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Mathematical Modeling of the Transmission Dynamics of Tuberculosis and its Control. (A case study of Ika General Hospital, Ankpa, Kogi State).

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Abstract: *In this research work, we formulated a mathematical model for the transmission dynamics of tuberculosis, a case study of Ika Christian Hospital, Ankpa L.G.A, Kogi State, Nigeria. The model which adopts a standard incidence formulation incorporates treatment and vaccination as control strategies.The Disease Free Equilibrium (DFE) statewas determined, which was shown to be locally asymptotically stable. The basic reproduction number of the model was determined using the next generation matrix approach. The Endemic Equilibrium (EE) state of the model wasalsoestablished and proved to be locally asymptotically stable using the trace and determinant method. The numerical solution of the basic reproduction number of the model shows thatthe disease tuberculosis, will be reduced or eliminated with time from the population as the value was less than one (1). Simulations of the model using the data we obtained from Ika Christian Hospital shows that the disease will be eradicated from the population with time by using vaccination and treatment as control intervention strategies.*

Keywords*: Tuberculosis, model, heterogeneous, transmission.*
AMS (2010)*: Subject Classification: 93A30*

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I. Introduction

Tuberculosis [TB] is an infectious disease which is caused by mycobacterium tuberculosis bacteria [1].It affects the lungs and virtually other parts of human body, it can also affect any age group.

Tuberculosis is an airborne disease that can be transmitted via the respiratory route. Most infections do not have symptoms in which case is called latent tuberculosis [1].Some proportion of the latent tuberculosis infections progress to active tuberculosis cases which is deadly if not treated. The symptoms includes chronic cough with blood containing sputum fever, night sweet and weight loss [1].When people who have active tuberculosis cough, spit, speak, sing or sneeze they propel and expel tuberculosis germs [2] which can infect human in contact. HIV / AIDS Patient and those that smoke are always at risk in contacting tuberculosis [1].

Diagnosis of active tuberculosis is based on chest x -ray as well as microscopic examination and culture of bodily fluid [3].Diagnosis can be done at most 2 hours and the test is presently recommended by the World Health Organization as the first diagnostic test in all person with the symptoms of tuberculosis [3].

The diseases tuberculosis can be prevented by screening of those at high risk, early detection of cases treatment and vaccination with Bacillus Calmelte – Guerin (BCG) vaccine,[4] [5], [6].

The group of people mostly affected with TB are medical health workers, social gathering, those attending to TB patient, transits with active TB patient.

Treatment of tuberculosis infection involves the use of multiple antibiotics over a prolong period of time (1). Antibiotics resistance is a growing problem with increasing rates of multiple drug resistant tuberculosis (MDR-TB) and extensively drug – resistant tuberculosis (XDR- TB) [1].

In treating tuberculosis (TB), it is important that patient are provided with adequate support, information and supervision by a trained or qualified health personnelon the need to take treatment serious without such support treatment adherence may be difficult. Most of tuberculosis cases can be cured when the drugs are given and taken properly.

Nidhi et al [7] proposed a mathematical model to study the dynamics of tuberculosis by. It was assumed that the rate at which the number of latently infected individual moves to recovery class (R) and again from recovery class to latent class is not equal. The possibility of existence of endemic equilibrium state was discussed and examined.

In 2013, Ibrahim et al [8] presented a mathematical model for the epidemiology of tuberculosis with estimate of the basic reproduction number. The basic reproduction number was determined and the disease free equilibrium state of the model was shown to be globally asymptotically stable if $Ro \leq 1$.Simulation of the model showed that a continuous vaccination would result in a more stable disease free equilibrium state.

Furthermore, Koriko & Yusuf [9] developed a model based on the SIRS model, the model equations were presented graphically, and simulation analysis result showed that the population dynamics for tuberculosis depends on the number of actively infected people in the population at the initial time. The authors showed that the disease free equilibrium was stabled why the endemic equilibrium state may not be stable depending on the various values of the model parameters.

The aim of this present research work is to develop a mathematical model of the transmission of dynamic of tuberculosis and its control in a heterogeneous population, a case study of Ika Christian Hospital, in Akpa Local Government of Kogi State, Nigeria.

The specific objectives of the work are as follows:

(i) To developa mathematical model for the transmission dynamics of tuberculosis.

