Modeling the health impact of water and sanitation service deficits on waterborne disease transmission

Rujira Chaysiri 1, Garrick E. Louis 2 and Wirawan Chinviriyasit 3*

*Correspondence: wirawon.chi@kmutt.ac.th
3Department of Mathematics, Faculty of Science, King Mongkut’s University of Technology Thonburi, Bangkok, Thailand
Full list of author information is available at the end of the article

Abstract
Cholera is a waterborne disease that continues to pose serious public health problems in many developing countries. Increasing water and sanitation coverage is a goal for local authorities in these countries, as it can eliminate one of the root causes of cholera transmission. The SIWDR (susceptible–infected–water–dumpsite–recovered) model is proposed here to evaluate the effects of the improved coverage of water and sanitation services in a community at risk of a cholera outbreak. This paper provides a mathematical study of the dynamics of the water and sanitation (WatSan) deficits and their public health impact in a community. The theoretical analysis of the SIWDR model gave a certain threshold value (known as the basic reproductive number and denoted $R_0$) to stop the transmission of cholera. It was found that the disease-free equilibrium was globally asymptotically stable whenever $R_0 \leq 1$. The unique endemic equilibrium was globally asymptotically stable whenever $R_0 > 1$. Sensitivity analysis was performed to determine the relative importance of model parameters to disease transmission and prevention. The numerical simulation results, using realistic parameter values in describing cholera transmission in Haiti, showed that improving the drinking water supply, wastewater and sewage treatment, and solid waste disposal services would be effective strategies for controlling the transmission pathways of this waterborne disease.

Keywords: Water and sanitation; Waterborne disease; Global stability; Lyapunov function; Cholera

1 Introduction
The most common waterborne diseases that pose a major public health risk in communities and continue to be leading causes for illnesses and deaths worldwide, especially in developing countries, include diarrhea, cholera, giardia, dysentery, Escherichia coli, salmonella, shigella, typhoid, and hepatitis A [1]. Waterborne diseases are caused by a lack of safe drinking water and proper sanitation, which is a major cause [2]. According to the World Health Organization (WHO), cholera is an infectious disease characterized by severe watery diarrhea and is caused by eating food or drinking water contaminated with the bacterium *Vibrio cholerae*. Diarrhea kills an estimated 829,000 people globally each year as a result of contaminated drinking water, sanitation, and hand hygiene [3].
addition, morbidity and mortality are worse in developing countries where communities lack the resources to provide sustained access to adequate water and sanitation services [4]. For instance, in 2010, Haiti suffered from a cholera outbreak with 665,000 cases and 8,183 deaths [5]. A cholera outbreak also occurred in Yemen in 2016 with more than 1.2 million cases and 2,500 deaths, of which 30% were children under the age of five [6]. In many low income countries, inadequate investment in water and sanitation infrastructure has perpetuated the problem. The inadequate investment resulted in illnesses among citizens. Loss of work productivity due to illnesses lowered individual income; hence, there was a lower national income. Consequently, low income countries had the least amount of money to invest in water and sanitation infrastructure that is necessary to combat the burden of waterborne diseases [7, 8]. Furthermore, open dumpsites in developing countries play an important role in the transmission of diseases [9, 10]. Open dumpsites can be an important and large reservoir of pathogens in these communities. Pathogens in open dumpsites can get into water sources through leachate, which contaminates unprotected groundwater sources, or through precipitation and runoff into surface water [11–13]. For example, open dumpsites in Yemen are located close to communities. This situation increases the health risks in these communities, especially from clinical waste, which has been demonstrated to contribute to cholera outbreaks in Yemen [14].

Since the connections between unclean water, sanitation, and health are well established [7, 15–17], combating waterborne diseases requires multiple strategies. These strategies include increasing access to safe drinking water and improved sanitation and cutting disease transmission pathways by having good hygiene practices [18]. According to the WHO, 94% of diarrhea cases are preventable by healthy environments, which include increasing access to clean water and sanitation services, and improved hygiene [19]. For sanitation services [7, 20], systematic reviews concluded that improved sanitation can reduce diarrhea cases by 32%–37%. This indicates that providing adequate safe drinking water and sanitation services reduces the incidence of acute infectious diarrhea [21–23]. Moreover, a study in Brazil and El Salvador found that improving sewerage coverage led to a significant reduction in diarrhea prevalence [24–27]. In this paper, therefore, the impact of improving water and sanitation services and the management of open dumpsites are explored as mechanisms to control waterborne disease transmission.

Mathematical models have been used to understand the transmission dynamics of waterborne disease, to improve control and prevention strategies, and to inform public health impacts [28–32]. Tien and Earn [28] presented the susceptible–infected–recovered (SIR) model with pathogen concentration, which was called the susceptible–infected–water–recovered (SIWR) model when applied to waterborne disease transmission. Their study showed that applying the SIR model rather than using the SIWR model underestimates the basic reproductive number and overestimates the infectious period when the pathogen decay rate is slow in the water compartment. Eisenberg et al. [29] showed that parameters in the SIWR model were globally identifiable. They estimated this by using the data from a cholera outbreak in Angola as a case study. Also, their study concluded that including environmental data was important when analyzing waterborne diseases. The SIWR model was further extended by having multiple patches within a common water resource. Their model assumed that the human-to-human transmission pathway occurs within a patch and the environment-to-human pathway happens across multiple patches with a shared water resource. Their study also explored the effects of heterogeneity on the final size of
an outbreak and the efficiency of interventions [30]. This type of spatially explicit model [31–33] was also used to investigate waterborne disease epidemics that account for local communities of susceptible and infected individuals in a spatially explicit arrangement of nodes linked by networks having different topologies. A network epidemic model of waterborne disease was studied by Wang and Cao [34] and Collins and Govinder [35]. These models considered the heterogeneity of different water sources that were contaminated. The optimal control of waterborne disease having multiple patches was also studied. Tuite et al. [36] proposed a gravity model to predict the sequence and timing of regional cholera epidemics in Haiti (2010) and suggested that adaptive strategies for vaccination may provide a modest reduction in morbidity and mortality in the economically challenged country. Collins et al. [37] studied the effect of heterogeneity in transmission due to socioeconomic status on the dynamics of waterborne diseases. Even though all the aforementioned studies have contributed immensely to improving the understanding of waterborne disease dynamics, other pathogen reservoirs that cause waterborne disease outbreaks have not been studied. In this paper, we address this research gap by formulating an appropriate mathematical epidemiological model that incorporates open dumpsites.

Another important issue of interest is the study of globally asymptotic properties of epidemiological models. This can help design effective control strategies intended to permanently reduce pathogen spread, or even break the chain of disease transmission. Understanding transient phenomena in potential epidemiological scenarios can also be important. It can help to prevent transitory epidemics that are triggered by external perturbations to a system, in which endemic transmission is not possible. That is, the disease-free equilibrium (DFE) is globally asymptotically stable [38]. This results in the threshold value of epidemiological models, which is called the basic reproductive number and denoted by $R_0$. The study conducted in [28] showed that estimates of $R_0$ and the infectious period can be sensitive to the relative contributions of the different transmission pathways and the pathogen’s lifetime in water. Further studies [2, 28, 37, 39, 40] and references (therein) confirmed that the sensitivity of $R_0$ is relevant for dynamical studies of waterborne diseases because the basic life history and epidemiological traits remain uncertain for many waterborne pathogens.

Based on the above discussion, this paper proposes a compartmental mathematical model to study the health impact of water and sanitation service deficits on the spread of waterborne diseases. The paper is organized as follows. In Sect. 2, the model proposed by Tien and Earn [28] is modified by incorporating water and sanitation services together with open dumpsites. In Sect. 3, the proposed model is analyzed to gain insights into the mechanisms of waterborne disease transmission and the control measures for waterborne diseases. The existence and the number of equilibria are established. The threshold value of the proposed model is derived by using the next generation method. The global stabilities of equilibria are analyzed by constructing the suitable Lyapunov functions and using LaSalle’s invariance principle. In Sect. 4, numerical simulations are illustrated to determine the appropriate parameter values used for the proposed model and to determine the parameters that are sensitive to disease prevalence and the impact of water and sanitation (WatSan) services. Finally, conclusions are given in Sect. 5.
2 System modeling

Individuals can be infected with waterborne disease if they come near to an open dump-site and contact waste directly [13]. This is because pathogens in open dumpsites can get into water sources through leachate and contaminate unprotected groundwater sources, or through precipitation that washes into surface water [11, 12]. Improving WatSan services is an effective means of reducing the number of infected individuals and the number of people who die each year as a result of diarrhea [41]. An increase in WatSan services would reduce the incidence of WatSan-related diseases, such as enteric diarrheal disease (EDD). Therefore, the system modeling included open dumpsites as reservoirs of pathogens and WatSan services, namely, the drinking water supply (DWS), wastewater and sewage treatment (WST), and municipal solid waste management (MSW). To investigate the impact caused by WatSan deficits on individual health and the state of the environment, we modified the model presented by Tien and Earn [28] with the following assumptions. We assumed that disease transmission occurred through two pathways, namely, a direct transmission pathway (human-to-human) and an indirect transmission pathway (environment-to-human). The indirect transmission pathway was formulated based on the SIR epidemic model for monitoring the dynamics of the sub-populations of susceptible individuals (S(t)), infectious individuals (I(t)), and recovered individuals (R(t)), so that the total population at time t is \( N(t) = S(t) + I(t) + R(t) \). The indirect transmission pathway consisted of the SIR epidemic model together with compartments \( W \) and \( D \). \( W \) and \( D \), respectively, represent pathogen concentration in water reservoirs and open dumpsites that are potential reservoirs of pathogens. Compartment \( D \) represents open dumpsites and other pathogen reservoirs that are not water reservoirs. Furthermore, to improve the WatSan services, let \( 0 \leq r_1 \leq 1 \), \( 0 \leq r_2 \leq 1 \), and \( 0 \leq r_3 \leq 1 \) represent the ratio of people who receive DWS, WST, and MSW services, respectively, in a community. A flow diagram of the formulated model, the susceptible–infected–water–dumpsite–recovered (SIWDR) model, is depicted in Fig. 1. The dynamics of each sub-population in Fig. 1 are described next.

