



Global Stability Analysis of Disease Free Equilibrium for Modeling the Dynamics of Bacteria Infection in Higher Institution Kaura Namoda, Nigeria

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ABSTRACT

Bacterial infection of the intestine is caused by ingestion of food or water containing vibrio cholera. Sanitary conditions in the environment play an important role. In this research work, we proposed a model to describe the transmission of the bacterial infection amongst the Students of Federal Polytechnic Kaura Namoda, Zamfara State. We also incorporated the effectiveness of drug usage and awareness for proper hygiene and sanitation. We obtained the invariant region and found that the model system is well posed mathematically and epidemiologically. The disease free occurs when the infection is free in the population or no epidemic in the population, while, endemic equilibrium occurs when the infection is persistence in the population. So, disease free and endemic equilibrium were determined. The effective reproduction number R_e is obtained and we established that disease free equilibrium is globally asymptotically stable if secondary case of the infection on average is less than a Unit ($R_e < 1$), using suitable Lyapunov function method.

Numerical simulations from figures show that Hygiene and Sanitation awareness, and Effectiveness of drug reduced the average number of the secondary cases of bacterial infection disease; on the other hand, Production of Bacterial infection, Human population and Contact rate increased the average number of the secondary case of the bacterial infection disease.

Keywords: Bacteria Infection, Disease free equilibrium, Effective reproduction number, Epidemiological model.

INTRODUCTION

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 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, Gonorrhoea, 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, Olayiwola *et al.*, 2018). Among the common are Ebola virus (Lasisi, Akinwande, Olayiwola *et al.*, 2018). Hepatitis B virus (Bertoletti, Maini, & Williams, 2003). The Human Immunodeficiency 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 explore the transmission dynamics and control of the infection.

Model Formulation

The model considers human population and the Bacterial host environment, the mathematical model systems are formulated using nonlinear incidence rate of ordinary differential equations. The work combined class of vaccination, drug effectiveness and awareness for proper hygiene and sanitation into our model. The population is subdivided into classes of five; class of susceptible (S): this class is exposed to bacterial infection. Class of infection (I); this class is individuals who have been infected, when treatment takes place, they move to class of recovery, meanwhile, with no treatment also contribute to bacterial infection population. Class of vaccination (V): this is group of individuals that are vaccinated against the disease. Class of recovery (R): this is group of individuals that have recovered from the disease and still go back to susceptible class and Class of Bacteria is K_B , as presented in Figure 1.

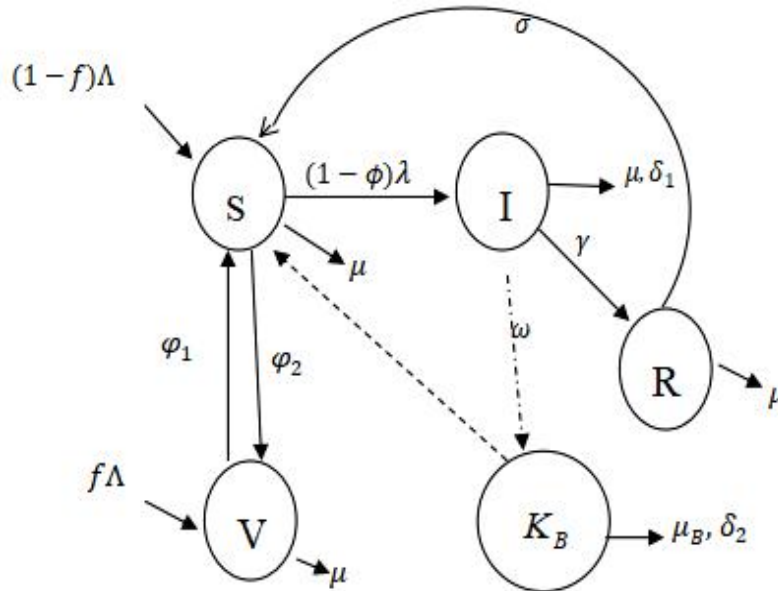


Figure 1: Schematic representation for the Bacterial transmission model

The transfer rates between the sub-classes are collection for several epidemiological parameters. The susceptible human population (S) is increase by recruitment rate Λ , the rate at which individuals are 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. class (S) shows that susceptible individuals get the infection from Bacteria. The tick lines show the movement of one class to another

