% of cholera infected individuals are healthy cholera pathogen carriers) during a cholera outbreak.
5. Recovery of severe cholera patients is through treatment and asymptomatic cholera pathogen carriers can heal untreated.
6. Recruitment into the population is through birth and immigration while the exit is through migration and natural and disease-related deaths.

These assumptions were used to draw the flow diagram in Figure 1, then used to derive the model’s system of differential equations, as follows:

$$\frac{dS}{dt} = q - v_1\phi S - (1-h)v_2SI - \delta S, \quad (5)$$

$$\frac{dI}{dt} = v_1\phi S + (1-h)v_2SI - (\delta + \varepsilon + r_1 + r_2 + h)I, \quad (6)$$

$$\frac{dC}{dt} = hI - (\theta + f + \delta)C, \quad (7)$$

$$\frac{dU}{dt} = (1-\tau)mI + (\theta + f)C + g_2T - (\delta + r_1 + r_2 + \mu + g_1)U, \quad (8)$$

$$\frac{dT}{dt} = \tau mI + g_1U - (\delta + \alpha + g_2 + r_2)T, \quad (9)$$

$$\frac{dR}{dt} = (r_1 + r_2)I + r_2T + (r_1 + r_2)U - \delta R, \quad (10)$$

$$\frac{dB}{dt} = (1-h)\eta I - \beta B. \quad (11)$$

## 2.2 Existence and uniqueness of solution of the model

Formulated mathematical models need to undergo a surety test to ascertain the existence and uniqueness of their solution. Using Lipschitz criteria as in the definition of basic terms, the existence and uniqueness of a solution of the model are established as follows:

Theorem 1 stated below, is from Derrick and Grossman (1981), also available in [9, 16], together with Theorem 2 are adopted to prove the existence and uniqueness of solution of the model.

**Theorem 1.** Suppose that  $D^1$  denotes the region

$$\begin{aligned} |t - t_0| \leq l, \quad \|a - a_0\| \leq m, \\ a = (a_1, a_2, \dots, a_n), \quad a_0 = (a_{10}, a_{20}, \dots, a_{n0}), \end{aligned}$$

and that

$$\|g(a_1, t) - g(a_2, t)\| \leq p \|a_1 - a_2\|. \quad (12)$$

If the pair  $(a_1, t)$  and  $(a_2, t)$  belong to  $D^1$ , where  $p$  is a positive constant, then there is a constant  $\delta > 0$  such that there exist a unique continuous vector solution  $a(t)$  of the system in the interval  $t - t_0 \leq \delta$ . This condition is satisfied provided that

$$\frac{\partial g_i}{\partial t_j}, \quad i = 1, 2, \dots,$$

is continuous and bounded in  $D^1$ , (where  $i$  represents equations in the system and  $j$  represents the compartments).

Now, considering the region  $0 \leq w \leq \mathbb{R}$ . Bounded solution of the system of equations (5)–(11) will be looked for in the same region whose partial derivatives satisfy  $\delta \leq w \leq \mathbb{R}$ , where  $\delta$  and  $w$  are positive constants, as in the theorem that follows.

**Theorem 2.** Let  $\mathbf{D}$  denotes the region  $0 \leq w \leq \mathbb{R}$ . Then the system of equations (5)–(11) has a unique solution if it is established that

$$\frac{\partial l_i}{\partial t_j}, \quad i = 1, 2, 3, 4, 5, 6, 7,$$

are continuous and bounded in  $\mathbf{D}$ .

The proof of the existence and uniqueness of solution of model two system of equations (5)–(11) are as follows:

*Proof.* Let

$$\begin{aligned} l_1 &= q - v_1\phi S - (1-h)v_2SI - \delta S, \\ l_2 &= v_1\phi S + (1-h)v_2SI - (\delta + \varepsilon + r_1 + r_2 + h)I, \\ l_3 &= hI - (\theta + f + \delta)C, \\ l_4 &= (1-\tau)mI + (\theta + f)C + g_2T - (\delta + r_1 + r_2 + \mu + g_1)U, \\ l_5 &= \tau mI + g_1U - (\delta + \alpha + g_2 + r_2)T, \\ l_6 &= (r_1 + r_2)I + r_2T + (r_1 + r_2)U - \delta R, \\ l_7 &= (1-h)\eta I - \beta B. \end{aligned}$$

Partial derivatives with respect to each state variables of the system (5)–(11) are obtained as follows:

For  $l_1$ ,

$$\begin{aligned} \left| \frac{\partial l_1}{\partial S} \right| &= |-v_1\phi - (1-h)v_2I - \delta| < \infty, & \left| \frac{\partial l_1}{\partial I} \right| &= |-(1-h)v_2S| < \infty, \\ \left| \frac{\partial l_1}{\partial C} \right| &= \left| \frac{\partial l_1}{\partial U} \right| = \left| \frac{\partial l_1}{\partial T} \right| = \left| \frac{\partial l_1}{\partial R} \right| = \left| \frac{\partial l_1}{\partial B} \right| = 0 < \infty. \end{aligned}$$

Similarly, for  $l_2, l_3, l_4, l_5, l_6,$  and  $l_7,$  all have the absolute values of their partial derivatives less than infinity.  $\square$

Solution of the system of equations (5)–(11) exists and is unique in  $\mathbb{R}^7$  since all the partial derivatives are bounded and defined.

### 2.3 Boundedness of solutions of the model

Let  $P_H$  be the human population of the model represented by equations (5)–(10). The variables and parameters used are nonnegative.

**Theorem 3.** All solutions of equations (5)–(10) of the model are bounded in  $\mathbb{R}_+^6,$  and solution to equation (11) is bounded in  $\mathbb{R}_+.$

*Proof.* Let

$$P_H = S + I + C + U + T + R. \quad (13)$$

Differentiating (13) with respect to  $t$  gives

$$\frac{dP_H}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dC}{dt} + \frac{dU}{dt} + \frac{dT}{dt} + \frac{dR}{dt}. \quad (14)$$

Simplifying (14) gives

$$\frac{dP_H}{dt} = q - \delta S - (\varepsilon - m + \delta)I - (\delta)C - (\mu + \delta)U - (\alpha + \delta)T - \delta R. \quad (15)$$

Taking and using any  $\rho > 0,$  multiplying the  $\rho$  by (13), then adding the result to respective sides of (15), and simplifying, thus we have

$$\rho(P_H) = \rho(S + I + C + U + T + R),$$

$$\begin{aligned} \frac{dP_H}{dt} + \rho P_H = & q - (\delta - \rho)S - (\varepsilon - m + \delta - \rho)I - (\delta - \rho)C - (\mu + \delta - \rho)U \\ & - (\alpha + \delta - \rho)T - (\delta - \rho)R. \end{aligned}$$

Let

$$A_q = (\delta - \rho)S - (\varepsilon - m + \delta - \rho)I - (\delta - \rho)C - (\mu + \delta - \rho)U - (\alpha + \delta - \rho)T - (\delta - \rho)R$$

with

$$\rho = \min \{ \delta, \varepsilon - m + \delta, \delta, \mu + \delta, \alpha + \delta, \delta \},$$

which means that  $\rho$  is the minimum value of  $\delta$ . Hence  $\delta - \rho \leq 0$ . Similarly,  $\rho$  is the minimum values of  $\varepsilon - m + \delta$ ,  $\mu + \delta$ , and  $\alpha + \delta$ . This implies  $\varepsilon - m + \delta - \rho \leq 0$ ,  $\mu + \delta - \rho \leq 0$ , and  $\alpha + \delta - \rho \leq 0$ .

Therefore

$$\frac{dP_H}{dt} + \rho P_H = q - A_q$$

but  $A_q \leq 0$ . Taking  $A_q = 0$  gives

$$\frac{dP_H}{dt} + \rho P_H \leq q. \quad (16)$$

Now, using the differential inequality theorem from Gronwall (1919) and the Brikhoff and Rota version as used in [7, 15, 21, 22], (16) gives

$$\frac{dP_H}{dt} \leq q - \rho P_H. \quad (17)$$

Separating the differential inequality (17) gives

$$\frac{dP_H}{q - \rho P_H} \leq dt. \quad (18)$$

Integrating the both sides of (18) gives

$$-\frac{1}{\rho} \ln (q - \rho P_H) \leq t + c. \quad (19)$$

Solving (19) in terms of  $c$  gives

$$-t - \frac{1}{\rho} \ln (q - \rho P_H) \leq c. \quad (20)$$

Evaluating (20) at  $t = 0$  and  $P_H = P_H(0)$  gives

$$-\frac{1}{\rho} \ln (q - \rho P_H(0)) \leq c. \quad (21)$$

Substituting (21) into (19) gives

$$-\frac{1}{\rho} \ln (q - \rho P_H) \leq t + \left( -\frac{1}{\rho} \ln (q - \rho P_H(0)) \right). \quad (22)$$

