

Spatiotemporal Dynamics of Tuberculosis Disease and Vaccination Impact in North Senatorial Zone Taraba State Nigeria

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Abstract: In this work, The researchers present Passively Immune Infant (V_m) -Susceptible class (S_m) - Infection class (I_m) -Recovery class (R_m) model to study the dynamic of tuberculosis transmission and vaccination impact in North Senatorial Zone, Taraba State, Nigeria. The compartment of the model is presented in a system of ordinary differential equations. Quantitative analysis of the model was done to investigate the equilibrium and stability of the model. An analytical approach was used to determine their Disease Free equilibrium and the Epidemic equilibrium state. The stability of the epidemic equilibrium is tested using Bellman and Cooke's theorem.

The model had two equilibrium position: The disease free equilibrium which was asymptotically stable for $R_e(\lambda) < 0$ and the endemic equilibrium which was locally asymptotically stable for as it satisfies the Bellman and Cooke's condition for stability i.e. $|J| > 0$.

Key words: Tuberculosis (TB), Vaccination, Infection, Equilibrium analysis, Stability analysis.

I. Introduction

Tuberculosis is mostly transmitted through air by infected persons coughing, sneezing or spitting with pulmonary tuberculosis. The probability of transmission per contact, per relevant unit time is general quite low. The bacteria may lodge in the person's lung and multiply. If the immune system in the lung is able to fight the bacteria and render it inactive (wall off) as a result of vaccination or treatment from TB, the person will develop latent TB which is not infectious and cannot harm others.

Infected individuals may remain asymptomatic over their entire live (Latent TB). Active TB (the clinical disease) can develop into pulmonary and extra pulmonary form. Extra pulmonary TB is common in children while pulmonary TB is frequent in adult. Mycobacterium tuberculosis, the casual agent of the disease is transmitted almost exclusively via pulmonary cases. Cases arising within Five (5) years from first infection are classified as primary tuberculosis while cases arising after five (5) years from first infection are known as secondary tuberculosis. The asymptomatic individual may remain at latent for a long period of time due to vaccine durations and the antibody defense deposit in the body unless cured by treatment with antibiotics such as *Ethambutol*, *Rifampicine*, *B6* e.t.c. if the bacteria later become active due to breakdown of the immune system, the person is symptomatic and infectious and can be cured (recovered) if treated otherwise die from the infection.

The person when cured becomes susceptible as he is likely to be re-infected on contact with an infected person. Recent research efforts have been geared towards studying the heterogeneous factors in the population with a view to incorporate them into the model. The efforts that have been made by researchers and their findings on various aspects of TB disease dynamics falls under the following subheadings.

- i. Transmission
- ii. Vaccination against tuberculosis
- iii. Protective efficacy of the vaccine and duration of immunity
- iv. Infection
- v. Treatment
- vi. Prevention

Most early transmission models were deterministic but recent resurgence of tuberculosis (TB) in develop countries and increase in cases of casualty contact and public transport suggested a model based on thorough understanding of the dynamics of the disease.

Castillo-Chavez and Feng (1998) also consider an age-structured model, in this case with age-dependent transmission rates. They include one form of latency, as in their earlier work, and this model does not

contain re-infection. They study vaccination, and define a vaccine-dependent R_0 threshold $R(\psi)$. They prove stability of the disease-free equilibrium when $R(\psi) < 1$, and the existence of an endemic steady state when $R(\psi) > 1$, and discuss analytically-determined optimal vaccination strategies. These turn out to be either vaccination at a single age, or vaccination in precisely two age classes.

Aparacio et al (2000) develop a generalized household model, which took close and casual contact into account. The house cluster comprises of social networks (Family member, office mates, classmates, any person who have prolong contact with an active case). The basic reproductive number for the model is

$$R_o = \frac{\beta_n k}{\beta + \gamma + \mu + k}$$

Where β the transmission rate, n is the size of the cluster, μ natural mortality rate, γ the total per-capita removal rate from infected and k is the progression rate to active TB. BCG-induced immunity develops about six weeks after vaccination. Experimental studies indicate that the mechanism of protection by BCG vaccination consists in reduction of the hematogenous spread of bacilli from the site of primary infection Smith & Harding (1979) mediated by memory *T lymphocytes* induced by the first exposure to BCG. There is no evidence that BCG reduces the risk of becoming infected with tuberculosis bacilli, but it prevents forms of tuberculosis depending on hematogenous spread of the bacillus Heimbeck (1929). This inhibition of the hematogenous spread of bacilli thus reduces the risk of immediate disease and of disease due to reactivation. Because there is reduction in risk of immediate disease, but not of infection, there is a difference in the protective effect of BCG, depending on the type of tuberculosis infection. Myint *et al.* (1987).