The number of susceptible individuals increases at the recruitment rate of \( \mu N \), where \( \mu \) is the birth rate (intrinsic growth rate). The birth rate is assumed to be equal to the natural death rate so that the population size \( N \) remains constant. Thus, the susceptible

![Figure 1](https://example.com/figure1.png)
Table 1 Description of state variables and units for SIWDR model (2.1)

| State variables | Descriptions | Units       |
|-----------------|--------------|-------------|
| S               | susceptible individual density | individuals km$^{-2}$ |
| I               | infected individual density | individuals km$^{-2}$ |
| R               | recovered individual density | individuals km$^{-2}$ |
| W               | pathogen concentration in water reservoir | cells ml$^{-3}$ |
| D               | pathogen concentration in dumpsite | cells kg$^{-1}$ |
| N               | total population density | individuals km$^{-2}$ |

Table 2 Description of model parameters and units for SIWDR model (2.1)

| Parameters | Descriptions | Units |
|------------|--------------|-------|
| $b_I$      | person–person contact rate | km$^2$ individuals$^{-1}$ month$^{-1}$ |
| $b_W$      | reservoir–person contact rate | ml$^3$ cells$^{-1}$ month$^{-1}$ |
| $b_D$      | dumpsite–person contact rate | kg cells$^{-1}$ month$^{-1}$ |
| $\gamma$  | infectious period | months |
| $\xi_W$   | pathogen lifetime in water reservoir | months |
| $\xi_D$   | pathogen lifetime in dumpsite | months |
| $\alpha_1$ | person–reservoir contact rate ‘shedding’ | cells ml$^{-3}$ month$^{-1}$ km$^2$ individuals$^{-1}$ |
| $\alpha_2$ | person–dumpsite contact rate | cells kg$^{-1}$ month$^{-1}$ km$^2$ individuals$^{-1}$ |
| $\psi$    | dumpsite–reservoir contact rate | kg ml$^{-3}$ month$^{-1}$ |
| $\mu$     | birth rate | month$^{-1}$ |
| $\mu_2$   | non-disease-related death rate | month$^{-1}$ |
| $r_1$      | service coverage of DWS | – |
| $r_2$      | service coverage of WST | – |
| $r_3$      | service coverage of MSW | – |

group decreases at the natural death rate $\mu$. This includes when they contact infected individuals at a rate $b_I S$, or through contact with contaminated water and infected waste at rates $(1 - r_1)b_W W$ and $(1 - r_3)b_D D S$, respectively. The parameters $b_I$, $b_W$, and $b_D$ represent the transmission rate from person to person, water to person, and dumpsite to person contact, respectively. The number of infected individuals increases when these individuals generate secondary infections in two ways. The first way is through direct contact with susceptible individuals at a rate $b_I S$. The second way is by first shedding the pathogen into water and solid waste compartments at rates $(1 - r_1)b_W W S$ and $(1 - r_3)b_D D S$, respectively. Susceptible individuals can subsequently come into contact with pathogens in these compartments. This population decreases at natural death rate $\mu$. The recovered population increases when the infectious individuals recover from infection at rate $\gamma$ and decreases at natural death rate $\mu$.

The pathogen shedding rates from infected individuals into water and dumpsite compartments are given by $(1 - r_2)\alpha_1 I$ and $(1 - r_2)\alpha_2 I$, respectively. The pathogen shedding rate from the dumpsite compartment into the water compartment is given by $(1 - r_3)\psi D$. The parameters $\xi_W$ and $\xi_D$ give the decay rate of pathogens in the water and dumpsite compartments, respectively.

Therefore, based on the above descriptions and Fig. 1, the SIWDR model for the pathways of infection in waterborne diseases is given by the following deterministic system of
nonlinear differential equations:

\[
\begin{align*}
\frac{dS}{dt} &= \mu N - (1 - r_1)b_1SW - (1 - r_3)b_DSD - b_I S - \mu S, \\
\frac{dI}{dt} &= (1 - r_1)b_1SW + (1 - r_3)b_DSD + b_I S - \gamma I - \mu I, \\
\frac{dW}{dt} &= (1 - r_2)\alpha_1I + (1 - r_3)\psi D - \xi_W W, \\
\frac{dD}{dt} &= (1 - r_2)\alpha_2I - (1 - r_3)\psi D - \xi_D D, \\
\frac{dR}{dt} &= \gamma I - \mu R,
\end{align*}
\]

(2.1)

where the descriptions of the model parameters and sample units in system (2.1) are given in Tables 1–2. Since the above model monitors human and pathogen populations, all associated parameters and state variables have nonnegative values.

3 Analysis of the model
3.1 Basic properties
Adding the first, second, and fifth equations of system (2.1) gives \(\frac{dN}{dt} = 0\). Thus, the total human population \(N\) is constant. For convenience of analysis, we rewrite system (2.1) in terms of new dimensionless quantities by setting the state variables:

\[
\begin{align*}
S &= \frac{S}{N}, \\
I &= \frac{I}{N}, \\
R &= \frac{R}{N}, \\
W &= \frac{\xi_W}{\alpha_1N}W, \\
D &= \frac{\xi_D}{\alpha_2N}D,
\end{align*}
\]

so that

\[
s + i + r = 1.
\]

(3.1)

The model parameters are replaced by

\[
\begin{align*}
\beta_I &= b_1N, \\
\beta_W &= \frac{b_W \alpha_1 N}{\xi_W}, \\
\beta_D &= \frac{b_D \alpha_2 N}{\xi_D},
\end{align*}
\]

Using the expression above, system (2.1) is reduced to

\[
\begin{align*}
\dot{s}(t) &= \mu - (1 - r_1)\beta_W Sw - (1 - r_3)\beta_D sd - \beta_1 si - \mu s, \\
\dot{i}(t) &= (1 - r_1)\beta_W Sw + (1 - r_3)\beta_D sd + \beta_1 si - \gamma i - \mu i, \\
\dot{w}(t) &= \xi_W (1 - r_2)i + \frac{(1 - r_3)\alpha_2 \xi_W \psi}{\alpha_1 \xi_D} - d - \xi_W w, \\
\dot{d}(t) &= \xi_D (1 - r_2)i - (1 - r_3)\psi \xi_D d,
\end{align*}
\]

(3.2)

where \(r = 1 - s - i\). This allows us to solve system (2.1) by studying system (3.2). Clearly, \(\lim_{t \to \infty} \sup(s + i) \leq 1\) implies \(\lim_{t \to \infty} \sup(\dot{\theta}(t)) \leq 1\). It follows from the fourth equation of (3.2) that

\[
\dot{d}(t) \leq \xi_D (1 - r_2)i - (1 - r_3)\psi \xi_D d,
\]
which implies \( \lim_{t \to \infty} \sup(d(t)) \leq \frac{\xi_D(1-r_2)}{(1-r_3)\psi + \xi_D} \). Thus, from the third equation of system (3.2), we have

\[
w' \leq \xi_W(1-r_2) + \frac{(1-r_3)\alpha_2\xi_W\psi}{\alpha_1\xi_D} \left( \frac{\xi_D(1-r_2)}{(1-r_3)\psi + \xi_D} \right) - \xi_W w
= \xi_W(1-r_2) \left( 1 + \frac{(1-r_3)\alpha_2\psi}{\alpha_1((1-r_3)\psi + \xi_D)} \right) - \xi_W w.
\]

It is easy to show that

\[
\lim_{t \to \infty} \sup(w(t)) \leq (1-r_2) \left( 1 + \frac{(1-r_3)\alpha_2\psi}{\alpha_1((1-r_3)\psi + \xi_D)} \right).
\]

Hence, the feasible region

\[
\Omega = \{ (s, i, w, d) : 0 \leq s + i \leq 1, w \leq w_c, \text{ and } d \leq d_c \}
\]

is positively invariant for model (3.2) with \( w_c = (1-r_2)(1 + \frac{(1-r_3)\alpha_2\psi}{\alpha_1((1-r_3)\psi + \xi_D)}) \) and \( d_c = \frac{\xi_D(1-r_2)}{(1-r_3)\psi + \xi_D} \). Thus, the existence, uniqueness, and continuation results for model (3.2) hold in \( \Omega \). All solutions that start in \( \Omega \) remain in \( \Omega \) for all \( t \geq 0 \) [42]. Hence, model (3.2) is mathematically and epidemiologically well posed, and it is sufficient to consider the dynamics of the flow generated by model (3.2) in \( \Omega \).