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

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

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

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

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

$$\frac{dK_B}{dt} = (1-\varepsilon)\omega I - \mu_B K_B - \delta_2 K_B \quad (5)$$

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

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

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:

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

As $t \rightarrow \infty$ in (8), the population size $N \rightarrow \frac{\Lambda}{\mu}$ which means that, $0 \leq \mu \leq \frac{\Lambda}{\mu}$. Thus, the feasible

solution set of the system equations of the model enters and remains in the region:

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

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

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

$$I = 0 \text{ or } \frac{\beta S \omega (1-\phi)(1-\varepsilon)}{(C\mu_B + C\delta_2 + (1-\varepsilon)\omega I)} - \gamma - (\mu + \delta_1) = 0 \tag{9}$$

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

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

(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, Olayiwola *et al.* (2018). Both $F(x)$ and $V(x)$ are obtained from the model equations (2) and (5), we get

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

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

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:

$$\text{And } v = \begin{pmatrix} (\gamma + \mu + \delta_1)I \\ (\mu_B + \delta_2)K_B \end{pmatrix}, V = \begin{pmatrix} \gamma + \mu + \delta_1 & 0 \\ 0 & \mu_B + \delta_2 \end{pmatrix}$$

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

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

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:

$$R_e^2 = \frac{(1-\varepsilon)\omega(1-\phi)\beta\{(1-f)\Lambda(\varphi_1 + \mu) + f\Lambda\varphi_1\}}{(\gamma + \mu + \delta_1)C(\mu_B + \delta_2)\{(\varphi_1 + \mu)(\varphi_2 + \mu) - \varphi_1\varphi_2\}} \quad (13)$$

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:

$$\frac{dL}{dt} = (1-\varepsilon)\omega\left\{\frac{\beta(1-\phi)K_B S}{C + K_B} - (\gamma + \mu + \delta_1)I\right\} + (\gamma + \mu + \delta_1)\{\omega I - (\mu_B + \delta_2)K_B\} \quad (15)$$

From (15) yields:

$$\frac{dL}{dt} = \frac{(\gamma + \mu + \delta_1)(\mu_B + \delta_2)CK_B}{C + K_B} \left\{R_e^2 - \frac{(C + K_B)}{C}\right\} \quad (16)$$

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 $\{(S, I, V, R, K_B) \in \Omega, \frac{dL}{dt} = 0\}$ 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 $R_e \leq 1$, hence, DFE is globally asymptotically stable in the feasible region Ω if $R_e \leq 1$

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:

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

NUMERICAL SIMULATIONS OF THE MODEL PARAMETERS

We carry out numerical simulation of the effective reproduction number on every sensitive and important parameter, using the

parameter values in Table 1. To examine the impact of each control on eradication of the Bacterial disease, we generate the following simulations with the below table.

Table 1: Symbols, Parameters definition and Values

Symbols	Definition of Parameters of the Model	Values
Λ	Human Populations rate	100/day /day
φ_1	Rate of losing immunity from vaccination individual	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	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 death rate	1.052/day
f	Unvaccinated individuals proportion	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.

