Application of the Susceptible Infectious Susceptible (sis) model for the appraisal of the disseminating potential of waterborne parasites in domestic water sources in Bamenda (Cameroon)

Abstract

This research presents a distributed continuous-time epidemic model, called a SUSCEPTIBLE INFECTIOUS SUSCEPTIBLE- SIS model to denote a waterborne disease spreading over a network of different domestic waters sources such as tap water, well water and spring water in the understanding of infectious pathogens in the Municipality of Bamenda. It involves multiple groups of individuals sharing a water source which comprise the population in the communities or quarter of Upstation mile 1, Nkwen, Mankon and Nsongwa in and around this urban area. A sufficient condition is obtained for the healthy state, at which all individuals are not infected and the water is not contaminated, to be globally asymptotically stable. The effects of the shared water source on the disease spreading are analyzed through the comparison of the basic reproduction number with the networked SIS model without water and demonstrated via simulations. It was realized that human activities and generally anthropogenic influences contaminate these water sources making individuals of this localities to become susceptible to waterborne infections. The characterization and identification of intestinal parasites reveal that , those who consume these comtaminated water were infected and these infected individuals intend recontaminate the environment including water bodies and or food thereby by making the population and uninfected individuals susceptible to waterborne diseases in the community..

I.INTRODUCTION

Waterborne pathogens have caused diseases and other health problems worldwide, especially in developing countries. Water systems (e.g., rivers, groundwater, and reservoirs) are important pathways for transmitting pathogens (Kough. 2015). Therefore, some recent studies have considered the role of a water compartment in epidemic dynamic processes (Vermeulen *et al.,* 2015). There are various routes through which these pathogens enter a human body. They might be transmitted directly by consumption of contaminated water or indirectly by eating food that has come in contact with contaminated water, or by using a device (toothbrush etc.) that has contaminated water. They may also enter while taking a

bath in contaminated water by penetrating skin, or through cut or wound on body (ECDC, 2009). Although a lot of measures are introduced by government to improve sanitation, new challenges still emerge.

A distributed continuous-time epidemic model, called networked SIWS (Susceptible-Infected Water-Susceptible) model, for an SIS type waterborne disease spreading over a network of multiple groups of individuals sharing a water source (Tien and Earn, 2010). A sufficient condition is obtained for the healthy state, at which all individuals are not infected and the water is not contaminated, to be globally asymptotically stable (Zhou et al., 2018). The effects of the shared water source on the disease spreading are analyzed through the comparison of the basic reproduction number with the networked SIS model without water and demonstrated through simulations.

Modeling can often be used to compare different water borne diseases (enteropathogenic Protozoa and Helminthic infections) in the same population of the city of Bamenda and its environs and or the same disease at different times (monthly and seasonal evealuations from November 2022 to July 2023). The model is used to compare waterborne disease control procedures such as screening for (*Nematode larvae, Ascaris lumbricoides, Balantidium coli , Entamoeba histolytica, Toxocara cati, Diphyllobothrium latum, Cryptosporidium parvum, Gardia lamblia, Fasciola hepatica, Hymenolepis nana, Schistosoma hematobium and Tenia saginata*), rescreening, tracing infectors, tracing infectees, post-treatment vaccination and general vaccination (Zhang et al., 2016). The modeling leads to a clear statement of the assumptions about the biological and sociological mechanisms which influence spread of gastroenteritis in Bamenda and its environs. The parameters used in an epidemiological model must have a clear interpretation such as a contact rate or a duration of infection. The objectives of this research is to proof the validity of the susceptibility of an infectious model for the spread of protozoans and helminthes in the municipality of Bamenda and its environs.

II-Methodological applications

II.1- Assumptions and Notation of modelling in Bamenda urban area.

The population of Bamenda and its environs under consideration is divided into disjoint classes which change with time *t.* The susceptible class to gastrointestinal water borne infections consists of those individuals who can incur the disease but are not yet infective. The infective class consists of those who are transmitting the disease to others. The removed class consists of those who are removed from the susceptible-infective interaction by recovery with immunity,

isolation, or death. The fractions of the total population in these classes are denoted by S(l), /(t) and R(t), respectively.