The best method for determining the protective efficacy of a vaccine is a prospective, randomized, double-blind, placebo-controlled trial. These studies are difficult and expensive, and have rarely been performed. WHO has recently sponsored studies to evaluate the protective efficacy of BCG immunization in infants and/or children by two low-cost methods: case-control studies Smith (1982, 1987) and contact studies Ten Dam (1987).

These studies have recently been reviewed by Milstien and Gibson (1989), who concluded that there is good evidence that the efficacy of modern BCG vaccines is in the range of 60% to 90% for disseminated tuberculosis or meningitis in young children, but somewhat lower for other forms of primary tuberculosis disease. They also found no evidence that one BCG preparation tested was more efficacious than any other under the conditions of the trials, and thus no evidence to support the choice of one preparation or manufacturer of BCG over another on the basis of protective efficacy. A recent matched case-control study in Bangkok Sirinavin *et al.* (1991) found an adjusted protective efficacy for neonatal BCG vaccination of 83%, and provided evidence to support the thesis that the accuracy of tuberculosis diagnosis, the types of tuberculosis, the length of time after vaccination, and the household tuberculosis exposure contribute to variation in the reported protective efficacy of neonatal BCG vaccination. There is a need for the development of a single *in vitro* test capable of predicting the induction of immune resistance of humans to infection or dissemination of *M. tuberculosis*. Despite the difficulty in interpretation of data, several trials have shown that efficacy of BCG is highest among the youngest recipients, and that it falls with increasing age at vaccination Fine *et al.* Tuberculosis Prevention Trial (1979).

It is therefore difficult to assess the impact of BCG vaccination programmes. However, when BCG immunization of newborns was stopped in Sweden, the incidence of the disease in infants raised six fold Romanus (1987). There are several publications which suggest that the protection BCG provides against tuberculosis is a function of the relative importance of disease due to endogenous reactivation as compared to re infection from the outside. Thus, BCG protects against hematogenous spread of infection Fine (1988), Ten Dam (1984). This suggestion predicts the greater protective efficacy seen against miliary tuberculosis and tuberculous meningitis, compared with pulmonary tuberculosis Smith (1987).

II. Prevention and Control for Tuberculosis

Dye *et al* (1998) also have developed an age-structured model, with the goal of exploring TB control under the DOTS strategy and under improved case detection and cure. Their model uses discrete time rather than a partial differential equation framework. It includes two latent classes (slow and fast) with a set portion of new infections moving into each, as well as re-infection. They validate their model by comparing their results to annual risk of infection data from the Netherland. They then simulate different epidemiological situations. They conclude that if tuberculosis is stable, and if HIV is not included in the model, then WHO targets of 70% case detection and 85% cure “would reduce the incidence rate by 11% (range 8-12) per year and the death rate by 12% (9-13) per year”. The effect would be smaller if TB were already in decline. They conclude that DOTS has greater potential today (in developing countries) than it did 50 years ago in developed countries, but that case detection and cure rates must be improved.

Salomon *et al.* (2006) extend this model, calibrating it to natural TB epidemics and then studying the effects of hypothetical new treatments with differing durations. In their model, drug resistance could result from transmission and acquisition. They first calibrate the model to represent TB in South-East Asia. They conclude that if more rapid treatments for TB were available, with reduced infectious periods, this could significantly reduce TB mortality and incidence, particularly if these become available soon.

There are, of course, other alternatively-structured models. Salpeter and Salpeter (1998) used TB incidence data to derive a function for the time delay from initial infection to active disease, and develop an integral equation for R_0 . This is not a dynamic epidemiological model for TB, but it is relevant to TB models because the variable delays and widely ranging estimates of R_0 are important. They report R_0 near 1, which was associated with time scales of approximately 100 years.