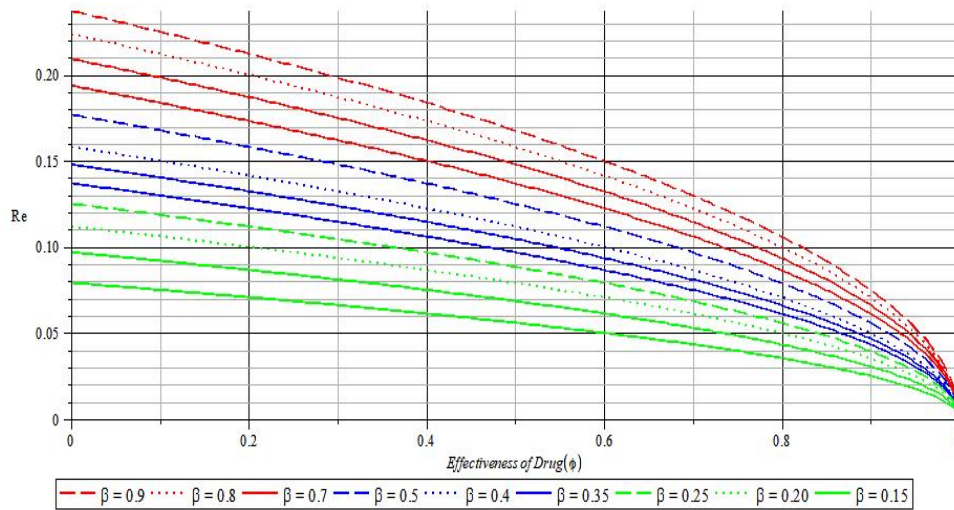


Figure 2: Effectiveness of Drug on Effective Reproduction Number

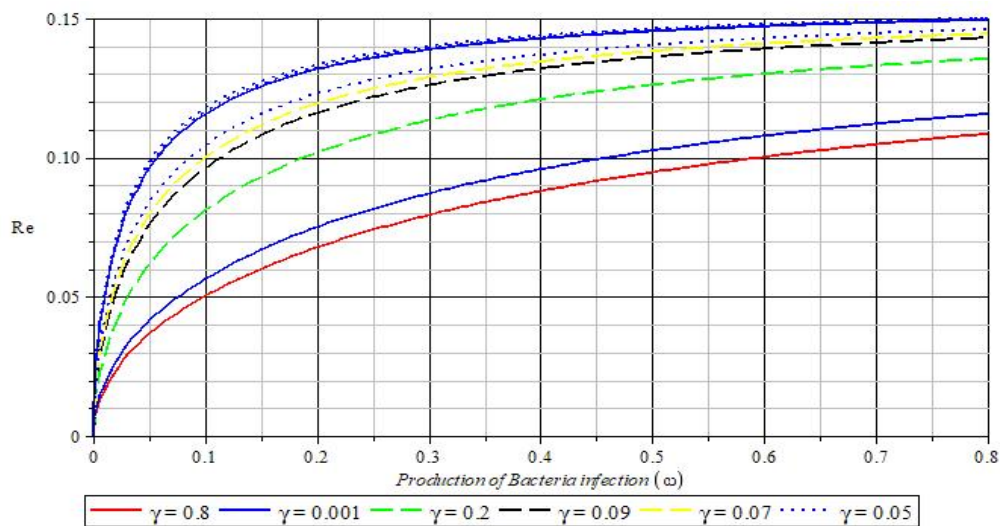


Figure 3: Bacterial Infection Production on Effective Reproduction Number

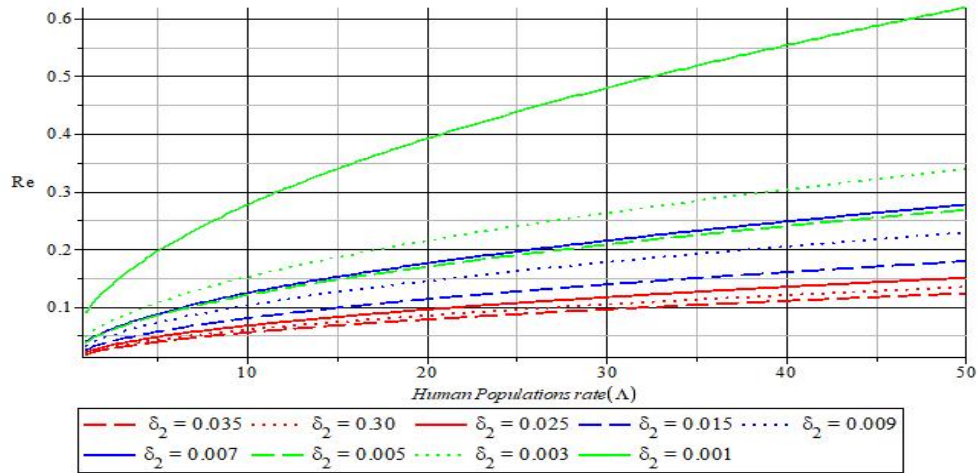


Figure 4: Human population on Reproduction Number

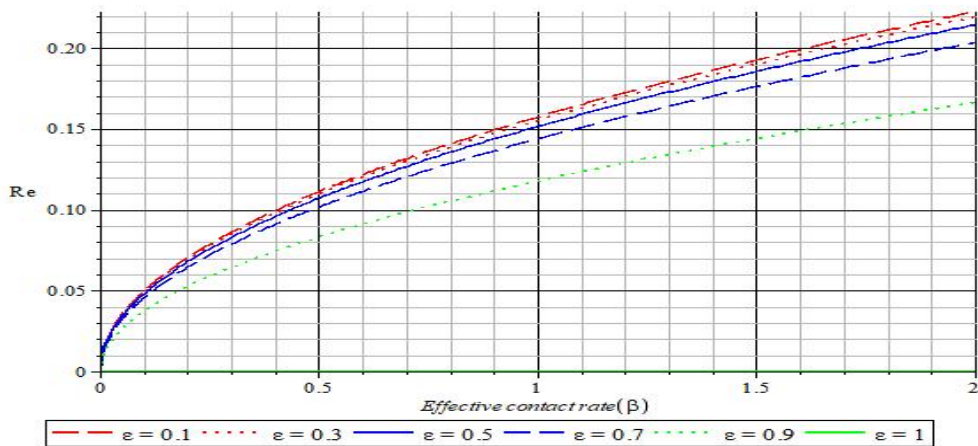


Figure 5: Contact Rate on Reproduction Number, Varying public awareness (good hygiene)

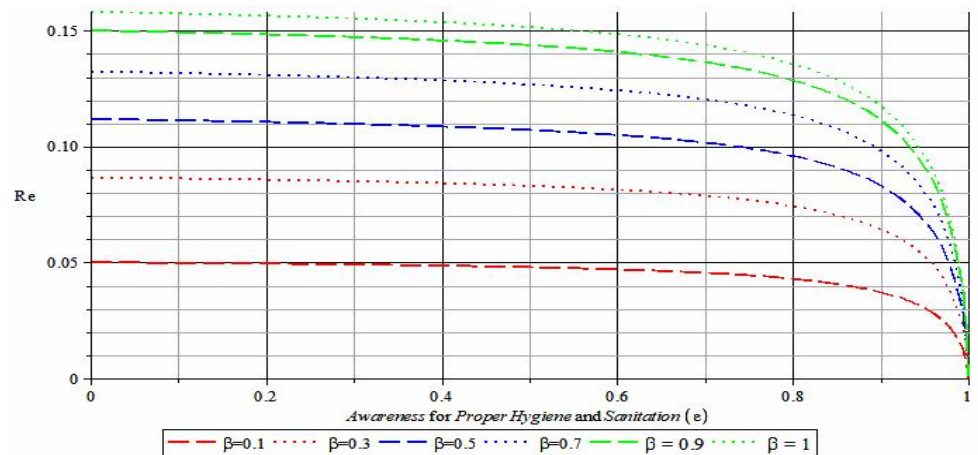


Figure 6: Hygiene and Sanitation awareness on Effective reproduction number



DISCUSSION

In figure 2, we simulated the effectiveness of drug on effective reproduction number, we simulate the effectiveness of drug with difference values of contact rate on effective reproduction number (Secondary cases of the Bacterial infection disease), we found in figure 2, that average secondary cases of disease reduces as drug usage increases, varying the contact rate, we observed that secondary cases of the bacterial disease reduces as contact rate reduces. This implies that the bacterial disease would not have a place in the population if we demarcate the region of the epidemic and implement drug regime. Figure 3 shows the simulation of Bacterial infection production with changing in recovery rate of infected person; it is observed that average secondary cases of infection increase as production of bacterial infection increases, varying the recovery rate, we established that as recovery of person increases, it also reduced the effective reproduction number which we called average secondary cases. This means, proper sanitation is good strategist to eradicate the production of the virus in the Community. In the figure 4, we observed that secondary cases of the bacterial disease increases as the population of the Community increases without a proper control strategy on how to eradicate the Bacterial disease and it is only the death rate of the bacterial which is from sanitation that reduces the secondary cases in the population. Figure 5 shows that, as contact rate rises, it also rises secondary cases of the bacterial infection, varying good hygiene compliance, it is observed that as compliance of good hygiene increases, it reduces the secondary

cases of bacterial disease and even gone to zero as compliance of good hygiene reach maximum (1). This means that with full compliance of good hygiene, there would not be secondary cases of the disease and the bacterial disease would not persist in the Community. In this figure 6, we observed that Hygiene and Sanitation awareness increases, it reduces the secondary cases of the bacterial disease, varying the contact rate we revealed that, it reduces the secondary cases as contact rate of the human population reduces.