In the epidemiological models here, the following assumptions are made:

I. The population of Bamenda and its environs considered has constant size *N* which is sufficiently large so that the sizes of each class is considered as continuous variables. If the model is to include vital dynamics, then it is assumed that births and natural deaths occur at equal rates and that all newborns are susceptible.Individuals are removed by death from each class at a rate proportional to the class size with proportionality constant μ which is called the daily death removal rate. This corresponds to a negative exponential age structure with an average lifetime of l/μ.

2. The population Bamenda and its environs is homogenously mixing. The daily contact rate λ , is the average number of adequate contacts per infective per day. An adequate contact of an infective is an interaction which results in infection of the other individual if he is susceptible. Thus the average number of susceptibles infected by an infective per day is λS, and the average number of susceptibles infected by the infective class with size *NI* per day in λSNI. The daily contact rate λ is fixed and does not vary seasonally. The type of direct or indirect contact adequate for transmission depends on the specific disease or reservoir characteristics of the causative agents . The number of cases per day $\lambda SN\ I$, which is called the incidence, is a mass action law since it involves the product of S and *I.*

3. Individuals recover and are removed from the infective class at a rate proportional to the number of infectives with proportionality constant y, called the daily recovery removal rate. The latent period is zero (it is defined as the period between the time of exposure and the time when infectiousness begins). Thus the proportion of individuals exposed (and immediately infective) at time t0 who are still infective at time t_0 +tis exp (- yt), and the average period of infectivity is l /y.

The removal rate from the infective class by both recovery and death is y + μ so that the death-adjusted average period of infectivity is l/(y +μ).Thus the average number of adequate contacts (with both susceptibles and others) of an infective during the infectious period is α= λ/(y + μ), which is called the contact number.(basic reproductive rate).

Since the average number of susceptibles infected by an infection during the infectious period is αS, the quantity αS is called the replacement number. Since recovery these water borne pathogens in Bamenda domestic water sources can easily propagate to affect uninfected individuals and the fact that there is little or no immunization of the population for these water borne pathogens, then the

model is called an SIS model, since individuals move from the susceptible class (exposed to biological and physico- chemical pollution or contaminating agents) to the infective class (those who ingested the contaminated water and or food to acquire the infection) and then back to the susceptible class upon recovery.

III. Results and Discussion

III. 1- The SIS Model for estimataion of dissemination of parasitic waterborne pathogens in domestic water sources in Bamenda and its environs.

In this case, an extension of the networked SIS model is done by adding a water compartment W by taking into consideration the different domestic water sources that was evaluated for enteropathogens, in which both person-person and person-water-person transmissions exist, and thus we call it the networked SIWS model. The way to model and interpret a networked SIS model is to regard each agent of gastroenteritis as a group of fully connected individuals and each agent's variable represents the proportion of infected individuals in the corresponding group of enteropathogenic organisms. The variables take values between zero and one. Since we are interested in studying the epidemic spreading of a waterborne disease over multiple groups of individuals, the interpretation and its corresponding model derivation fits our purpose.

The main contributions of this model are three-fold. First, we propose a networked model for SIS-type waterborne diseases, called networked SIWS model, for a system consisting of multiple groups of individuals (Upstation, Nkwen, Nsongwa and Mnakon) with a shared water resource. Second, we obtain a sufficient condition for the healthy state to be globally asymptotically stable. Third, we compare the basic reproduction number per quarter of domestic water source of the networked SIWS model with that of the networked SIS model for non-waterborne diseases, and provide a set of simulations to demonstrate the behavior of the networked SIWS model differing from the networked SIS model.

