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Effects of Poor Sanitation and Public Awareness in Modeling Bacterial Infection amongst the Students of a Tertiary Institution in Kaura Namoda, Zamfara State, Nigeria

***LASISI, NO; SULEIMAN, F**

Department of Statistics, Federal Polytechnic, Kaura Namoda, Zamfara State, Nigeria

**Corresponding Author Email: nurudeenlasisi2009@yahoo.com*

**ORCID: https://orcid.org/0000-0002-5022-2790*

**Tel: 08131810027*

Co-Author Email: fssalai@gmail.com

ABSTRACT: Acute bacterial infection of the intestine is caused by ingestion of food or water containing vibrio cholera. The symptoms include acute water diarrhea and vomiting which can result in severe dehydration or water loss. Sanitary conditions in the environment play an important role. Hence, the objective of this paper as to evaluate the effects of poor sanitation and public awareness in modeling bacterial infection amongst the students of a tertiary institution in Kaura Namoda, Zamfara State, Nigeria. We incorporated effectiveness of drug and awareness for proper hygiene and sanitation into our model. The disease free and endemic equilibrium were determined. The effective reproduction number R_e was showed. Numerical results of the dynamics system of the transmission of bacterial infection were presented and we found that as the effective contact rate increases, the effective reproduction number increases. Also as the effectiveness of compliance of good hygiene increases, the effective reproduction number decreases by varying the contact rate. More so, as production rate of acute diarrhea bacteria increases, it increases the secondary cases of the infected individuals.

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Bacterial infection is one of the most common reported illnesses in developing Country, according to World Health Organization (WHO), an acute bacterial infection of the intestine caused by ingestion of food

**Corresponding Author Email: nurudeenlasisi2009@yahoo.com*
**ORCID: https://orcid.org/0000-0002-5022-2790*
**Tel: 08131810027*

or water containing vibrio cholera (Blanca and Christina, 2002). The symptoms include acute water diarrhoea and vomiting which can result in severe dehydration or water loss. More so, sanitary conditions in the environment play an important role (Codecco, 2001). Bacteria are living organisms that have only one cell and under a microscope, they look like balls, rods, or spirals. They are so small that 1,000 lines can fit on a pencil eraser (Codecco, 2001). Most types do not make person sick and many types are very useful (Blanca and Christina, 2002). Some of them help digest food, destroy disease-causing cells and provide the body with the necessary vitamins. Bacteria are also used to make healthy foods like yogurt and cheese (Tien and Earn, 2010).

But infectious bacteria can make someone sick and reproduced rapidly in body (Blanca and Christina, 2002). Many of the chemicals released are called toxins, which can damage tissue and make person sick (Pascual, Bouma and Dobson, 2002). The most deadly bacterial infections are Tuberculosis, Cholera, Botulism, MRSA Infection, Meningitis, Gonorrhea, Bubonic Plague, Syphilis (Bertoletti, Maini, and Williams, 2003) and Antibiotics are the usual treatment (Mabel, Juliet and James, 2022). Nonliving reservoirs Air can become contaminated by dust or human respiratory secretions containing pathogenic bacteria.

Bacteria do not multiply in the air itself, but may be transported by air currents to areas more conducive to their growth. Infections acquired through the air are characterized as airborne. The classic airborne bacterial infection is tuberculosis (Codecco, 2001).

Mathematical models have played an important role to the dynamics of both transmission and infectious of individuals (Lasisi, Akinwande and Olayiwola *et al*., 2018). Among the common are Ebola virus (Lasisi, Akinwande and Olayiwola *et al*., 2018). Hepatitis B virus (Bertoletti, Maini and Williams, 2003). The HumanImmunodeficiency virus (HIV) (Abdulrahman, Akinwande, Awojoyogbe and Abubakar, 2013; Akinwande, 2006). These models have been useful to study the control of the both transmission and virus kinetics in order to provide a quantitative understanding and create public awareness of the infection, while Codecco (2001); Pascual, Bouma and Dobson (2002); Jensen, Faruque, Mekalanos and Levin (2006); Tien and Earn (2010); Misra and Singh (2012); Lasisi, Akinwande and Oguntolu, (2020) have designed mathematical models to explored the transmission dynamics and control of the infection.

