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# Global Dynamics of a Water-Borne Disease Model with Multiple Transmission Pathways

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## Abstract

We propose and analyze a water born disease model introducing water-to-person and person-to-person transmission and saturated incidence. The disease-free equilibrium and the existence criterion of endemic equilibrium are investigated. Trans critical bifurcation at the disease-free equilibrium is obtained when the basic reproductive number is one. The local stability of both the equilibria is shown and a Lyapunov functional approach is also applied to explore the global stability of the system around the equilibria. We display the effects of pathogen contaminated water and infection through contact on the system dynamics in the absence of person-to-person contact as well as in the presence of water-to-person contact. It is shown that in the presence of water-to-person transmission, the model system globally stable around both the disease-free and endemic equilibria. Lastly, some numerical simulations are provided to verify our analytical results.

**Keywords:** Water-borne disease, epidemic model, basic reproductive number, global stability, transcritical bifurcation

**MSC 2010:** 92D30, 37N25, 34D23

## 1. Introduction

In mathematical epidemiology it is interesting to study the water-borne disease spreading primarily through contaminated water. In recent years, people have been facing the major problems that are related to water quantity or quality issues. Water-borne diseases like hepatitis, cholera, dysentery and typhoid are the more common infectious diseases that affect large populations in the tropical regions. These diseases are spread either directly or through flies or filth. Hepatitis A and hepatitis E viruses, while unrelated to one another, are both transmitted via the faecal oral route, most often through contaminated water and from person-to-person. Nasser (1994) discussed prevalence and fate of hepatitis A virus in water and which is transmitted primarily through person-person contact, with contaminated water providing a secondary transmission route. Cholera is a diarrhoeal illness caused by infection of the intestine with the bacterium Vibrio cholerae. Most intestine diseases are infectious and transmitted through faecal washed (see Hopkins et al. (1986), Laursen et al. (1994), Hrudey et al. (2003)). Pathogens-which include virus, bacteria, protozoa and parasitic worms-are disease producing agents found in the faeces or infected persons. These pathogens directly travel through water sources or through person with handling water and food.

Now, outbreaks of water-borne diseases are at the top of the list among other infectious diseases whereas cholera is the most frequent disease followed by acute diarrhea and typhoid fever (see WHO 2002, http://www.who.int/whr/2002/overview/en/index.html). A life-threatening situation has been happened in Sunderbans, Calcutta, India- when Cyclone Alia hit the Bay of Bengal at midnight on $26^{th}$ May 2009 and in just 15 minutes everything was flooded. A lack of hygiene and sanitation makes people more susceptible to diarrhoeal diseases and 100,000 diarrheoal cases resulting in 31 deaths have been reported by the government. Incorporating a class for severe infections as well as a class for mild or in apparent infections into a two-path cholera model, King et al. (2008) produced long likelihood estimates using 1900's mortality data in 26 Bengal districts and indicate that the districts of Bogra and Calcutta are well described by the two-path model, yet differ greatly in several parameter estimates, most particularly the estimated proportion of infections resulting in severe symptoms.

In epidemiological model, the disease transmission rate plays an important role in incidence form. The bilinear incidence rate $\beta SI$ and the standard incidence rate $\beta SI / N$ were assumed in most of the epidemic model, where $S$ and $I$ are the susceptible and infected individuals respectively, $N$ is the total population and $\beta$ is the disease transmission rate. Several authors (see Anderson and May (1978), Ruan and Wang (2003), Korobeinikov and Maini (2004), Zhang and Teng (2008)) have suggested that the disease transmission procedure may follow saturation incidence form. Kar and Mondal (2011) elucidated global dynamics of delayed *SIR* epidemic model introducing the incidence term is of saturated form with the susceptible. They investigated the local and global stability of the system around the endemic equilibrium, and also presented how the basic reproduction number varies with the saturation factor. Assuming the force of infection for human population interaction as saturation form, Cai et al. (2009) studied the global dynamics of dengue epidemic mathematical model. They investigated global stability of disease-free equilibrium and endemic equilibrium based on Lyapunov functional approach. Zhang et al. (2008) analyzed delayed *SIR* model with nonlinear incidence rate which is saturated with the

susceptible and found that the global dynamics of the system around the disease-free equilibrium are completely determined by the values of the threshold value $R_0$ and time delay.

In this paper, the disease transmission process will be regarded as multiple ways viz. 'water-to-person' transmission and 'person-to-person' transmission. Tien and Earn (2010) discussed multiple transmission path ways and disease dynamics in a waterborne pathogen model including a pathogen compartment into the classical *SIR* model. Both person-to-person transmission and water-to-person transmission were included as bilinear incidence form and their analysis illustrated how multiple transmission routes and persistence in a reservoir outside of human hosts can affect fundamental characteristics such as the basic reproductive number and epidemic growth rate. Kistemann et al. (2002) found that floods make extremely large contributions to load the bacterial and parasite in drinking water reservoirs. Their results showed that substantial shares of the total microbial loads in watercourses and in drinking-water reservoirs result from rainfall and extreme runoff events. The dynamics of floods during runoff events correspond well with drastic increases in turbidity.

So, if different types of pathogens initially load in water, then a high infection rate will be taken place in a community. In this paper, we consider the disease transmission rate from water-to-person as saturation incidence rate of the form $\beta_W SW/(\alpha_1 + W)$, where $W$ denotes the concentration of pathogens in a water source, $\beta_W$ is the transmission coefficient and $\alpha_1$ is the saturation factor. Moreover, from a practical point of view, the disease transmission rate from person-to-person is assumed saturation incidence rate instead of the bilinear incidence rate in Tien and Earn (2010) and the force of infection is in this version $\beta SI/(\alpha_2 + S)$ which is saturated with the susceptible.

Rest of this paper is arranged as follows. In section 2, *SIWR* model is constructed for illustrating the multiple transmission pathways and some basic results are provided. Also, the existence of equilibria and some simulations are investigated in this section. Section 3 includes the bifurcation analysis at the disease-free equilibrium, local stability of both the equilibria, and the global stability of the system around the endemic equilibrium and then some numerical results are provided in this section. We discuss a sub model in section 4 when only water-to-person transmission is applied to the original model system. The objective of this paper is to compare the original model to that sub model. In section 4, it is discussed that both the disease-free and endemic equilibria are globally asymptotically stable and in addition some numerical simulations are presented in this section. Concluding remarks close the paper by section 5.

## 2. *SIWR* Model for Water-Borne Disease

We consider the following system of differential equations as follows:

$$\begin{aligned} \frac{dS}{dt} &= \mu N - \frac{\beta_W SW}{\alpha_1 + W} - \frac{\beta SI}{\alpha_2 + S} - \mu S, \\ \frac{dI}{dt} &= \frac{\beta_W SW}{\alpha_1 + W} + \frac{\beta SI}{\alpha_2 + S} - (\gamma + \mu) I, \end{aligned} \tag{1}$$

$$\frac{dW}{dt} = \sigma I - \delta W,$$
$$\frac{dR}{dt} = \gamma I - \mu R,$$

where $S$, $I$ and $R$ are respectively denoted as the density of susceptible, infected and recovered individuals within the population, $W$ represents the pathogen concentration in water source, the total constant population is $N = S + I + R$, $\beta_W$ and $\beta$ are the disease transmission coefficients for water-to-person and person-to-person contact respectively. If $A$ is the total recruitment rate, then we obtain $dN/dt = A - \mu N$, where $\mu$ is the natural death rate. In our paper, as the total population $N$ is constant, so $\mu N$ is the total recruitment rate. In this scenario the birth rate is equal to the natural death rate $\mu$. Also, $\alpha_1$ and $\alpha_2$ are the saturation factors, $\gamma$ is the recovery rate, that is, an individual who acquire an infection stay in the infected class during a period of time $\gamma^{-1}$ and $(\gamma + \mu)^{-1}$ indicates the mean time spent in the infected class, $\sigma$ is the pathogen shedding rate per day per infected individual, $\delta$ is the pathogen particle inactivation rate in water.