III. Model Formulation

The model is going to be divided into four classes of population under study namely;

- i. The passively immune infant
- ii. Susceptible Class
- iii. Infected Class
- iv. Recovery Class

Based on this, the researchers divided the populations into four compartments represented as:

Model Diagram

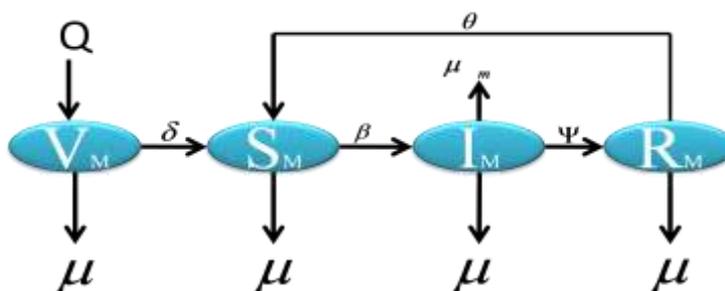


Figure 1: Schematic representation of VSIR Model

Individuals are recruited into V_m class via natural birth at the rate Q through passive vaccination, the population of the V_m decreases due to natural death at rate μ and movement of the individuals into S_m as a result of wearing out of vaccines used in passive vaccination at the rate δ . The population of the S_m increases due to coming of individuals from the V_m and R_m classes at the rates δ and θ . The population of the S_m class decreases due to the movement of individuals into the infected class at the rate β and natural death at a rate μ . The population of the I_m decreases due to treatment against TB at the rate ψ and natural death at the rate μ and death as result of TB infection at the rate μ_m . The population of the R_m increases due to movement of individuals at the rates ψ from I_m and decreases due to movement of individuals into the S_m at the rate θ and natural death at rate μ .

Definition of parameters

Q = Natural Birth rate

V_m = Passively immune infant at the time t

S_m = Susceptible class at the time t

I_m = Infected class at time t

R_m = Removed class at time t

μ = Natural death rate

δ = Rate of the duration of vaccine efficacy

β = TB contact rate

μ_m = Death as a result of TB infection

ψ = Rate of recovery from TB infection

θ = Rate at which individuals become susceptible

Model Equation

The model above is thus described by the following set of ordinary differential equations

$$\frac{dV_m}{dt} = Q - (\mu + \delta)V_m \tag{1}$$

$$\frac{dS_m}{dt} = \delta V_m - (\mu + \beta I_m)S_m + \theta S_m \tag{2}$$

$$\frac{dI_m}{dt} = \beta S_m I_m - (\mu + \mu_m + \psi)I_m \tag{3}$$

$$\frac{dR_m}{dt} = \psi I_m - (\mu + \theta)R_m \tag{4}$$

IV. Equilibrium State of the Model

Let $V_m = X_1, S_m = X_2, I_m = X_3, R_m = X_4$

Equations 1-4 becomes,

$$\frac{dX_1}{dt} = Q - (\mu + \delta)X_1 \tag{5}$$

$$\frac{dX_2}{dt} = \delta X_1 - (\mu + \beta X_3 - \theta)X_2 \tag{6}$$

$$\frac{dX_3}{dt} = \beta X_2 X_3 - (\mu + \mu_m + \psi)X_3 \tag{7}$$

$$\frac{dX_4}{dt} = \psi X_3 - (\mu + \theta)X_4 \tag{8}$$

Disease Free Equilibrium (DFE)

From (5)

$$\frac{dX_1}{dt} = Q - (\mu + \delta)X_1$$

$$\Rightarrow Q - (\mu + \delta)X_1 = 0$$

$$\therefore X_1 = \frac{Q}{(\mu + \delta)} \tag{9}$$

From (6)

$$\Rightarrow \frac{dX_2}{dt} = \delta X_1 - (\mu + \beta X_3 - \theta)X_2$$

$$\Rightarrow \delta X_1 - (\mu + \beta X_3 - \theta)X_2 = 0$$

$$\therefore X_2 = \frac{\delta X_1}{(\mu + \beta X_3 - \theta)} \tag{10}$$

Substituting (9) into (10) we have,

$$\Rightarrow X_2 = \frac{\delta \left(\frac{Q}{(\mu + \delta)} \right)}{(\mu + \beta X_3 - \theta)}$$

$$\therefore X_2 = \frac{\delta Q}{(\mu + \delta)(\mu + \beta X_3 - \theta)}$$

$$X_2 = \frac{\delta Q}{(\mu + \delta)(\mu - \theta)} \tag{11}$$

From (7)

