ANALYSIS OF EFFECT OF OVERCROWDING ON THE SPREAD OF TUBERCULOSIS USING DETERMINISTIC MODELLING APPROACH: A CASE STUDY OF INTERNALLY DISPLACED RIVERSIDE SETTLEMENTS BY HYDRO ELECTRIC GENERATING POWER STATIONS OF NIGERIA.

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Abstract

In this study, the effects of population density on the dynamics of tuberculosis were discussed using deterministic modeling approach . A mathematical model was formulated that incorporated the size of the area occupied by the population being studied. The results suggested that the observed trend in tuberculosis incidence was partly as a result of explosive population growth due to urbanization and resettlements. In the qualitative analysis of the model, the disease free steady state was analyzed for stability using Rowth Hurwitz theorem. The analysis showed that there exists a globally stable disease-free equilibrium point. The disease-free equilibrium point will be globally asymptotically stable provided the characteristic area per individual is greater than the product of the probability of survival from latent stage into the infectious stage and the number of latent infections produced by a typical infectious individual during the mean infectious period. The results of numerical simulation were remarkably in line with those of the qualitative analysis of the model.

Key words: Tuberculosis, Population density, Equilibrium State and Stability Analysis.

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1.0. INTRODUCTION

Tuberculosis (TB) is a contagious bacteria disease caused by inhaling the tubercle bacillus in the droplet nucleus form. Like the common cold, it spreads through the air. An infected person may have latent TB infection or active TB infection. Only actively infected people who are sick with TB bacilli in their lungs are infectious. When infectious people cough, sneeze, talk or spit, they propel TB germs, known as bacilli, into the air. A person needs only to inhale a small number of these to be infected (World health Organization, 2007). A latent TB infected person does not show any symptoms of the disease and cannot infect others, though may live as long as possible without it degenerating into active TB (Sepkowitz, 1996).

The spread of Tuberculosis and other infectious diseases in the last decade has been closely linked with environmental and social structures that are used as defensive mechanism against disease. It is a proved fact from the previous epidemiological studies that there exists a direct relationship of tuberculosis with poverty and under development. Tuberculosis occurs in the poorest and most underdeveloped countries of the world and within societies, it occurs in the most socio economically deprived sectors, where factors contributing to its spread exist in overcrowding, malnutrition and lack of access to Health care services. Overcrowding increases the chance of infection by prolonged exposure (Miranda, 2003, Adetunde, 2009 and Gannon, 2000).

Niger state in Nigeria, houses two hydro electric generating power stations. Electric current is generated by the force of flowing water from a dam built to accumulate water from River Niger. The accumulated water is then discharged under controlled speed which turns the blades of turbines thereby generating electric current which is then distributed to the nation.

However, when there is excess accumulation of water in the dam as a result of heavy rainfall, the dam releases excess water which over flows the river bank; the riverside settler are displaced from their place of settlement and are forced to find safety in camps until the water level subsides.

The over flow of the Niger river bank have forced occupants of riverine settlements into safety camps as internally displaced people. Most of these camps are overcrowded with extremely poor

sanitation. Overcrowding, Cold, Sleeping rough and poor sanitary conditions ensures rapid transmission of Tuberculosis within the camp and treatment of tuberculosis which lasts for approximately eight months is quite costly in areas far from hospitals. (Dorsey and Opeitum, 2002). Preventive care and treatment of tuberculosis is less available and less extensive and effective due to low access to quality health care services and because of this, some cases go untreated and those affected often die. There are very limited spaces in these camps for human activities and the tents that people occupy are not properly ventilated to ensure proper sanitation. Displaced people are at a greater risk of becoming infected with TB due to close contact with infectious individuals. In this study the researcher investigate the effect of population density on the dynamics of tuberculosis.