### 3.2 Disease-free equilibrium and basic reproductive number

#### 3.2.1 Local stability

In the absence of infection and with adequate water and sanitation services \((i = 0, w = 0 \text{ and } d = 0)\), system (3.2) has a unique disease-free equilibrium (DFE) denoted by

\[
\epsilon_0(s^0, i^0, w^0, d^0) = (1, 0, 0, 0). \tag{3.3}
\]

According to the next generation matrix and using the notation in [43], system (3.2) is rewritten as

\[
x'(t) = \mathcal{F} - (\mathcal{V}^- - \mathcal{V}^+), \tag{3.4}
\]

where \( x = (i, w, d, s)^T \),

\[
\mathcal{F} = \begin{pmatrix}
\hat{r}_1 \beta_W s w + \hat{r}_3 \beta_D s d + \beta_i s i \\
0 \\
0 \\
0
\end{pmatrix},
\]

\[
\mathcal{V}^- = \begin{pmatrix}
\gamma i + \mu i \\
\xi_W w \\
(\hat{r}_3 \psi + \xi_D)d \\
Q_1
\end{pmatrix}, \quad \mathcal{V}^+ = \begin{pmatrix}
0 \\
\xi_W \hat{r}_2 i + \frac{Q_2}{\alpha_1 \xi_D} d \\
\xi_D \hat{r}_2 i \\
\mu
\end{pmatrix}
\]

with \( Q_1 = \hat{r}_1 \beta_W s w + \hat{r}_3 \beta_D s d + \beta_i s i + \mu s \) and \( Q_2 = \hat{r}_3 \alpha_2 \xi_W \psi \), respectively. Clearly, \( x_0 = (0, 0, 0, 1) \) is a disease-free equilibrium of system (3.4), which is identical to \( \epsilon_0 \) of system
(3.2). It is easy to show that system (3.4) satisfies assumptions (A1)–(A5) in [43]. Further, the derivatives $DF(x_0)$ and $DV(x_0)$ are partitioned as

$$DF(x_0) = \begin{pmatrix} F & 0 \\ 0 & 0 \end{pmatrix} \quad \text{and} \quad DV(x_0) = \begin{pmatrix} V & 0 \\ J_3 & J_4 \end{pmatrix},$$

where the matrices $F$ (for the new infection terms) and $V$ (for the transition terms) are given, respectively, by

$$F = \begin{pmatrix} \beta I \tilde{r}_1 \beta W \tilde{r}_3 \beta D \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \quad \text{and} \quad V = \begin{pmatrix} \gamma + \mu & 0 & 0 \\ -\xi W \tilde{r}_2 & \xi W & -\frac{\gamma \alpha_2 \xi W \psi}{\alpha_1 \xi D} \\ -\xi D \tilde{r}_2 & 0 & \tilde{r}_3 \psi + \xi D \end{pmatrix},$$

where $\tilde{r}_1 = 1 - r_1$, $\tilde{r}_2 = 1 - r_2$, and $\tilde{r}_3 = 1 - r_3$. Defining $R_0 = \rho(FV^{-1})$, where $\rho$ is the spectral radius of matrix $FV^{-1}$, we get

$$R_0 = \frac{\alpha_1 (\tilde{r}_3 \psi + \xi D) (\beta I + \tilde{r}_1 \tilde{r}_2 \beta W) + \tilde{r}_2 \tilde{r}_3 (\alpha_1 \beta D \xi D + \tilde{r}_1 \alpha_2 \beta W \psi)}{\alpha_1 (\mu + \gamma) (\psi \tilde{r}_3 + \xi D)}.$$  

(3.6)

According to Theorem 1 in [43], the following result is established.

**Lemma 3.1** The DFE $\varepsilon_0$ of system (3.2) is locally asymptotically stable (LAS) if $R_0 < 1$ and unstable if $R_0 > 1$.

The local stability that resulted in Lemma 3.1 implies that for $R_0 < 1$ the disease dies out and for $R_0 > 1$ the infected population is not reduced to zero. Thus, the quality that $R_0 > 1$ is the threshold value of model (3.2). Furthermore, the result in Lemma 3.1 verifies that if the initial sizes of the sub-groups of the model are within the basin of attraction of $\varepsilon_0$, the total number of infected people in the population can be reduced to zero whenever $R_0 < 1$. Moreover, it was found that the value of $R_0$ depends on the ratios of people who receive the DWS, WST, and MSW services: $r_1$, $r_2$, and $r_3$, respectively. This theoretical determination of conditions of these ratios can make $R_0$ independent of the initial sizes of the five state variables. This is of great public health interest, which is later illustrated by analyzing the global stability.

### 3.2.2 Global stability of DFE

We claim the following result.

**Theorem 3.2** The DFE $\varepsilon_0$ of model (3.2) is globally asymptotically stable (GAS) in $\Omega$ if $R_0 < 1$.

**Proof** We introduce a new function

$$L = i + A_W w + A_D d,$$
where \( A_W = \frac{\bar{r}_1\bar{r}_W}{\bar{r}_W} \) and \( A_D = \frac{\bar{r}_3(\alpha_1\beta_W+\bar{r}_1\alpha_2\beta_W)}{\alpha_1\xi_D(\psi\bar{r}_3+\bar{r}_3)} \). The derivative of \( L \) with the solutions of (3.2) is

\[
L'(t) = (s-1)(\bar{r}_1\beta_Ww + \bar{r}_3\beta_Dd) + (\mu + \gamma)\left(\frac{\bar{r}_2\bar{r}_3(\alpha_1\beta_D+\bar{r}_1\alpha_2\psi) + \alpha_1(\bar{r}_3\psi + \xi_D)(\beta_Ws + \bar{r}_1\bar{r}_3\beta_W)}{\alpha_1(\mu + \gamma)(\psi\bar{r}_3 + \xi_D)} - 1\right)i \\
\leq (\mu + \gamma)(R_0 - 1)i.
\]

It is obvious that \( L'(t) \leq 0 \) if \( R_0 \leq 1 \). Furthermore, \( L'(t) = 0 \) if \( i = 0 \). The Lyapunov–LaSalle theorem [44] implies that all paths in \( \Omega \) approach the largest positively invariant subset of set \( \{(s,i,d,w) | i=0\} \). On the boundary of \( \Omega \), where \( i = 0 \), we have \( r'(t) = -\mu r \), so \( r = r(0)e^{-\mu t} \to 0 \) as \( t \to \infty \), and \( d'(t) = -(\bar{r}_3\psi + \xi_D)d \). Then \( d(t) = d(0)e^{-(\bar{r}_3\psi + \xi_D)t} \to 0 \) as \( t \to \infty \). Thus, we easily obtain \( \lim_{t \to \infty} w(t) = 0 \). Hence, all solution paths in \( \Omega \) approach the DFE \( \varepsilon_0 \). This completes the proof of this theorem.

### 3.3 Endemic equilibrium (EE)

In the presence of infection \( (i \neq 0) \) and insufficient water and sanitation services \( (w \neq 0 \) and \( d \neq 0) \), model (3.2) has a unique EE, which is given by \( \varepsilon_1 = (s^*, i^*, w^*, d^*, r^*) \) where:

\[
\begin{align*}
\frac{s^*}{R_0} &= 1, \\
\frac{i^*}{\mu + \gamma} &= \frac{\mu}{(\mu + \gamma)R_0}, \\
d^* &= \left(\frac{\bar{r}_2\bar{r}_3(\alpha_1\beta_D + \bar{r}_1\alpha_2\psi) + \alpha_1(\bar{r}_3\psi + \xi_D)(\beta_Ws + \bar{r}_1\bar{r}_3\beta_W)}{\alpha_1(\mu + \gamma)(\psi\bar{r}_3 + \xi_D)}\right)i^*, \\
w^* &= \left(\frac{\bar{r}_2\alpha_1(\bar{r}_3\psi + \xi_D) + \bar{r}_2\bar{r}_3\alpha_2\psi}{\alpha_1(\bar{r}_3\psi + \xi_D)}\right)i^*.
\end{align*}
\]

Clearly, \( i^* > 0 \) (corresponding to the EE, see (3.7) whenever \( R_0 > 1 \), \( i^* = 0 \) when \( R_0 = 1 \) (corresponding to the DFE, see (3.3)) and \( i^* < 0 \) (meaningless) whenever \( R_0 < 1 \).

#### 3.3.1 Local stability

Linearizing model (3.2) around its unique EE is analyzed by the use of the center manifold theory [45] as described in Theorem 4.1 [46]. The following theorem, thus, is established.