Model Formulation: The model equations are formulated using ordinary differential equations with nonlinear incidence rate called force of infection. We incorporated vaccination class, effectiveness of drug and awareness for proper hygiene and sanitation into our model. The population is divided into five classes: susceptible class (S): this class includes the individuals at risk for acute diarrheal infection after infected it then move to Infected class with thick arrow line. Infected class (I): this includes an individuals who have been infected and shows symptom of the infection, after treatment it then move to recovery class, without treatment it contribute to bacteria population with thick arrow directed to Bacteria class.

Vaccination (V): this is individual who vaccinated against the infection. Recovery class (R): this class includes all individuals that have recovered from the infection and move back to susceptible and Concentration of Bacteria is K_B, as shown in *Figure 1*, K_B is interact with population S as it shown with dash arrow and become infected I with tick line, I recovered and move to Recovery class R, meanwhile, R have only temporary recovery, it then move back to S, while S is vaccinated and move to vaccination class and vaccination individuals become susceptible (S) after loss of immunity.

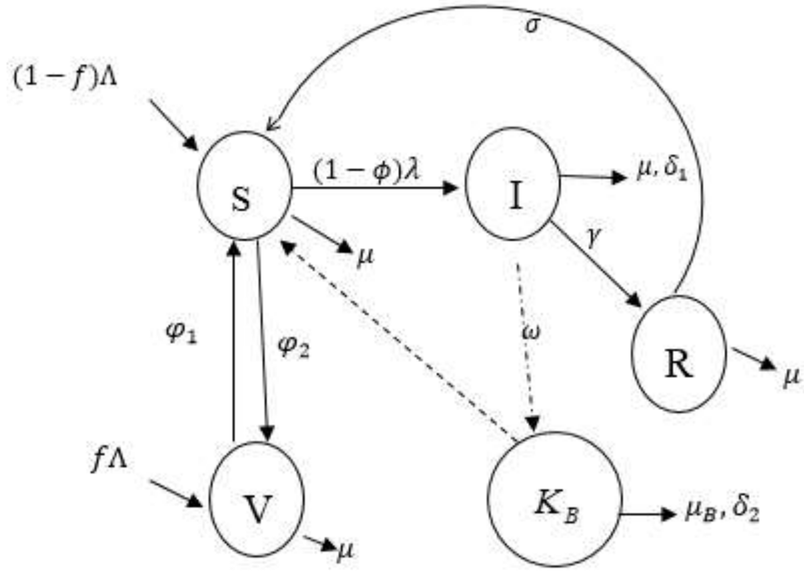

Fig 1: Schematic representation for the Bacterial transmission model

The transfer rates between the sub-classes are collection of several epidemiological parameters. The susceptible human population (S) is increase by recruitment rate Λ, the rate at which individuals is vaccinated is φ_2 and f is the proportion of individuals who are vaccinated. The proportion of unvaccinated individuals is (1-f) and φ_1 is the rate of losing immunity from vaccination individuals. Also μ is the natural death rate which is applicable to all the classes. Bacteria (K_B) interact with S and become infected with force of infection $\beta K_B/(C+K_B)$, it then move to infected class (I), where β is the effective contact rate, also, K is the concentration of the bacteria in contaminated environment, and $K_B/(C+K_B)$ is the probability of individuals in consuming foods or drinks contaminated caused by bacteria, the rate at which infected individuals die as a result of disease is δ_1 and ε is the effectiveness of compliance of good hygiene and ϕ is the effectiveness of drug. Meanwhile, The rate at which individuals recovered from I class as a result of treatment from infection is γ, there is no permanent recovery from the infection, recovery (R) individuals move back to susceptible class at the rate of σ. Population of Bacteria (K_B) increase at the rate of ω, the mortality rate of bacteria is μ_B and the rate of sanitation which lead to death of bacteria is δ_2. The model flow diagram is shown in *figure 1*. The dash line from Bacteria class (K_B) to susceptible class (S) shows that susceptible individuals get the infection from Bacteria. The tick lines show the movement of one class to another class.