$$\Rightarrow \frac{dX_3}{dt} = \beta X_2 X_3 - (\mu + \mu_m + \psi) X_3$$

$$\Rightarrow \beta X_2 X_3 - (\mu + \mu_m + \psi) X_3 = 0$$

$$[\beta X_2 - (\mu + \mu_m + \psi)] X_3 = 0$$

Therefore, either,

$$X_3 = 0 \tag{12}$$

OR

$$\Rightarrow \beta X_2 - (\mu + \mu_m + \psi) = 0$$

$$X_2 = \frac{(\mu + \mu_m + \psi)}{\beta} \tag{13}$$

Substituting (12) into (10) gives,

$$\therefore X_2 = \frac{\delta Q}{(\mu + \delta)(\mu - \theta)} \tag{14}$$

Substituting (12) into (8) gives,

$$X_4 = 0$$

Hence, the disease free equilibrium (DFE) states are as follows

$$(X_1, X_2, X_3, X_4) = \left(\frac{Q}{(\mu + \delta)}, \frac{\delta Q}{(\mu + \delta)(\mu - \theta)}, 0, 0 \right)$$

Epidemic Equilibrium State (EE)

Substituting (8) into (11)

$$\therefore X_3 = \frac{\delta Q}{(\mu + \delta)(\mu + \mu_m + \psi)} - \frac{(\mu - \theta)}{\beta} \tag{15}$$

Recall from (8)

$$\Rightarrow \psi X_3 - (\mu + \theta) X_4 = 0$$

$$X_4 = \frac{\psi X_3}{(\mu + \theta)} \tag{16}$$

Substituting (13) into (14)

$$X_4 = \frac{\psi \delta Q}{(\mu + \delta)(\mu + \theta)(\mu + \mu_m + \psi)} - \frac{\psi(\mu + \theta)}{\beta(\mu + \theta)}$$

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Hence, the Epidemic Equilibrium (EE) states are as follows:

$$X_1 = \frac{Q}{(\mu + \delta)}$$

$$X_2 = \frac{(\mu + \mu_m + \psi)}{\beta}$$

$$\therefore X_3 = \frac{\delta Q}{(\mu + \delta)(\mu + \mu_m + \psi)} - \frac{(\mu - \theta)}{\beta}$$

$$X_4 = \frac{\psi \delta Q}{(\mu + \delta)(\mu + \theta)(\mu + \mu_m + \psi)} - \frac{\psi(\mu + \theta)}{\beta(\mu + \theta)}$$

IV. Stability Analysis

$$|J| = \begin{bmatrix} -(\mu + \delta) & 0 & 0 & 0 \\ \delta & -(\mu + \beta X_3 - \theta) & -\beta & 0 \\ 0 & \beta X_3 & \beta X_2 - (\mu + \mu_m + \psi) & 0 \\ 0 & 0 & \psi & -(\mu + \theta) \end{bmatrix}$$

$$-(\mu + \delta) - \lambda \left(-(\mu + \beta X_3 - \theta) - \lambda \begin{bmatrix} \beta X_2 - (\mu + \mu_m + \psi) - \lambda & 0 \\ \psi & -(\mu + \theta) - \lambda \end{bmatrix} + \beta \begin{bmatrix} \beta X_3 & 0 \\ 0 & -(\mu + \theta) - \lambda \end{bmatrix} \right) = 0$$

$$-(\mu + \delta) - \lambda (-(\mu + \beta X_3 - \theta) - \lambda) (\beta X_2 - (\mu + \mu_m + \psi) - \lambda) (-(\mu + \theta) - \lambda) + [\beta(-\beta X_3(\mu + \theta) - \lambda)] = 0$$

$$-(\mu + \delta) - \lambda (-(\mu + \beta X_3 - \theta) - \lambda) [(\beta X_2 - (\mu + \mu_m + \psi) - \lambda) + \beta^2 X_3] (-(\mu + \theta) - \lambda) = 0$$

Disease Free Stability Analysis

The characteristic equation above takes the form:

$$-(\mu + \delta) - \lambda (-(\mu + \beta X_3 - \theta) - \lambda) [(\beta X_2 - (\mu + \mu_m + \psi) - \lambda) + \beta^2 X_3] (-(\mu + \theta) - \lambda) = 0 \quad (18)$$