Set

$$\bar{S} = \frac{S}{N}, \ \bar{I} = \frac{I}{N}, \ \overline{W} = \frac{\delta}{\sigma N} W \text{ and } \bar{R} = \frac{R}{N}.$$

For simplicity, we ignore the upper scripts and then the system (1) is transformed to the following system:

$$\begin{aligned}
\frac{dS}{dt} &= \mu - \frac{\beta_W SW}{a + W} - \frac{\beta SI}{b + S} - \mu S, \\
\frac{dI}{dt} &= \frac{\beta_W SW}{a + W} + \frac{\beta SI}{b + S} - (\gamma + \mu) I, \\
\frac{dW}{dt} &= \delta (I - W), \\
\frac{dR}{dt} &= \gamma I - \mu R,
\end{aligned} \tag{2}$$

where

$$a = \frac{\delta \alpha_1}{\sigma N}, \ b = \frac{\alpha_2}{N}.$$

In this paper, a simple extension of the classical *SIR* model is considered by adding a extra compartment *W* (a waterborne pathogen). We also assume that those individuals who have been recovered from the waterborne infection will not be infected again by the same disease. Thereby,

$S$ and $W$ are independent of $R$. As $R = N - S - I$ and $S$, $W$ are independent of $R$, so, the dynamical behaviour of $S$, $I$ simply give the dynamical behaviour $R$. Therefore, we do not consider the last equation of (2) in our discussion. Now, we shall study the following nonlinear ordinary differential equations:

$$\begin{aligned}
\frac{dS}{dt} &= \mu - \frac{\beta_W SW}{a+W} - \frac{\beta SI}{b+S} - \mu S, \\
\frac{dI}{dt} &= \frac{\beta_W SW}{a+W} + \frac{\beta SI}{b+S} - (\gamma + \mu) I, \\
\frac{dW}{dt} &= \delta(I - W).
\end{aligned} \tag{3}$$

For the bounded ness and persistence of the system (3), we state the following two important lemmas.

**Lemma 1.**

For any $\varepsilon > 0$, the set $\Theta = \{(S, I, W) \,|\, S, I, W \geq 0, S + I < 1, W \leq \varepsilon + 1\}$ is a positive invariant subset of $R^3$.

***Proof:***

The proof is evidently true. □

**Lemma 2.**

For every positive solution $(S(t), I(t), W(t))$, the system (3) is uniformly persistent (Kuang, 1993).

***Proof:***

From the first equation of the system (3), we obtain

$$\frac{dS(t)}{dt} \geq -\mu S(t),$$

which implies that $\liminf_{t\to\infty} S(t) \geq S(0) > 0$. Similarly, from the other equations of the system (3), we can show that

$$\liminf_{t\to\infty} I(t) \geq I(0) > 0, \ \liminf_{t\to\infty} W(t) \geq W(0) > 0.$$

Hence, the system is uniformly persistent for every positive solution. □

***Note* 1:**

From the lemma 1, we can conclude that all solutions starting in the positive invariant set $\Theta$ stay in $\Theta$, whereas the lemma 2 indicates the existence of positive solution of the system (3).

### 2.1. Equilibria and their Existence Criteria

Equilibrium analysis of the model (3) is stated in the following theorem:

**Theorem 1.**

The system (3) has two equilibria, namely

(i) disease-free equilibrium $D^0 = (1,0,0)$,

and

(ii) unique positive equilibrium $D^e = \left(S^e, I^e, W^e\right)$,

where $I^e = \frac{\mu(1-S^e)}{\mu+\gamma}$, $W^e = I^e$ and $S^e = \frac{-x_2 + \sqrt{x_2^2 - 4x_1x_3}}{2x_1}$.

The second equilibrium exists if $R_1 > 1$, where

$$R_1 = \frac{(\gamma+\mu)(\beta_W+\mu)}{\mu\beta}$$

and the other symbols are stated in the proof.

***Proof:***

We prove the existence of equilibria as follows. Setting right hand side of the system (3) to zero, we obtain

$$\mu - \frac{\beta_W SW}{a+W} - \frac{\beta SI}{b+S} - \mu S = 0,$$

$$\frac{\beta_W SW}{a+W} + \frac{\beta SI}{b+S} - (\gamma+\mu)I = 0,$$

$$I - W = 0.$$

From these three equations, it can be concluded that

(i) The existence of the disease-free equilibrium $D^0 = (1, 0, 0)$ is obvious.

(ii) The endemic equilibrium is $D^e = \left(S^e, I^e, W^e\right)$,

where

$$I^e = \frac{\mu(1 - S^e)}{\mu + \gamma},\ W^e = I^e,$$

and $S^e$ is a positive root of the following equation:

$$x_1 S^2 + x_2 S + x_3 = 0, \tag{4}$$

where

$$x_1 = (\gamma + \mu)(\beta_W + \mu) - \mu\beta,$$
$$x_2 = a\mu(\gamma + \mu)((\beta - \gamma - \mu) + \beta\mu + (b-1)\mu(\gamma + \mu) + b\beta_W(\gamma + \mu), \text{ and}$$
$$x_3 = -b(\gamma + \mu)\{\mu + a(\gamma + \mu)\}.$$

If $R_1 > 1$, then $x_1 > 0$ and therefore, there is only one sign change in the coefficients of the equation (4). By Descartes' rule of signs, the number of positive root of (4) is exactly one. Therefore,

$$S^e = \frac{-x_2 + \sqrt{x_2^2 - 4x_1x_3}}{2x_1}.$$

Hence, the theorem 1(ii) is proved. □

For the system (3), the basic reproductive number is defined as

$$R_0 = \frac{1}{\gamma + \mu}\left[\frac{\beta}{b+1} + \frac{\beta_W}{a}\right]. \tag{5}$$

***Note* 2:**

From the theorem 1, we obtain a threshold parameter $R_1$ which provides the existence of endemic equilibrium of the system (3). Moreover, it is an increasing function of water-to-person transmission coefficient and inversely proportional to the person-to-person contact rate. If the total mortality of infected individual decreases that is if the mean infectious period increases, the value of $R_1$ decreases. Now, we want to remind that this threshold value isn't the basic

reproductive number. Comparing the basic reproductive number $R_0$ with another threshold value $R_1$, it is cleared that if $R_1 > R_0$, then $R_0 > 1$ implies that the system has unique endemic equilibrium. But, when $R_0 > R_1$, then $R_1 > 1$ provides the existence of unique endemic equilibrium of the system (3) and in this case trivially the basic reproduction number is above one.

From the expression of $R_0$, it is observed that $R_0$ increases while the mean time is very long and it decreases when both the saturation factors increase. Variation of basic reproductive number with the saturation factors and recovery rate are plotted in figures 1 and 2 respectively taking the simulated parameter values which are depicted in table 1. In figure 1(i-ii), it is found that the disease will extinct if the value of the pathogen induced saturation factor is above 1.667 where as the basic reproductive number decreases with the saturation factor $b$ very slowly and the disease may wipe out from the community for too much high value of the susceptible induced saturation factor. Also, the basic reproductive number has a sharp decrease is depicted in figure 2 and it is found that when the value of the recovery rate is greater than 26.18, then this noticeably shows that no other recover individuals become infected again. The effect of both the saturation factors on the basic reproductive number is shown in figure 3 and in this figure, it is observed that the basic reproduction number $R_0 = 0.9866$ when the values of $a = 1.593$ and $b = 0.08814$. As the value of the basic reproduction number is below one, the disease-free equilibrium is locally asymptotically stable and so the disease may not persist in the population.