(ii) To incorporate control strategies that can help eradicating the pandemic disease.

(iii) To conduct sensitivity analysis of the model to know the parameters to be targeted by the medical personnels.

(iv) To come up with recommendations that can help in controlling disease.

This study is significant as humanity will always entertain any contribution for the prevention, cure and even eradication of the disease in human history. The knowledge will be welcomed in the medical world, policy maker will also use the acquired knowledge from this work on the need for tuberculosis vaccine, its treatment and the isolation of those with tuberculosis cases for the control of the disease, tuberculosis. The research work will also add to the existing or current literatures in tuberculosis.

(1.1) BRIEF HISTORY OF IKA CHRISTIAN HOSPITAL

Ika Christian Hospital is a private owned hospital located in Ankpa Local Government Area of Kogi State, Nigeria, It was established in 1961 by a Canadian missionary, Raymon Dibble, but the facility is currently managed by his granddaughter, Mrs. Lois Wheeler, a nurse, after the demise of her father, Spencer Dibble who was a bible translator. Raymond acquired the land for the hospital in 1952 but had been in Nigeria as a Christian missionary since 1952. The hospital recruits and trains her own personnel in different medical fields to serve her purpose.

The motivation for the establishment of the hospital is for physical and spiritual healing. The aim is to win souls by treating their illnesses through preaching the gospel of Jesus Christ. The hospital is known for treatment of tuberculosis cases. Patient comes from all parts of the country as -information of her efficiency in handling the disease spreads.

II. Model Assumptions

(1) The population is assumed to be homogeneous

(2) We assumed that the population of the Susceptible class is been recruited by birth and emigration

(3)We assumed that all age group can be infected with tuberculosis

(4) We assumed that a proportion of the susceptible class are vaccinated

(5) We assumed that some of the susceptible class are illiterate and may not want to go for vaccination because of myths

(6) That the infected class also present themselves for treatment

(7) Asymptomatic class, that is, suspected cases are quarantined.

(8) Symptomatic class, that is, those who have developed clinical symptoms are isolated

(9) Some infected people do not want to rely on orthodox medicine

(10) We assume that all compartments may die naturally

(11) That there is disease induced death in the infected classes

2.1 MODEL FORMULATION & DESCRIPTION

We formulated our model based on the standard SEIR model where the population was divided into nine (9) compartments comprises of the Susceptible class(S), Exposed class(E), Vaccinated class(V), Infected class(I),Infected but treated class (I_T), Infected but not treated class (I_N),Quarantined class(Q), Isolated class(J) and the Removed class(R). A deterministic model of the form $SEVII_T I_N QJR$ was formulated based on the stated assumptions.

The Susceptible class (S) was recruited by birth and by emigration (undetected entry of individuals into the community) at the levels of (θ) and (λ) respectively. The class increases by the incoming of the recovered

individuals into the population at the rate of (ω) and also by the rate at which the vaccinated becomes susceptible again due to vaccine failure at the rate of $(1-\gamma)$. It reduces at the rate at which people are exposed at the level of (ψ) and also by the rate at which some proportion of the population are vaccinated at the level of (β).The class finally reduces by natural death at the rate of (μ).

The Exposed class (E) increases due to the incoming of the susceptible individuals that have exposed themselves to tuberculosis but have not yet developed clinical symptoms at the rate of (ψ).The population reduces due to the rate at which the exposed becomes infected with active tuberculosis and moved to the infected class at the rate of (ϕ) and also by the rate at which they are quarantined at the rate of (η), The class finally reduces naturally by death at the rate of (μ).

The Quarantined class (Q) , the class is generated at the rate at which the exposed are quarantined at the level of (η), we assumed that all quarantined individuals are asymptomatically infective who will go on to develop symptoms and then moved to the isolated class at the rate of (σ_1). The class reduces naturally by death at the rate of (μ).

The vaccinated class is recruited with the proportion of those vaccinated from the Susceptible class at the level of (β). The class reduces by the rate at which some individuals who are vaccinated becomes susceptible again due to vaccine failure at the rate of $(1-\gamma)$and also by the rate at which some vaccinated cases recover due to the vaccine efficacy at the rate of (γ).The class finally reduces by natural death at the rate of (μ).

