ASIAN JOURNAL OF MATHEMATICS AND APPLICATIONS
Volume 2017, Article ID ama0437, 18 pages
ISSN 2307-7743
http://scienceasia.asia

MODELING THE TRANSMISSION OF URINARY TRACT INFECTION (UTI) IN HUMAN POPULATION

XAVERY IDAN MBENA[1,*], DAMIAN KAJUNGURI[1,3], JOSEPH SSEBULIBA[2]

Abstract. Urinary tract infection (UTI) surrounds the urinary tract system and is mainly transferred between populations through sex and contact contaminated with UTIs. A mathematical model is used to examine the transmission of the UTIs. The disease free equilibrium and the basic reproduction number (R_0) were established using the next generation matrix method. The basic reproduction number was used to ascertain local and global stabilities for the disease free equilibrium. Sensitive parameters that regulate the dynamics of UTI transmission were identified. Numerical simulations were used to verify the analytical solutions of the model. Results show that drying-out stagnant water, disinfection and sterilization of items in recognition to water and other environment like flows, toilets and bath-houses, may be one of the effective measure for controlling the prevalence and incidence of UTIs in human population.

1. Introduction

A urinary tract infection (UTI) is a situation where one or more sections of the urinary system (the kidneys, ureters, urethra, and bladder) is infected. UTIs are the most common of all bacterial infections and can happen at any time in the life of an individual. Approximately 95% of cases of UTIs are caused by bacteria that typically reproduce at the opening of the urethra and move up to the bladder. Often, bacteria spread to the kidney from the bloodstream [24, 22]. It had been known that people were suffering from UTIs before antibiotics were discovered in the early twentieth century; the truth is, nevertheless, there was no real treatment before antibiotics were invented. Doctors recommended a number of tinctures, ointments and special meals/diets to deal with the symptoms, in cases in which the infection spread to the kidneys and bladder and elsewhere, they were honestly helpless. As a last-minute effort, they operated to drain pus from the infected kidneys and hoped the patient would survive. Treatment did not basically change until antibiotics arrived on the scene [21]. The infection customarily begins at the opening of the urethra where the urine goes out of the human body and moves upward into the urinary tract.
UTIs are mostly caused by bacteria and to the small extent by viruses and fungi [8]. The

Key words and phrases. Water and environment, Prevalence, xponentially, UTI transmission, Asymptotically.

bacteria usually associated with UTI in hospitals is Uropathogenic Escherichia Coli (UPEC), signifying more than 80% of infections, the remaining pathogens include Klebsiella species, other coliforms, staphylococci, Enterococcus faecalis and Pseudonzonas aeruginosa [27]. Candida species like Candida albicans are chiefly cause of fungal UTIs specifically in immunosuppressed patients and in those with in-dwelling catheters [10, 18]. Early diagnosis with treatment of UTI is recommended because missed or postponed diagnosis of UTI may cause the failure of appropriate treatment and possibly lead to long-term consequences, including renal scarring, hypertension, and chronic renal failure [11].

UTI transmission dynamics is complicated by the multiple interactions between the human host, the pathogen and the environment, which contribute to both human-to-human and human to environment to human transmission pathways. A deep understanding of the disease transmission dynamics would provide important strategies to the effective prevention and control. Mathematical modeling, simulation and analysis offer a promising way to look into the nature of UTI dynamics. It is very important to know the life cycles of the pathogens causing UTI. It has been discovered that some pathogens live in human the body and others in aquatic environments. Pathogens like E. coli, klebsiella pneumonia, normally live in human bodies and pathogens like Pseudomonas aeruginosa, Acinetobacter baumannii, Legionella spp., Aeromonas spp., Mycobacterium avium are ubiquitous indigenous aquatic organisms that can both survive and multiply in water-bath, soil, moist floor, etc. [16]. Furthermore, some pathogens like virus species live in host cells, viruses cannot live by themselves and they need other living cells for reproduction. Viral diseases are quite different from bacterial and fungi diseases, they cannot be treated by antibiotics [25].
The infection to human comes when the healthy person engages with infected human or infested water and other environment.
Signs and symptoms frequently associated with UTIs are a repeated or intense urge to urinate and/or even though little comes out when you do; a burning feeling when you urinate; aching or pressure sores in your back or beneath the abdomen; dark, cloudy, bloody, or abnormal-smelling urine, feeling exhausted, illness [17]. Antibiotic treatment is generally effective for eradication of the infecting strain; however, literature reviews document of increasing antibiotic resistance, allergic reaction to certain pharmaceuticals, variation on normal gut flora, and failure to avoid recurrent infections represent significant barriers to curative measures.

2. Model description and formulation

The model system of this study is divided into five major epidemiological classes: Susceptible male S_m, Infected male I_m, Susceptible female S_f, Infected female I_f, Water and other environmental W_e. The total population at any time is denoted by $N(t)$; Susceptible male

and female individuals to both sexes are $S_m(t)$ and $S_f(t)$ respectively; male and female individuals infected with UTI at any time, are $I_m(t)$ and $I_f(t)$ respectively. This means that $N(t) = S_m(t) + S_f(t) + I_m(t) + I_f(t)$

Male infection rate from female and female infection rate from male are β_f and β_m respectively. Susceptible male and female also can get the disease from water and other environment (We) at any time at the rates β_w and β respectively. The UTI infection rates between male and female from environment is not the same. Females are more vulnerable than males because of their morphology (i.e. $\beta > \beta_w$). The constant per capita recruitment rates into the susceptible population is Λ and suppose α , ϕ are per capita natural death rate and, water and other environment dying-out rate respectively. P and $1 - P$are the proportion of male and female newborn individuals respectively, and ρ is the Male and female shedding constant rate to water and other environment.

2.1. Model assumptions

(i) Individuals who have frequent and long interactions with infectious individuals and infectious water and other environment experience a high risk of UTI infection.

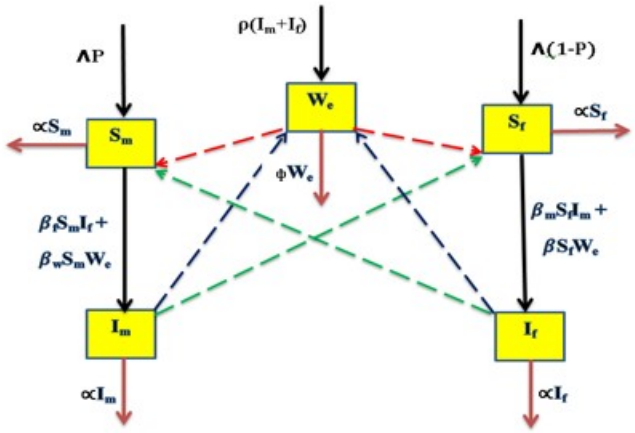
(ii) Individuals are mixing homogeneously, that is, all susceptible individuals are equally likely to be infected by infectious individuals or infectious water and environment in case of contact.