2.0. MATERIALS AND METHODS

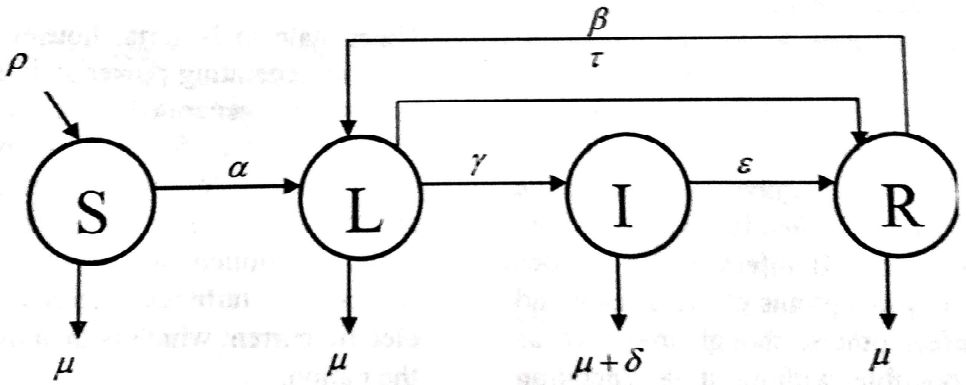
Presentation of the Model

To help analyze the density dependence of the dynamics of tuberculosis. the researcher make use of the mechanism of transmission as in the model of (Song et al, 2006) and formulate a mathematical model incorporating a parameter for the size of the area occupied by the population being studied in the transmission of tuberculosis.

The author assumed that all immigrants and new born babies are uninfected and therefore join the susceptible category, the author further assumed that there is homogeneous mixing of population where every one in the population under study have equal chance of been infected by infectious individuals through contact. The initial population of 1,500 individual was considered and distributed over the whole settlement (in the specified area).

2.1. Model flow diagram

Figure: 1 Schematic presentation of the model



2.2. Model Equations.

Based on the assumption and interrelations between the variables and parameters as described above in the compartmental model, the researcher described the model for controlling the spread of Tuberculosis in a density dependent population with the following set of ordinary differential equations.

$$\frac{dS}{dt} = \rho - \mu S - \frac{\alpha SI}{A} \quad (2.1)$$

$$\frac{dL}{dt} = \frac{\alpha SI}{A} - (\mu + \gamma + \tau)L + \frac{\beta RI}{A} \quad (2.2)$$

$$\frac{dI}{dt} = \gamma L - (\mu + \delta + \varepsilon)I \quad (2.3)$$

$$\frac{dR}{dt} = \tau L + \varepsilon I - \mu R - \frac{\beta RI}{A} \quad (2.4)$$

Where P=S+L+I+R

2.3 Model parameters and variables

A – Total area of the camp.

ρ – Recruitment rate.

S (t) – Susceptible class

L (t) – Latent class

I (t) – Infectious class.

R(t) – Recovered class.
μ - Natural mortality rate.
δ - Tuberculosis induced death.
α - Susceptible contracting rate.
β - Recovered individuals' contracting rate.
γ - Progression rate to active Tuberculosis
τ - Latent recovery rate.
ε - Infectious recovery rate.

The susceptible population changes as a result of recruitment into the camp at the rate ρ. This is further reduced by the natural mortality rate μ and infection through respiratory contact with the infected population. In the same way the latently infected population dynamics depends on the population of susceptible that got infected by the infectious population and this is reduced by the natural death, break down from latent class to the infectious class and recovery of the latently infected individuals. It is also important to note that there is possibility of the recovered individuals becoming infected again after recovery, this however adds to the population of the latent class. The infectious class depends on the break down from latent class to Infectious class and this is reduced by natural death rate, Tuberculosis induced deaths and recovery from the infectious class. The recovered class depends on the population of recovered latent and recovered Infectious individuals. This diminishes by natural death and re infection after recovery through respiratory contacts with infectious individuals.

3. Methods of Solutions

The researcher applies two different methods to obtain solutions to the model. The analytical solutions at equilibrium states and the numerical solutions using

$$\rho - \mu w - \frac{\alpha w y}{A} = 0$$

Euler's numerical method and the results displayed using Maple mathematical software package.

3.1. Equilibrium Solutions.

Let the equilibrium states be given by {(S(t), L(t), I(t), R(t)}= {w, x, y, z}= {0, 0, 0, 0}Equations (2.1) to (2.4) give(3.1)

$$\frac{\alpha w y}{A} - (\mu + \gamma + \tau)x + \frac{\beta z y}{A} = 0 \qquad (3.2)$$

$$\gamma x - (\mu + \delta + \varepsilon)y = 0 \qquad (3.3)$$

$$\tau x + \varepsilon y - \mu z - \frac{\beta z y}{A} = 0 \qquad (3.4)$$

3.1. Zero equilibrium state

For as long as there is continuous movement into the camp, the population will not be wiped off by Tuberculosis. Hence there exists no zero equilibrium state. i.e. (w, x, y, z) $\neq$ (0,0,0,0).