⇒ Either,

$$\therefore \lambda_1 = -(\mu + \delta) \quad (19)$$

OR

$$\therefore \lambda_2 = -(\mu - \theta) \quad (20)$$

OR

$$\lambda_4 = \beta X_2 - (\mu + \mu_m + \psi)$$

$$\lambda_4 = \frac{\beta \delta Q}{(\mu + \delta)(\mu - \theta)} - (\mu + \mu_m + \psi) \quad (21)$$

$$\lambda_4 = \frac{\beta \delta Q}{(\mu + \delta)(\mu - \theta)} < (\mu + \mu_m + \psi)$$

This implies the for λ_4 to be asymptotically stable, $(\frac{\beta \delta Q}{(\mu + \delta)(\mu - \theta)}) < (\mu + \mu_m + \psi)$ where

$\frac{\beta \delta Q}{(\mu + \delta)(\mu - \theta)}$ is the number of susceptible individuals produced.

OR

$$\lambda_3 = -(\mu + \theta) \quad (22)$$

Since then $R_e(\lambda_1) < 0, R_e(\lambda_2) < 0, R_e(\lambda_3) < 0, R_e(\lambda_4) < 0$, then it is asymptotically stable.

Stability Analysis of the Epidemic Equilibrium

When applying Bellman and Cooke's (1963), the result of Bellman and Cooke's theorem state that, if $\Delta(iy)$, $y \in I$ is separated into real and imaginary parts $\Delta(iy) = F(y) + iG(y)$ and all zeros of $\Delta(z)$ have negative real parts then the zeros of $F(y)$ and $G(y)$ are real and alternative then:

$$F(0)G'(0) - F'(0)G(0) > 0 \tag{23}$$

(23) is applied to the characteristic equation (18). We consider (18) in the form

$$H(\lambda) = 0$$

And we obtain:

$$H(\lambda) = -(\mu + \delta) - \lambda(-(\mu + \beta X_3 - \theta) - \lambda) \left[(\beta X_2 - (\mu + \mu_m + \psi) - \lambda) + \beta^2 X_3 \right] - (\mu + \theta) - \lambda \tag{24}$$

Also, by applying Bellman and Cooke's theorem on above, setting $\lambda = iw$, we have $H(iw) = F(w) + iG(w)$

Where $F(w)$ and $iG(w)$ are real and imaginary part of $H(iw)$.

Substituting $\lambda = iw$ (24) to have

$$H(iw) = -(\mu + \delta) - iw(-(\mu + \beta X_3 - \theta) - iw) \left[(\beta X_2 - (\mu + \mu_m + \psi) - iw) + \beta^2 X_3 \right] - (\mu + \theta) - iw$$

$$\therefore F(w) = (\mu + \delta)(\mu + \beta X_3 - \theta) - (\mu + \theta) \left[\beta X_2 - (\mu + \mu_m + \psi) + \beta^2 X_3 \right] + 2w^2$$

$$G(w) = w \left[(\mu + \delta) + (\mu + \beta X_3 - \theta) + (\mu + \theta) - \beta X_2 + (\mu + \mu_m + \psi) - \beta^2 X_3 \right]$$

$$F'(w) = 4w$$

$$G'(w) = (\mu + \delta) + (\mu + \beta X_3 - \theta) + (\mu + \theta) - \beta X_2 + (\mu + \mu_m + \psi) - \beta^2 X_3$$

Setting $w = 0$

$$F(0) = (\mu + \delta)(\mu + \beta X_3 - \theta) - (\mu + \theta) \left[\beta X_2 - (\mu + \mu_m + \psi) + \beta^2 X_3 \right]$$

Substituting the values of X_2 and X_3 into $F(0) = 0$ we have,

$$F(0) = 2\mu^2 - (2\mu + \delta - \mu_m - \psi)\theta + \mu\mu_m + \mu\psi + \frac{\delta Q \beta (\mu + \delta - \beta(\mu + \theta))}{(\mu + \mu_m + \psi)(\mu + \delta)} - (\mu - \theta) [(\mu + \delta) + \beta(\mu + \theta)] - (\mu + \theta)(\mu + \mu_m + \psi) \tag{25}$$

$$F'(0) = 0 \tag{26}$$

$$G'(0) = (\mu + \delta) + (\mu + \beta X_3 - \theta) + (\mu + \theta) - \beta X_2 + (\mu + \mu_m + \psi) - \beta^2 X_3$$