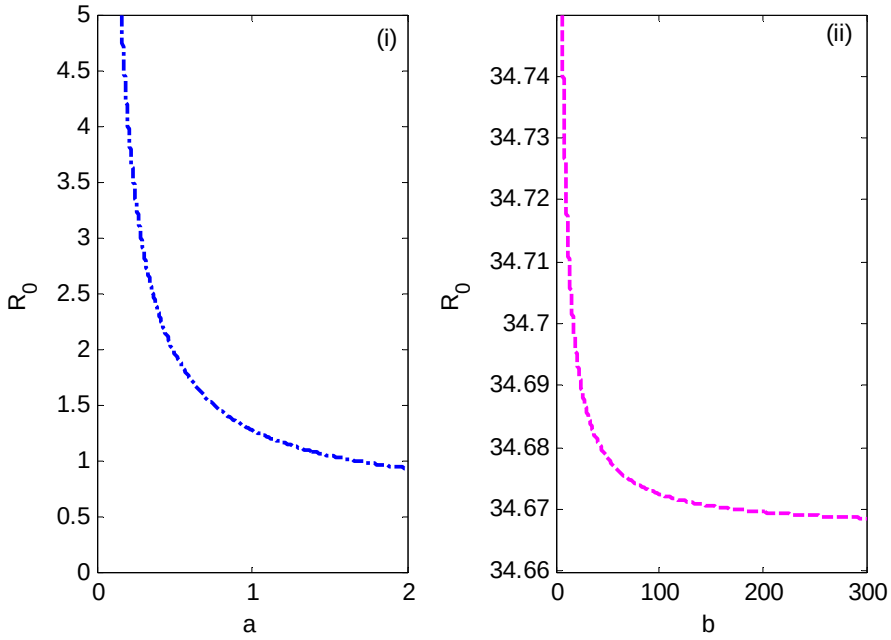


**Figure 1 (a-b):** We plot the basic reproductive number as a function of saturation factors

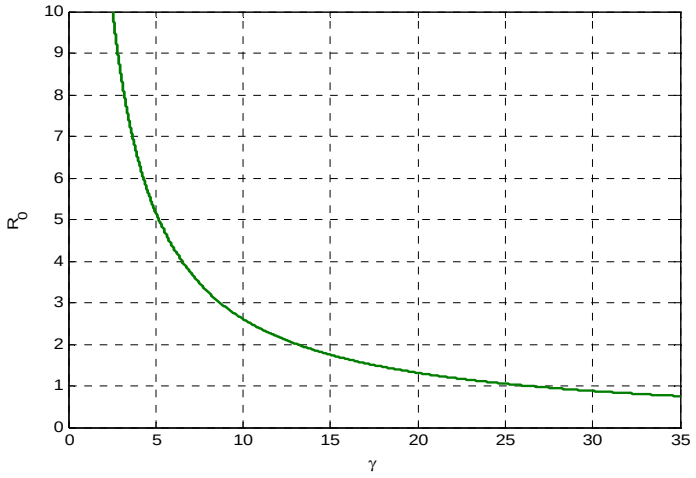


**Figure 2.** Variation of basic reproductive number with recovery rate $\gamma$

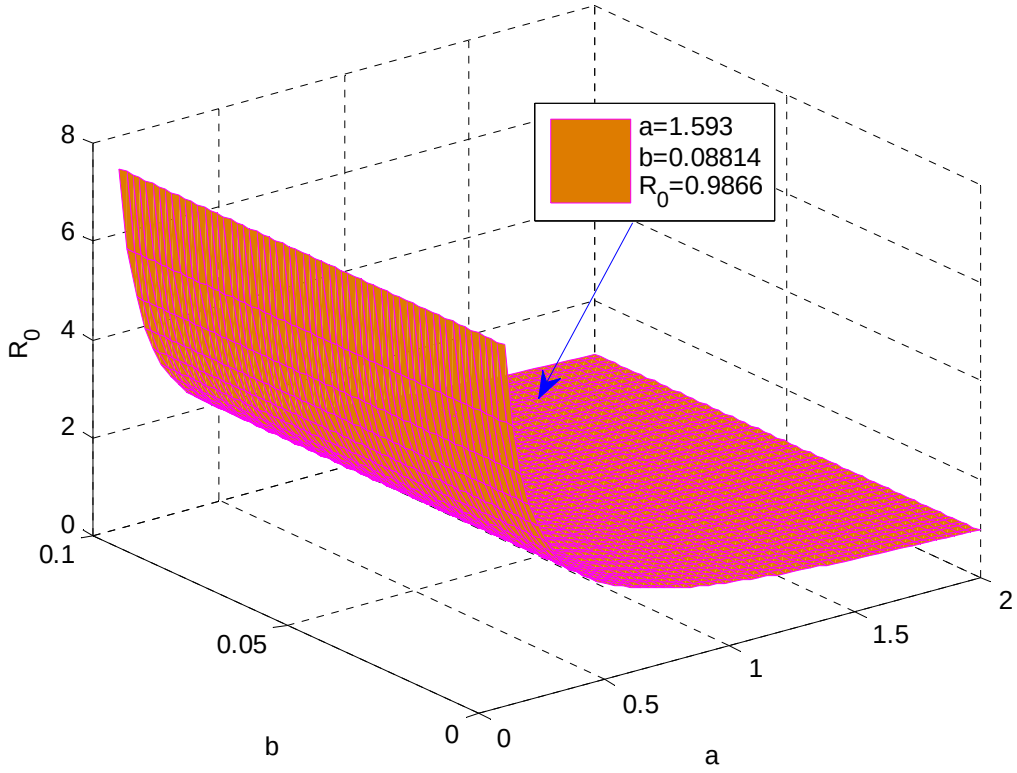


**Figure 3.** Surface plot of basic reproductive number as a function of saturation factors

**Table 1.** The values assigned for the model's parameters

| |
|---|
| $\mu = 0.1,\ \beta_W = 0.52,\ \beta = 0.45,\ a = 0.02,\ b = 0.03,\ \gamma = 0.65,\ \delta = 0.4$ |

## 3. Stability Criteria and Bifurcation Analysis

**Theorem 2.**

The disease-free equilibrium $D^0 = (1,0,0)$ is locally asymptotically stable when $R_0 < 1$ and unstable when $R_0 > 1$.

***Proof:***

The characteristic equation for the system (3) at the disease-free equilibrium is

$$(\lambda + \mu)(\lambda^2 + a_1\lambda + a_2) = 0, \tag{6}$$

where

$$a_1 = \gamma + \mu + \delta - \frac{\beta}{b+1},$$
$$a_2 = \delta\left(\gamma + \mu - \frac{\beta}{b+1}\right) - \frac{\delta\beta_W}{a}.$$

If $R_0 < 1$, then both $a_1$ and $a_2$ are positive. So, the equation (6) has one negative real root and two other roots with negative real parts. Hence, the disease-free equilibrium is locally asymptotically stable when $R_0 < 1$ and unstable when $R_0 > 1$. □

***Note* 3:**

The above theorem shows that the stability of the disease-free equilibrium changes when the basic reproductive number passes through one. In this scenario the system exhibits trans critical bifurcation at disease-free equilibrium when the basic reproductive number is equal to one which is proved in the next theorem.

**Theorem 3.**

The system (3) undergoes trans critical bifurcation at $D^0 = (1,0,0)$ when $R_0 = 1$.