(iii) Infectious males and females contribute pathogens equally to water and other environment.

(iv) The human natural death rate is the same in all populations and UTI does not cause death.

TABLE 2.1. Model Variables and Parameters

Variables & Parameters	Description
N	Total population size.
S_m	Susceptible male individuals.
S_f	Susceptible female individuals.
I_m	Infectious male individuals.
I_f	Infectious female individuals.
W_e	Infectious W & E.
Λ	Per capita birth rate.
P	Proportion of male newborn individuals
$1-P$	Proportion of female newborn individuals.
β_f	Male infection rate from female.
β_m	Female infection rate from male.
β_w	Male infection rate from W & E.
β	Female infection rate fromW & E.
ρ	Male and female shedding rate to W & E.
α	Human natural death rate.
ϕ	W & E dying-out rate.



$$\frac{dS_m}{dt} = \Lambda P - \left(\beta_f S_m I_f + \beta_w S_m W_e\right) - \alpha S_m \quad (2.1)$$

$$\frac{dI_m}{dt} = \beta_f S_m I_f + \beta_w S_m W_e - \alpha I_m \quad (2.2)$$

$$\frac{dS_f}{dt} = \Lambda(1-P) - \left(\beta_m S_f I_m + \beta S_f W_e\right) - \alpha S_f \quad (2.3)$$

$$\frac{dI_f}{dt} = \beta_m S_f I_m + \beta S_f W_e - \alpha I_f \quad (2.4)$$

$$\frac{dW_e}{dt} = \rho\left(I_m + I_f\right) - \phi W_e \quad (2.5)$$

FIGURE 2.1. Compartmental and Mathematical models for transmission of UTI

The model systems (2.1)-(2.5) satisfies the following initial conditions,

$$S_m \geq 0,\ I_m \geq 0,\ S_f \geq 0,\ I_f \geq 0, \text{ and } W_e \geq 0.$$

The total number of human population is given by;

$$N = S_m + I_m + S_f + I_f.$$

3. Model System Analysis

For the disease free equilibrium points $(DFE)\, D^*$, we assume there are no pathogens, no infectious individuals and no infectious water and other environment. We resolve the model system (2.1)-(2.5) by setting the infectious compartments to zero, that is $I_m = I_f = W_e = 0$ and the results evaluated are as follows

$$\Lambda P - (\beta_f S_m I_f + \beta_w S_m W_e) - \alpha S_m = 0,$$

$$S_m^* = \frac{\Lambda P}{\alpha}. \tag{3.1}$$

$$\Lambda\,(1-P) - (\beta_m S_f I_m + \beta S_f W_e) - \alpha S_f = 0,$$

$$S_m^* = \frac{\Lambda\,(1-P)}{\alpha}. \tag{3.2}$$

Therefore the disease free equilibrium points are

$$D^* = \left(\frac{\Lambda P}{\alpha}, 0, \frac{\Lambda\,(1-P)}{\alpha}, 0, 0\right). \tag{3.3}$$

3.1. The basic reproduction number, R_o The basic reproduction number can be used to analyze the stability of equilibrium points [2]. The basic reproduction number R_o is the probable number of secondary cases formed by a typical infective individual presented into an entirely susceptible population, without any control measure. The disease-free equilibrium is said to be locally asymptotically stable if and only if $0 < R_o < 1$ and unstable if $R_o > 1$. A general method for calculating R_o is the next generation method [5]. Using the method defined by **(author?)** [30];**(author?)** [31]. Now
$\mathcal{F}_1(I_f, W_e) = (\beta_f S_m I_f + \beta_w S_m W_e)$, $\mathcal{F}_2(I_m, W_e) = (\beta_m S_f I_m + \beta S_f W_e)$
and $\mathcal{F}_3(I_f, I_f, W_e) = 0$;
$\mathcal{V}_1 = \alpha I_m$, $\mathcal{V}_2 = \alpha I_f$ and $\mathcal{V}_3 = -\rho(I_m + I_f) + \phi W_e$.
Therefore

$$F = \begin{bmatrix} 0 & \beta_f S_m & \beta_w S_m \\ \beta_m S_m & 0 & \beta S_f \\ 0 & 0 & 0 \end{bmatrix} \tag{3.4}$$

and

$$V = \begin{bmatrix} \alpha & 0 & 0 \\ 0 & \alpha & 0 \\ -\rho & -\rho & \phi \end{bmatrix}. \tag{3.5}$$

Hence,

$$FV^{-1} = \begin{bmatrix} \frac{\beta_w \Lambda P \rho}{\alpha^2 \phi} & \frac{\beta_f \Lambda P \rho}{\alpha^2} + \frac{beta_w \Lambda P \rho}{\alpha^2 \phi} & \frac{\beta_w \Lambda P}{\alpha \phi} \\ \frac{\beta_m \Lambda (1-P) \rho}{\alpha^2} + \frac{\beta \Lambda (1-P) \rho}{\alpha^2 \phi} & \frac{\beta \Lambda (1-P) \rho}{\alpha^2 \phi} & \frac{\beta \Lambda (1-P)}{\alpha \phi} \\ 0 & 0 & 0 \end{bmatrix}. \tag{3.6}$$

According to **(author?)** [4] the largest or dominant eigenvalue obtained is picked as the basic reproduction number R_o.

Then,

(3.7)

$$\boldsymbol{R_o} = \frac{\Lambda}{2\Phi\alpha^2}\Bigg(-\rho(P-1)\beta + P\rho\beta_w + \Bigg(\Big(\big((\beta-\beta_w)^2\rho^2 - 4\Phi\big(\beta\beta_f + \beta_m\beta_w\big)\rho - 4\Phi^2\beta_f\beta_m\big)P^2 + \big(-2\beta(\beta-\beta_w)\rho^2 + 4\Phi\big(\beta\beta_f + \beta_m\beta_w\big)\rho + 4\Phi^2\beta_f\beta_m\big)P + \beta^2\rho^2\Big)^{1/2}\Bigg)\Bigg).$$

3.2. Local stability of disease-free equilibrium point D^* The local stability of the disease free equilibrium point is examined using trace-determinant or eigenvalues criteria of the Jacobian matrix which is defined as a matrix of all first-order partial derivative of the model system. The equilibrium point is said to be locally asymptotically stable if the Jacobian matrix estimated at that point gives a negative trace and a positive determinant or has negative eigenvalues **(author?)** [13]. We aim to prove the following Theorem.