The Isolated class (J) is recruited by the incoming of those infected with active TB at the level of (σ_2) and also by the incoming of those that have been quarantined and now developed symptoms and then moved to the isolated class at the rate of (σ_1). The class reduces by disease induced death and by natural death at the rates of $(\delta \text{ and } \mu)$respectively.

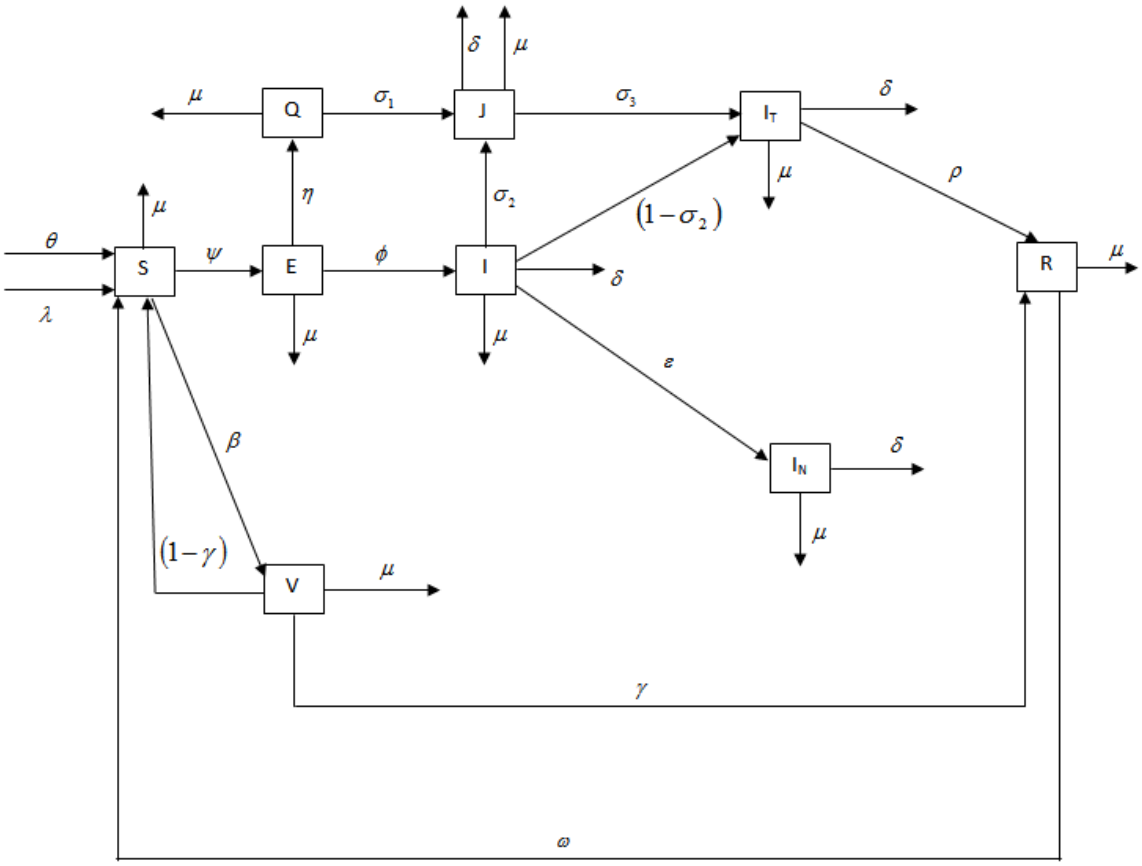
The Infected class (I) increases only through the incoming of the exposed individuals at the rate of (ϕ) .The population reduces due to the isolation of some proportion of the infected individuals at the rate of (σ_2) .The class was reduced by the rate at which a proportion of the infected are treated at the rate of $(1-\sigma_2)$ and moved to the Infected but treated class, some infected individuals who refused to go for treatment also reduces the population of the infected class as they moved to the Infected but non -treated class at the rate of (ε). The class reduces due to death caused by the disease at the rate of (δ) and naturally at the rate of (μ).

The infected but treated class (I_T) population increases by the incoming of the Isolated class who are taken for treatment at the rate of (σ_3) and also by the incoming of the infected population that accept to go for treatment at the rate of $(1-\sigma_2)$. The class reduces at the rate by which some recovered due to treatment and moved to the Recovered class at the rate of (ρ). The population further reduces by disease induced death rate at the rate of (δ) and natural death at the rate of (μ).