***Proof:***

The Jacobian matrix at the disease-free equilibrium is

$$J(D_0)=\begin{pmatrix} -\mu & -\frac{\beta}{b+1} & -\frac{\beta_W}{a} \\ 0 & \frac{\beta}{b+1}-\gamma-\mu & \frac{\beta_W}{a} \\ 0 & \delta & -\delta \end{pmatrix}. \tag{7}$$

Let

$$f=\begin{pmatrix} \mu-\frac{\beta_W SW}{a+W}-\frac{\beta SI}{b+S}-\mu S \\ \frac{\beta_W SW}{a+W}+\frac{\beta SI}{b+S}-(\gamma+\mu)I \\ \delta(I-W) \end{pmatrix}=\begin{pmatrix} f_1 \\ f_2 \\ f_3 \end{pmatrix} \text{ and } X=(S,I,W)^T.$$

The Jacobian matrix (7) has a geometrically simple zero eigenvalue with left eigenvector $\psi=\begin{pmatrix} 0 & 1 & \frac{\beta_W}{a\delta} \end{pmatrix}$ and right eigenvector $\phi=\begin{pmatrix} -\frac{\gamma+\mu}{\mu} & 1 & 1 \end{pmatrix}^T$.

From the above discussion, we obtain

$$\psi\left(D_X D_{\beta_W} f\right)_{D_0}=1/a>0$$

and

$$\begin{aligned}\psi\left((D_{XX} f)(\phi,\phi)\right)&=\left(\psi\sum_{i=1}^{3} e_i\phi^T\left(D_X(D_X f_i)^T\right)\phi\right)_{D_0}\\ &=-2\left[\frac{b\beta(\gamma+\mu)}{\mu(b+1)^2}+\frac{\beta_W(\gamma+\mu)}{a\mu}+\frac{\beta_W}{a^2}\right]\\ &<0.\end{aligned}$$

Hence, the system (3) undergoes trans critical bifurcation at the disease-free equilibrium $D_0$ (Guckenheimer and Holmes, 1983). □

**Theorem 4.**

The endemic equilibrium point $D^e=\left(S^e,I^e,W^e\right)$ is locally asymptotically stable if $R_1>1$.

***Proof:***

The characteristic equation for the system (3) at the endemic equilibrium is

$$\lambda^3 + A\lambda^2 + B\lambda + C = 0, \tag{8}$$

where

$$A = bp + vS^e + vI^e + \delta + \mu,$$
$$B = (u+\delta)vI^e + v\mu S^e + v^2 S^e I^e + \delta\mu + \delta(vS^e - aq) + bp(u + vS^e + \delta),$$
$$C = \delta\{bp(u + vS^e) + uvI^e + v^2 S^e I^e + \mu(vS^e - aq)\},$$
$$p = \frac{\beta I^e}{(b+S^e)^2},\ q = \frac{\beta_W S^e}{(a+I^e)^2},\ u = \frac{\beta S^e}{b+S^e},\ v = \frac{\beta_W}{a+I^e}.$$

Now,

$$vS^e - aq = \frac{\beta_W S^e I^e}{(a+I^e)^2},$$
$$AB - C = (bp + vS^e + vI^e + \mu)B + \delta^2(vS^e - aq + vI^e + \mu) + aq\delta\mu.$$

Therefore, all the coefficients of (8) are positive and $AB - C > 0$. Hence, the Routh-Hurwitz condition is satisfied. Thus, the theorem. □

To investigate the global stability (see Li et al. (2001), McCluskey (2006)) of the system around the endemic equilibrium, we give an analytical proof in the next theorem using Lyapunov functional approach.

**Theorem 5.**

For $R_1 > 1$, the endemic equilibrium $D^e$ of system (3) exists and is globally asymptotically stable (GAS) provided $2ab(\gamma + \mu) > a\beta(2b + I^e) + b\beta_W(3S^e + a)$ and $F > 0$, where the symbol is stated in the proof of this theorem.

***Proof:***

We define a Lyapunov function as follows:

$$V(S,I,W) = d_1(S - S^e)^2 + d_2(I - I^e)^2 + d_3(W - W^e)^2, \tag{9}$$

where $d_1$, $d_2$ and $d_3$ are arbitrary positive constants to be chosen afterward.

The time derivative of $V(S,I,W)$ along the solution path is given by

$$\frac{dV}{dt} = 2d_1\frac{d(S-S^e)}{dt} + 2d_2\frac{d(I-I^e)}{dt} + 2d_3\frac{d(W-W^e)}{dt}$$
$$\leq -2d_1\left[\frac{\beta_W W}{a+W} + \frac{b\beta I^e}{(b+S^e)(b+S)} + \mu - \beta\right](S-S^e)^2$$
$$-\left[2d_2\left(\gamma+\mu-\beta-\frac{\beta_W S^e}{a}\right) - \delta d_3 - d_1\beta\right](I-I^e)^2$$
$$+2\left[d_2\left(\beta_W + \frac{\beta I^e}{b}\right) - d_1\beta\right](S-S^e)(I-I^e) - \left[\delta d_3 - \frac{d_2\beta_W S^e}{a}\right](W-W^e).$$

Let us choose

$$d_1 = \frac{1}{2},\ d_2 = \frac{b\beta}{2(\beta I^e + b\beta_W)},\ d_3 = \frac{b\beta\beta_W S^e}{2a\delta(\beta I^e + b\beta_W)}.$$

Then,

$$\frac{dV}{dt} \leq -F(S-S^e)^2 - G(I-I^e)^2,$$

where

$$F = \frac{\beta_W W}{a+W} + \frac{b\beta I^e}{(b+S^e)(b+S)} + \mu - \beta,$$

$$G = \frac{2ab\beta(\gamma+\mu) - a\beta^2(2b+I^e) - b\beta\beta_W(3S^e+a)}{2a(\beta I^e + b\beta_W)}.$$

Thus, $\dot{V}(S,I,W) \leq 0$, provided $2ab(\gamma+\mu) > a\beta(2b+I^e) + b\beta_W(3S^e+a)$ and $F > 0$, with equality only at $(S=S^e,\ I=I^e,\ W=W^e)$. Hence, the system (3) is globally asymptotically stable around its endemic equilibrium. □

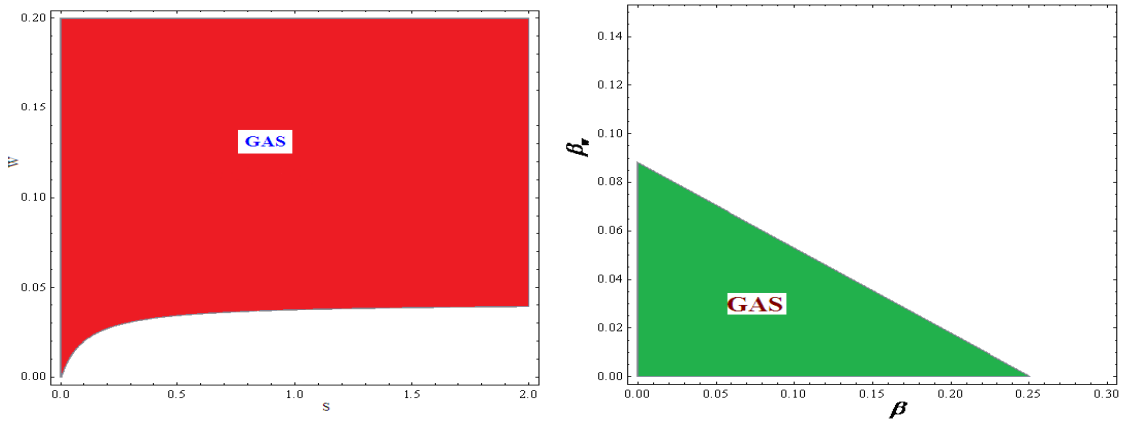


**Figure 4:** The global stability regions in $(S, W)$ and $(\beta, \beta_W)$ – plane respectively are presented for the endemic equilibrium. The numerical values for the other parameters are chosen from Table 1

**Remark 1.**

From the above theorem, it can be concluded that the disease surely persist in the total population as the endemic equilibrium globally stable in our feasible region.