Theorem 3.1. *The disease free equilibrium point (D^*) whenever it occurs is locally asymptotically stable if $R_o < 1$ and unstable if $R_o > 1$.*

Proof

The Jacobian matrix given below is obtained at disease free equilibrium point, (D^*) for model system (2.1) - (2.5)

$$JD^*\big|_{\left(\frac{\Lambda P}{\alpha},0,\frac{\Lambda(1-P)}{\alpha},0,0\right)} = \begin{bmatrix} -\alpha & 0 & 0 & -\frac{\Lambda P\beta_f}{\alpha} & -\frac{\Lambda P\beta_w}{\alpha} \\ 0 & -\alpha & 0 & \frac{\Lambda P\beta_f}{\alpha} & \frac{\Lambda P\beta_w}{\alpha} \\ 0 & -\frac{\Lambda(1-P)\beta_m}{\alpha} & -\alpha & 0 & -\frac{\Lambda(1-P)\beta}{\alpha} \\ 0 & \frac{\Lambda(1-P)\beta_m}{\alpha} & 0 & -\alpha & \frac{\Lambda(1-P)\beta}{\alpha} \\ 0 & \rho & 0 & \rho & -\phi \end{bmatrix}. \tag{3.8}$$

From above Jacobian matrix we find that elements $a_{11} = \lambda_1 = -\alpha$ and $a_{33} = \lambda_2 = -\alpha$ are among five eigenvalues of the matrix equation (3.8) and they all have purely negative real parts. Therefore, the matrix equation (3.8) is reduced to sub-matrix equation (3.9) by

eliminating the rows and columns the eigenvalues λ_1 and λ_2 belong.

$$J(D^*)_1 = \begin{bmatrix} -\alpha & \frac{\Lambda P\beta_f}{\alpha} & \frac{\Lambda P\beta_w}{\alpha} \\ \frac{\Lambda(1-P)\beta_m}{\alpha} & -\alpha & \frac{\Lambda(1-P)\beta}{\alpha} \\ \rho & \rho & -\phi \end{bmatrix}. \tag{3.9}$$

With the aid of Maple software, we evaluated and obtained the characteristic polynomial for matrix equation (3.9) as $\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0$
where

$$a_1 = \frac{2\alpha^3 + \alpha^2\phi}{\alpha^2},$$

$$a_2 = \frac{\Lambda^2 P^2 \beta_f \beta_m - \Lambda^2 P \beta_f \beta_m + \Lambda P \alpha \beta \rho - \Lambda P \alpha \rho \beta_w - \Lambda \alpha \beta \rho + \alpha^4 + 2\alpha^3 \phi}{\alpha^2},$$

$$a_3 = \frac{1}{\alpha^2}\left(\Lambda^2 P^2 \beta \rho \beta_f + \Lambda^2 P^2 \phi \beta_f \beta_m + \Lambda^2 P^2 \rho \beta_m \beta_w - \Lambda^2 P \beta \rho \beta_f - \Lambda^2 P \phi \beta_f \beta_m \right.$$
$$\left. - \Lambda^2 P \rho \beta_m \beta_w + \Lambda P \alpha^2 \beta \rho - \Lambda P \alpha^2 \rho \beta_w - \Lambda \alpha^2 \beta \rho + \alpha^2 \phi\right).$$

According to Routh-Hurwitz stability criterion the eigenvalues $\lambda_1, \lambda_2, \lambda_3, \lambda_4$ and λ_5 have negative real parts if and only if all coefficients satisfy the following criteria:

$$H_1 = a_1 > 0,$$
$$H_2 = a_1 a_2 - a_3 > 0,$$
$$H_3 = a_1 a_2 a_3 + a_1 a_5 - a_1^2 a_4 - a_3^2 > 0,$$

Hence, all roots (eigenvalues) of the characteristic polynomial of our Jacobian matrix have negative real parts [9]. From this we can conclude that the disease free equilibrium point is locally asymptotically stable under these extreme conditions.

3.3. Global stability of disease-free equilibrium point D^* In our model system (2.1) - (2.5), it can be recognized that the disease-free equilibrium point is locally asymptotic stable whenever $R_o < 1$ and unstable when $R_o > 1$. In this section, we have adopted two conditions that if agreed, also give the assurance for the global asymptotic stability of the disease-free state [3]. First, the system of differential equations (2.1) - (2.5) must be written in the form:

$$\frac{dX}{dt} = F(X, I), \tag{3.10}$$

$$\frac{dI}{dt} = G(X, I);\ G(X, 0) = 0, \tag{3.11}$$

where $X \in R^r, I \in R^n, r, n, \geq 0$, and $G(X, 0) = 0$. The components of X indicate the number of susceptibles, and other classes of non-infected population. The components of I

represent the number of infected population liable to transmitting the UTI. $D^* = (X^*, 0)$, denotes the disease-free equilibrium point of this system. The fixed point $D^* = (X^*, 0)$ is a globally asymptotically stable equilibrium point for this system provided that $R_o < 1$ (i.e. locally asymptotically stable) and the following two conditions are satisfied:

(H1) $\frac{d\mathrm{X}}{dt} = F(\mathrm{X}, I)$, X^* is globally asymptotically stable,

(H2) $\frac{d\mathrm{I}}{dt} = G(\mathrm{X}, I) = QI - \hat{G}(\mathrm{X}, I)$, $\hat{G}(\mathrm{X}, I) \geq 0$ for $(\mathrm{X}, I) \in \Omega$,

where the Jacobian $Q = \left(\frac{\partial G}{\partial I}\right)(X^*, 0)$ is an M-matrix (the off diagonal elements of Q are non negative) and Ω is the invariant region. For this case the disease free equilibrium point is now signified as $D^* = (X^*, 0), X^* = \left(\frac{\Lambda P}{\alpha}, 0, \frac{\Lambda(1-P)}{\alpha}, 0, 0\right)$. If the model system (2.1) - (2.5) fulfills the conditions (H1) and (H2), then conferring to **(author?)** [3] the following theorem holds.