Multiplying (22) by  $-\rho$  gives

$$\ln(q - \rho P_H) \leq -\rho t + \ln(q - \rho P_H(0)). \quad (23)$$

Taking exponent of (23) gives

$$q - \rho P_H \leq e^{-\rho t} (q - \rho P_H(0)) \quad (24)$$

as  $t \rightarrow \infty$ ,

$$e^{-\rho t} \rightarrow 0.$$

Also,

$$e^{-\rho t} (q - \rho P_H(0)) \rightarrow 0,$$

$$q - \rho P_H \leq 0,$$

so  $P_H \leq \frac{q}{\rho}$ . All state variables and parameters of the model are positive. Hence the sum  $P_H$  is greater than zero. Therefore

$$0 < P_H \leq \frac{q}{\rho}.$$

This cumulatively implies the system of equations (5)–(10) is bounded and its solutions enter into the region

$$H_h = \left\{ (S, I, C, U, T, R) \in \mathbb{R}_+^6 : 0 < P_H \leq \frac{q}{\rho} \right\}. \quad (25)$$

Hence, the solution of equations (5)–(10) is bounded and belongs to feasible region  $H_h$ . On the other hand, the nonhuman population is described by equation (11). Its boundedness is sought as follows.

Let  $P_B$  denote the number of cholera pathogens, so that equation (11) can be written as

$$\frac{dP_B}{dt} = (1 - h)\eta I - \beta P_B.$$

From (13)  $\frac{dP_H}{dt} \leq q - \rho P_H$ . Since  $P_H \leq \frac{q}{\rho}$ , then from the same (13), it implies that  $I \leq \frac{q}{\rho}$ .

Therefore

$$\frac{dP_B}{dt} \leq \frac{q}{\rho}(1 - h)\eta - \beta P_B$$

holds and can be written as

$$\frac{dP_B}{\frac{\eta q}{\rho}(1 - h) - \beta P_B} \leq dt. \quad (26)$$

Integrating the both sides of (26) gives

$$-\frac{1}{\beta} \ln \left( \frac{q\eta}{\rho} (1-h) - \beta P_B \right) \leq t + c_1. \quad (27)$$

Evaluating (27) at  $t = 0$  and  $P_B = P_B(0)$  gives

$$-\frac{1}{\beta} \ln \left( \frac{q\eta}{\rho} (1-h) - \beta P_B(0) \right) < c_1. \quad (28)$$

Substituting (28) into (27) gives

$$-\frac{1}{\beta} \ln \left( \frac{q\eta}{\rho} (1-h) - \beta P_B \right) \leq t - \frac{1}{\beta} \ln \left( \frac{q\eta}{\rho} (1-h) - \beta P_B(0) \right). \quad (29)$$

Multiplying both sides of (29) by  $-\beta$  gives

$$\ln \left( \frac{q\eta}{\rho} (1-h) - \beta P_B \right) \leq -\beta t + \ln \left( \frac{q\eta}{\rho} (1-h) - \beta P_B(0) \right). \quad (30)$$

Taking exponent of both sides of (30) gives

$$\frac{q\eta}{\rho} (1-h) - \beta P_B \leq e^{-\beta t + \ln \left( \frac{q\eta}{\rho} (1-h) - \beta P_B(0) \right)},$$

which can be written as

$$\frac{q\eta}{\rho} (1-h) - \beta P_B \leq e^{-\beta t} \left( \frac{q\eta}{\rho} (1-h) - \beta P_B(0) \right).$$

Then, as  $t \rightarrow \infty$ ,  $e^{-\beta t} \rightarrow 0$ , and also,  $e^{-\beta t} \left( \frac{q\eta}{\rho} (1-h) - \beta P_B(0) \right) \rightarrow 0$ .

it is obtained that

$$\begin{aligned} P_B &\leq \frac{q\eta}{\beta\rho} (1-h), \\ \Rightarrow P_B &\rightarrow \frac{q\eta}{\beta\rho} (1-h). \end{aligned}$$

Therefore

$$0 < P_B \leq \frac{q\eta}{\beta\rho} (1-h).$$

The nonhuman population equation solution is hence bounded and enters the region

$$H_b = \left\{ B \in \mathbb{R}_+, 0 < P_B \leq \frac{q\eta}{\beta\rho} (1-h) \right\}. \quad (31)$$