### 3.1. Simulations

In this section, the numerical results are used to confirm and visualize our analytical findings. For this purpose a simulated set of parameters value are chosen, given in table 1. Then the system (3) has a unique endemic equilibrium $(0.106679, 0.11911, 0.11911)$ and the basic reproduction number $R_0 = 35.2492 > 1$. Also, the another important threshold parameter $R_1 = 10.33 < R_0$. The eigenvalues of the system at the endemic equilibrium are $-0.444341$, $-0.492871 \pm 0.382799i$. Therefore, the endemic equilibrium is locally asymptotically stable (see figure 5) because of the negative real parts of the eigenvalues. The figure 5 indicates that the susceptible individuals decrease in numbers within small time interval and then increases, and converge to the endemic point. It has been also observed in Figure 5 that both the infected individuals and pathogen concentration increase sharply during a period of time and at the end of time of our noticeable time period both these decrease and then comes together to their endemic point. Biologically the Figure 5 indicates that the disease spreads into the total population within a small period of time and after then the disease becomes endemic. The bifurcation diagram is presented in Figure 6 and it has been understood that our model system will be globally stable around the endemic equilibrium.

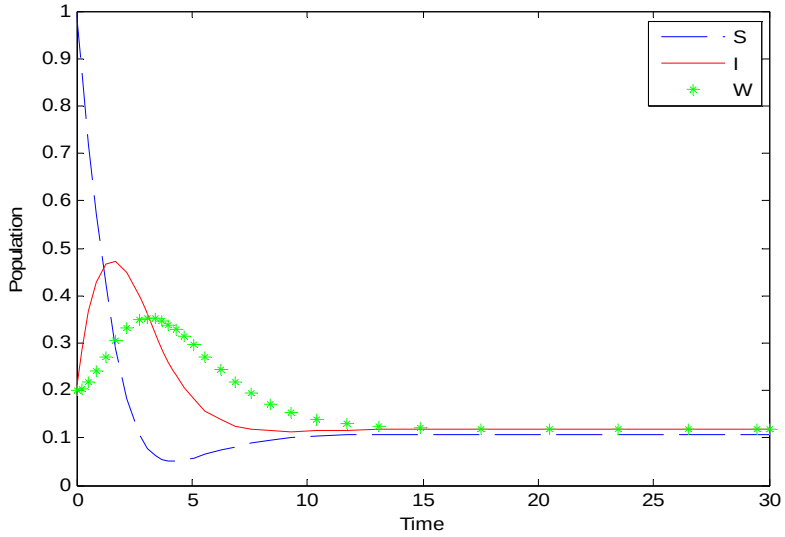


**Figure 5.** Solution curves for susceptible individuals, infected individuals and pathogen concentration

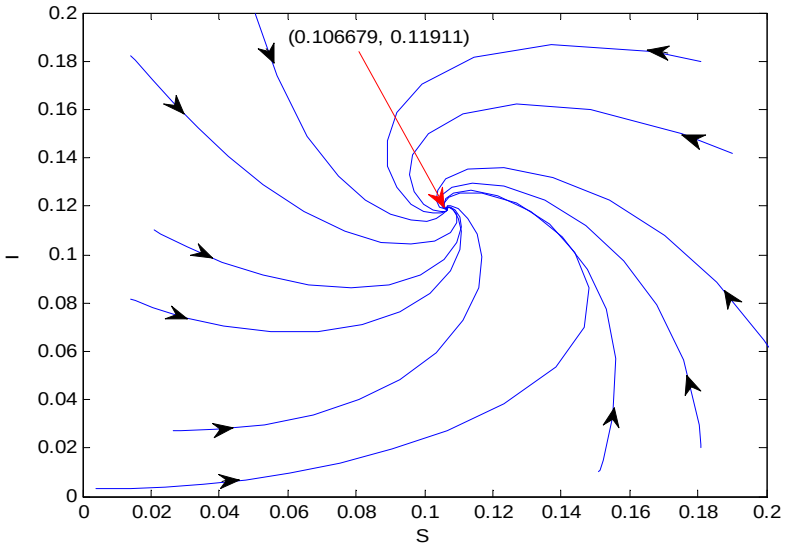


**Figure 6.** Bifurcation diagram in $(S, I)$ -plane indicates that the endemic equilibrium is globally asymptotically stable

## 4. Model in Absence of Person-to-Person Transmission

In this section, we will concern about such a waterborne disease model in which the disease is transmitted only through contaminated water to the person. In many urban or municipal areas (e.g. Kolkata, Howrah etc. in West Bengal, India), most of the water supply pipe lines are not in good position being used for long times. Thereby, different types of harmful virus viz. Rota, Hepatitis, Parvo etc., bacteria viz. Cholera, Salmonella, Escherisia Coli are mixed into the drinking water through the leakage of pipe lines. So, drinking water gets contaminated from the supply source of water and spreading various infections in population. In recent years, arsenic in West Bengal (India) available geogenically resulting 50% to 60% rural population of about 5 million people are victim of arsenic containing water. So, our model system (3) will be analyzed in this section when the susceptible individuals become infected only through contact with contaminated water.

Then, the system (3) reduces to

$$\begin{aligned}
\frac{dS}{dt} &= \mu - \frac{\beta_W SW}{a+W} - \mu S, \\
\frac{dI}{dt} &= \frac{\beta_W SW}{a+W} - (\gamma+\mu)I, \\
\frac{dW}{dt} &= \delta(I-W).
\end{aligned} \tag{10}$$

The basic reproductive number for the system (10) is defined by

$$R_0^{WP} = \frac{\beta_W}{a(\gamma + \mu)}. \tag{11}$$

Clearly, the basic reproductive number is an increasing function of the disease transmission rate and also of the mean time. It is found that $R_0^{WP}$ decreases if the saturation factor $a$, that measures the inhibitory effect, increases. Moreover, if we compare both the basic reproductive numbers in (5) and (11), then it is seen that $R_0 > R_0^{WP}$. It indicates that both the type of transmissions is possible, when $R_0^{WP} > 1$ and the disease will persist in a population.

### 4.1. Equilibria and Stability Criteria

**Theorem 6.**

The system (10) has also disease-free equilibrium $(S^0, I^0, W^0) = (1, 0, 0)$ and it is locally asymptotically stable for $R_0^{WP} < 1$ and unstable for $R_0^{WP} > 1$.

***Proof:***

The proof is trivially true. □

**Theorem 7.**

(a) If the basic reproductive number $R_0^{WP} > 1$, then the system (10) has unique endemic equilibrium point $E^* = (S^*, I^*, W^*)$, where

$$S^* = \frac{a\gamma + a\mu + \mu}{\beta_W + \mu}, \quad I^* = W^* = \frac{a\mu\left(R_0^{WP} - 1\right)}{\beta_W + \mu}.$$

(b) The endemic equilibrium is locally asymptotically stable if $R_0^{WP} > 1$.

***Proof:***

(a) One can easily verify this theorem by equating the right side of all the equations of system (10) to zero.

(b) The result is true using theorem 3. □

**Theorem 8.**

If $R_0^{WP} < 1$, then the disease-free equilibrium of the system (10) is GAS in $\Theta_0 = \{(S^0, I^0, W^0) \in \Theta : I^0 = 0 = W^0\}$.