Theorem 3.2. *The equilibrium point $D^* = (X^*, 0)$ of the model system (2.1) - (2.5) is globally asymptotically stable if $R_o \leq 1$ and the conditions (H1) and (H2) are met.*

Proof

We initiate our proof by outlining $X = (S_m, S_f)$ and $I = (I_m, I_f, W_e)$. We look on the two vector valued functions $F(X, I)$ and $G(X, I)$. We adopt the method done by **(author?)** [14]. By considering the equation $\frac{dX}{dt} = F(X, 0)$, from condition (H1), we obtain

$$\begin{cases} \frac{dS_m}{dt} = \Lambda P - \alpha S_m, \\ \frac{dS_f}{dt} = \Lambda (1 - P) - \alpha S_f. \end{cases} \tag{3.12}$$

$X^* = \left(\frac{\Lambda P}{\alpha}, \frac{(\Lambda(1-P))}{\alpha}\right)$ is globally asymptotically stable equilibrium point for the reduced system model equations $\frac{dX}{dt} = F(X, 0)$.

We then compute $G(X, I) = \left(\frac{\partial G}{\partial I}\right)(X^*, 0)\, I - \hat{G}(X, I)$ and show that $\hat{G}(X, I) \geq 0$;

$$\left(\frac{\partial G}{\partial I}\right)(X^*, 0) = \begin{pmatrix} -\alpha & \frac{\Lambda P \beta_f}{\alpha} & \frac{\Lambda P \beta_w}{\alpha} \\ \frac{\Lambda(1-P)\beta_m}{\alpha} & -\alpha & \frac{\Lambda(1-P)\beta}{\alpha} \\ \rho & \rho & -\phi \end{pmatrix}, \tag{3.13}$$

this is an M-matrix with non-negatives off diagonal elements. Then,

$$\left(\frac{\partial G}{\partial I}\right)(X^*, 0)\, I = \begin{pmatrix} -\alpha I_m + \beta_f \beta_f \frac{\Lambda P \beta_f}{\alpha} + \beta_w W_e \frac{\Lambda P \beta_w}{\alpha} \\ \beta_m I_m \frac{\Lambda(1-P)\beta_m}{\alpha} + -\alpha I_f + \beta W_e \frac{\Lambda(1-P)\beta}{\alpha} \\ \rho I_m + \rho I_f + -\phi W_e \end{pmatrix}. \tag{3.14}$$

Hence,
$\hat{G}(X,I) = \left(\frac{\partial G}{\partial I}\right)(X^*,0)\,I - G(X,I),$

$$\hat{G}(X,I) = \begin{pmatrix} 0 \\ 0 \\ 0 \end{pmatrix}. \tag{3.15}$$

Therefore, $\hat{G}(X,I) \geq 0$. In this case the Theorem 3.2 has been proved and we can conclude that the disease free equilibrium point is globally asymptotically stable under these extreme circumstances.

3.4. The existence of endemic equilibrium point E^* The state where the disease cannot be completely eliminated but remains in the population is called the endemic equilibrium state.Furthermore, the endemic equilibrium point is said to be locally stable when $R_o > 1$ and unstable when $0 < R_o < 1$ [28]. For the disease to continue in the population, the susceptible classes and the Infectious classes must not be zero at equilibrium state [14]. In other words, the endemic equilibrium state is given as

$$E^* = \left(S_m^*, I_m^*, S_f^*, I_f^*, W_e^*\right) \neq (0,0,0,0,0),$$

with the fact that $S_m^* > 0$, $I_m^* > 0$, $S_f^* > 0$, $I_f^* > 0$ and $W_e > 0$.
Initially, we evaluated the variables W_e^* and S_m^* from model system (2.1) - (2.5) respectively as follows

$$W_e^* = \frac{\rho\left(I_m^* + I_f^*\right)}{\phi}, \tag{3.16}$$

$$I_m^* = \frac{\alpha I_f^*\left(\alpha\phi + \beta\rho I_f^*\right) - \Lambda(1-P)\left(\beta\rho I_f^*\right)}{\Lambda(1-P)\left(\beta_m\phi + \beta\rho\right) - \alpha I_f^*\left(\beta_m\phi + \beta\rho\right)}, \tag{3.17}$$

$$I_f^* = \frac{\alpha I_m^*\left(\alpha\phi + \beta_w\rho I_m^*\right) - \Lambda P\left(\beta_w\rho I_m^*\right)}{\Lambda P\left(\beta_f\phi + \beta_w\rho\right) - \alpha I_m^*\left(\beta_f\phi + \beta_w\rho\right)}. \tag{3.18}$$

For the positivity, existence and uniqueness of endemic equilibrium point, the following conditions in above equations must hold,
$\alpha I_f^*\left(\alpha\phi + \beta\rho I_f^*\right) > \Lambda(1-P)\left(\beta\rho I_f^*\right)$, $\Lambda(1-P)\left(\beta_m\phi + \beta\rho\right) > \alpha I_f^*\left(\beta_m\phi + \beta\rho\right)$,
$\alpha I_m^*\left(\alpha\phi + \beta_w\rho I_m^*\right) > \Lambda P\left(\beta_w\rho I_m^*\right)$, and $\Lambda P\left(\beta_f\phi + \beta_w\rho\right) > \alpha I_m^*\left(\beta_f\phi + \beta_w\rho\right)$.
Furthermore, we can produce polynomials for I_m^* and I_f^* by substitution of I_f^* into I_m^* and vice versa respectively [33]. We obtain the following polynomial equations

(3.19)
$$A_1 I_m^4 + A_2 I_m^3 + A_3 I_m^2 + A_4 I_m = 0 \text{ and } B_1 I_f^4 + B_2 I_f^3 + B_3 I_f^2 + B_4 I_f = 0,$$

I_m^* and I_f^* are solutions of the above equations with coefficients of $A_1, A_2, A_3, A_4, B_1, B_2, B_3$, and B_4.