Altogether, the feasible solutions of the system of equations (5)–(11) are bounded and enter the region

$$H = \left\{ (S, I, C, U, T, R) \in \mathbb{R}_+^6, B \in \mathbb{R}_+, S, I, C, U, T, R, B \geq 0, \right. \\ \left. 0 < P_H \leq \frac{q}{\rho}, 0 < P_B \leq \frac{q\eta}{\beta\rho}(1-h) \right\}. \quad (32)$$

□

### 3 Results of the model using DTM

Let

$$S = s(t), \quad I = i(t), \quad C = c(t), \quad U = u(t), \quad T = t_1(t), \quad R = r(t), \quad B = b_1(t),$$

$$G_1 = \delta + \varepsilon + r_1 + r_2 + h,$$

$$G_2 = \delta + \mu + r_1 + r_2 + g_1,$$

$$G_3 = \delta + \alpha + g_2 + r_2,$$

$$G_4 = \delta + \theta + f,$$

$$G_5 = \theta + f,$$

$$G_6 = \eta - \eta h,$$

$$G_7 = v_1 \phi,$$

$$G_8 = v_2 - v_2 h,$$

$$G_9 = m\tau,$$

$$G_{10} = m - m\tau.$$

The system of ordinary differential equations of the model is now written as

$$\frac{ds(t)}{dt} = q - G_7 s(t) - G_8 s(t)i(t) - \delta s(t), \quad (33)$$

$$\frac{di(t)}{dt} = G_7 s(t) + G_8 s(t)i(t) - G_1 i(t), \quad (34)$$

$$\frac{dc(t)}{dt} = hi(t) - G_4 c(t), \quad (35)$$

$$\frac{du(t)}{dt} = G_{10} i(t) + G_5 c(t) + g_2 t_1(t) - G_2 u(t) \quad (36)$$



$$\frac{dt_1(t)}{dt} = G_9 i(t) + g_1 u(t) - G_3 t_1(t), \quad (37)$$

$$\frac{dr(t)}{dt} = G_5 c(t) + r_1 u(t) + r_2 t_1(t) - \delta r(t), \quad (38)$$

$$\frac{db_1(t)}{dt} = G_6 i(t) - \beta b_1(t). \quad (39)$$

Let  $S(b)$ ,  $I(b)$ ,  $C(b)$ ,  $U(b)$ ,  $T(b)$ ,  $R(b)$ , and  $B(b)$  denote the differential transformation of  $s(t)$ ,  $i(t)$ ,  $c(t)$ ,  $u(t)$ ,  $t_1(t)$ ,  $r(t)$ , and  $b_1(t)$ , respectively. Applying the fundamental operations of DTM provided in Table 1, the following recurrence relations are obtained:

$$S(b+1) = \frac{1}{b+1} \left( qL(b,0) - (G_7 + \delta)S(b) - G_8 \sum_{a=0}^b (S(a)I(b-a)) \right), \quad (40)$$

$$I(b+1) = \frac{1}{b+1} \left( G_7 S(b) + G_8 \sum_{a=0}^b (S(a)I(b-a)) - G_1 I(b) \right), \quad (41)$$

$$C(b+1) = \frac{1}{b+1} (hI(b) - G_4 C(b)), \quad (42)$$

$$U(b+1) = \frac{1}{b+1} (G_{10} I(b) + G_5 C(b) + g_2 T(b) - G_2 U(b)), \quad (43)$$

$$T(b+1) = \frac{1}{b+1} (G_9 I(b) + g_1 U(b) - G_3 T(b)), \quad (44)$$

$$R(b+1) = \frac{1}{b+1} (G_5 C(b) + r_1 U(b) + r_2 T(b) - \delta R(b)), \quad (45)$$

$$B(b+1) = \frac{1}{b+1} (G_6 I(b) - \beta B(b)). \quad (46)$$

Substituting the initial conditions

$$s(0) = 900, i(0) = 50, c(0) = 500, u(0) = 25, t_1(0) = 22, r(0) = 3, b_1(0) = 600$$