Based on the above schematic representation and assumptions of the models, the equations governing the dynamics of the Acute diarrhea infection are given as:

$$\frac{dS}{dt} = (1-f)\Lambda + \varphi_1 V + \sigma R - (1-\phi)\lambda S - \varphi_2 S - \mu S \quad (1)$$

$$\frac{dI}{dt} = (1-\phi)\lambda S - \gamma I - (\mu + \delta_1)I \quad (2)$$

$$\frac{dV}{dt} = f\Lambda + \varphi_2 S - \varphi_1 V - \mu V \quad (3)$$

$$\frac{dR}{dt} = \gamma I - \sigma R - \mu R \quad (4)$$

$$\frac{dK_B}{dt} = (1-\varepsilon)\omega I - \mu_B K_B - \delta_2 K_B$$

(5)

Where, $N = S + I + V + R$

And $\lambda = \frac{\beta K_B}{C + K_B}$ (6)

The Model Analysis

Invariant Region. To obtain the invariant region, we considered the total human population (N), where $N = S + I + V + R$. Then, the differentiation of N with respect to time leading to:

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dV}{dt} + \frac{dR}{dt} \quad (7)$$

Then we have:

$$N \le \frac{\Lambda}{\mu} - \{\frac{\Lambda - \mu N_0}{\mu}\}e^{-\mu t} \quad (8)$$

As $t \to \infty$ in (8), the population size $N \to \frac{\Lambda}{\mu}$ which means that, $0 \le \mu \le \frac{\Lambda}{\mu}$. Thus, the feasible solution set of the system equations of the model enters and remains in the region:

$$\Omega = \{(S, I, V, R) \in \Re^4 : N \le \frac{\Lambda}{\mu}\}$$

Therefore, the model system is well posed mathematically and epidemiologically. Hence, it is sufficient to study the dynamics of the basic model in region Ω.

The Disease Free Equilibrium (DFE). To find the disease free equilibrium, we set the equations (1)-(6) to zero (0) and solve simultaneously, we make K_B in (5) subject of the expression and substitute into (2), we have

$$I\{\frac{\beta S\omega(1-\phi)(1-\varepsilon)}{C((\mu_B + \delta_2) + (1-\varepsilon)\omega I)} - \gamma - (\mu + \delta_1)\} = 0$$

$I = 0$ or

$$\frac{\beta S\omega(1-\phi)(1-\varepsilon)}{(C\mu_B + C\delta_2 + (1-\varepsilon)\omega I)} - \gamma - (\mu + \delta_1) = 0$$

(9)

Since

$I = 0$, then it implies $K_B = 0, R = 0$. Therefore, the disease free equilibrium

$$DFE(E_0) = (\frac{(1-f)\Lambda(\varphi_1 + \mu) + f\Lambda\varphi_1}{(\varphi_1 + \mu)(\varphi_2 + \mu) - \varphi_1\varphi_2}, 0, \frac{f\Lambda\mu + \Lambda\varphi_2}{(\varphi_1 + \mu)(\varphi_2 + \mu) - \varphi_1\varphi_2}, 0, 0)$$

(10)

The Effective Reproduction Number (R_e). The effective reproduction number (R_e) is the secondary infection cases infected on average per person, to obtain the basic reproduction number, we used the next generation matrix which is the approach adopted by Lasisi, Akinwande and Olayiwola *et al*. (2018). Both $F(x)$ and $V(x)$ are obtained from the model equations (2) and (5), we get

$$I^I = \frac{\beta K_B(1-\phi)S}{C + K_B} - \gamma I - (\mu + \delta)I$$

$$K_B^1 = (1-\varepsilon)\omega I - \mu_B K_B - \delta_2 K_B$$

Therefore, $F(x)$ is the inflow of the infected class while $V(x)$ is the outflow of the infected class, we have the following:

$$f = \begin{pmatrix} f_1 \\ f_2 \end{pmatrix} = \begin{pmatrix} \frac{(1-\phi)\beta K_B}{C+K_B} S \\ (1-\varepsilon)\omega I \end{pmatrix},$$

$$F = \begin{pmatrix} 0 & \frac{C(1-\phi)\beta}{(C+K_B)^2} S \\ (1-\varepsilon)\omega & 0 \end{pmatrix}$$

The Jacobian matrix of f and v evaluated at DFE are given by F and V, we get:

And

$$v = \begin{pmatrix} (\gamma+\mu+\delta_1)I \\ (\mu_B+\delta_2)K_B \end{pmatrix},$$

$$V = \begin{pmatrix} \gamma+\mu+\delta_1 & 0 \\ 0 & \mu_B+\delta_2 \end{pmatrix}$$