**Theorem 3.3** The unique EE \( \varepsilon_1 \) of the model (3.2) is LAS if \( R_0 > 1 \) and is close to 1.

**Proof** The Jacobian of system (3.2) evaluated at \( \varepsilon_0 \) is given by

\[
J(\varepsilon_0) = \begin{bmatrix}
-\mu & -\beta_1 & -\bar{r}_1\beta_W & -\bar{r}_3\beta_D \\
0 & \beta_W - (\mu + \gamma) & \bar{r}_1\beta_W & \bar{r}_3\beta_D \\
0 & \bar{r}_2\xi_W & -\xi_W & \bar{r}_3\alpha_2\psi \xi_D \\
0 & \bar{r}_2\xi_D & 0 & -\bar{r}_3 + \xi_D
\end{bmatrix}.
\]

Suppose, that \( \beta_1 = \beta_1^* \) is a bifurcation parameter. Solving \( R_0 = 1 \) for \( \beta_1 \) gives

\[
\beta_1^* = (\gamma + \mu) - \frac{\bar{r}_2(\bar{r}_1\beta_W(\bar{r}_3\alpha_2\psi + \alpha_1(\bar{r}_3\psi + \xi_D)) + \bar{r}_3\alpha_1\beta_D\xi_D)}{\alpha_1(\bar{r}_3\psi + \xi_D)}.
\]

It was found that system (3.2) with \( \beta_1 = \beta_1^* \) has at least one non-hyperbolic equilibrium point. Thus, the centre manifold theory [45] as described in Theorem 4.1 [46] is used to
analyze the LAS of EE $\varepsilon_1$ of the system (3.2) with $\beta_I = \beta_I^*$. The right eigenvector of $J(\beta_I^*)$ with $\beta_I = \beta_I^*$ is given by $w = [w_1, w_2, w_3, w_4, w_5]^T$, where

$$w_1 = -\frac{(\gamma + \mu)(\tilde{r}_3 \psi + \xi_D)w_4}{\tilde{r}_2 \mu \xi_D}, \quad w_2 = \frac{(\tilde{r}_3 \psi + \xi_D)w_4}{\tilde{r}_2 \xi_D},$$

$$w_3 = \frac{(\alpha_1(\tilde{r}_3 \psi + \xi_D) + \tilde{r}_3 \alpha_2 \psi)w_4}{\alpha_1 \xi_D}, \quad \text{and} \quad w_4 > 0.$$

Further, $J(\beta_I^*)$ with $\beta_I = \beta_I^*$ has a left eigenvector $v = [v_1, v_2, v_3, v_4, v_5]$, where

$$v_1 = v_5 = 0, \quad v_3 = \frac{\tilde{r}_1 \beta_D w_2}{\xi_w}, \quad v_4 = \frac{\tilde{r}_3 (\alpha_1 \beta_D \xi_D + \tilde{r}_1 \alpha_2 \psi \beta w) v_2}{\alpha_1 \xi_D (\tilde{r}_3 \psi + \xi_D)}, \quad \text{and} \quad v_2 > 0.$$

For system (3.2), the associated non-zero partial derivatives of the function $f_i(s, i, w, d, r)$, $i = 1, \ldots, 5$ on the right-hand side of each equation are given by

$$\frac{\partial^2 f_i}{\partial x_1 \partial x_2} = -\beta_I^*, \quad \frac{\partial^2 f_i}{\partial x_1 \partial x_3} = -\tilde{r}_1 \beta_D, \quad \frac{\partial^2 f_i}{\partial x_1 \partial x_4} = \tilde{r}_1 \beta_D,$$

$$\frac{\partial^2 f_i}{\partial x_1 \partial x_5} = -1, \quad \frac{\partial^2 f_i}{\partial x_2 \partial \beta_I^*} = 1,$$

for variables $x_1 = s$, $x_2 = i$, $x_3 = w$, and $x_4 = d$.

Using the above expressions, parameters $a$ and $b$ as given in Theorem 4.1 [46] are computed.

$$a = \sum_{k,j=1}^{5} v_k w_j \frac{\partial^2 f_k}{\partial x_k \partial x_j}(0, 0)$$

$$= -\frac{2 v_2 w^2 \xi_D (\alpha_1 \xi_D (\beta_I + \tilde{r}_1 \beta_D \xi_D) + \tilde{r}_3 \psi \beta w))}{\mu \alpha_1 (\tilde{r}_2 \xi_D)^2},$$

and

$$b = \sum_{k,j=1}^{5} v_k w_j \frac{\partial^2 f_k}{\partial x_k \partial \phi}(0, 0) = \frac{C_1 v_2 w_4}{\tilde{r}_2 \xi_D},$$

where $C_1 = \tilde{r}_3 \psi + \xi_D$ and $C_2 = \gamma + \mu$. Clearly, $a < 0$ and $b > 0$. Hence, by Theorem 4.1 in [46], the unique EE $\varepsilon_1$ of the system (3.2) exists and is LAS whenever $R_0 > 1$ and $\beta_I^* < \beta_I$ with $\beta_I$ close to $\beta_I^*$.

This theorem indicates that if $R_0 > 1$, the EE $\varepsilon_1$ is locally asymptotically stable (that is, the disease will persist in the community if the initial sizes of the four-state variables are within the vicinity of $\varepsilon_1$). If the EE $\varepsilon_1$ is globally asymptotically stable, then any trajectories
tend toward the attractor of $\varepsilon_1$, of system (3.2), regardless of the initial conditions. This means that the attracting basin of trajectories in the dynamical system is the state space $[47]$. In a biological sense, this property gives the theoretical determination of conditions that can cause $R_0 > 1$, irrespective of the initial sizes of the five state variables that increase the risk of a disease outbreak. The Lyapunov function is constructed and LaSalle’s invariance principle, which is the criterion for the asymptotic stability of an autonomous dynamical system $[48–51]$, is applied to prove the global stability of $\varepsilon_1$. The following theorem, therefore, is established.

**Theorem 3.4** The unique EE $\varepsilon_1$ of model (3.2) is globally asymptotically stable (GAS) in $\Omega$ whenever $R_0 > 1$.

**Proof** Consider the Lyapunov function

$$V(t) = A_1 \left( s - s^* - s^* \ln \left( \frac{s}{s^*} \right) + i - i^* - i^* \ln \left( \frac{i}{i^*} \right) \right) + \frac{1}{\xi_w} \left( w - w^* - w^* \ln \left( \frac{w}{w^*} \right) \right)$$

$$+ A_2 \left( d - d^* - d^* \ln \left( \frac{d}{d^*} \right) \right),$$

where

$$A_1 = \frac{\tilde{r}_2}{\tilde{r}_1 \beta_w w^* s^*} \left( \tilde{r}_3 \alpha_2 \psi + \frac{\tilde{r}_2 \beta_D i^*}{\tilde{r}_2 \tilde{r}_1 \beta w d^*} \right),$$

$$A_2 = \frac{\tilde{r}_3 d^*}{\tilde{r}_2 \tilde{r}_1 \tilde{r}_1 \xi_d \alpha_2 \psi + \frac{\tilde{r}_2 \beta_D i^*}{\tilde{r}_2 \tilde{r}_1 \beta w d^*} \tilde{r}_2 \tilde{r}_1 \tilde{r}_1 \xi_d \beta w d^*}.$$  

The derivative of $V(t)$ and the solutions of system (3.2) are given by

$$\frac{dV}{dt} = A_1 \left( 1 - \frac{s^*}{s} \right) (\mu - \tilde{r}_1 \beta_w s w - \tilde{r}_3 \beta_D s d - \beta_1 s - \mu s)$$

$$+ A_1 \left( 1 - \frac{i^*}{i} \right) (\tilde{r}_1 \beta_w s w + \tilde{r}_3 \beta_D s d + \beta_1 s - \gamma i - \mu i)$$

$$+ \xi_w \left( 1 - \frac{w^*}{w} \right) \left( \tilde{r}_2 i + \frac{\tilde{r}_3 \alpha_2 \psi}{\alpha_1 \xi_d} d - w \right)$$

$$+ A_2 \left( 1 - \frac{d^*}{d} \right) (\xi_d \tilde{r}_2 i - (\tilde{r}_3 \psi + \xi_d) d). \quad (3.8)$$

At steady state when $R_0 > 1$, and from the first four equations of system (3.2), we have

$$\mu = \tilde{r}_1 \beta_w s^* w^* + \tilde{r}_3 \beta_D s^* d^* + \beta_1 s^* i^* + \mu s^*, \quad (\gamma + \mu) i^* = \tilde{r}_1 \beta_w s^* w^* + \tilde{r}_3 \beta_D s^* d^* + \beta_1 s^* i^*,$$

$$w^* = \tilde{r}_2 i^* + \frac{\tilde{r}_3 \alpha_2 \psi}{\alpha_1 \xi_d} d^*, \quad (\tilde{r}_3 \psi + \xi_d) d^* = \xi_d \tilde{r}_2 i^*. \quad (3.9)$$
Substituting expressions (3.9) into (3.8) and rearranging give

\[
\frac{dV}{dt} = A_1 \left( \beta_1 s^* i^* + \mu s^* \right) \left( 2 - \frac{s^*}{s} - \frac{s}{s^*} \right) + A_1 \beta_1 \psi (s^* w^*) \left( 2 + \frac{w}{w^*} - \frac{s^*}{s} - \frac{i}{i^*} - \frac{i^*}{s^* i w^*} \right) + A_1 \beta_3 \beta_2 \psi (s^* w^*) \left( 3 - \frac{s^*}{s} - \frac{s i^* d^*}{s^* i d^*} - \frac{id^*}{i^* d} \right) + \tilde{\gamma}_2 \left( \frac{3}{3} \frac{s^*}{s} - \frac{s^*}{s} + \frac{i^*}{i^*} - \frac{i^*}{s^* i w^*} \right) + \frac{\tilde{\gamma}_2 \psi d^*}{\xi d^*} \left( 4 - \frac{s^*}{s} - \frac{s^* i w^*}{s^* i w^*} - \frac{w^* d}{w d^*} - \frac{d}{d i^*} \right). \]