***Proof:***

Using the approach of Mwasa and Tchuenche (2011), consider an average Lyapunov function of the form

$$V_1(S,I,W) = S^{h_1} I^{h_2} W^{h_3}, \quad \text{with} \quad h_i > 0; \quad i = 1,2,3. \tag{12}$$

Taking time derivative of (12), we have

$$\begin{aligned}\frac{\dot{V}_1}{V_1} &= h_1 \frac{\dot{S}}{S} + h_2 \frac{\dot{I}}{I} + h_3 \frac{\dot{W}}{W} \\ &= h_1\left[\frac{\mu}{S} - \mu - \frac{\beta_W W}{a+W}\right] + h_2\left[\frac{\beta_W W}{a+W}\cdot\frac{S}{I} - (\gamma+\mu)\right] + h_3\left[\delta\frac{I}{W} - \delta\right] \\ &\le h_1\left(\frac{\mu}{S} - \mu\right) + h_2\big(-(\gamma+\mu)\big) + h_3(-\delta).\end{aligned}$$

Thus,

$$\frac{\dot{V}_1}{V_1} \le -h_1\frac{\mu}{S}(S-1) - h_2(\gamma+\mu) - h_3\delta.$$

Since $S^0 = 1$, so $\dot{V}_1 \le 0$ for $S > S^0$ and the equality holds at $(S^0, I^0, W^0) = (1,0,0)$. Hence, the disease-free equilibrium is GAS for $S > S^0$. □

**Theorem 9.**

If $R_0^{WP} > 1$, then the system (10) is GAS around endemic equilibrium in $\Theta \setminus \Theta_0$ provided $2(\gamma+\mu)W > \delta I$ and $(a+W^*)SW \le (a+W)S^*W^*$.

***Proof:***

Using the approach of Korobeinikov (2006), we define a Lyapunov function as

$$V_2(S,I,W) = S^*\left(\frac{S}{S^*} - \ln\frac{S}{S^*}\right) + I^*\left(\frac{I}{I^*} - \ln\frac{I}{I^*}\right) + W^*\left(\frac{W}{W^*} - \ln\frac{W}{W^*}\right). \tag{13}$$

This function is defined and continuous for all $S, I, W > 0$ and satisfies

$$\frac{\partial V_2}{\partial S} = 1 - \frac{S^*}{S}, \ \frac{\partial V_2}{\partial I} = 1 - \frac{I^*}{I}, \ \frac{\partial V_2}{\partial W} = 1 - \frac{W^*}{W}.$$

Hence, it is easy to see that $E^*$ is the only extremum and the global minimum of the function in the positive octant $R_+^3$. Consequently, the function (13) is indeed a Lyapunov function (see Lyapunov (1992)). Taking time derivative of (13) along the positive solution of the system (10), we obtain

$$\begin{aligned}
\dot{V}_2 &= \left(1-\frac{S^*}{S}\right)\dot{S} + \left(1-\frac{I^*}{I}\right)\dot{I} + \left(1-\frac{W^*}{W}\right)\dot{W} \\
&= \left(1-\frac{S^*}{S}\right)\left(\mu - \mu S - \frac{\beta_W SW}{a+W}\right) + \left(1-\frac{I^*}{I}\right)\left(\frac{\beta_W SW}{a+W} - (\gamma+\mu)I\right) + \left(1-\frac{W^*}{W}\right)\delta(I-W) \\
&= \left(1-\frac{S^*}{S}\right)\left(\mu S^* + \frac{\beta_W S^* W^*}{a+W^*} - \mu S - \frac{\beta_W SW}{a+W}\right) \\
&\quad + \left(1-\frac{I^*}{I}\right)\left(\frac{(\gamma+\mu)(a+W^*)I^* SW}{(a+W)S^*W^*} - (\gamma+\mu)I\right) + \delta\left(1-\frac{W^*}{W}\right)\left((I-I^*)-(W-W^*)\right) \\
&\le -\left(\mu + \frac{\beta_W W}{a+W}\right)\frac{(S-S^*)^2}{S} - \left(\frac{2(\gamma+\mu)W - \delta I}{2W}\right)\frac{(I-I^*)^2}{I} - \frac{\delta}{2}\frac{(W-W^*)^2}{W}.
\end{aligned}$$

Therefore, $\dot{V}_2(S,I,W) \le 0$, provided $2(\gamma+\mu)W > \delta I$ and equality holds at $(S=S^*, I=I^*, W=W^*)$. Hence, the endemic equilibrium is GAS. □

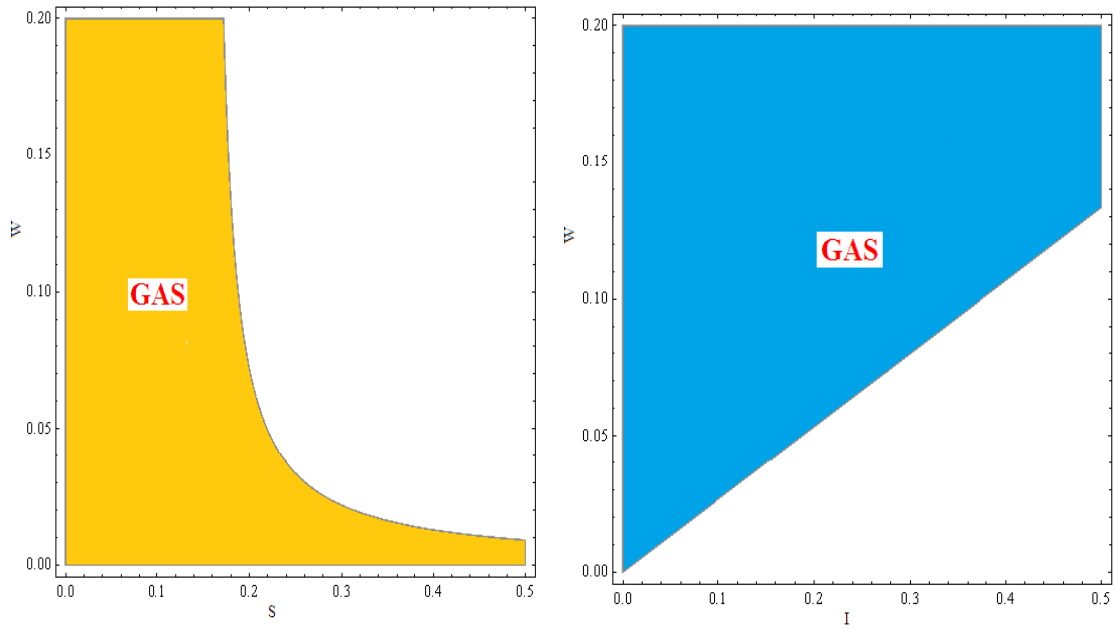


**Figure 7.** The global stability regions in $(S, W)$ and $(I, W)-$ plane respectively are presented for the endemic equilibrium. The numerical values for the other parameters are chosen from table 1.

**Remark 2.**

From the theorem 8, it is confirmed that the population may be totally disease free in presence of water-to-person disease transmission while the basic reproductive number is below one and when its value above one, the theorem 9 shows that our system is globally asymptotically stable around the endemic equilibrium in the regions, presented in figure 6. It implies that the disease may not extinct totally from the community.

## 5. Sensitivity Analysis

To determine the robustness of the model system, it is useful to carry out a sensitivity analysis of the system with respect to some important parameters. The disease prevalence is directly related to the endemic equilibrium. The most sensitive parameters in our water dynamics are the multiple contact rates, the decay rate of pathogen in the water source and both the saturation factors. So, we discuss the sensitivity analysis of the system (3) varying the disease transmission coefficients $\beta$, $\beta_w$, the decay rate of pathogen $\delta$ and the saturation factors $a$, $b$. In figure 9, we see that the number of infected and the pathogen populations are directly proportional with the force of infection $\beta$, but the susceptible population is inversely proportional with the force of infection. If the time is taken as days, then it is found that the number of infected individuals increase fast within one and half days where as the pathogen concentration increases rapidly after two days.