The characteristics equation of FV^{-1} is obtained with the inverse of V as:

$$\left|(FV^{-1})-\lambda I\right| = \begin{pmatrix} -\lambda & \frac{(1-\phi)\beta}{C(\mu_B+\delta_2)} S^0 \\ \frac{(1-\varepsilon)\omega}{\gamma+\mu+\delta_1} & -\lambda \end{pmatrix} = 0 \quad (11)$$

The dominant eigenvalues of FV^{-1} which is the spectral radius give:

$$\lambda = +\sqrt{\frac{(1-\varepsilon)\omega}{(\gamma+\mu+\delta_1)} \frac{(1-\phi)\beta}{C(\mu_B+\delta_2)} S^0} \quad (12)$$

Therefore, the basic reproduction number (R_0) after substitution of S^0 is given as:

$$R_e^2 = \frac{(1-\varepsilon)\omega(1-\phi)\beta\{(1-f)\Lambda(\varphi_1+\mu)+f\Lambda\varphi_1\}}{(\gamma+\mu+\delta_1)C(\mu_B+\delta_2)\{(\varphi_1+\mu)(\varphi_2+\mu)-\varphi_1\varphi_2\}}$$

(13)

Global Stability of DFE

Theorem 1: The disease free equilibrium is globally asymptotically stable if $R_0 < 1$

Proof: To show this theorem, we construct suitable Lyapunov function is given by:

$$L = \omega I + (\gamma+\mu+\delta_1)K_B \quad (14)$$

We differentiate (14) with respect to t and substitute (1) - (5) into the differentiation, we get:

$$\frac{dL}{dt} = (1-\varepsilon)\omega\{\frac{\beta(1-\phi)K_B S}{C+K_B} - (\gamma+\mu+\delta_1)I\} + (\gamma+\mu+\delta_1)\{\omega I - (\mu_B+\delta_2)K_B\}$$

(15)

From (15) yields:

$$\frac{dL}{dt} = \frac{(\gamma+\mu+\delta_1)(\mu_B+\delta_2)CK_B}{C+K_B}\{R_e^2 - \frac{(C+K_B)}{C}\}$$

(16)

So if $R_e < 1$ then $\frac{dL}{dt} < 1$ or if $K_B = 0 \Rightarrow \frac{dL}{dt} = 0$. Hence, L is Lyapunov function on Ω and largest compact invariant set in $\{(S,I,V,R,K_B) \in \Omega, dL/dt = 0\}$ is the singleton $(S,0,V,0,0)$. Therefore, by Lasalle's invariance principle (16),that all the solution of the model equations (1) - (6) with initial condition in the region which approach the DFE at time tends to infinity when $R_e \le 1$, hence, DFE is globally asymptotically stable in the feasible region Ω if $R_e \le 1$

The Endemic Equilibrium: The endemic equilibrium state is denoted by $E^* = (S^*, I^*, V^*, R^*, K_B{}^*)$ and this occurs when the infection is persistence in the population. To obtain this, we equate the system of equations (1) - (6) to zero and we have the following:

$$E^* = \left\{\frac{(\gamma+\mu+\delta_1)\{(\mu_B+\delta_2)C+\omega I^*\}}{\beta(1-\emptyset)\omega}, I^* > 0, \frac{f\Lambda\beta(1-\varepsilon)(1-\emptyset)+\varphi_2(\gamma+\mu+\delta_1)\{(\mu_B+\delta_2)C+(1-\varepsilon)\omega I^*\}}{\beta(1-\varepsilon)\omega(1-\emptyset)(\varphi_1+\mu)}, \frac{\gamma I^*}{(\sigma+\mu)}, \frac{(1-\varepsilon)\omega I^*}{(\mu_B+\delta_2)}\right\} \quad (17)$$

Numerical Results and Discussion: The model is simulated using the parameter values in Table 1, to assess the effect of different strategies considered in this study, which are educational awareness, environmental sanitations and hygiene, and treatment of individuals.