It follows from the arithmetic mean is greater than or equal to the geometric mean that the following inequalities hold:

\[
\begin{align*}
2 - \frac{s^*}{s} - \frac{s}{s^*} & \leq 0, \\
3 - \frac{s^*}{s} - \frac{s i^* d^*}{s^* i d^*} - \frac{id^*}{i^* d} & \leq 0, \\
3 - \frac{s^*}{s} - \frac{s i^* w^*}{s^* i w^*} - \frac{i^* w}{i^* w} & \leq 0, \\
4 - \frac{s^*}{s} - \frac{s^* i w^*}{s^* i w^*} - \frac{w^* d}{w d^*} - \frac{d}{d i^*} & \leq 0, \\
\end{align*}
\]

(3.10)

for \( s \geq 0, i \geq 0, d \geq 0, w \geq 0 \). Using the inequalities in (3.10) and that all the model parameters are nonnegative, it is clear that \( \frac{dV}{dt} \leq 0 \) whenever \( R_0 > 1 \). Furthermore, \( \frac{dV}{dt} = 0 \) if and only if \( s = s^*, i = i^*, d = d^*, \) and \( w = w^* \). Hence, \( V(t) \) is a Lyapunov function in \( \Omega \). By LaSalle’s invariance principle \([52]\), it is found that \( s \to s^*, i \to i^*, d \to d^* \), and \( w \to w^* \) as \( t \to \infty \). Hence, the EE (3.1) of model (3.2) is GAS. That is, every solution of model (3.2) with initial condition in \( \Omega \) approaches \( \varepsilon_1 \) as \( t \to \infty \) whenever \( R_0 > 1 \). \( \square \)

### 4 Numerical simulations

#### 4.1 Estimation values of model parameters

From the outbreak size of cholera in Haiti in 2010, the death toll was approximately 9,700, and there were over 800,000 suspected cases \([53]\). Model (3.2) used this cholera outbreak as a case study. The model was simulated with \( N = 2,200,000 \), which is the approximate population in the capital of Haiti in 2010 \([54]\). The parameter values are given in Table 3 for
Table 3 Parameters values used in model (3.2)

| Parameters | Nominal values | Ranges        | Sources       |
|------------|----------------|---------------|---------------|
| $\mu$      | 0.00005363     | 0–0.0012      | [55, 56]      |
| $\beta_I$  | 0.43           | 0.3–3         | [29, 36]      |
| $\beta_W$  | 9.49           | 0.0003–30     | [29, 36, 57]  |
| $\beta_D$  | 6.54           | 0.0003–30     | [29, 36, 57]  |
| $\gamma$   | 12.10          | 1.5–30        | [29, 36, 58, 59] |
| $\alpha_1$ | 3.89           | 0.3–300       | [57, 58, 60]  |
| $\alpha_2$ | 19.93          | 0.3–300       | [57, 58, 60]  |
| $\psi$     | 11.32          | 0.3–300       | [57, 58, 60]  |
| $\xi_W$    | 2.63           | 0.6857–10     | [36, 57]      |
| $\xi_D$    | 2.58           | 0.3477–12     | [61]          |
| $r_1$      | 0.635          | 0.0–1.0       | [62, 63]      |
| $r_2$      | 0.225          | 0.0–1.0       | [62, 63]      |
| $r_3$      | 0.1            | 0.0–1.0       | [64]          |

Figure 2 Comparison of the cumulative predicted infection data produced by model (3.2) and the cumulative real data from Haiti, 2010 [65]. Red points denote real data and the blue line denotes predicted data.

predicting the number of infected individuals. The results are then used to compute the cumulative number of predicted infected individuals by solving the differential equation

$$\frac{dC}{dt} = kI,$$

(4.1)

where $k = 0.99$ is the rate of progression in the monthly infectious state. With all values given in Table 3, the basic reproductive number is $R_0 = 1.2406$, which is within the range (1.06, 2.78) [36, 66]. This value indicates that cholera persists in the community and can result in a disease outbreak, as guaranteed by Theorem 3.4. As shown in Fig. 2, the cumulative predicted data are compared with the cumulative real cholera data in Haiti 2010, which are reported by the Pan American Health Organization (PAHO) [65]. Figure 2 shows that the cumulative predicted data produced by model (3.2) are close to the real data generated by using a statistical measure, the coefficient of determination, $R^2 = 0.9946$. This study result verifies that model (3.2) can be used to investigate the cholera transmission in Haiti (2010). Moreover, the values of model parameters given in Table 3 are appropri-
ate for model (3.2). These values are used to investigate the influential factors that can mitigate the 2010 cholera outbreak in Haiti in the next sections.

4.2 Sensitivity analysis of the basic reproductive number

Sensitivity analysis [67] is a tool for determining the relative importance of the different factors responsible for disease transmission. The initial disease transmission is directly related to the basic reproductive number $R_0$. We computed the sensitivity indices of $R_0$ in a model with parameter $p$, defined by using partial derivatives

$$\Upsilon_{R_0}^p = \frac{\partial R_0}{\partial p} \times \frac{p}{R_0}. \quad (4.2)$$

Table 4 shows the order of sensitivity indices of $R_0$ in relation to thirteen model parameters, evaluated at the baseline values given in Table 3. The values are arranged from the most to the least sensitive parameters. A negative value means that increasing a model parameter value decreases the value of $R_0$, resulting in a decrease in the infected individual density. In contrast, a positive value means that increasing a model parameter value increases the value of $R_0$, resulting in an increase in the infected individual density. The model parameters ($\beta_W, \alpha_1, \alpha_2, \psi, \beta_D, \beta_I$) affect the increase in the initial transmission, and $\beta_W$ is the most sensitive parameter. In contrast, the model parameters ($r_1, \gamma, r_2, r_3, \xi_W, \xi_D, \mu$) affect the decrease in the initial disease transmission, and $r_1$ is the most sensitive parameter. Regarding the coverage of DWS, WST, and MSW services, the coverage of DWS service ($r_1$) is the most sensitive parameter, followed by the coverage of WST service ($r_2$) and the coverage of MSW service ($r_3$). This result indicates that improving the coverage of the DWS service is the most effective intervention to control a waterborne disease outbreak.

Although an increase in the coverage of DWS is the most effective strategy, choosing only one strategy to control a waterborne outbreak might not be an effective strategy. Next, the results of combining two of these model parameters on the value of $R_0$ are explored using the contour plots of $R_0$ (see Fig. 3). The results show that an increase in the coverage $r_1, r_2,$ and $r_3$ services decreases the value of $R_0$ to be less than unity. However, it is observed that if the coverage of $r_1$ is 0.65, the coverage of $r_2$ should be increased by at least 0.38 (compared to the baseline value $r_2 = 0.225$) to decrease $R_0$ to be less than 1 (see

| Model parameters | Sensitivity indices $\Upsilon_{R_0}^p$ |
|------------------|-----------------------------------|
| $r_1$            | -1.5831                           |
| $\gamma$         | -1.0000                           |
| $\beta_W$        | 0.9099                            |
| $\alpha_1$       | 0.7311                            |
| $\alpha_2$       | 0.7311                            |
| $r_2$            | -0.2820                           |
| $r_3$            | -0.2820                           |
| $\psi$           | 0.0987                            |
| $\xi_W$          | -0.0987                           |
| $\xi_D$          | -0.0987                           |
| $\beta_D$        | 0.0614                            |
| $\beta_I$        | 0.0286                            |
| $\mu$            | $-4.4322 \times 10^{-6}$         |
Figure 3 Contour plots of the surface \( \mathcal{R}_0 \) as the function of the services coverage \( r_1, r_2, \) and \( r_3 \). The values of the other model parameters are given in Table 2.
Fig. 3(a)). In this case, an increase in the coverage of the $r_5$ service has the least impact in decreasing the value of $R_0$ (see Figs. 3(b) and 3(c)). In summary, this study suggests that the WatSan services coverage would be a crucial factor to mitigate the health impact of the 2010 cholera outbreak in Haiti.

Generally, coverages of DWS, WST, and MSW services can decrease the initial disease transmission and interrupt the transmission of waterborne diseases.

4.3 Impacts of WatSan services
The 2013–2022 plan to combat cholera in Haiti has been put in place [68]. The plan is to spend approximately $1.6 billion U.S. dollars to improve WatSan service coverage. The budget allocation for improving the coverage of DWS, WST, and MSW services was set to be 49.5%, 28.1%, and 22.4%, respectively. This budget allocation satisfies the result obtained in Sect. 4.2. Our research results show that the coverage of DWS, WST, and MSW services significantly impacts the control of waterborne diseases.

In 2010, the government proposed that every citizen must have improved water supplies and sanitation services. However, only 1.5% of water supplies and only 12.5% of sanitation facilities improved from 2010 to 2017. According to the 2017 UNICEF report [69], the proportion of the Haitian population with improved water supplies and sanitation services was only 65% and 35% of the total population, respectively. In Haiti, the progress of WatSan service improvement was not done according to the 10-year plan proposed by the Haitian government [68]. Therefore, insufficient WatSan service provisions were likely the cause of the persistent cholera outbreaks in Haiti.

In this section, the impacts of improving WatSan services are investigated as strategies to interrupt the transmission pathways of a cholera outbreak. Model (3.2) was simulated to determine the fraction of infected individuals and the fraction of pathogens in water reservoirs and dumpsites with varying percentages of coverage of the DWS service ($r_1$), WST service ($r_2$), and MSW service ($r_3$). The other parameters used in the simulation are given in Table 2. The dynamic behavior of the fraction of infected individuals and the fraction of pathogens in water reservoirs and dumpsites is shown in Figs. 4–6.