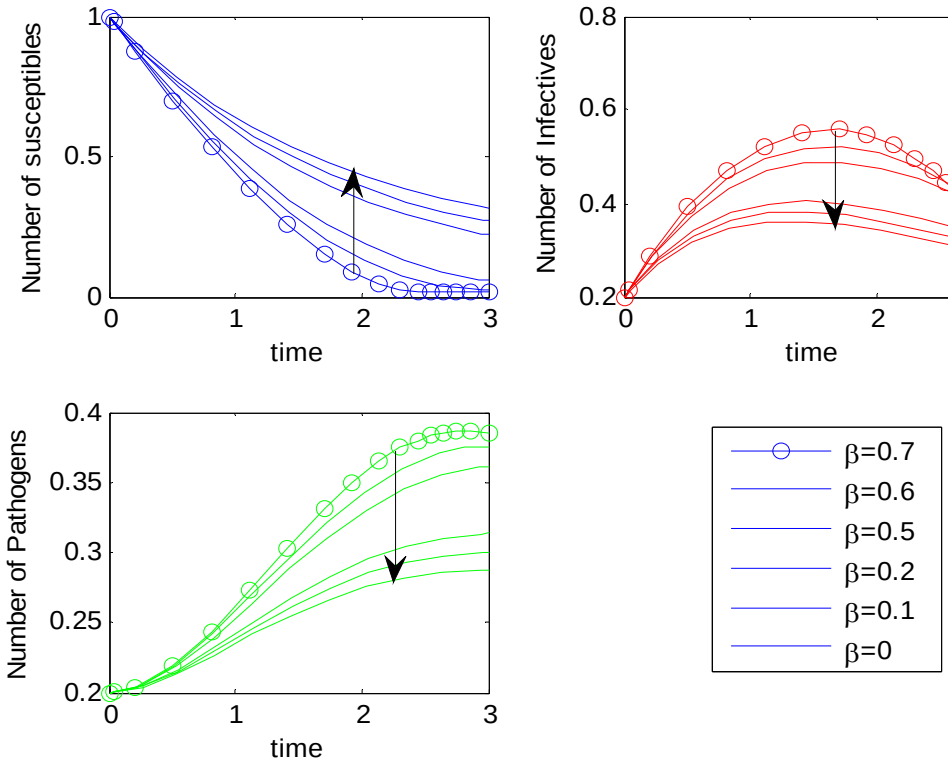


**Figure 9.** Sensitivity of the system (3) for different values of person-to-person transmission

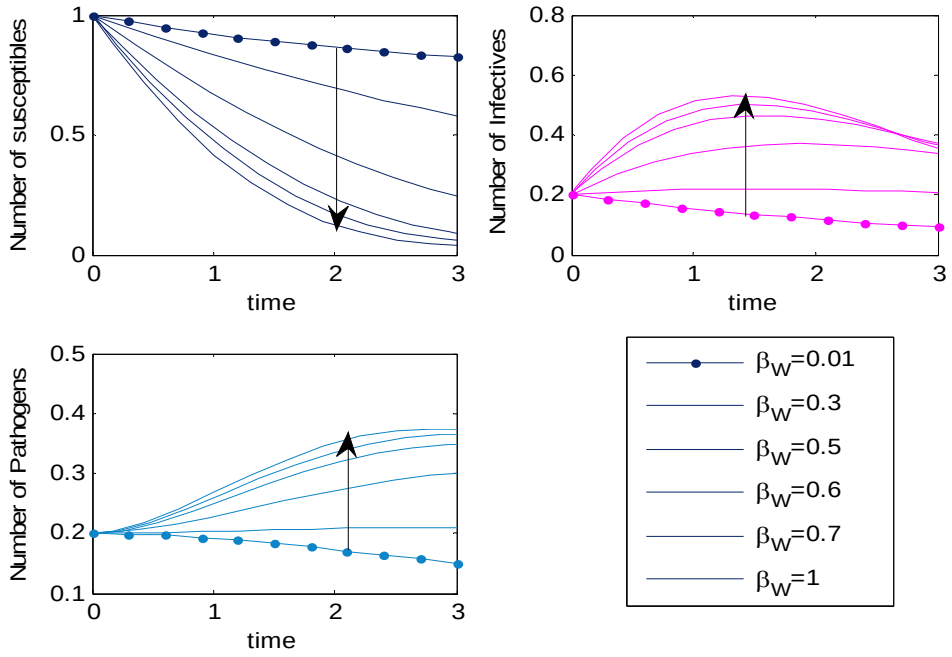


**Figure 10.** Sensitivity of the system (3) due to the effect of water-to-person transmission

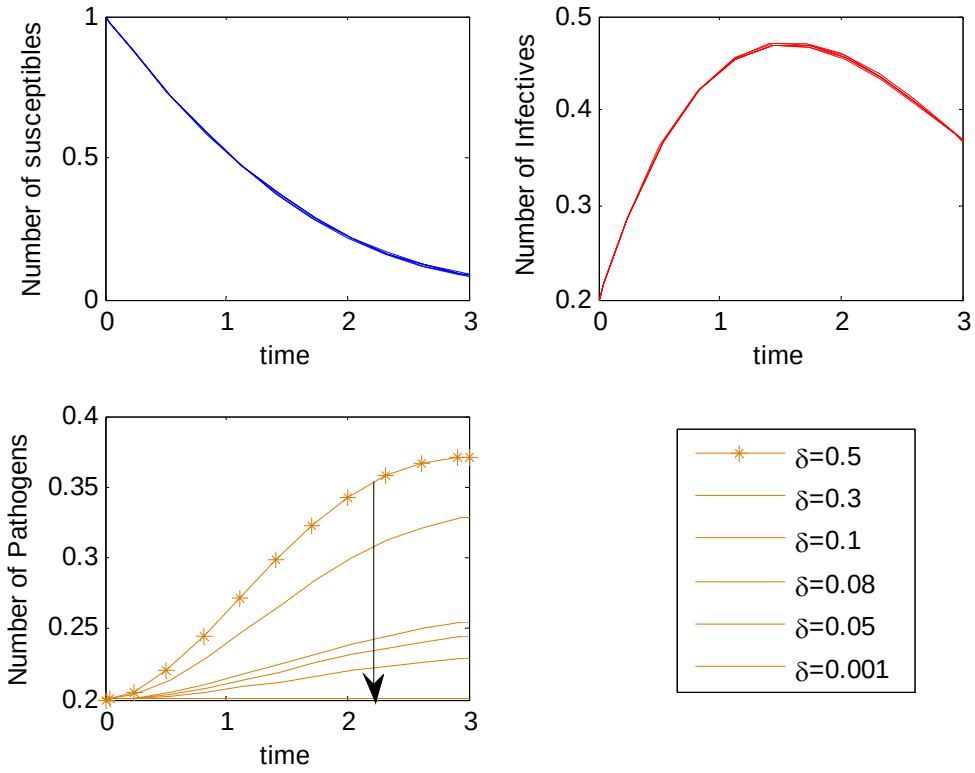


**Figure 11.** Sensitivity of the system (3) for different values of pathogen decay rate

The Figure (10) represents the variation of susceptibles, infected and pathogen concentration with the different values of water-to-person disease transmission rate. The number of infected and the pathogen populations are directly proportional with the force of infection $\beta_W$, but the susceptible population is inversely proportional with the force of infection as it is noticed in Figure 10. In Figure 11, we see that the pathogen concentration gradually decreases when the decay rate of pathogen decreases. Further it is noticeable from figure 11 that the pathogen decay rate doesn't have so much impact on susceptible and infected populations. From Figures (12) & (13), it is observed that the number of susceptible individuals is directly proportional to both the saturation factors where as the number of infected individuals and pathogen concentration is inversely proportional to that saturation factors. It has also been noticed that the number of susceptible individuals much more increase in Figure 12 than in Figure 13 when both the saturation factors are equal to one. In this scenario the number of infected individuals and the number of pathogens decrease quickly in Figure 12 than in Figure 13.