(3.20)

$$(A_1 I_m^3 + A_2 I_m^2 + A_3 I_m + A_4) I_m = 0 \text{ and } \left(B_1 I_f^3 + B_2 I_f^2 + B_3 I_f + B_4\right) I_f = 0.$$

It follows that, I_m^* , $I_f^* = 0$, and

$$A_1 I_m^{*3} + A_2 I_m^{*2} + A_3 I_m^* + A_4, B_1 I_f^{*3} + B_2 I_f^{*2} + B_3 I_f^* + B_4 = 0. \tag{3.21}$$

For the endemic equilibrium point to exist, the solutions of (3.21) must be real and positive. Also, since I_m^* , $I_f^* = 0$, the viral free steady state is said to be in neutral state (i.e. exists an infection or no infection) [23]. From these conditions, we conclude that the endemic equilibrium solution is stable when $R_o > 1$, and it exhibits persistence of UTI transmission in the population.

4. Numerical Results, Simulation and Discussion

Basic model simulations are important aspects in Mathematical modeling. We understand the behaviour of UTI dynamics when an endemic situation persists and demonstrate how susceptible sub-populations interact with infected sub-populations. We identified the effects of the most positive and negative sensitive parameters with respect to basic reproduction number (R_o). The simulation of the model system of the equations has been plot to examine the dynamic forces of UTI in the entire population when there is no intervention.

4.1. Parameter estimation The total population of consideration is an area sparingly populated with a density of 51 persons per square kilometre with deviation across the regions in Tanzania [19]. We estimate the proportion of male and female newborn individuals, taking into consideration that the life expectancy at birth of the male and female newborn individuals in Tanzania [15] are 63.5% (out of all male newborns) and 66.4% (out of all female newborns) respectively which are equivalent to $P = 0.4888$ and $1 - P = 0.5112$. **(author?)** [6] revealed that the cumulative incidence rate of UTI was three times greater in girls (females) by 6.6% than boys (males) by 1.8%. In this study we estimated the male infection rate from female, β_f as 0.0000018 and the female infection rate from male, β_m is 0.0000053. UN-Water and the Interagency Network on Women and Gender Equality as a programme under UN-Water policy which is tasked with responsibilities of access to water, sanitation and health. It has been verified that women have a primary concern for management of domestic water supply, sanitation and health [32]. We estimated that women (females) have access to water and other environment twice as much as men (males). In this case we estimate the female infection rate, β from water and other environment to be 0.0000044 and for male infection rate, β_w from water and other environment to be 0.0000021.

TABLE 4.1. Model Variables and Parameters and their description

Item	Value	Unit	Source
N	51,000		[19]
S_m	22,000		[7]
S_f	16,000		[7]
I_m	3,000		[7]
I_f	10,000		[7]
W_e	15,000		Estimated
Λ	0.00027	day^{-1}	Assumed
P	0.4888		Estimated
$1-P$	0.5112		Estimated
β_f	$1.8\text{x}10^{-6}$	day^{-1}	Estimated
β_m	$5.3\text{x}10^{-6}$	day^{-1}	Estimated
β_w	$2.1\text{x}10^{-6}$	$\text{ml}^3\text{Cells}^{-1}\text{day}$	Estimated
β	$4.4\text{x}10^{-6}$	$\text{ml}^3\text{Cells}^{-1}\text{day}$	Estimated
ρ	0.1072	$\text{Cellsml}^{-3}\text{day}^{-1}\text{km}^2\text{person}^{-1}$	[29]
α	$1.25\text{x}10^{-4}$	day^{-1}	Assumed
ϕ	0.0333	day^{-1}	[29]

4.2. Sensitivity analysis of R_o Sensitivity analysis expresses how significant each parameter is in disease transmission. Sensitivity analysis plays a central role in epidemiological modeling. With the help of Maple 18 software we noticed that reproduction number for disease free equilibrium (DFE), R_o is 0.208 while for endemic equilibrium (EE), R_o is 3463. In this paper we adopt the method of determining the sensitivity analysis as used by [26, 20]. The basic reproduction number R_o of UTI depends on nine parameters, we deduce an analytical expression for its sensitivity to every parameter using the normalized forward sensitivity indices of R_o with respect to parameters p_j involved in R_o as shown below:

$$\Upsilon_{p_j}^{R_o} = \frac{\partial R_0}{\partial p_j} \times \frac{p_j}{R_0}. \tag{4.1}$$

For instance, the sensitivity indices of R_o with respect to Λ is calculated as

$$\Upsilon_{\Lambda}^{R_o} = \frac{\partial R_0}{\partial \Lambda} \times \frac{p_j}{R_0} = 1. \tag{4.2}$$

Other differentials produces long expressions, to determine the sensitivity index of the respective parameter we substitute the parameter values specified in Table 4.1. Following the same technique we can obtain the sensitivity indices for $\Upsilon_{\beta_m}^{R_o}$, $\Upsilon_{\beta_f}^{R_o}$, $\Upsilon_{\beta_w}^{R_o}$, $\Upsilon_{\beta}^{R_o}$, $\Upsilon_{\alpha}^{R_o}$, $\Upsilon_{\phi}^{R_o}$, $\Upsilon_{\rho}^{R_o}$ and $\Upsilon_{P}^{R_o}$ as tabulated in the Table 4.2. The dynamics of these sensitivity analysis done corresponding to $R_0 = 3463$ for EE. From Table 4.2 we observe that the most sensitive

TABLE 4.2. Model Variables and Parameters

Parameter	Sensitivity index	Parameter	Sensitivity index
Λ	+ 1.0000	β	+0.5760
P	-0.2635	ρ	+0.8761
β_f	+0.0540le	α	-0.0636
β_w	+0.3001	ϕ	-0.8761
β_m	+0.0669		

positive parameter is the Per capita birth rate (Λ). The following parameters in their positive descending order of sensitivity are: male and female shedding rate to water and other environment (ρ), male infection rate from water and other environment (β_w), female infection rate from water and other environment (β), female infection rate from male (β_m), male infection rate from female (β_f) and human mortality/death rate (α). The positive sign of the stated parameters indicates that decreasing (increasing) one of these parameters while keeping other parameters constant drops (rises) the value of the basic reproduction number. Taking an example for the sensitivity indices of R_o with respect to β is 0.5760, this indicates that increasing female infection rate from water and other environment by 50%, increases the value of basic reproduction number by 57% and hence increases the existence of the UTI and vice versa. Conversely, water and other environment dying-out rate (ϕ) is the most negative parameter. Other parameters with negative sensitivity indices are human mortality/death rate (α) and proportion of male newborn individuals (P). This implies that increasing (decreasing) this parameter while keeping the other parameters constant decreases (increases) the value of basic reproduction number R_o and hence decreases (increases) the persistence of UTI.