Substituting the values of X_2 and X_3 into $G(0) = 0$ we have,

$$G'(0) = 3\mu + \delta + \psi + \mu_m - (1 - \beta)(\mu - \theta) + \frac{(\beta - \beta^2)\delta Q}{(\mu + \delta)(\mu + \mu_m + \psi)} \tag{27}$$

$$G(0) = 0 \tag{28}$$

We have from Bellman and Cooke's theorem (1963) that:

The condition $R_e(\lambda) < 0$; is given by the inequality:

$$F(0)G'(0) - F'(0)G(0) > 0 \tag{29}$$

The equation (29) is the stability condition for the Epidemic Equilibrium. But from (26) and (27) we have

$$F'(0) = 0 \text{ and } G(0) = 0$$

Hence, equation (29) becomes

$$F(0)G'(0) > 0$$

Let $|J| = F(0)G'(0)$ (30)

Then the Epidemic equilibrium state is stable when $J > 0$.

V. Results and Discussion

Data collected from Tuberculosis and Leprosy unit, Ministry of Health, Taraba state, Nigeria, was used to obtain the simulation for the model.

Using hypothetical values pertinent to Taraba State, the researchers obtain the following results using Maple software as shown below:

Q	β	θ	μ	Ψ	δ	μ_m	J	REMARKS
0.03923	0.1	0.5	0.01433	1.0	0.02	0.15	0.0699087	STABLE
0.03923	0.2	0.5	0.01433	0.9	0.02	0.15	0.0688880	STABLE
0.03923	0.3	0.5	0.01433	0.8	0.02	0.15	0.0666962	STABLE
0.03923	0.4	0.5	0.01433	0.7	0.02	0.15	0.0633377	STABLE
0.03923	0.5	0.5	0.01433	0.6	0.02	0.15	0.0587936	STABLE
0.03923	0.6	0.5	0.01433	0.5	0.02	0.15	0.0530335	STABLE
0.03923	0.7	0.5	0.01433	0.4	0.02	0.15	0.0460057	STABLE
0.03923	0.8	0.5	0.01433	0.3	0.02	0.15	0.0376138	STABLE
0.03923	0.9	0.5	0.01433	0.2	0.02	0.15	0.0143337	STABLE
0.03923	1.0	0.5	0.01433	0.1	0.02	0.15	0.0156232	STABLE
0.03923	1.1	0.5	0.01433	0.0	0.02	0.15	-0.0002277	UNSTABLE

Figure 2: Result of stability analysis of Epidemic Equilibrium State.

Simulations

The first example is concern with the estimation properties of the VSIR model given by equation (5) – (8) in the presence of low contraction, while the second example is concern with the estimation properties of the VSIR model given by equation (5) – (8) in the presence of high contraction. The VSIR model is described by the following parameters taken from TB outbreak in North Senatorial zone of Taraba state. $Q= 0.03923$, $\beta =0.1$ & 0.01 , $\mu =0.01433$, $\mu_m =0.15$, $\theta=0.5$, $\delta = 0.02$, $\psi =0.23$. The initial conditions are given by $V(0) =419,359$, $S(0)=838,712$, $I(0)=536$, $R(0)=427$.