3.2 Disease free equilibrium state.

The disease free equilibrium state is the state of complete eradication of tuberculosis. At this state there will be no latent and infectious TB classes. This implies that (x, y) = (0, 0). Substituting this into equations (3.1) to (3.4) gives $E_0 = (\frac{\rho}{\mu}, 0, 0, 0)$, The author define S_0 as the asymptotic carrying capacity of the population.

3.3. Stability analysis of the Disease Free Equilibrium State.

To investigate the stability of the disease free equilibrium state $E_0 = (\frac{\rho}{\mu}, 0, 0, 0)$, we obtain the Jacobian matrix at E_0, This gives,

$$J_{E_0} = \begin{pmatrix} -\mu & 0 & -\beta\left(\frac{\rho/\mu}{A}\right) & 0 \\ 0 & -(\mu+\gamma+\tau) & \beta\left(\frac{\rho/\mu}{A}\right) & 0 \\ 0 & \gamma & -(\mu+\delta+\varepsilon) & 0 \\ 0 & \tau & \varepsilon & -\mu \end{pmatrix}$$

If λ_i are the eigen values of the Jacobian matrix at the disease free equilibrium state, then

$$(\lambda+\mu)^2 \det\begin{pmatrix} -(\mu+\gamma+\tau)-\lambda & \beta\left(\frac{\rho/\mu}{A}\right) \\ \gamma & -(\mu+\delta+\varepsilon)-\lambda \end{pmatrix} = 0 \Rightarrow (\lambda+\mu)^2 = 0 \quad (4.1)$$

Or

$$\det\begin{pmatrix} -(\mu+\gamma+\tau)-\lambda & \beta\left(\frac{\rho/\mu}{A}\right) \\ \gamma & -(\mu+\delta+\varepsilon)-\lambda \end{pmatrix} = 0 \qquad (4.2)$$

From (4.1)

$\lambda_1 = \lambda_2 = -\mu$

To determine the nature of eigen values in (4.2), we apply Routh-Hurwitz theorem.

Let B=$\begin{pmatrix} -(\mu+\gamma+\tau) & \beta\left(\frac{\rho/\mu}{A}\right) \\ \gamma & -(\mu+\delta+\varepsilon) \end{pmatrix}$

Then from B, we have

Det (B)= $(\mu+\gamma+\tau)(\mu+\delta+\varepsilon) - \gamma\beta\left(\frac{\rho/\mu}{A}\right)$

And

Trace (B) $= -(\mu+\gamma+\tau) \ -(\mu+\delta+\varepsilon)$, obviously Trace (B) <0 since all the parameters are positive.

For the determinant of B to be positive, we should have

$$\left(\frac{\rho/\mu}{A}\right) < \left(\frac{\mu+\gamma+\tau}{\gamma}\right)\left(\frac{\mu+\delta+\varepsilon}{\beta}\right)$$

Hence the disease free equilibrium state will be stable if

$\left(\frac{A}{\rho/\mu}\right) > \left(\frac{\gamma}{\mu+\gamma+\tau}\right)\left(\frac{\beta}{\mu+\delta+\varepsilon}\right)$ where $\left(\frac{A}{\rho/\mu}\right)$ is the area occupied by the individual also referred to as characteristic area, $\left(\frac{\gamma}{\mu+\gamma+\tau}\right)$ is the probability of survival from latent infection to infectious stage and $\left(\frac{\beta}{\mu+\delta+\varepsilon}\right)$ is the number of latent infections produced by a typical infectious individual during mean infectious period.

The term $\left(\frac{\gamma}{\mu+\gamma+\tau}\right)\left(\frac{\beta}{\mu+\delta+\varepsilon}\right)$ defines the critical area size required for eradication of Tuberculosis.

3.4. Expected population after eradication of the disease

The disease will be completely eradicated when there are no more Latent and infectious individuals. This implies that L(t) = I(t) = 0. In this case, the total population is

P = S + R.

Hence the model equations will be reduced to

$$\frac{dS}{dt} = \rho - \mu S$$

$$\frac{dR}{dt} = -\mu R$$

Solving these equations gives

$$S(t)\ \frac{\rho}{\mu} + \left(S_0 - \frac{\rho}{\mu}\right)e^{-\mu t} \text{ and}$$

$$R(t) = R_0 e^{-\mu t}$$

Where S_0 and R_0 are the initial number susceptible and recovered individuals respectively.