Consider an SIS type waterborne disease spreading over a network consisting of n > 1 groups of individuals, labeled 1 to n, and a water compartment shared among the n groups. The water compartment can be contaminated by infected individuals shedding the pathogen into it. We simulate the water compartment W as a reservoir-like water system with homogeneous water quality, assuming instantaneous pathogen diffusion process in W. An individual may be infected either by contact with contaminated water or by contact with infected individuals only in its own and neighboring groups around the domestic water sources in

Upstation, Nkwen, Nsomgwa and Mankon considered .Neighbour relationships among the n groups are described by a directed graph G on n vertices with an arc (or a directed edge) from vertex j to vertex i whenever the individuals in group i can be infected by those in group j. Thus, the neighbour graph G has self-arcs at all n vertices, and the directions of arcs in G represent the directions of epidemic contagion. It is assumed that G is strongly connected. We also assume that each group has bidirectional connection with the water compartment, which implies that each group can contaminate the water if it has infected individuals, and the individuals in each group can in turn get infected by the water if it is contaminated. Let Ii (t) and Si (t) respectively denote the number of infected and susceptible individuals in group i at time $t \geq 0$. We assume that the total number of individuals in each group i, denoted by Ni, does not change over time. In other words, $Si(t) + Ii(t) = Ni$ for all $i \in [n]$ and $t \geq 0$, which implies that the birth and death rates for each group are equal. Such an assumption simplifies the model and has been adopted in (Fall et al., 2007). We leave the relaxed, and more realistic, scenarios without this assumption as future work. Associate with each group i several parameters: curing rate γi , birth rate μi , death rate $\bar{\mu} i$, person-to-person infection rates αij (with the understanding that $\alpha ij > 0$ whenever group j is a neighbor of group i and $\alpha ij = 0$ otherwise), and water-to-person infection rates αiw. Since Ni is constant, there holds $\bar{\mu} i = \mu i$. We assume that individuals are susceptible at birth even if their parents are infected. The evolution of the numbers of infected and susceptible individuals in each quarter of Bamenda and its environs i is as follows: 360 for *Entamoeba histolytica*, 178 for *Balantidium coli*. 199 for *Gardia lamblia* and 210 *Cryptosporidium parvum* for Protozoa infections while for Helminthic infections 218 for *Ascaris lumbricoides,* 219 for *Hymenolepis nana,* 192 for *Diphyllobothrium latum,* 178 for *Schistosoma hematobium,* 256 for *Tenia saginata,* 159 for *Toxocara cati and* 108 for *Fasciola hepatica.*

III.2 Mathematical basis of SIS model data analysis.

$$\begin{aligned}\dot{S}_i(t) &= \mu_i N_i - \bar{\mu}_i S_i(t) + \gamma_i I_i(t) - \sum_{j=1}^{n} \alpha_{ij} \frac{S_i(t)}{N_i} I_j(t) \\ &\quad -\alpha_{iw} W(t) S_i(t) \\ &= (\mu_i + \gamma_i) I_i(t) - \sum_{j=1}^{n} \alpha_{ij} \frac{S_i(t)}{N_i} I_j(t) \\ &\quad -\alpha_{iw} W(t) S_i(t), \qquad (1) \\ \dot{I}_i(t) &= -\gamma_i I_i(t) - \bar{\mu}_i I_i(t) + \sum_{j=1}^{n} \alpha_{ij} \frac{S_i(t)}{N_i} I_j(t) \\ &\quad +\alpha_{iw} W(t) S_i(t) \\ &= (-\gamma_i - \mu_i) I_i(t) + \sum_{j=1}^{n} \alpha_{ij} \frac{S_i(t)}{N_i} I_j(t) \qquad (2) \\ &\quad +\alpha_{iw} W(t) S_i(t),\end{aligned}$$