From the 2010 cholera outbreak data in Haiti, we set the starting values of $r_1$, $r_2$, and $r_3$ to be 65%, 35%, and 10%, respectively. The fraction of infected individuals (Figs. 4–6(top)) uses fixed values of two out of three of the other variables ($r_1 = 65\%$, $r_2 = 35\%$, or $r_3 = 10\%$). The lines in each graph show that a change in the coverage of DWS service ($r_1$) has the greatest impact in reducing the fraction of infected individuals. In Figs. 4–6(middle), the lines in each graph show that a change in the coverage of WST service ($r_2$) has the greatest impact in reducing the fraction of pathogens in water reservoirs. In Figs. 4–6(bottom), the lines in each graph show that a change in the coverage of WST service ($r_2$) has the greatest impact in reducing the fraction of pathogens in dumpsites. Figure 4 shows that the fraction of infected individuals, the fraction of pathogens in water reservoirs, and the fraction of pathogens in dumpsites decrease with an improvement in DWS service coverage. However, with a coverage of 65%, the outbreak seems to maintain a steady state of the fraction of infected individuals (noted by the blue horizontal line in Fig. 4(top)). Relatedly, the fraction of pathogens in water reservoirs and the fraction of pathogens in dumpsites (in this case) also seem to stay constant (noted by the blue horizontal line in Figs. 4(middle) and 4(bottom)). Note that with varying $r_1$, the peaks in Fig. 4(bottom) occur approximately at the same position. This is because changing the coverage of DWS service ($r_1$) does not
Figure 4 Dynamic behavior of the fraction of infected individuals (top), fraction of pathogens in water reservoirs (middle), and fraction of pathogens in dumpsites (bottom), with $r_2 = 35\%$, $r_3 = 10\%$, and varying $r_1$ from 65\%, 70\%, 80\%, 90\%, and 100\%
Figure 5 Dynamic behavior of the fraction of infected individuals (top), fraction of pathogens in water reservoirs (middle), and fraction of pathogens in dumpsites (bottom), with $r_1 = 65\%$, $r_3 = 10\%$, and varying $r_2$ from 35\%, 40\%, 60\%, 80\%, and 100\%
Figure 6 Dynamic behavior of the fraction of infected individuals (top), fraction of pathogens in water reservoirs (middle), and fraction of pathogens in dumpsites (bottom), with $r_1 = 65\%$, $r_2 = 35\%$, and varying $r_3$ from 10\%, 20\%, 40\%, 60\%, 80\%, and 100\%
directly affect the fraction of pathogens in dumpsites. Similarly, Figs. 5 and 6 show that at starting values of \( r_2 = 35\% \) and \( r_3 = 10\% \), respectively, the outbreak seems to maintain a steady state of the fraction of infected individuals, fraction of pathogens in water reservoirs, and fraction of pathogens in dumpsites.

Figure 7 displays the dynamic behavior of the fraction of infected individuals and the fraction of pathogens in water reservoirs and dumpsites with varying the coverage of two or three services at a time. Looking at the extreme case where \((r_1, r_2, r_3) = (80, 70, 50)\), the fraction of infected individuals (Fig. 7(top)) and the fraction of pathogens in water reservoirs (Fig. 7(middle)) are at the lowest. However, this does not reduce the fraction of pathogens in dumpsites (Fig. 7(bottom)) to be at the lowest. The cases of \((r_1, r_2, r_3) = (65, 70, 10)\) and \((80, 70, 10)\) have lower peaks than those of \((r_1, r_2, r_3) = (80, 70, 50)\). This shows that an increase in the coverage of MSW service \( r_3 \) or improvement in waste or garbage management does not reduce the fraction of pathogens in dumpsites within the first 150 months. The fraction of pathogens will eventually be reduced to zero, but over a long time. In other words, better MSW management means that garbage or the pathogens in the garbage are less likely to get into water reservoirs, and a disease is less likely to be transmitted to individuals via water. This improvement cuts the transmission pathway where pathogens in dumpsites can get into water, but not the fraction of pathogens in dumpsites.

Consequently, an increase in the coverage of WST service \( r_2 \) has a greater impact (short-term) on the fraction of pathogens in dumpsites than an increase in the coverage of MSW service \( r_3 \). WST has a direct impact on the fraction of pathogens in dumpsites. In many developing countries, wastewater and dumpsites are in direct contact with each other. Thus, wastewater treatment reduces the fraction of pathogens in dumpsites.

The results can be explained using Fig. 1. An increase in the coverage of DWS service \( r_1 \) also affects the fraction of pathogens in dumpsites. Figure 1 shows that a change in \( r_1 \) affects the fraction of infected individuals, which affects the quantity of infected garbage/waste. Also, Fig. 1 shows that a change in the coverage of MSW service \( r_3 \) has the least impact on the fraction of pathogens in dumpsites, as it does not affect compartment \( D \) directly. Better solid waste management means that pathogens in garbage are less likely to get into compartment \( W \). This leads to fewer pathogens in water reservoirs, which yields a smaller fraction of infected individuals and less infected garbage.

Figure 7(bottom) also shows that the fraction of pathogens in dumpsites is at the lowest when \((r_1, r_2, r_3) = (80, 70, 10)\). This implies that an increase in the coverage of DWS service \( r_1 \) and WST service \( r_2 \) affects the fraction of pathogens in dumpsites. Overall, an increase in variable \( r_1 \) or \( r_2 \) alone has less impact on the fraction of infected individuals. With increased coverage of DWS and WST services, the 10% coverage of MSW service \( r_3 \) is enough to improve the situation of a cholera outbreak in Haiti according to Fig. 7(bottom) for approximately the first 100 months. This is an important implication.

In Fig. 7(bottom), the cases of \((r_1, r_2, r_3) = (65, 70, 10)\) and \((80, 70, 10)\) result in the lowest peaks. This implies that a change in the coverage of WST service \( r_2 \) may have the greatest impact on the fraction of pathogens in dumpsites. An increase in the coverage of DWS service \( r_1 \) delineates these two lines (see 50 to 150 months). For \((r_1, r_2, r_3) = (65, 70, 50)\) and \((80, 70, 50)\) where \( r_2 = 70 \), the peaks are the second lowest. For \((r_1, r_2, r_3) = (65, 35, 10)\) and \((80, 35, 10)\), where \( r_2 = 35 \), the peaks occur approximately at the same position and at the highest. That is, it takes longer to reduce the fraction of pathogens in dumpsites.
Figure 7: Effects of improving DWS, WST, and MSW services coverage \( r_1 \), \( r_2 \), and \( r_3 \) on the fraction of infected individuals and the fraction of pathogens in water reservoirs and dumpsites that are predicted by model (3.2)
that have less improvement in DWS. In summary, the coverage of WST service \((r_2)\) plays a significant role in reducing the fraction of pathogens in dumpsites. The same pattern happens for any of the two lines where \(r_2\) and \(r_3\) are the same with only a change in \(r_1\). Peaks at any of the two lines occur at the same position. However, the decline in the fraction of pathogens (afterward) depends on the \(r_1\) value.

Figure 7 (bottom) illustrates that when \(r_1\) and \(r_2\) are the same, with only a change in \(r_1\), there are intersections between any two selected observed lines. The coverage of MSW service \((r_3)\) has a long-term impact. It reduces the fraction of pathogens significantly after approximately 170 months. In contrast, the coverage of DWS service \((r_1)\) changes the fraction of pathogens significantly from about 25 to 100 months (the time after where the peak occurs). Consequently, decision makers must consider short-term and long-term goals depending on the budget at that time. The timing of eliminating a disease may be of concern.

Figure 8 confirms that there is a threshold value. When fixing the value of \(r_3\), the coverage of DWS service \((r_1)\) and the coverage of WST service \((r_2)\) must be maintained. At the line where \((r_1, r_2, r_3) = (60, 30, 10)\), the fraction of infected individuals, the fraction of pathogens in water reservoirs, and the fraction of pathogens in dumpsites are out of control.

This study suggests that controlling and preventing waterborne diseases could be achieved by improving the service coverage of WST and MSW, to be sufficient, to protect water reservoirs and dumpsites from contamination. However, improving the service coverage of the DWS service could have the most impact among all of the WatSan services. Therefore, the WatSan service impact is explored as follows.

The results obtained in Table 5 are used as the projection of the fraction of the infected population for the next five years to cope with the 10-year plan which ends in 2022. The current data of coverage of WatSan services in 2017 from UNICEF [69] are used as the current situation (scenario 1). In scenario 1, the coverage of DWS, WST, and MSW services is 65%, 35%, and 10%, respectively. These percentages correspond to the values of \(r_1 = 0.65\), \(r_2 = 0.35\), and \(r_3 = 0.10\), respectively. These values, with other parameter values given in Table 4, lead to \(R_0 = 1.0073\). In addition, the percentage reduction in the infected population is calculated by comparing the fraction of the infected population to the baseline, computed from the parameter values in Table 4. There is a 66.0674% reduction in the fraction of the infected population for scenario 1. By fixing all parameter values in scenario 1 except the coverage of each WatSan service, the threshold service coverage for each WatSan service that makes \(R_0 = 1\) is 65.28% of the service coverage of DWS \((r_1)\), or 35.49% of the service coverage of WST \((r_2)\), or 13.95% of the service coverage of MSW \((r_3)\).