4.3. Numerical Simulations of the basic model The simulation of the model system (2.1) - (2.5) is plotted to examine the dynamic forces of UTI in the entire population when there is no disease intervention. We used MATLAB software to perform the simulation. The Runge-Kutta 4th order method was employed. The initial values of susceptible males, infected males, susceptible females and infected females are 22, 3, 16 and 10 respectively. The number of males are infected by 12% and females by 40% [7]. The total population of water and other environment was estimated to be 15. The total population in square kilometres as shown in table 4.3. The research case study is Sombetini ward located in Arusha city with a total population of 48,268 per 2012 census [19]. We approximated this population to be 51,000 in the year 2017. The total population of Sombetini as used in simulation as shown in table 4.3.

TABLE 4.3. Population under consideration

Item	Population in square kilometres	Population of Sombetini
Susceptible male	22	22,000
Infected male	3	3,000
Susceptible female	16	16,000
Infected female	10	10,000
Water and other environment	15	15,000

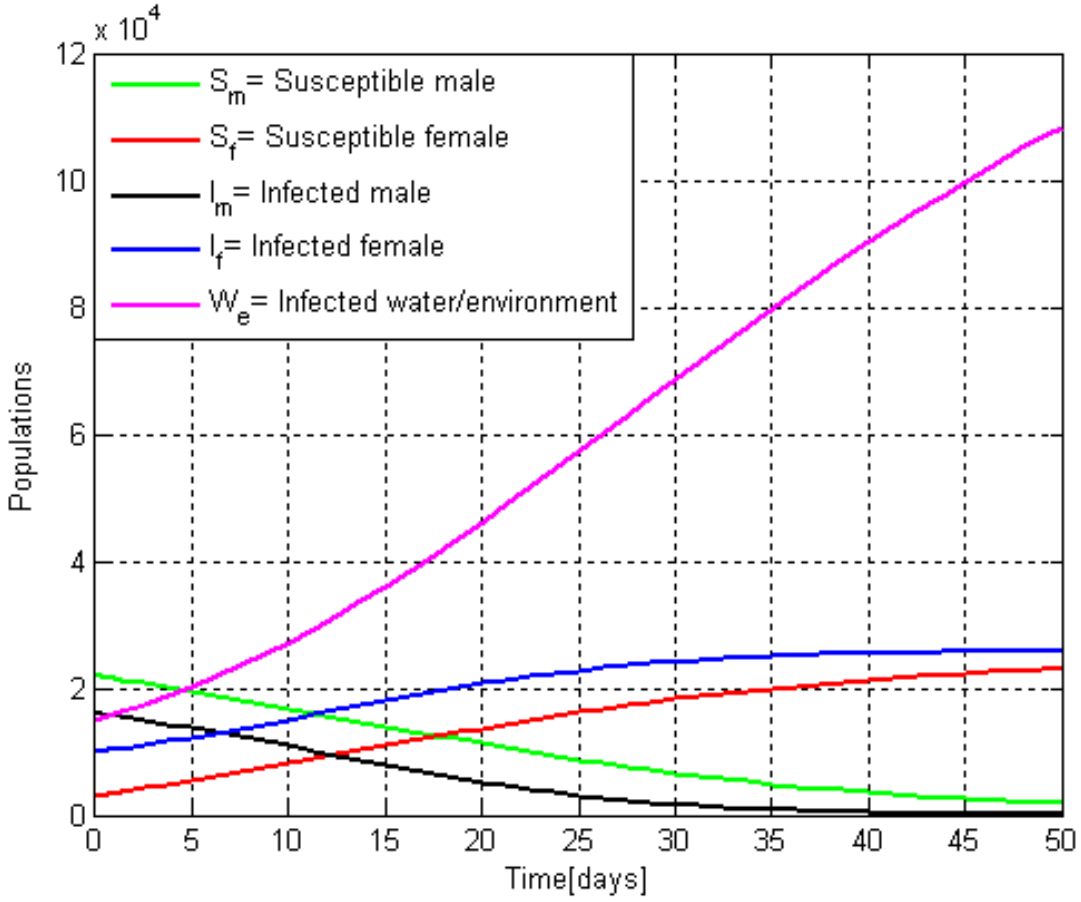


FIGURE 4.1. Characteristics of five model system variables

Figures 4.1 shows the dynamics in humans and, water and other environment populations, displaying the characteristics of five state variables S_m, I_m, S_f, I_f and W_e with given time when there is no control. The susceptible male and susceptible female sub-populations fall exponentially to achieve endemic equilibrium point. The sub-populations of susceptible male and female decrease exponentially as they die naturally and as they get infections from severely infected humans or from the UTI pathogens living in water and other environment

and finally reach the endemic equilibrium point. The sub-populations of infected male and infected female caused by UTI infections rise exponentially to a certain point in time then decline to reach endemic equilibrium point as they undergo a natural death. The decrease in susceptible male and female sub-populations lead into the growth of the insignificantly infected male and female that raise exponentially after sometime they start diminishing because of natural death and as they gain more infection from infected male and female and infected water and other environment to join the severely infected male and female class then finally reach endemic equilibrium point. The severely infected male and female advance exponentially to a certain point in time then start deteriorating due to natural death and lastly achieves endemic equilibrium point. The population of pathogens in water and other environment increases exponentially with time to a certain point and then declines to reach endemic equilibrium point as they die naturally. They die-out when the water and other environment is dried-up and as they burrow into the earth. The lines of susceptible male and female sub-populations decrease and extend approaching to zero asymptotically meaning that some susceptible male and female sub-populations will not all get infection or die naturally.

Another important aspect in epidemiology is the area which concerned with the existence of disease in populations. Public health professionals and epidemiologists use each measure of disease frequency for particular purposes. Prevalence and Incidence are greatest useful for valuing the effectiveness of plans that try to stop disease from occurring in the entire population [1]. Researchers who investigate the sources of disease prefer to examine new cases (incidence) over prevailing ones (prevalence) since they are usually concerned in exposures that advance to emerging the disease. Prevalence it combines incidence and survival to obscures underlying relationships.

In figures 4.2 and 4.3 we present the numerical outcomes with respect to the negatively sensitive parameter to R_o, which is ϕ (dying-out rate of pathogens in water and other environment) that affect disease prevalence and incidence (negatively) based on sensitivity analysis and numerical outcomes of the general UTI model. Figure 4.2 shows that the disease prevalence to human sub-populations (Male and Female) increases as the pathogens in

water and other environment dying-out rate decreases and vice versa.