The Infected but non-treated class (I_N) population increases due to the incoming of the infected individuals who refused to go for treatment at the rate of (ε) but reduces due to disease induced death rate at the rate of (δ) and natural death at the rate of (μ).

The Removed class (R) increases by the incoming of those that responded to treatment and therefore recover at the rate of (ρ). The class also increases due to the incoming of those vaccinated against the disease and become free due to the effectiveness of the vaccine and therefore moved to the Recovered class at the rate of (γ). The population finally decreases due to natural death at the rate of (μ).

2.2 MODEL FLOW DIAGRAM



$$Where\ \psi = \frac{S(\alpha_1 I + \zeta_1\alpha_2 I_T + \zeta_2\alpha_3 I_N + \zeta_3\alpha_4 E + \zeta_4\alpha_5 Q + \zeta_5\alpha_6 J)}{N}$$

Figure 1.0 Model flow diagram

2.3MATHEMATICAL MODEL

$$\frac{dS}{dt} = (\theta + \lambda) + \omega R + (1-\gamma)V - (\psi + \beta + \mu)S \quad \ldots\ldots(2.1)$$

$$\frac{dE}{dt} = \psi S - (\eta + \phi + \mu)E \quad \ldots\ldots(2.2)$$

$$\frac{dV}{dt} = \beta S - [(1-\gamma) + \mu]V \quad \ldots\ldots(2.3)$$

$$\frac{dI}{dt} = \phi E - [\sigma_2 + (1-\sigma_2) + \varepsilon + \delta]I \quad \ldots\ldots(2.4)$$

$$\frac{dI_T}{dt} = (1-\sigma_2)I + \sigma_3 J - (\delta + \rho + \mu)I_T \quad \ldots\ldots(2.5)$$

$$\frac{dI_N}{dt} = \varepsilon I - (\delta + \mu)I_N \quad \ldots\ldots(2.6)$$

$$\frac{dQ}{dt} = \eta E - (\sigma_1 + \mu)Q \quad \ldots\ldots(2.7)$$

$$\frac{dJ}{dt} = \sigma_2 I + \sigma_1 Q - (\sigma_3 + \delta + \mu)J \quad \ldots\ldots(2.8)$$

$$\frac{dR}{dt} = \rho I_T + \gamma V - (\omega + \mu)R \quad \ldots\ldots(2.9)$$

$$Where\ \psi = \frac{S(\alpha_1 I + \zeta_1\alpha_2 I_T + \zeta_2\alpha_3 I_N + \zeta_3\alpha_4 E + \zeta_4\alpha_5 Q + \zeta_5\alpha_6 J)}{N}$$

2.4 MODEL VARIABLES AND PARAMETERS

Table 1: Variable used and descriptions

S/N	VARIABLES	DESCRIPTIONS
1	S	Susceptible class
2	E	Exposed class
3	I	Active tuberculosis infected class
4	V	Vaccinated class
5	Q	Quarantine class
6	J	The isolated class
7	I_T	Infected but treated class
8	I_N	Infected but non-treated class
9	R	Removed class

Table 2: Parameters used and descriptions

S/N	PARAMETERS	DESCRIPTION
1	θ	Recruitment rate through birth
2	λ	Recruitment rate through emigration
3	α	Contact rate of the susceptible and the infected
4	ϕ	Rate at which the Exposed class becomes infected
5	β	Rate at which the Susceptible are vaccinated
6	$(1-\gamma)$	Rate at which the Vaccinated becomes exposed due to vaccine failure
7	γ	Rate at which the Vaccinated recovers due to the vaccine efficiency
8	η	Rate at which the exposed are quarantined
9	σ_1	Rate at which the quarantined are isolated after showing symptoms of TB
10	σ_2	Rate at which the infected are isolated
11	σ_3	Rate at which the isolated are taken for treatment
12	$(1-\sigma_2)$	Rate at which the infected go for treatment
13	ρ	Rate at which the infected but treated recovers
14	ε	Rate at which the Infected refuses to go for treatment
15	μ	Natural death rate for all the classes
16	δ	Disease induced death rate for the infectious class
17	ω	Rate at which the recovered becomes susceptible again.
18	ψ	Force of infection
19	α_1	Contact rate between the Susceptible and the infected
20	α_2	Contact rate between the Susceptible and the infected but on treatment
21	α_3	Contact rate between the Susceptible and the infected but not on treatment
22	α_4	Contact rate between the Susceptible and the exposed
23	α_5	Contact rate between the Susceptible and the Quarantined class
24	α_6	Contact rate between the Susceptible and the Isolated class
25	ζ_1	Modification parameter for transmission between the Susceptible and Infected but on treatment class
26	ζ_2	Modification parameter for transmission between the Susceptible and the non-treated infected class
27	ζ_3	Modification parameter for transmission between the Susceptible and Exposed class
28	ζ_4	Modification parameter for transmission between the Susceptible and Quarantined class
29	ζ_5	Modification parameter for transmission between the Susceptible and Isolated class

