

## **The Mathematical Models of the Dynamical Behaviour of Tuberculosis Disease in the Upper East Region of the Northern Part of Ghana: A Case Study of Bawku**

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**Abstract:** This study considers a Mathematical model of the dynamical behaviour of Tuberculosis disease in the Upper East region of the Northern part of Ghana. The equilibrium points of the model system are found and their stability is investigated. The model exhibits two equilibria namely, the disease-free and the endemic equilibrium. Using stability theory and computer simulation, it is observed that population determine the infection rate of tuberculosis hence the higher the population density, the greater the risk of instability of the disease free equilibrium.

**Key words:** Mathematical model, stability, free equilibrium, endemic equilibrium, simulation, tuberculosis

### INTRODUCTION

Tuberculosis which is deadly disease is on the rise and revisiting both developed and developing world. Globally, it is the leading cause of death than any other infectious disease like malaria, HIV, Schistosomiasis, Typhoid fever etc. Tuberculosis kill more adults each year, more women die of tuberculosis than other disease.

*Mycobacterium tuberculosis* is the bacteria that causes Tuberculosis (TB) and has been present in the human population since 2400BC. TB is an airborne infection that primarily affects the lungs. TB can be spread by coughing, sneezing, laughing or singing repeated exposure to someone with TB disease is generally necessary for infection to take place. TB is always spread by person(s) with tuberculosis of the lungs who are not been treated, these people cough out tuberculosis in droplet(s) nuclei into the air. This droplet(s) nuclei are then inhale into the respiratory tract of another person. The mycobacterium tuberculosis then starts to grow. If the organism grows, they cause a small area of bronchopneumonia, the organism then spread to the lymph node in the chest. They then carried by the lymph into the blood. The blood spreads to the lymph nodes in the chest. The blood spreads the organisms to the whole body in the 1-2 months whiles it is happening the body slowly developing immunity to the organism, when this immunity has properly developed usually the body kills the organism and the infection heals otherwise not healed.

According to Miranda (2003) he emphasized that the resurgence of tuberculosis and other infectious diseases

in the last decade has been closely linked with environmental and social changes that compromised peoples immune systems and the social structures that are used to defend against disease; in 1993, World Health Organization (WHO, 2003) declared tuberculosis as a global emergency in which approximately one third of the whole world is attacked by the tuberculosis. It was revealed that 99% of tuberculosis death all over the world occurs in developing countries.

Waalder *et al.* (1962) were the first people that's started mathematical modeling of tuberculosis, transmission dynamics of tuberculosis, their model comprise of a linear system of difference equations. The importance of their research is that it provided researchers with the basics starting point in the modeling tuberculosis dynamics in communities. In ReVelle *et al.* (1967) extended the work of Waalder by introducing a cluster transmission model and incorporated both the linear and logistics growth rates.

Most recently several investigation have concern themselves with modeling density-dependence dynamic of tuberculosis disease, notable and then are Gao and Hethcote (1992), Roberts and Jowett (1996) and Asematimaba (2005) to mention a few.

This study is an extension of the study of Asmatimba (2005) to the mathematical model of the dynamical behaviour of tuberculosis disease in Bawku District in the Upper East Region of Ghana, this is mainly to give suggestions on who to check, control, minimized, preventing and eradicate tuberculosis in Bawku Municipality.

Bawku Municipality is in the Upper East Region of Ghana. It is located at the extreme Northeastern part of the Upper East Region. It shares international borders with Burkina Faso to the North and Togo to the East. Since this municipality share common boarders with these two neighbouring Countries, it has certain Political and Social challenges. Some families have members in these neighbouring Countries which leads to free movement of People and this bring about high population density and health risk which is associated with overcrowding. There is high level of disease incidence, hence there is that need to look into the health condition of the people living in Bawku Municipality.

**THE MODEL**

We consider the model of Song *et al.* (2002) and Asematimaba (2005) and we have the following assumptions.