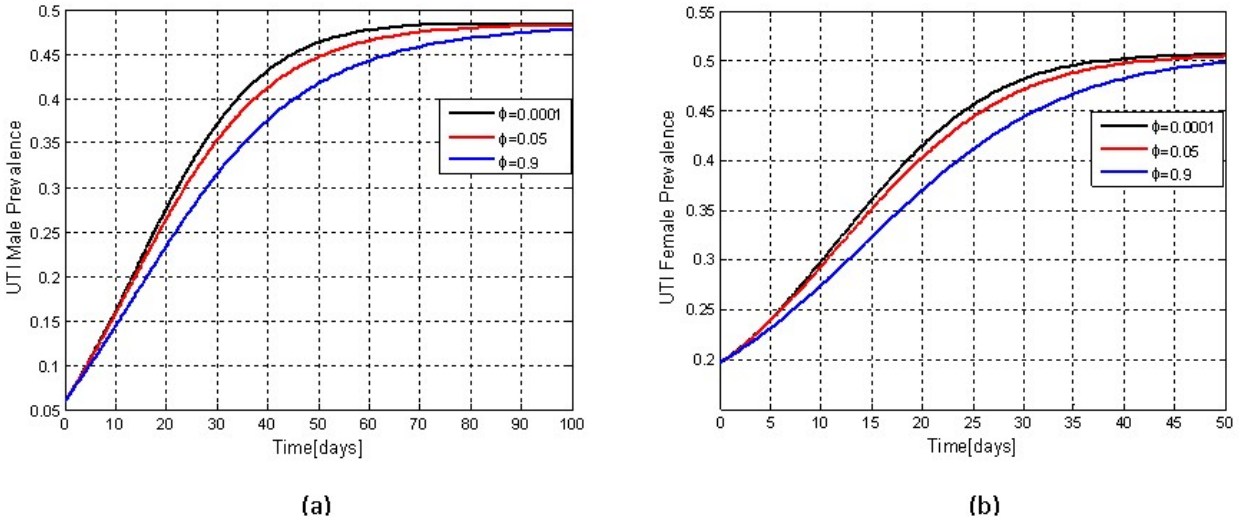


FIGURE 4.2. Disease prevalence with respect to variation of water and other environment dying-out rate.

In figure 4.3, we noticed that disease incidence to the human sub-populations on the increase or decrease of water and other environment dying-out rate, ϕ. From the figure we deduced that when the pathogens dying-out rate is very big or small, the incidence increases sharply for male and female sub-populations within the first 15 and 12 days respectively, then drops exponentially approaching zero asymptotically. The incidence drops sharply for male and female sub-populations within the first 3 and 1 days respectively.

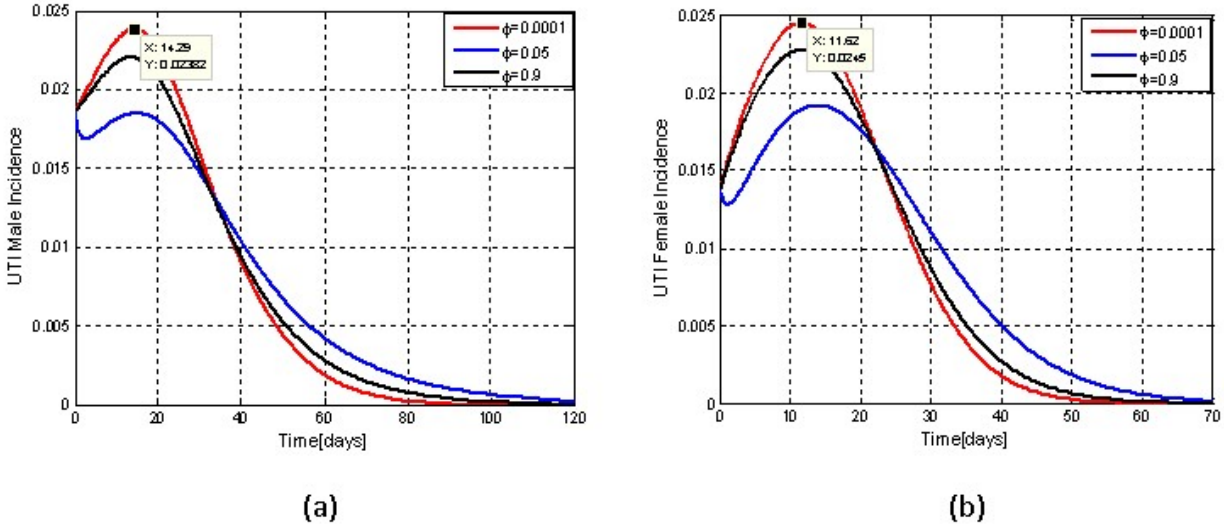


FIGURE 4.3. Disease incidence with respect to variation of water and other environment dying-out rate.

The incidence then starts to increase exponentially within 12 and 14 days respectively before starting to decrease exponentially. Figures 4.2 and 4.3 depict positive impact of drying out

and/or treating water and other environment on UTI prevalence and incidence as one way of eradicating the infections in the human population.

5. Conclusion

In this paper one major issue that surrounds UTIs modeling is conferred. We found that the infectious water and other environment is the reservoir and habitats for bacteria and other UTI pathogens and is a major source of UTIs transmission route. Increased awareness of water and other environment as a possible major source of UTIs transmission route is needed [12]. We are not intending to marginalize other transmission paths, awareness should be taken even to them.

We recommend the following for reduction and prevention on the prevalence and incidence of UTIs in human population: Applying a rational approach to disinfection and sterilization of items in recognition to water and other environment like floors, toilets and bathhouses. Others are drying out unnecessary standing water, treating water bodies (reservoir) like swimming pools and sewages regularly with recommended antibiotics. Domestic animals are among carriers of UTI pathogens, precautions on health care should be taken in consideration when providing services. Also avoid staying with wet clothes for long time especially to children and practicing sex to a man/woman who you dont know their health status. Education campaign and implementing prevailing infection control strategies to the entire population is necessary. Attend hospital treatments when you notice the UTI symptoms, this can help to reduce the intensity of infections.

References

[1] A. Aschengrau, G. R. Seage, et al., *Essentials of epidemiology in public health*, Jones & Bartlett Publishers, 2013.

[2] Castillo-Chavez, *Mathematical approaches for emerging and reemerging infectious diseases: models, methods, and theory*, Springer, 2002.