CONCLUSION

In this research work, a deterministic model for the dynamics of Bacterial Infection Disease is proposed. The qualitative analysis of the model equations is shown. We obtained equilibrium states of the model equations and the global stability conditions for disease free equilibrium are well established. We also showed the effective reproduction number. Numerical simulations from figures show that Hygiene and Sanitation awareness, and Effectiveness of drug reduced the average number of the secondary cases of bacterial infection disease, on the order hand, Production of Bacterial infection, Human population and Contact rate increased the average number of the secondary case of the bacterial infection disease.

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REFERENCES

- Abdulrahman, S., Akinwande, N. I., Awojoyogbe, O. B., and Abubakar, U. Y. (2013). Sensitivity analysis of the parameters of A mathematics model of Hepatitis B virus transmissions. *Universal Journal of Applied mathematics*, Vol. 1, Issue 4, p.230-240.
- Akinwande, N.I. (2006). A Mathematical Model of the Dynamics of the HIV/AIDS Disease Pandemic. *Journal of Nigeria Mathematical Society*, Vol. 25, p. 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 Research*, Vol. 60, Issue 2, p. 61– 66.
- Blanca Ochoa, MD and Christina M. Surawicz, MD, MACG (2002). University of Washington School of Medicine, Seattle, WA – Published October. Updated April 2007. Updated December 2012.
- Culshaw, Rebecca V., & Shigui Ruan (2000). A delay-differential equation model of HIV infection of CD4+ T-cells. *Journal of Mathematical Biosciences*, Vol. 165, p. 27-39.
- Codecco, C. T. (2001). Endemic and Epidemic dynamics of Cholera: the role of the aquatic reservoir. *BMC Infection Disease*, Vol. 1, Issue 1.
- Jensen, M. A., Faruque S.M., Mekalanos, J. J. And Levin B.R. (2006). Modelling the role of bacteriophage in the control of Cholera outbreaks. *P. Natl. Acad. Sci. USA*, Vol. 103, Issue 12, p.4652-4657
- Lasisi, N. O., Akinwande, N. I. & Oguntolu, F. A. (2020). Development and exploration of a Mathematical Model for Transmission of Monkey-Pox in Humans. *Journal of Mathematical Models in Engineering*, Vol. 6, Issue 1, p. 23-33. <https://doi.org/10.21595/mme.2019.21234>
- Lasisi N. O., Akinwande N. I., Olayiwola R. O., et al. (2018). Mathematical Model for Ebola Virus Infection In Human With Effectiveness of Drug Usage. *J. Appl. Sci. Environ. Manage*, Vol. 22, Issues 7, p. 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. and 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 A. K and Singh V. (2012). A delay mathematical model for the spread and control of water borne diseases. *J. Theor. Bio.*, Vol. 301, p.49-56.
- Mwasa and Tchuenche. (2011). Mathematical analysis of a cholera model with public health interventions. *Biosystems*, Vol. 105, p. 190-200.
- Pascual M., Bouma M. J. and Dobson A.P. (2002). Cholera and climate: Revisiting



the quantitative evidence. *Microbes. Infect.*, Vol. 4, p. 237-245

Tilahun G. T., Makinde O. D. and D. Malonza (2017). Modelling and Optimal Control of Typhoid Fever Disease with Cost-Effective Strategies. *Computational and*

Mathematical Method in Medicine, Vol. 2017, p. 1-16.

Tien J. H. And Earn D.J.D. (2010). Multiple transmission pathways and disease dynamics in a waterborne pathogen mode. *Bull. Math. Biol.* Vol. 72, p. 1506-1533.