All people are likely to be infected by infectious individual in case of contact:

- Bawku Municipality has a fixed area size and only the population size is varying.
- All immigrants and newborn are uninfected hence they join the susceptible group.
- The population size is  $N(t)$  at time  $t$ , is divided into four subclasses of susceptible  $S(t)$ , infective  $I(t)$ , (also assumed to be infectious), latently infected/exposed individuals  $L(t)$ , recovered/tested individuals  $T(t)$  and  $A$  the total area of Bawku Municipality.

**In the model the following parameters are used:**  $\lambda$  is the recruitment rate,  $\mu$  is the per capital natural mortality rate,  $d$  is the tuberculosis-induced mortality rate,  $\beta_1$ , is the probability that a susceptible individual becomes infected by one infectious individual per contact per unit time.  $\beta_2$  is the probability that a recover individual becomes infected by one infectious individual per contact per unit time,  $k$  is the rate of progression to active tuberculosis,  $r_2$  is the recovery rate of the latent class,  $r_1$  is the recovery rate of the infectious class and  $c$  is the per capital contact rate.

In view of the above assumptions and inter-relations between the parameters, we have the following system of differential equations:

$$\frac{dS}{dt} = \lambda - \mu S - \beta_1 CS \frac{1}{A} \tag{1}$$

$$\frac{dL}{dt} = \beta_1 CS \frac{1}{A} + \beta_2 CR \frac{1}{A} - (\mu + K + r_2)L \tag{2}$$

$$\frac{dI}{dt} = KL - (\mu + d + r_1)I \tag{3}$$

$$\frac{dR}{dt} = r_1I + r_2L - \mu R - \beta_2 CR \frac{1}{A} \tag{4}$$

**ANALYSIS OF THE MODEL**

In this study, we present the results of stability analysis of the equilibrium point.

**Equilibrium of the model:** The governing system of equations of the model-[(i.e. Eq. 1-4)] has two non-negative equilibrium points namely:

- $E_0 (\lambda/\mu, 0,0,0)$  the disease-free equilibrium; Here we have  $I^* = L^* = 0$  and we define  $S_0 = \lambda/\mu$  as the asymptotic carrying capacity of the population.
- $E^* (S^*, L^*, I^*, T^*)$  the endemic equilibrium.

**Stability of the equilibria:** Now to determine the stability of  $E_0$  and  $E^*$ , the following variational Jacobian matrices are computed corresponding to equilibrium points  $E_0$  and  $E^*$ :

$$M_0 = \begin{pmatrix} -\mu & 0 & -\beta_1 \left( \frac{\lambda/\mu}{A} \right) & 0 \\ 0 & -(\mu+k+r_2) & \beta_1 \left( \frac{\lambda/\mu}{A} \right) & 0 \\ 0 & k & -(\mu+d+r_1) & 0 \\ 0 & r_2 & r_1 & -\mu \end{pmatrix}$$

$$M^* = \begin{pmatrix} -\left( \mu + \beta_1 c \frac{I^*}{A} \right) & 0 & -\beta_1 \frac{S^*}{A} & 0 \\ \beta_1 c \frac{I^*}{A} & -(\mu+k+r_2) & \beta_1 c \frac{S^*}{A} + \beta_2 c \frac{R^*}{A} & \beta_2 c \frac{I^*}{A} \\ 0 & k & -(\mu+d+r_1) & 0 \\ 0 & r_2 & r_1 - \beta_2 c \frac{R^*}{A} & -\mu - \beta_2 c \frac{I^*}{A} \end{pmatrix}$$

From  $M_0$  it is clear that  $E_0$  is asymptotically stable provided

$$\frac{A}{\lambda/\mu} > \left( \frac{k}{\mu+k+r_2} \right) \left( \frac{\beta_1 c}{\mu+d+r_1} \right)$$

i.e.,  $R_0 > 1$ , the disease dies out and wipe out under condition the equilibrium  $E^*$  does not exist. If  $R_0 > 1$ , then  $E^*$  exists and the infection is maintained in the population; hence to minimize the disease the population of the infections and latently infected must be very small, this implies that  $dL/dt < 0$  and  $dI/dt < 0$ . With these conditions we can minimize the disease if and only if

$$A > \left( \frac{\beta_1 c S + \beta_2 c T}{\mu + k + r_2} \right) \left( \frac{k}{\mu + d + r_1} \right)$$