[3] C. Castillo-Chavez, Z. Feng, and W. Huang, *On the computation of r0 and its role on global stability, 2002*, Math. la. asu. edu/chavez/2002/JB276. pdf, (2002).

[4] O. Diekmann, *The construction of next-generation matrices for compartmental epidemic models*, Journal of the Royal Society Interface, (2009), p. rsif20090386.

[5] O. Diekmann, J. A. P. Heesterbeek, and J. A. Metz, *On the definition and the computation of the basic reproduction ratio r0 in models for infectious diseases in heterogeneous populations*, Journal of mathematical biology, 28 (1990), pp. 365–382.

[6] B. Foxman, *Epidemiology of urinary tract infections: incidence, morbidity, and economic costs*, The American journal of medicine, 113 (2002), pp. 5–13.

[7] ——, *Urinary tract infection syndromes: occurrence, recurrence, bacteriology, risk factors, and disease burden*, Infectious disease clinics of North America, 28 (2014), pp. 1–13.

[8] B. Foxman and P. Brown, *Epidemiology of urinary tract infections: transmission and risk factors, incidence, and costs*, Infectious disease clinics of North America, 17 (2003), pp. 227–241.

[9] A. A. Gebremeskel and H. E. Krogstad, *Mathematical modelling of endemic malaria transmission*, American Journal of Applied Mathematics, 3 (2015), pp. 36–46.

[10] M. Grabe, R. Bartoletti, T. Bjerklund Johansen, T. Cai, M. Çek, B. Köves, K. Naber, R. Pickard, P. Tenke, F. Wagenlehner, et al., *Guidelines on urological infections. european association of urology (eau)*, 2015.

[11] S. Hansson and U. Jodal, *Dimercapto-succinic acid scintigraphy instead of voiding cystourethrography for infants with urinary tract infection*, The Journal of urology, 172 (2004), pp. 1071–1074.

[12] D. Johnson, L. Lineweaver, and L. M. Maze, *Patients bath basins as potential sources of infection: a multicenter sampling study*, American Journal of Critical Care, 18 (2009), pp. 31–40.

[13] J. Kahuru, L. Luboobi, and Y. Nkansah-Gyekye, *Stability analysis of the dynamics of tungiasis transmission in endemic areas*, Asian Journal of Mathematics and Applications, 2017 (2017).

[14] T. Kinene, L. S. Luboobi, B. Nannyonga, and G. G. Mwanga, *A mathematical model for the dynamics and cost effectiveness of the current controls of cassava brown streak disease in uganda*, Journal of Mathematical and Computational Science, 5 (2015), p. 567.

[15] H. Masanja, D. de Savigny, P. Smithson, J. Schellenberg, T. John, C. Mbuya, G. Upunda, T. Boerma, C. Victora, T. Smith, et al., *Child survival gains in tanzania: analysis of data from demographic and health surveys*, The Lancet, 371 (2008), pp. 1276–1283.

[16] M. Middelboe, *Bacterial growth rate and marine virus–host dynamics*, Microbial Ecology, 40 (2000), pp. 114–124.

[17] S. J. Midthun, *Criteria for urinary tract infection in the elderly: variables that challenge nursing assessment*, Urologic Nursing, 24 (2004), p. 157.

[18] D. Minardi, G. dAnzeo, D. Cantoro, A. Conti, and G. Muzzonigro, *Urinary tract infections in women: etiology and treatment options*, Int J Gen Med, 4 (2011), pp. 333–343.

[19] T. NBS, *Population and housing census: population distribution by administrative areas*, Ministry of Finance, Dar es Salaam, (2012).

[20] R. C. Ngeleja, L. S. Luboobi, and Y. Nkansah-Gyekye, *Modelling the dynamics of bubonic plague with yersinia pestis in the environment*, Communications in Mathematical Biology and Neuroscience, 2016 (2016), pp. Article–ID.

[21] J. C. Nickel, *Management of urinary tract infections: historical perspective and current strategies: part 2modern management*, The Journal of urology, 173 (2005), pp. 27–32.

[22] L. E. Nicolle, *Catheter-related urinary tract infection*, Drugs & aging, 22 (2005), pp. 627–639.

[23] M. J. Ongala Jacob Otieno and O. Paul, *Mathematical model for pneumonia dynamics with carriers*, International Journal of Mathematical Analysis, 7 (2013), pp. 2457–2473.

[24] P. C. Pappas, *Laboratory in the diagnosis and management of urinary tract infections*, Medical Clinics of North America, 75 (1991), pp. 313–325.

[25] S. A. Rahman, *Study of Infectious Diseases by Mathematical Models: Predictions and Controls*, PhD thesis, The University of Western Ontario, 2016.

[26] H. S. Rodrigues, M. T. T. Monteiro, and D. F. Torres, *Sensitivity analysis in a dengue epidemiological model*, in Conference Papers in Science, vol. 2013, Hindawi Publishing Corporation, 2013.

[27] S. M. Soto, *Importance of biofilms in urinary tract infections: new therapeutic approaches*, Advances in Biology, 2014 (2014).

[28] A. Ssematimba, J. Mugisha, and L. S. Luboobi, *Mathematical models for the dynamics of tuberculosis in density-dependent populations: The case of internally displaced peoples camps (idpcs) in uganda*, (2005).

[29] J. H. Tien and D. J. Earn, *Multiple transmission pathways and disease dynamics in a waterborne pathogen model*, Bulletin of mathematical biology, 72 (2010), pp. 1506–1533.

[30] P. Van Den Driessche, *Some epidemiological models with delays*, tech. report, 1994.

[31] P. Van den Driessche and J. Watmough, *Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission*, Mathematical biosciences, 180 (2002), pp. 29–48.

[32] U. Water, *Gender, water and sanitation: A policy brief*, UN, New York, (2006).

[33] J. Zhang, J. Jia, and X. Song, *Analysis of an seir epidemic model with saturated incidence and saturated treatment function*, The Scientific World Journal, 2014 (2014).

[1]Department of Applied Mathematics and Computational Science, Nelson Mandela African Institution of Science and Technology, P.O. Box 447 Arusha, Tanzania

[2]Department of Mathematics, Makerere University, P.O. Box 7062 Kampala, Uganda

[3]Department of Mathematics, Kabale University, P.O. Box 317 Kabale, Uganda

*Correspondence: idanx@nm-aist.ac.tz