A little improvement in each WatSan service while maintaining the other services would alleviate the disease outbreak. The experiments are extended by increasing or decreasing the percentage of \(r_1\), \(r_2\), and \(r_3\) by 5% from scenario 1, as shown in Table 5. Scenarios 2–8 decrease \(r_1\), \(r_2\), and \(r_3\), while scenarios 9–15 increase \(r_1\), \(r_2\), and \(r_3\) by 5% from scenario 1. It is found that decreasing \(r_1\), \(r_2\), or \(r_3\) from scenario 1 while maintaining the other services could result in a reduction in the infected population that is worse than scenario 1.

\(R_0\) in scenarios 2–8 are higher than \(R_0\) in scenario 1. In scenarios 9–15, increasing \(r_1\), \(r_2\), or \(r_3\) from scenario 1 while maintaining the other services could result in a percentage reduction in the infected population that is better than scenario 1. In addition, \(R_0\) in scenarios 9–15 is less than \(R_0\) in scenario 1. The impact of decreasing \(r_1\), \(r_2\), or \(r_3\) is greater
Figure 8: Effects of improving DWS and WST services coverage ($r_1$ and $r_2$) on the fraction of infected individuals and the fraction of pathogens in water reservoirs and dumpsites that are predicted by model (3.2).
Table 5  Impact of WatSan services on the percentage reduction in the infected population

| Scenarios | \( r_1 \) | \( r_2 \) | \( r_3 \) | % reduction of infected population | \( R_0 \) |
|-----------|------------|------------|------------|-----------------------------------|--------|
| 1         | 0.6500     | 0.3500     | 0.1000     | 66.0674                           | 1.0073 |
| 2         | 0.6000     | 0.3500     | 0.1000     | 36.0778                           | 1.1370 |
| 3         | 0.6500     | 0.3000     | 0.1000     | 48.4736                           | 1.0158 |
| 4         | 0.6500     | 0.3500     | 0.0500     | 60.0196                           | 1.0158 |
| 5         | 0.6000     | 0.3000     | 0.1000     | 8.5914                            | 1.2217 |
| 6         | 0.6000     | 0.3500     | 0.0500     | 32.9038                           | 1.1466 |
| 7         | 0.6500     | 0.3000     | 0.0500     | 46.1522                           | 1.0912 |
| 8         | 0.6000     | 0.3000     | 0.0500     | 3.3407                            | 1.2321 |
| 9         | 0.7000     | 0.3500     | 0.1000     | 75.5579                           | 0.8776 |
| 10        | 0.6500     | 0.4000     | 0.1000     | 70.6619                           | 0.9325 |
| 11        | 0.6500     | 0.3500     | 0.1500     | 63.0107                           | 0.9980 |
| 12        | 0.7000     | 0.4000     | 0.1000     | 80.0980                           | 0.8128 |
| 13        | 0.7000     | 0.3500     | 0.1500     | 76.2678                           | 0.8695 |
| 14        | 0.6500     | 0.4000     | 0.1500     | 71.6331                           | 0.9239 |
| 15        | 0.7000     | 0.4000     | 0.1500     | 80.5796                           | 0.8053 |

than the impact of increasing \( r_1 \), \( r_2 \), or \( r_3 \) when comparing the percentage reduction in the infected population for scenarios 2–8 and 9–15. The result in scenario 5 shows that decreasing \( r_1 \) and \( r_2 \) decreases the reduction in the infected population significantly. Scenario 12 demonstrates that increasing \( r_1 \) and \( r_2 \) increases the reduction in the infected population remarkably. However, the impact on the reduction in the fraction of the infected population is greater for scenario 5 (from 66.0674 % to 8.5914%) than for scenario 12 (from 66.0674 % to 80.0980%).

Thus, it is concluded that maintaining the service coverage of \( r_1 \) and \( r_2 \) is important to prevent the transmission of waterborne diseases. Figure 8 shows the impact of improving and reducing \( r_1 \) and \( r_2 \). Reducing both services could make water reservoirs and dumpsites more contaminated. This impacts the fraction of the infected population. Our study shows that maintaining the service coverage of MSW (\( r_3 \)) while improving the service coverage of DWS and WST (\( r_1 \) and \( r_2 \)) could have the most health impact on a community. Thus, our study suggests that shifting the budget away from improving the service coverage of MSW to just maintaining this service is a desirable policy. In addition, the shifted budget could be used for improving the service coverage of DWS and WST instead. According to the 2013–2022 plan to combat cholera in Haiti, the budget allocation for improving the service coverage of DWS, WST, and MSW is 49.5%, 28.1%, and 22.4%, respectively. This allocation is suitable for fighting cholera. However, to efficiently eliminate cholera from Haiti, our study recommends that the allocated budget of 22.4% to improve MSW should be reduced. This reduction in the budget should be used to improve the service coverage of DWS and WST by emphasizing the improvement of DWS.

As described earlier, proposed model (2.1) is suitable when there are other pathogen reservoirs that are important in the disease transmission process. This study extends the Tien and Earn (2010) model that takes into account only one pathogen reservoir, i.e., a water reservoir [28]. This study finds that environmental factors play an important role in controlling and preventing disease outbreaks. Considering the water compartment (\( W \)) and dumpsite compartment (\( D \)) is more effective than considering only compartment \( W \) if dumpsites in a particular community are contaminated. Additionally, our mathematical model quantifies the qualitative data that many researchers [41] have agreed upon. Wat-
San services do indeed prevent and control waterborne diseases. According to this paper, WatSan services should not be given equal priority.

5 Conclusions

This paper presents the modified SIR model by incorporating access to WatSan services and the prevalence of open dumpsites as pathogen enablers. The proposed model is rigorously analyzed to gain insights into its dynamic behavior. Then, it is used to investigate the health impact of WatSan service deficits on waterborne disease transmission. Using cholera as our case study, our analysis yielded some useful new insights into the dynamics and control of waterborne diseases. The basic reproductive number ($R_0$) of the model is derived and given in Equation (3.6) by using the next generation method. It was found that the disease-free equilibrium is globally asymptotically stable whenever $R_0 \leq 1$ as guaranteed by Theorem 3.2. When $R_0 > 1$, the endemic equilibrium (EE) of the proposed model exists and is globally asymptotically stable, which is consistent with Theorem 3.4. These theoretical studies reveal that the disease dies out when $R_0 \leq 1$ and the disease becomes endemic when $R_0 > 1$. Therefore, control strategies that set $R_0 \leq 1$ would generate public health benefits in making strategy decisions after infectious diseases appear. The appropriate parameter values of the model were obtained by comparing the predicted data with real data from the 2010 cholera outbreak in Haiti. These parameter values were used to investigate the most sensitive model parameters in controlling and preventing cholera outbreaks. It was found that $\beta_W$ is the most sensitive parameter to the initial transmission, and $r_1$ is the most sensitive parameter to the initial decrease in disease transmission. The coverage of DWS services is the most sensitive parameter to the initial decrease in disease transmission in relation to the coverage of WST and MSW services. In other words, this study verifies that improving the coverage of DWS services is the most effective intervention to control a cholera outbreak. Analyzing the contour plots of surface $R_0$ as a function of the service coverages ($r_1$, $r_2$, and $r_3$) demonstrates that improving all the service coverages (DWS, WST, and MSW) would certainly decrease the initial disease transmission. Moreover, analyses of single and double control strategies showed that control strategies that involve the combined improvement of multiple water and sanitation services are better at controlling the initial disease transmission and reducing the overall infections than single strategies.

Therefore, this study suggests that to control the spread of infectious waterborne diseases like cholera, the first priority should be to improve the access to safe drinking water supplies. As funding and other resources permit, the effectiveness of control measures would subsequently be increased by adding improved treatment of contaminated water sources and improved management of open dumpsites to the improved drinking water supply. To some degree these results are intuitive. Certainly, to improve access to safe drinking water supplies, the treatment of contaminated water sources would be required. By similar reasoning, improved management of open dumpsites could reduce the extent of leachate contamination of groundwater and control harmful runoff into surface water bodies that serve as sources for drinking water supplies. Thus, prioritizing the access to safe drinking water supplies is likely to require improved treatment of contaminated water bodies and improved management of community dumpsites as effective strategies for cutting the transmission pathways of waterborne diseases like cholera. This is an important implication and a contribution of this study.
The comparative results of the effects of each service are illustrated in Table 5 for the case of enteric diarrheal disease (EDD). In this case, we found that improving DWS and WST services yields a higher reduction in EDD than improvements in the MSW service. This result suggests that WatSan service managers should prioritize investment in DWS and WST services over the MSW service in a strategy to control the incidence of EDD from waterborne sources. The budget for infrastructure services may be limited, as it is in many low- and middle-income countries. Therefore, this strategy could be useful in budget allocation for improving WatSan services to manage outbreaks of waterborne diseases. Furthermore, model (2.1) is formulated by incorporating the state variables of WatSan services and the incidence of EDD. Thus, if reliable data on the parameters of a particular disease and the unit costs of WatSan services are available, model (2.1) can be applied to design optimization algorithms for budget allocation in improving WatSan services, to minimize the size of the infected population during disease outbreaks. This is the focus of our future work.

Acknowledgements
The authors would like to express their gratitude to the anonymous referee for helpful suggestions and comments which led to improvements in our original manuscript.

Funding
This research is supported by the Institute for the Promotion of Teaching Science and Technology (IPST), Thailand, under the Research Fund for DPST (Development and Promotion of Science and Technology Talents Project) Graduate with First Placement (Grant no. 033/2559).