**RESULTS AND DISCUSSION**

We give numerical simulation of the equilibrium and stability conditions of the governing equations of the model (i.e., Eq. 1-4). Following Asematimba (2005), Song *et al.* (2002), Styblo (1991), Feng *et al.* (2001), Castillo-Chavez and Song (2002) and WHO (2003) below are some of the parameter values used  $\Lambda = 3.805$ ,  $\mu = 0.0185$ ,  $r_1 = r_2 = 1.5$ ,  $\beta_1 = \beta_2 = 2.0$ ,  $c = 2$ ,  $d = 0.365$ ,  $k = 0.00396$ ,  $R^* = 300$ ,  $I^* = 90$ ,  $S^* = 5000$ ,  $L^* = 1000$ ,  $T = 1000$ .

The results of numerical simulation are shown graphically in Fig. 1-4.

In Fig. 1 the distribution of susceptible population with time in respect to the area occupied by the susceptible population is shown. We observe that the susceptible population increases as time increases, this is as a result of the recruitment rate,  $\Lambda$ , through birth and immigration. Also as the size of the area occupied is increased the number of susceptible will also increase because of the reduced disease incidence and recruitment.

Figure 2 shows the effect of size of the area occupied,  $A$  on the latent or exposed population with respect to time. It is observed that when the area occupied by the latent or exposed population increases with time, there is decrease in the number of latent population, this is as a result of the fact that when the area is big the contact rate of the susceptible with infectious individuals will be small. We further observe that with lower population density, the number of exposed individual increases.

It is observed from Fig. 3 that the number of infectious individuals declines in a small time interval irrespective of the area size. It is also seen that the infected individuals declined irrespective of the area size. This can be as a result of congestion in the community, which may lead to higher rate of infection and hence a big number of susceptible become infected and progress to infectious stages.

Figure 4 has to do with the effect of size of the area occupied,  $A$ , on the treated population with respect to

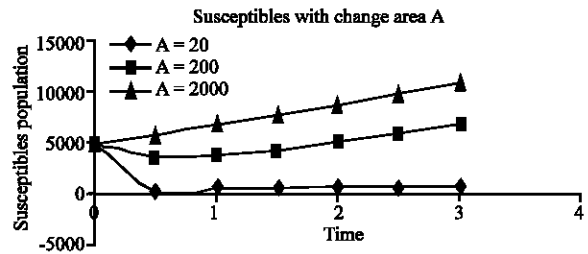


Fig. 1: The effect of size of the area occupied,  $A$ , on the susceptible population with respect to time

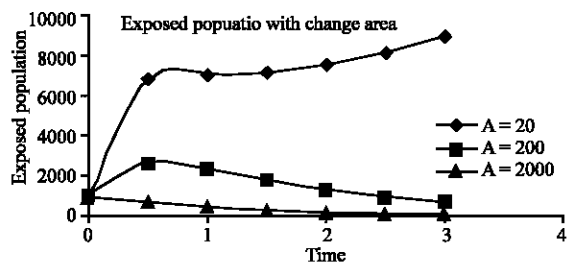


Fig. 2: The effect of size of the area occupied,  $A$ , on the exposed population with respect to time

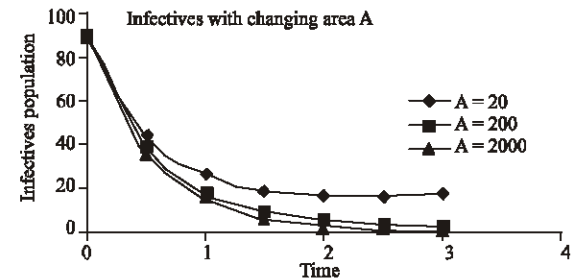


Fig. 3: The effect of the size of the area occupied,  $A$ , on the infective population with respect to time