Table 1. Symbols, Parameters definition and Values

Symbols	Definition of Parameters of the Model	Values
Λ	Human Populations rate	100/day
φ_1	Rate of losing immunity from vaccination individuals	0.55/day
σ	Rate of recovered humans to become Susceptible	0.003/day
φ_2	Rate at which individuals are vaccinated	0.45/day
β	Exposure to contaminated food and water(Effective contact rate)	0.9/day
ϕ	Compliance rate of waters & foods Hygiene	0.6/day
γ	Recovery rate of infected humans	0.002/day
ω	Production of Bacteria infection from infected humans	Assumed
μ	Natural human mortality	0.0247/day
δ_1	Disease induced dearth rate	0.052/day
f	Proportion of unvaccinated individuals	0.075/day
C	Concentration of the bacteria in contaminated water	50,000Lit/day
μ_B	Mortality rate for bacteria	0.001/day
δ_2	Water Sanitation lead to dearth of Bacteria	0.05/day
ε	Compliance of good hygiene	(0, 1)

(Mabel *et al.*, 2022)

The data for numerical simulations with respect to each of the epidemiological parameters are given in Table 1 and Using Maple 17 Software for the graphical representation of effective reproduction numbers and model simulations with parameter values of the model equations are shown below,

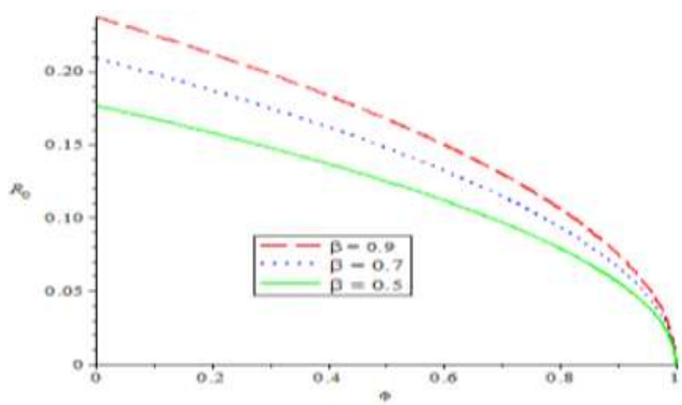

Fig 2. Varying the rate of exposure to contaminated foods and waters (β) on reproduction number

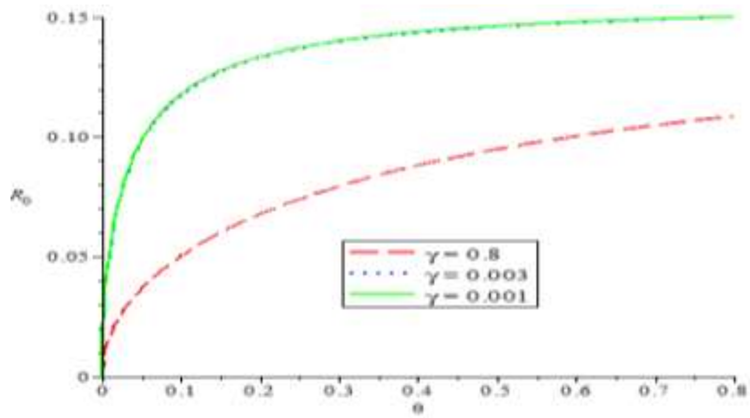

Fig 3. Production rate of Bacteria infection on reproduction number

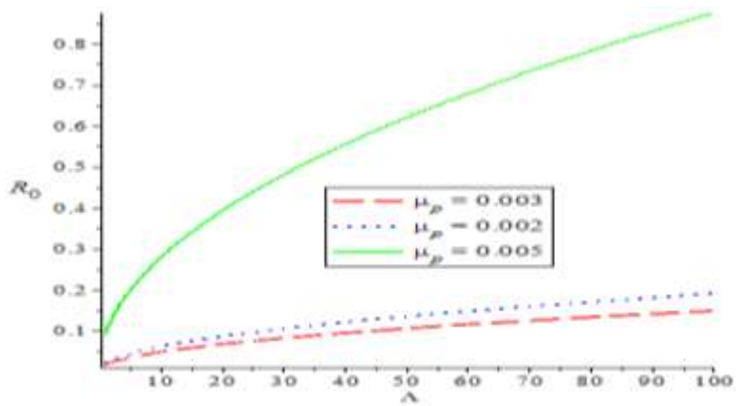

Fig 4. Effect of recruitment rate (Λ) on the basic reproduction number

Figure 3 shows the effect of compliance of good hygiene on effective reproduction number R_e, it is observed from figure 2 that, as compliance rate of good hygiene (ϕ) increases, the effective reproduction number decreases (R_e). Varying the rate of effective contact rate (β), we observed that as effective contact rate decreases, it also decreases the effective reproduction number. It is observed from figure 3 that as production rate of Bacteria infection (ω) from infected human increases, it increases the effective reproduction number R_e, varying the recovery rate (γ), it was discovered that as recovery parameter increases, it decreases the effective reproduction number. Meanwhile, figure 4 shows the effect of recruitment rate (Λ) on the effective reproduction number R_e, it is observed that as recruitment rate (Λ) increases, it increases the effective reproduction number R_e .