Availability of data and materials
The authors declare that all data and material in the paper are available and verifiable.

Competing interests
The authors declare that they have no competing interests.

Authors’ contributions
RC and GEL conceived this research. RC developed the SIWDR model, conducted the numerical simulations, performed analysis and drafted the manuscript. GEL revised the manuscript. WC participated in the interpretation of the data, and revised the manuscript. All authors read and approved the final manuscript.

Author details
1 School of Management Technology, Sirindhorn International Institute of Technology, Thammasat University, Pathum Thani, Thailand. 2 Department of Engineering Systems and Environment, University of Virginia, Charlottesville, Virginia, USA. 3 Department of Mathematics, Faculty of Science, King Mongkut’s University of Technology Thonburi, Bangkok, Thailand.

Publisher’s Note
Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Received: 5 March 2021 Accepted: 17 August 2021 Published online: 06 September 2021

References
1. Griffiths, J.K.: Waterborne diseases. In: International Encyclopedia of Public Health, 2nd edn., pp. 388–401. Elsevier, Amsterdam (2017).
2. Sharma, S., Kumar, N.: Dynamics of a waterborne pathogen model under the influence of environmental pollution. Appl. Math. Comput. 346, 219–243 (2019).
3. World Health Organization: Drinking-water (2020).
   https://www.who.int/news-room/fact-sheets/detail/drinking-water. Accessed 23 May 2021
4. World Health Organization (WHO, and UNICEF): Global water supply and sanitation assessment 2000 report (2000).
   http://www.who.int/water_sanitation_health/monitoring/jmp2000.pdf. Accessed 23 May 2021
5. Centers for Disease Control and Prevention: Cholera in Haiti (2014). https://www.cdc.gov/cholera/haiti/index.html.
   Accessed 23 May 2021
6. United Nations: Yemen: as cholera surges again, UN and partners double down on vaccination efforts (2018).
   https://news.un.org/en/story/2018/10/1022062. Accessed 23 May 2021
7. Fewtrell, L., Kaufmann, R.B., Kay, D., Enanoria, W., Haller, L., Colford, J.M. Jr.: Water, sanitation, and hygiene interventions to reduce diarrhoea in less developed countries: a systematic review and meta-analysis. Lancet Infect. Dis. 5(1), 42–52 (2005)
32. Mari, L., Bertuzzo, E., Righetto, L., Casagrandi, R., Gatto, M., Rodriguez-Iturbe, I., Rinaldo, A.: Modelling cholera epidemics: the role of waterways, human mobility and sanitation. J. R. Soc. Interface 9(67), 376–388 (2012)
33. Gatto, M., Mari, L., Bertuzzo, E., Casagrandi, R., Righetto, L., Rodriguez-Iturbe, I., Rinaldo, A.: Generalized reproduction numbers and the prediction of patterns in waterborne disease. Proc. Natl. Acad. Sci USA 109(48), 19703–19708 (2012)
34. Wang, Y., Cao, J.: Global dynamics of a network epidemic model for waterborne diseases spread. Appl. Math. Comput. 237, 474–488 (2014)
35. Collins, O.C., Govinder, K.S.: Incorporating heterogeneity into the transmission dynamics of a waterborne disease model. J. Theor. Biol. 356, 133–143 (2014)
36. Tuite, A.R., Tien, J., Eisenberg, M., Earn, D.J., Ma, J., Fisman, D.N.: Cholera epidemic in Haiti, 2010: using a transmission model to explain spatial spread of disease and identify optimal control interventions. Ann. Intern. Med. 154(9), 593–601 (2011)
37. Collins, O.C., Robertson, S.L., Govinder, K.S.: Analysis of a waterborne disease model with socioeconomic classes. Math. Biosci. 269, 86–93 (2015)
38. Mari, L., Casagrandi, R., Bertuzzo, E., Rinaldo, A., Gatto, M.: Conditions for transient epidemics of waterborne disease in spatially explicit systems. R. Soc. Open Sci. 6, 181517 (2019)
39. Mwasa, A., Tchuene, J.M.: Mathematical analysis of a cholera model with public health interventions. Biosystems 105, 190–200 (2011)
40. Wang, Y., Cao, J.: Global stability of a multiple infected compartments model for waterborne diseases. Commun. Nonlinear Sci. Numer. Simul. 19, 3753–3765 (2014)
41. Keusch, G.T., Fontaine, O., Bhargava, A., Boschetti, C., Bhutta, Z.A., Gotuzzo, E., Laxminarayan, R., Diarrheal diseases In: Jamison, J.T., Breman, J.G., Measham, A.R. (eds.) Disease Control Priorities in Developing Countries, 2nd edn., pp. 371–388. Oxford University Press, New York (2006)
42. Hethcote, H.M.: The mathematics of infectious diseases. SIAM Rev. 42, 599–693 (2000)
43. Van den Driessche, P., Watmough, J.: Further notes on the basic reproduction number. In: Brauer, F., van den Driessche, P., Wu, J. (eds.) Mathematical Epidemiology, pp. 159–178. Springer, Berlin (2008)
44. Hale, J.K.: Ordinary Differential Equations. Wiley, New York (1969)
45. Carr, J.: Applications of Centre Manifold Theory. Springer, New York (2012)
46. Castillo-Chavez, C., Song, B.: Dynamical models of tuberculosis and their applications. Math. Biosci. Eng. 1(2), 361–404 (2004)
47. Zhou, T.: Global stability. In: Dubitzky, W., Wolkenhauer, O., Cho, K.H., Yokota, H. (eds.) Encyclopedia of Systems Biology, p. 842. Springer, New York (2013)
48. Korobeinikov, A., Wake, G.C.: Lyapunov functions and global stability for SIR, SIRS, and SIS epidemiological models. Appl. Math. Lett. 15(8), 955–960 (2002)
49. Vargas-De-León, C.: On the global stability of SIS, SIR and SIRS epidemic models with standard incidence. Chaos Solitons Fractals 44(12), 1106–1110 (2011)
50. Roop-O, P., Chinviriyasit, W., Chinviriyasit, S.: The effect of infection function in backward bifurcation for malaria model with temporary immunity. Math. Biosci. 265, 47–64 (2015)
51. Nudée, K., Chinviriyasit, S., Chinviriyasit, W.: The effect of backward bifurcation in controlling measles transmission by vaccination. Chaos Solitons Fractals 123, 400–412 (2019)
52. LaSalle, J.P.: The Stability of Dynamical Systems. Hamilton Press, New Jersey (1976)
53. Centers for Disease Control and Prevention: Eliminating cholera transmission from Haiti (2018). https://www.cdc.gov/globalhealth/countries/haiti/what/eliminating-cholera-haiti.html. Accessed 23 May 2021
54. World Bank: Population in largest city (2020). https://data.worldbank.org/indicator/EN.URB.LCTY. Accessed 23 May 2021
55. The World Bank: Birth rate, crude (per 1,000 people)—Haiti (2019). https://data.worldbank.org/indicator/SP.DYN.CBRT.IN?locations=HT. Accessed 23 May 2021
56. The World Bank: Death rate, crude (per 1,000 people)—Haiti (2019). https://data.worldbank.org/indicator/SP.DYN.CDRT.IN?locations=HT. Accessed 23 May 2021
57. Fung, I.C.H.: Cholera transmission dynamic models for public health practitioners. Emerg. Theme Epidemiol. 11(1), 1 (2014)
58. King, A.A., Ionides, E.L., Pascual, M., Bouma, M.J.: Inapparent infections and cholera dynamics. Nature 454(7206), 877–880 (2008)
59. World Health Organization: Cholera (2012). http://www.unicef.org/cholera/Cholera/Chapter_1_intro/01_WHO_Cholera_Fact_sheet_187_July_2012.pdf. Accessed 23 May 2021
60. Hendrix, T.R.: The pathophysiology of cholera. Bull. N.Y. Acad. Med. 47(10), 1169–1180 (1971)
61. UNICEF: Cholera toolkit (2013). http://www.unicef.org/cholera/Cholera-Toolkit-2013.pdf. Accessed 23 May 2021
62. World Health Organization: Progress on drinking-water and sanitation—2012 Update (2012). http://www.who.int/media-centre/files/JMPreport2012.pdf. Accessed 23 May 2021
63. World Health Organization: 25 Years progress on sanitation and drinking water (2015). http://apps.who.int/nr/iris/bitstream/handle/10665/177752/9789241509145_eng.pdf. Accessed 23 May 2021
64. Pan American Health Organization: Atlas of cholera outbreak in La Hispaniola 2010–2016 (2016). https://www3.paho.org/hq/images/Atlas_IHR/CholeraHispaniola/atlashtml. Accessed 23 May 2021
65. Mukandavire, Z., Smith, D.L., Morris, J.G. Jr.: Cholera in Haiti: reproductive numbers and vaccination coverage estimates. Sci. Rep. 3, 997 (2013)
66. Ministry of Public Health and Population National Directorate for Water Supply and Sanitation, Republic of Haiti: National plan for the elimination of cholera in Haiti 2013–2022 (2013). https://www.paho.org/hq/mdmdocuments/2013/cholera-haiti-nat-plan-elim-2013-2022.pdf. Accessed 23 May 2021
67. UNICEF: Progress on household drinking water, sanitation and hygiene, 2000–2017 (2019). https://data.unicef.org/resources/progress-drinking-water-sanitation-hygiene-2019/. Accessed 23 May 2021