Where $W(t)$ denotes the pathogen concentration in the water reservoir which evolves as

$$\dot{W}(t) = -\delta_w W(t) + \sum_{k=1}^{n} \zeta_k I_k(t) \qquad (3)$$

where δ_w denotes the decay rate of pathogen in the water, and ζ_k denotes the person to water contact rate of group k. Note that (1) and (2) implies that $\dot{S}_i(t) + \dot{I}_i(t) = 0$, which is consistent with the assumption that N_i is a constant.To simplify the model and for the purpose of analysis, we change the variables of the model as follows. First, we denote the portion of infected individuals in each group i by $x_i(t)$, and thus,

$$x_i(t) = \frac{I_i(t)}{N_i}$$

Second, define a new variable as

$$z(t) = \frac{\delta_w}{\sum_{k=1}^{n} \zeta_k N_k} W(t),$$

which can be regarded as an index describing the waterborne pathogen concentration. Set the following parameters: which can be regarded as an index describing the waterborne pathogen concentration. Set the following parameters:

$$\delta_i = \gamma_i + \mu_i, \quad \beta_{ij} = \alpha_{ij} \frac{N_j}{N_i}, \quad \beta_{iw} = \frac{\alpha_{iw}}{\delta_w} \sum_{k=1}^{n} \zeta_k N_k .$$

Then, from (1) and (2), it follows that

$$\dot{x}_i(t) = -\delta_i x_i(t) + (1 - x_i(t))\left(\sum_{j=1}^{n} \beta_{ij} x_j(t) + \beta_{iw} z(t)\right) \quad (4)$$

To proceed, let

$$c_i = \frac{\zeta_i N_i}{\sum_{k=1}^{n} \zeta_k N_k}. \quad (5)$$

Then, from (3), it follows that

$$\dot{z}(t) = \delta_w \left(-z(t) + \sum_{k=1}^{n} c_k x_k(t)\right). \quad (6$$

We impose the following assumptions on the system parameters .

Study the following continuous-time networked system with specified initial conditions:

$$\dot{x}_i(t) = -\delta_i x_i(t) + (1 - x_i(t))\left(\sum_{j=1}^{n} \beta_{ij} x_j(t) + \beta_{iw} z(t)\right) \quad (7)$$

$x_i(0) \in [0,1]$, $i \in [n]$,

where δi , δw, βij , βiw, and ci are model parameters satisfying Assumption 1. The above n + 1 differential equations can be combined into one equation in a compact form. Toward this end, let x(t) be the state vector in IRn whose ith entry is xi(t), D be the n × n diagonal matrix whose ith diagonal entry is δi , B be the n × n matrix whose ijth entry is βij , X(t) be the n × n diagonal matrix whose ith diagonal entry is xi(t), b be the vector in IRn whose ith entry is βiw, and c be the vector in IRn whose ith entry is δwci . Then, from (7) and (8), it can be verified that:

$\dot{x}(t) = (-D + B - X(t)B)x(t) + (I - X(t))bz(t)$, (9)

$\dot{z}(t) = -\delta_w z(t) + c^{>} x(t)$, (10)

III.3- Mechanism of propagation of the infectious forms with respect to the SIS model.

The chosen applicable SIS model with regards to study were presented in form of an illustration showing showing the two way contamination scenarios where healthy individuals were susceptible to water borne enteropathogenic protozoa and Helminths when exposed to contaminated domestic water sources. While on the other hand , the infected individuals keep on contaminating other

water bodies and making other members of the community vulnerable to waterborne infections as as seen in illustration in figure 1.

Figure 1 : **Schematic illustration of SIS model that was applicable for this study.**

Shaded circles represent activated individuals in each domestic water samplying point. Arrows mean the possibility of propagation of infections. If the nodes to which the arrows point are susceptible and the source nodes are infected, then the propagation of gastroenteritis due to enterpathogenic Helminths and protozoa occurs Table I.

Table 1 : Properties of SIS Model

Model	Contacts	Active individual	Outbreak threshold	Equilibrium density of infected
1a	all neighbors	sender	vanish	Same as Epidemic Spreading in Scale-Free Networks.
1b	all neighbors	receiver	vanish	lower than Model 1a
1c	all neighbors	hybrid	vanish	intermediate of 1a and 1b
2a	one neighbor	sender	finite	lower than Model 2b
2b	one neighbor	receiver	finite	same as well-mixed case
2c	one neighbor	hybrid	vanish	

We have analyzed the spreading phenomena on scale-free networks using six SIS models with different contact and propagation mechanisms. Figure 2 **s**hows a decent match between theoretical predictions and numerical results. In Models 1a, b, c, the theory is not as accurate as in Models 2a, b, c. This is because the theory neglects the probability of an individual to re-infect the neighbor that had previously infected it (Parshani 2010). This probability increases with the rate λ.Figure 2.