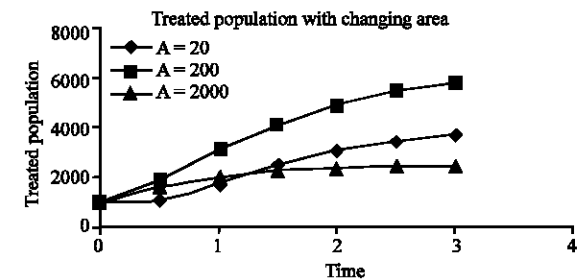


Fig. 4: The effect of the size of the area occupied,  $A$ , on the infective population with respect to time

time. We observe that there is decrease in the treated as the area occupied increases.

## CONCLUSION

In the study, a mathematical model of the dynamical behaviour of tuberculosis in Bawku in the Upper East Region of the Northern part of Ghana is studied. By analyzing the model, we have found a threshold parameter  $R_0$ . It is noted that when  $R_0 < 1$  then the epidemic will die out and when  $R_0 > 1$  the disease will persist in the population and become endemic. The model has two non-negative equilibria namely  $E_0 (\lambda/\mu, 0,0,0)$ , the disease free equilibrium and  $E^* (S^*, L^*, I^*, T^*)$  the endemic equilibrium. It is found that population density determines the infection rate of tuberculosis because of the level of respiratory contact in a community due to a high population density. Conclusively, the higher the population density, the greater the risk of instability of the disease-free equilibrium which implies that there is a possibility of an epidemic in Bawku district.

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## REFERENCES

- Asematimaba Amos, 2005. Mathematical models for the dynamic of Tuberculosis in Density-Dependent populations: The case of Internally Displaced Peoples' Camps (IPDCS) in Uganda M.Sc. dissertation, Makerere University Uganda.
- Castillo-Chavez, C. and F. Zhilan, 1997. To treat or not to treat: The case of tuberculosis. *J. Math. Biol.*, 35: 629-656.
- Castillo-Chavez, C. and B. Song, 2002. An overview of Dynamical Models of tuberculosis. Technical Report of BSCB, Cornell University, Ithaca, BU-1607-M. pp: 1-63.
- Feng, Z., W. Huang and C. Castillo-Chavez, 2001. On the role of variable latent periods in mathematical models for tuberculosis. *J. Dynamics and Differential Eq.*, 13: 425-452.
- Gao, L.Q. and H.W. Hethcote, 1992. Disease transmission model with density-dependent demographics. *J. Math. Biol.*, 30: 717-731.
- Miranda, D., 2003. Tuberculosis: Facts, challenges and courses of Action. Health Alert Asia-Pacific Edition, (HAIN-Health Action Information Network).
- ReVelle, C.S., W.R. Lynn and F. Feldman, 1967. Mathematical models for the economic allocation of tuberculosis control activities in developing countries. *Am. Rev. Respir. Dis.*, 96: 893-909.
- Roberts, M.G. and J. Jowett, 1996. An SEI model with density-dependent demographics and epidemiology. *IMA J. Math. Applied Med. Biol.*, 13: 245-257.
- Song, B., C. Castillo-Chavez and J.P. Aparicio, 2002. Tuberculosis models with fast and slow dynamics: The role of close and casual contacts. *Math Biosci.*, 180: 187-205.
- Styblo, K., 1991. Selected papers, epidemiology of tuberculosis. Royal Netherlands Tuberculosis Assoc., 24: 55-62.
- Waalder, H., A. Geser and S. Anderson, 1962. The use of Mathematical Models in the study of epidemiology of Tuberculosis. *Am. J. Public Health*, 52: 1002-1012.
- World Health Organisation, 2003. Tuberculosis Fact Sheet: Global Tuberculosis Program, Geneva.