and the values of the parameters

$$q = 0.1, \delta = 0.0101, v_1 = 0.5, v_2 = 0.03, \theta = 0.38, h = 0.6, \beta = 0.8, \eta = 10, \\ f = 0.7, g_1 = 0.08, g_2 = 0.2, r_1 = 0.8, r_2 = 0.9, \epsilon = 0.1, \mu = 0.1, \alpha = 0.08, \\ m = 0.4, \tau = 0.5, \phi = 0.000599,$$

into DTM conditions (40) to (46). Using Maple 18, we obtain

$$\begin{aligned}
S(1) &= -549.25955, & I(1) &= 419.76455, \\
C(1) &= -515.05, & U(1) &= 507.1475, \\
S(2) &= -2099.094692, & I(2) &= 1596.031182, \\
C(2) &= 406.6573675, & U(2) &= -716.84851, \\
S(3) &= -4396.37805, & I(3) &= 3121.246753, \\
C(3) &= 171.4405043, & U(3) &= 709.150619, \\
S(4) &= -2483.196769, & I(4) &= 613.668424, \\
C(4) &= 421.4651895, & U(4) &= -129.7781802, \\
S(5) &= 15561.71779, & I(5) &= -15852.50219, \\
C(5) &= -18.24762974, & U(5) &= 170.74677881, \\
S(6) &= 56847.30013, & I(6) &= -50505.80978, \\
C(6) &= -1581.934929, & U(6) &= -585.9512738, \\
T(1) &= -14.1822, & R(1) &= 579.7697, & B(1) &= 320, \\
T(2) &= 70.7014731, & R(2) &= -84.577827, & B(2) &= 3230.1164, \\
T(3) &= 59.23884417, & R(3) &= -23.26776309, & B(3) &= 7650.80193, \\
T(4) &= 152.6203129, & R(4) &= 201.506551, & B(4) &= 1095.82663, \\
T(5) &= -13.8564008, & R(5) &= 97.33658518, & B(5) &= 210.966696, \\
T(6) &= -523.3916988, & R(6) &= 17.2393537, & B(6) &= -42301.46807, \\
& & R(7) &= -378.3539126, & B(7) &= -110607.3974,
\end{aligned}$$

The series solutions of the transformed equations  $b$  ranges from 1 to 6 or 7:

$$\begin{aligned}
s(t) &= \sum_{a=0}^b t^b S(b) = 900 - 549.25955t - 2099.094692t^2 - 4396.37805t^3 \\
&\quad - 2483.196769t^4 + 15561.71779t^5 + 56847.30013t^6 + \dots, \\
i(t) &= \sum_{a=0}^b t^b I(b) = 50 + 419.76455t + 1596.031182t^2 + 3121.246753t^3 \\
&\quad + 613.668424t^4 - 15852.50219t^5 - 50505.80978t^6 + \dots, \\
c(t) &= \sum_{a=0}^b t^b C(b) = 500 - 515.05t + 406.6573675t^2 + 171.4405043t^3 \\
&\quad + 421.4651895t^4 - 18.24762974t^5 - 1581.934929t^6 + \dots, \\
u(t) &= \sum_{a=0}^b t^b U(b) = 25 + 507.1475t - 716.84851t^2 + 709.150619t^3 \\
&\quad - 129.7781802t^4 + 170.74677881t^5 - 585.9512738t^6 + \dots,
\end{aligned}$$

$$\begin{aligned}
t_1(t) &= \sum_{a=0}^b t^b T(b) = 22 - 14.1822t + 70.7014731t^2 + 59.23884417t^3 \\
&\quad + 152.6203129t^4 - 13.8564008t^5 - 523.3916988t^6 + \dots, \\
r(t) &= \sum_{a=0}^b t^b R(b) = 3 + 579.7697t - 84.577827t^2 - 23.26776309t^3 \\
&\quad + 201.506551t^4 + 97.33658518t^5 + 17.2393537t^6 \\
&\quad - 378.3539126t^7 + \dots, \\
b_1(t) &= \sum_{b=0}^7 t^b B(b) = 600 + 330t + 3226.6364t^2 + 7662.06045t^3 \\
&\quad + 10960.11186t^4 + 214.6803636t^5 - 42259.50383t^6 \\
&\quad - 110607.3974t^7 + \dots.
\end{aligned}$$

The Maple 18 plots of  $s(t)$ ,  $i(t)$ ,  $c(t)$ ,  $u(t)$ ,  $t_1(t)$ ,  $r(t)$ , and  $b_1(t)$  are displayed in Figures 2–6.