Conclusion: In this study, we presented an improved model for the transmission of Bacterial transmission disease; we incorporated the effectiveness of compliance of good hygiene. From the analysis, it was found that, the Disease Free Equilibrium is globally asymptotically stable if $R_e \leq 0$ and Endemic equilibrium state was obtained. Graphically, we found that as compliance rate of good hygiene (ϕ) increases, the effective reproduction number decreases (R_e) and we observed that as effective contact rate decreases, it also decreases the effective reproduction number. Meanwhile, we observed the effect of recruitment rate (Λ) on the effective reproduction number R_e , it is observed that as recruitment rate (Λ) increases, it increases the effective reproduction number R_e . Therefore, the treatment regime against bacterial infection in a population would be a good approach to effectively control or eradicate the acute diarrhea infection.

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REFERENCES

Abdulrahman, S; Akinwande, NI; Awojoyogbe, OB; Abubakar, UY (2013). Sensitivity analysis of the parameters of A mathematics model of Hepatitis B virus transmissions. *Universal J. Appl. mathematics*. 1(4): 230-240.

Akinwande, NI (2006). A Mathematical Model of the Dynamics of the HIV/AIDS Disease Pandemic. *J. Nig. Mathematical Soc.* 25: 99-108.

Bertoletti, A; Maini, M; Williams, R (2003). Role of hepatitis B virus specific cytotoxic T cells in liver damage and viral control, *Antiviral Res.* 60(2): 61–66.

Blanca Ochoa, MD; Christina MS; MACG MD (2002). University of Washington School of Medicine, Seattle, *WA – Published* October. Updated April 2007. Updated December 2012.

Culshaw, RV; Shigui, R (2000). A delay-differential equation model of HIV infection of CD4+ T-cells. *J. Mathematical Biosci*. 165: 27-39.

Codecco, CT (2001). Endemic and Epidemic dynamics of Cholera: the role of the aquatic reservoir. *BMC Infection Disease*. 1(1): 4-19

Jensen, MA; Faruque SM; Mekalanos, JJ; Levin BR (2006). Modelling the role of bacteriophage in the control of Cholera outbreaks. *P. Natl. Acad. Sci*. USA. 103(12): 4652-4657

Lasisi, NO; Akinwande, NI; Oguntolu, FA (2020). Development and exploration of a Mathematical Model for Transmission of Monkey-Pox in Humans. *J. Mathematical Models in Engineering*. 6(1): 23-33. DOI: https://doi.org/10.21595/mme.2019.21234

Lasisi, NO; Akinwande, NI; Olayiwola, RO, *et al*. (2018). Mathematical Model for Ebola Virus Infection In Human With Effectiveness of Drug Usage. *J. Appl. Sci. Environ. Manage*. 22(7): 1089–1095. DOI: https://dx.doi.org/10.4314/jasem.v22i7.16. http://ww.bioline.org.br/ja or https://www.ajol.info/index.php/jasem

Mabel, T; Juliet, E; James O (2022). Review of the Bacterial infection in a poor Student Hostel environment of Federal Polytechnic Kaura Namoda, Nigeria. *JBAS*, 1: 7-10

Misra, AK; Singh V (2012). A delay mathematical model for the spread and control of water borne diseases. *J. Theor. Bio.* 301: 49-56.

Mwasa M; Tchuenche, N (2011). Mathematical analysis of a cholera model with public health interventions. *Biosystems*. 105: 190-200.

Pascual, M; Bouma, MJ; Dobson, AP (2002). Cholera and climate: Revisiting the quatitative evidence. *Microbes. Infect.* 4: 237-245

Tilahun, GT; Makinde, OD; Malonza, D (2017). Modeling and Optimal Control of Typhoid Fever Disease with Cost-Effective Strategies. *Computat. Math. Meth. Med.* 2017: 1-16.

Tien, JH; Earn DJD (2010). Multiple transmission pathways and disease dynamics in a waterborne pathogen mode. *Bull. Math. Biol*. 72: 1506-1533