$S(t) \approx \frac{\rho}{\mu}$, where $\frac{\rho}{\mu}$ is the asymptotic carrying capacity of the total population.

Also $R(t) \to 0$ whenever $t \to \infty$ hence the whole population will be comprising of only susceptible individuals.

3.4 Data Analysis and Numerical Simulations.

Nigeria still have problem of adequate record keeping, this however made it impossible to obtained the required real-life data to process the model, hence hypothetical values were used for the parameters in the model to have insight into the dynamics of the model as in (Koriko and Yusuf, 2008; Adetunde, 2009 and Sirajo, 2009).

The system equations was solved numerically using Euler's Numerical method and using Maple mathematical package, we obtained numerical simulations for the model using the following values for the parameters and the results displayed in graphical forms.

ρ :Recruitment rate =1,500 per year, (Aparicio *et. al.*, 2000)

μ : Natural mortality rate = .0222,

δ : Tuberculosis induced death = 0.365, (Snider *et. al.*, 1994).

α - Susceptible contracting rate. = 2 , (Song *et. al.*, 2002).

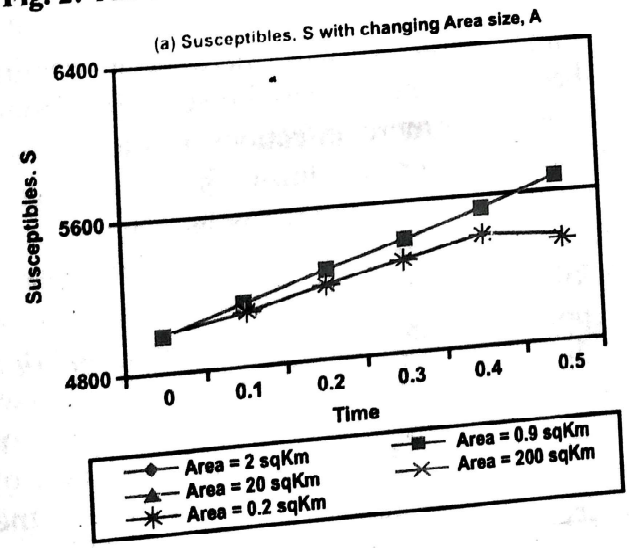
β - Recovered individuals' contracting rate.= 2, (Song *et. al.*, 2002).

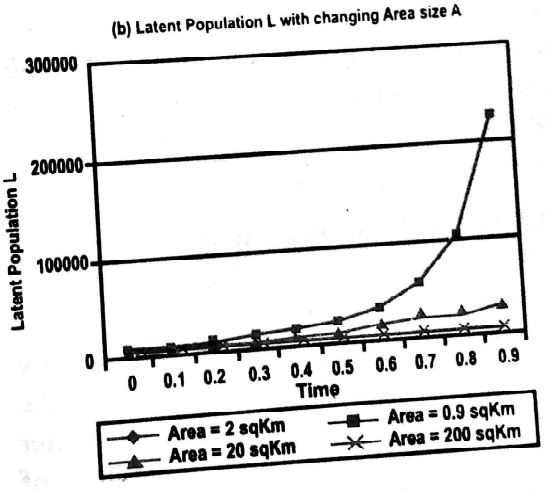
γ - Progression rate to active Tuberculosis = 0.00396, (Aparicio *et. al.*, 2000).

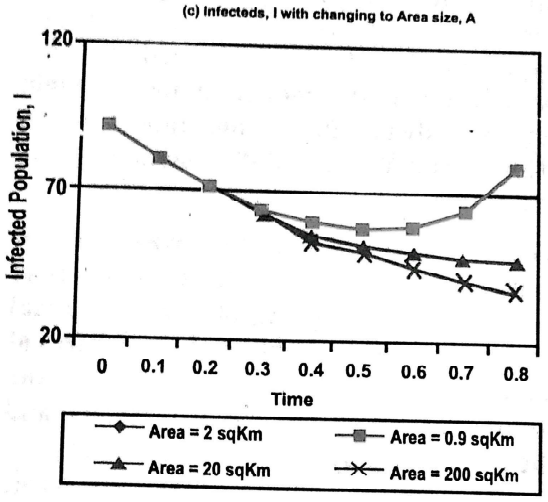
τ - Latent recovery rate= 1.5, (Styblo,1991).