## 4 Conclusion

The model proposed how hygiene consciousness of individuals influences the susceptible and infected individuals. A high rate of  $h$ , which is hygiene consciousness of individuals, increases number of susceptible individuals while the low rate of  $h$  decreases their number, for details, see Figures 3, 4 and 5. The untreated *Vibro Cholerae* carriers, are seen physically as healthy, because they do not manifest any symptoms of cholera, but spread the pathogen without knowing. The rate of freedom of individuals from *Vibro Cholerae* was determined by their hygiene consciousness (see Figure 6). In Figure 3, the number of susceptible individuals reduced from 900 to about 100 individuals in less than a week, infection reduced to zero in about half of a week, the peak of the infection is 350 out of 900 susceptible individuals when the rate of hygiene consciousness is 63%. While the number of susceptible individuals reduced from 900 to 90 individuals, in about two weeks' time, infection reduced to zero in two weeks, while the peak of infection is 200 out of 900 susceptible individuals, when the rate of hygiene consciousness is 82%. The number of

susceptible individuals dropped from 900 to 400 individuals, in about a week, infection reduced from 50 to zero individuals in the same period, peak of the infection is 50 out of 900 susceptible, when the rate of hygiene consciousness is 99%, as in Figure 5. Reduction of hygiene consciousness results in the prolongation of the cholera illness period to about two weeks and the maximum number of infected individuals reach 200.

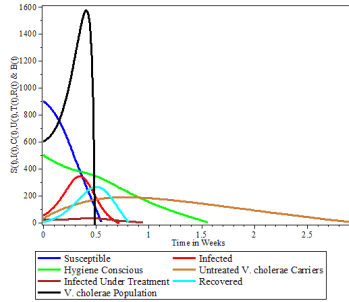


Figure 2: Combined dynamics of the seven compartments of the model plotted against time.

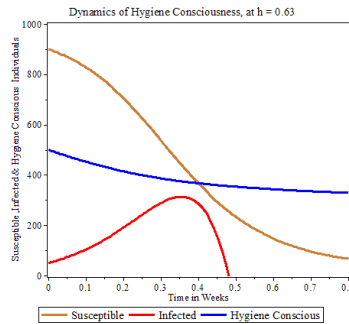


Figure 3: Dynamics of hygiene consciousness, its effect on susceptible and infected individuals at  $h = 0.63$ . As higher hygiene consciousness is maintained, number of individuals susceptible to cholera drastically reduced, and cholera infection stopped.

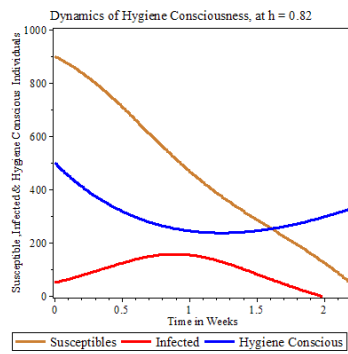


Figure 4: Dynamics of hygiene consciousness, its effect on susceptible and infected individuals at  $h = 0.82$ . Maintaining higher rate of hygiene consciousness, will make the number of individual susceptible to cholera reduced drastically and cholera spread and infection stopped immediately.

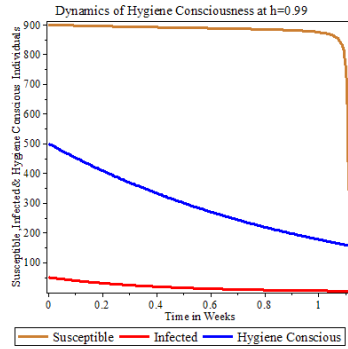


Figure 5: Dynamics of hygiene consciousness, its effect on susceptible and infected individuals at  $h = 0.99$ . Applying and maintaining about 100% hygiene consciousness, in societies, will within just about 10 days, make the number of susceptible individuals to approach zero as well as the rate of cholera infection.

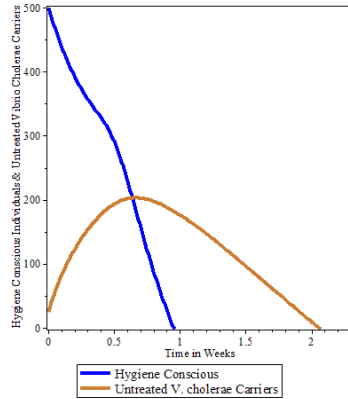


Figure 6: Effect of hygiene consciousness on untreated *Vibrio cholerae* bacteria carriers.

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