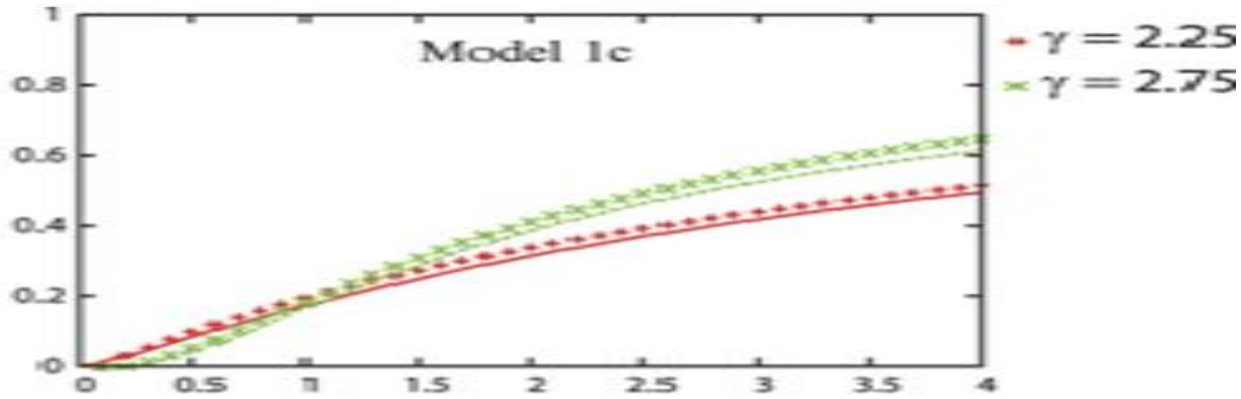

Figure- 2: The density of infected individuals ρ* is plotted as a function of λ for the six different models, when γ is 2.25 (red) or 2.75 (green).

The curves show the theoretical predictions, while the crosses represent the numerical results . In the numerical simulations, the system size is set to N = 100000 and each point is obtained by averaging over 10000 unit time after 10000 relaxation time on 10 different network realizations. Error bars are smaller than the size of the data point symbols. In this study, we formulated a model for water borne diseases using system of differential equations. We divided the human population into five compartments and took a sixth compartment for pathogen population. Relation for basic reproduction number, R0, is established and disease free equilibrium is analyzed. Analysis result shows that the disease free equilibrium is locally asymptotically stable if R0<1. Sensitivity analysis is done using parametric values for typhoid. This tells us that the most crucial parameters which escalate disease spread are environment-to-person transmission rate and pathogen population. The parameters that have negative impact on the disease spread are pathogen death rate and the rate moving out from symptomatically infected class.

CONCLUSION

We have obtained the basic reproduction number for the networked model and shown that the healthy state is globally asymptotically for

gastreoenteritis, stable if the number is less than or equal to one as reflected on the 360 for *Entamoeba histolytica*, 178 for *Balantidium coli*. 199 for *Gardia lamblia* and 210 *Cryptosporidium parvum* for Protozoa infections while for Helminthic infections 218 for *Ascaris lumbricoides,* 219 for *Hymenolepis nana,* 192 for *Diphyllobothrium latum,* 178 for *Schistosoma hematobium,* 256 for *Tenia saginata,* 159 for *Toxocara cati and* 108 for *Fasciola hepatica.* Sensitivity analysis results indicate that contaminated environment is more responsible for the spread of these diseases. Simulation results show that population in infected (I) class increases very fast and parallel to it, pathogen population also shoots up. So, it concludes that we need to take special care of the individuals in this class by means of starting treatment as soon as we identify them, proper and extra careful sanitation and disinfection of patient's belongings, rooms and toilets. We also need to treat the water and drainage of the area where patient reside so that the disease bacteria will not contaminate the environment of that area.

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