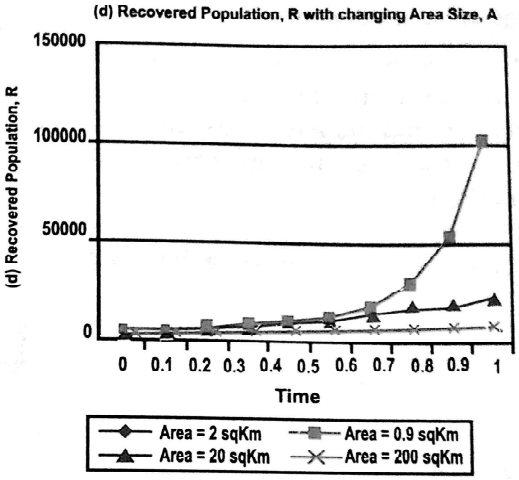
ε - Infectious recovery rate= 1.5, (Styblo,1991).

Fig. 2: The effect of size of the area occupied, *A* on the sizes of the different epidemiological classes









4. RESULTS AND DISCUSSION

In this research, the effect of population density on the dynamics of tuberculosis is discussed. It should be noted that population density determines the level of respiratory contact in a community and this level directly determines the infection rate of airborne diseases like tuberculosis. Overcrowding is most in urban areas and now settlement camps such as internally displaced peoples' camps and refugee camps. In order to carry out the investigations, a mathematical model was formulated that incorporated the size of the area occupied by the population being studied and areas with very high population densities were chosen for estimation of parameters. The results suggested that the observed trend in tuberculosis incidence was partly as a result of explosive population growth due to urbanization and resettlements (for the case of Internally Displaced People's Camps).

In the qualitative analysis of the model, the existence of steady states and their stabilities were analyzed. The analysis showed that a disease-free equilibrium point existed and was found to be globally asymptotically stable provided the characteristic area per individual is greater than the product of the probability of survival from latent stage into the infectious stage and the number of latent infections produced by a typical infectious individual during the mean infectious period. Based on the results of the findings in this study, if tuberculosis is to be eradicated, it is recommended that the characteristic area per individual to be 0.25 square kilometres.

The effect of size of the area occupied, A on the sizes of the different epidemiological classes is studied using A = 0.2 square kilometres, 0.9 square kilometres, 2 square kilometres, 20 square kilometres, 200 square kilometres. We observe in Fig. 2a that when the size of the area occupied, A is reduced from 2 square kilometres to 0.2 square kilometres thereby increasing the population density, the population will first increase because of the recruitment rate, ρ through birth and immigration. It will there after decline because of the increased disease incidence. There are more infections resulting from the close contact due to high population density. When the area is increased to say 20 square kilometres or 200 square kilometres, there is a slight deviation in the population sizes in the two cases, implying that there is a threshold area size. However, in both cases, member of susceptible class will increase because of the reduced disease incidence and recruitment.

Figure 2b shows that for a smaller area size, $A = 0.9$ square kilometres hence higher population density, the increase in population size of the Latently infected individuals shall be faster than in all the other cases. This is as a result of increased infection rate due to a higher contact rate of the susceptible class with the infectious individuals. We further observe that with lower population density (when the area is bigger say, $A = 20$ square kilometres or 200 square kilometres), the number of Latent individuals increases, though slowly.

It is observed in Fig. 2c that irrespective of the area size, the number of Infectious individuals declines in a small time interval, though at different rates. After a while, we observe that the population increases in all the cases, with a higher and faster increase when the population density is higher. It is further clearly observed that, at some point when for other area sizes the number of infected individuals is declining, it is on the rise when the area is so small. This is because in a congested community, there is a higher rate of Infection and hence a big number of susceptible individuals becomes infected and progress to Infectious stage.

A delay in the increase of the population size of Recovered individuals is observed in Fig. 2d when the area is bigger ($A = 200$ square kilometres and 20 square kilometres). However, an increase is observed in all the cases. These increments occur in the period when the number of infected individuals is declining as observed in the first phases.

The results of numerical simulation where remarkably in line with those of the qualitative analysis of the model in that, they both emphasized the dependence of tuberculosis incidence on the level population density in the community hence the area occupied by the population under study. Increments in the magnitude of the recruitment rate were observed to come hand in hand with an increased disease incidence and this is because of the increase in the level of contact due to a higher population density.

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