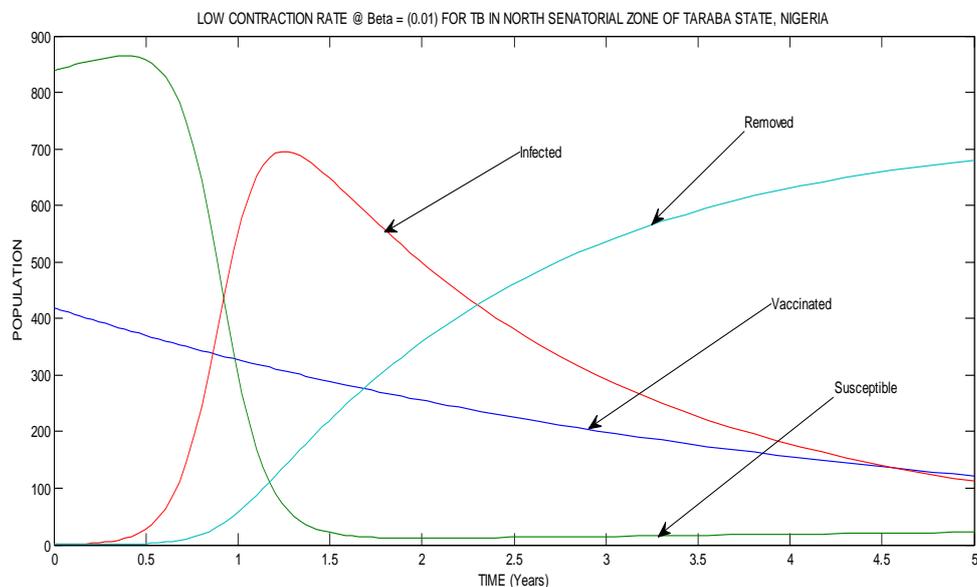


Figure 3: Graphical profile showing low contraction rate

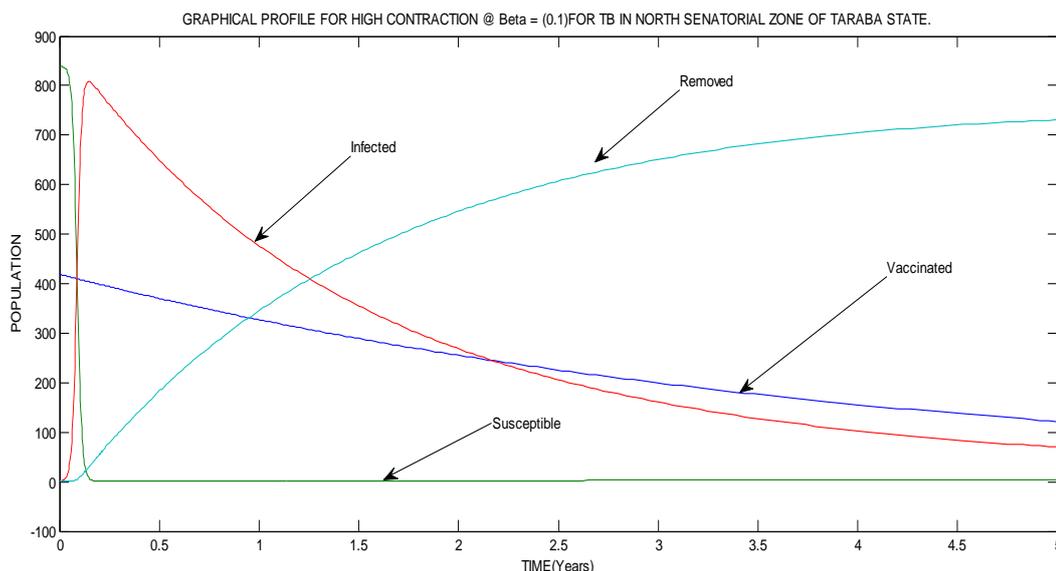


Figure 4: Graphical profile showing high contraction rate

Figure 3 shows the graphical profile of the VSIR model for $\beta = 0.01$ (low contraction rate). It was observed that the population of the infected class increases steadily until when $T = 1.3$, then it decreases steadily. While figure 4 shows the graphical profile of the VSIR model for $\beta = 0.1$ (high contraction rate). It was observed that the population of the infected class increase exponentially until $T = 0.25$ then, decreases steadily.

VI. Conclusion

Conclusively, we will like to conclude that, Progression to active TB among the population is epidemiologically significant and interventions should focus on both vaccinations and treatment of infected persons, therefore, effort should be made to minimize the contraction rate for the sustenance of North Senatorial Zone of Taraba state. Anti-tuberculosis, treatment of adults is crucial in controlling the epidemic and intervention measures such as frequent vaccination and adhering to TB treatment should be proposed and implemented, they should target progression to active TB for those infected.

Recommendations:

Tuberculosis is an airborne disease and therefore prevention and control of tuberculosis could be achieved if the following measures are adopted:

1. Ensure good, adequate, hygiene measures of health are put in place.
2. Preventive vaccines such as Becillus Calmette Guerin (BCG) should be administered to particularly children and pregnant mothers.
3. All health care centres involve in TB treatment should adopt the Direct Observation Treatment short course (DOTS) strategy for treatment of patients.
4. Government should create awareness and sensitize the public on transmission and spread of TB.
5. Prevent or control exposure to infected animals and infected persons.
6. Government should embark on contact tracing for newly infected individuals, so that they can be placed on TB treatment therapy immediately.

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