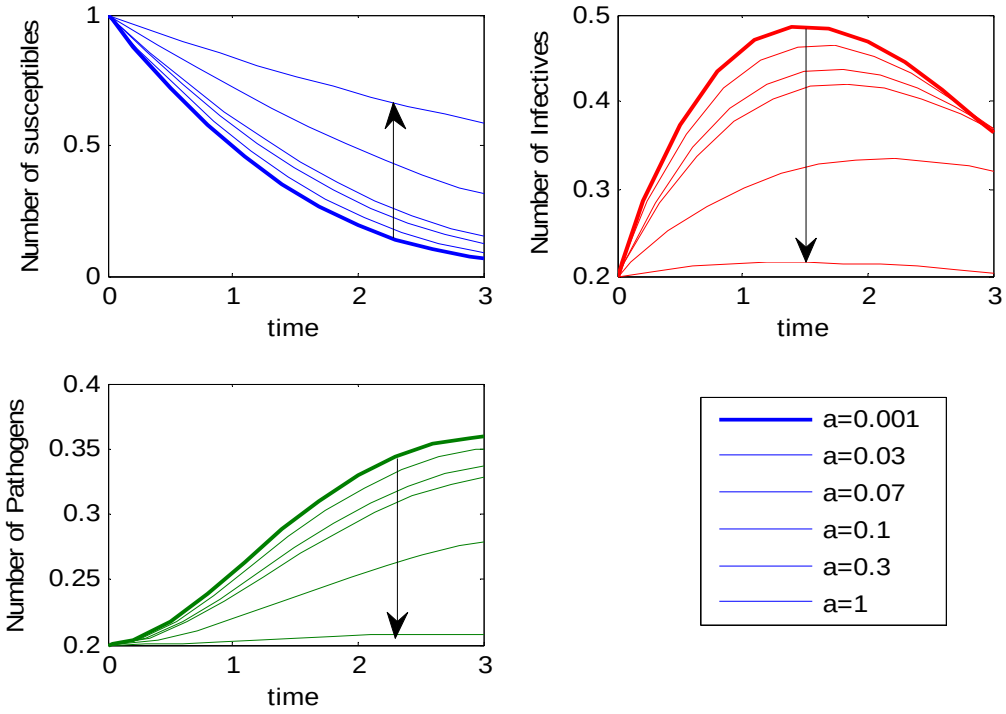


**Figure 12.** Sensitivity of the system (3) for different values of pathogen induced saturation factor

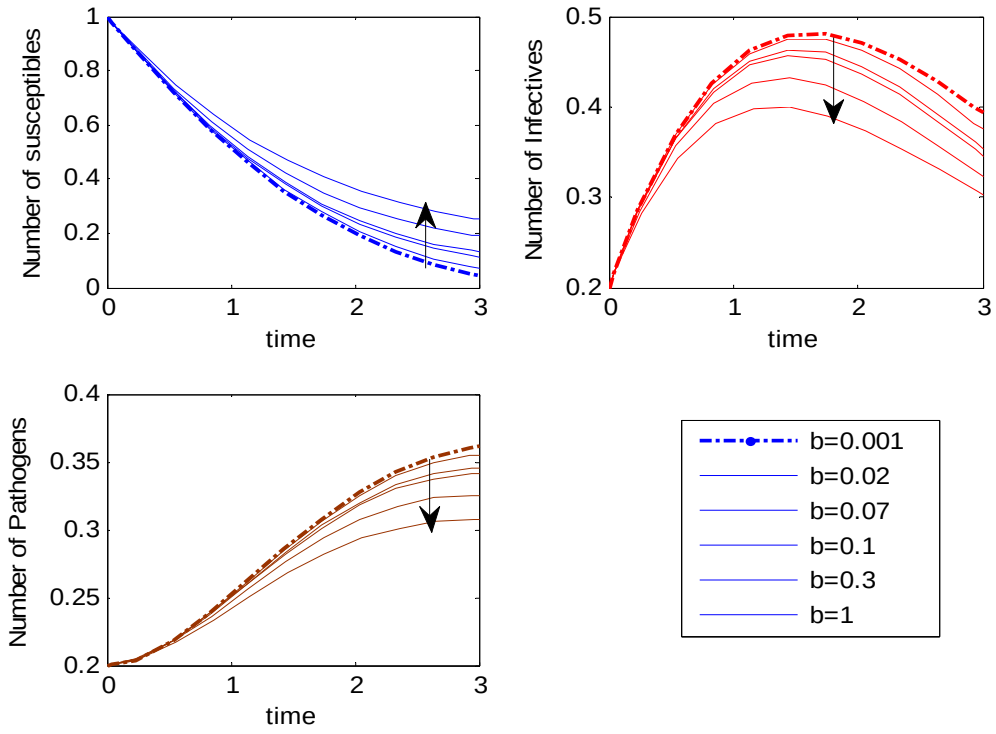


**Figure 13.** Sensitivity of the system (3) for different values of $b$.

## 6. Conclusion

In this paper, we have presented a water-borne disease epidemic model including multiple transmissions namely, water-to-person and person-to-person transmission. We also provided a positive invariant set for the system (3) and then proved that the system is uniformly persistence. Ensuring the global asymptotic stability of the unique endemic equilibrium, we obtained sufficient conditions expressing in terms of the parameters as well as also in terms of the state variables of the system. Also, the main model is compared with the sub model which is elucidated respectively in different sections. These are provided an example of the application of the method to a classical water dynamics-like model including peculiar non-linear incidence rate.

Our model system has two equilibria namely; disease-free equilibrium which always exists and endemic equilibrium which uniquely exists under some threshold conditions. For the system (3), we have investigated the basic reproductive number $R_0$ and it is an increasing function of both the disease transmission rate. It is found that the disease-free equilibrium is locally asymptotically stable when the basic reproductive number is below one and if its value above one the disease-free equilibrium is unstable but, when its value equal to one the system undergoes trans critical bifurcation at the disease-free equilibrium. The local stability and global stability of the endemic equilibrium is also investigated considering a Lyapunov function.

Furthermore, we elucidated the model system in absence of person-to-person transmission rate that is in presence of water-to-person transmission rate and this sub-model also has two

equilibria. The basic reproductive number $R_0^{WP}$ is obtained for the sub-model, which is less than the multiple transmissions induced basic reproductive number. It is observed that the disease-free equilibrium is globally asymptotically stable while $R_0^{WP} < 1$ and this means that the any new population will not infected by the disease. The sub-model has unique endemic equilibrium and locally asymptotically stable when the basic reproductive number is greater than one. Moreover, using Lyapunov functional approach, we have seen that the sub-model is global asymptotically stable around the endemic equilibrium and it is evident that the disease may not be removed from the community in presence of water-to-person transmission.

Our *SIWR* model may be fitted for different waterborne diseases. Particularly, this model can be fixed for the cholera disease because of the disease transmission process is chosen as water-to-person and person-to-person (see Goh et al. (1990)). These two types of transmission are very crucial in cholera disease, which depends upon the length of time that the Vibrio cholerae bacteria can persist in the water compartment. Global stability of our system around the endemic equilibrium indicates that the disease surely persist in the community.

Mwasa and Tchuenche (2011) discussed a cholera model with public health interventions including only water-to-person transmission only. The analysis of their model without any intervention showed that cholera may emerge infinitely many times. We studied the sensitivity analysis of the system in refer to some crucial model parameters. There it is seen that the number of infected individuals and concentration of pathogens are directly proportional to the two type disease transmission rate. We also found that if the person-to-person contact is not applied (see figure 9), then the disease may be transmitted initially through the contaminated reservoir and within a very tiny time this disease spreads into the population.

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