III. Model Analysis

3.1INVARIANT REGION

Theorem 1:

The solutions of the model are feasible for all $t > 0$, if they enter the invariant region, $\Omega \in R^{9}_{+}$.

Proof:

We shall first show that all the feasible solutions are uniformly bounded in a proper subset of the region, $\Omega \in R^{9}_{+}$.

We let

$\Omega = \{S(t), E(t), V(t), I(t), I_T(t), I_N(t), Q(t), J(t), R(t),\}$ be any solution of the model given by

$\{S(t), E(t), V(t), I(t), I_T(t), I_N(t), Q(t), J(t), R(t),\}$

$V(0) > 0 \in R^{9}_{+}$

Where $\{S(0) > 0, E(0) > 0, V(0) > 0, I(0) > 0, I_T(0) > 0, I_N(0) > 0, Q(0) > 0, J(t) > 0, R(0) > 0,\}$ with non-negative initial conditions.

From the model we have that the total population will be

$N = S + E + V + I + I_T + I_N + Q + J + R$

$$\Rightarrow \frac{dN}{dt} = (\theta + \lambda) - \mu N - \delta(I + I_T + I_N + J) \quad \text{(2.10)}$$

At equilibrium $\frac{dN}{dt} = 0$ and also in the absence of the disease

$I = I_T = I_N = Q = 0$ it follows that $-\delta(I + I_T + I_N + J) > 0$ this implies that $\frac{dN}{dt} = (\theta + \lambda) - \mu N$

$$\therefore \frac{dN}{dt} + \mu N \leq (\theta + \lambda) \quad \text{(2.11)}$$

Using $I.F = \ell^{\int Pdt}$ and $Y.IF = \int QIFdt$

but $\frac{dy}{dt} + py = Q$

Where $y = N,\ p = \mu, Q = (\theta + \lambda)$

I.F= $\ell^{\int \mu dt}$

$I.F = \ell^{\mu t}$

$\therefore Y.IF = \int QIFdt$ becomes

$N.\ell^{\mu t} = \int [(\theta + \lambda).]\ell^{\mu t}$

$N.\ell^{\mu t} = [(\theta + \lambda)].\frac{\ell^{\mu t}}{\mu} + c$

Divide all by $\ell^{\mu t}$

$$\Rightarrow N_{(t)} = \frac{[\theta + \lambda]}{\mu} + c\ell^{-\mu t} \quad \text{.....(2.12)}$$

at $t = 0, S(o) = So$ and $N(o) = No$

$$N(o) = \frac{[\theta + \lambda]}{\mu} + c \qquad \therefore c = No - \frac{(\theta + \lambda)}{\mu}$$

$\therefore$ equation (2.12) becomes

$$N(t) = \frac{\theta + \lambda}{\mu} + \left[No - \frac{(\theta + \lambda)}{\mu} \right] \ell^{-\mu t} \quad \text{.....(2.13)}$$

Applying the inequality theorem by Birkhurf and Rota (1982) on differential equations yields the following results;

$$0 \le N(t) \le \frac{(\theta + \lambda)}{\mu} \quad \text{as } t \to \infty$$

The total population approaches $\frac{(\theta + \lambda)}{\mu}$ *as* $t \to \infty$

Where, $\frac{(\theta + \lambda)}{\mu}$ is the carrying capacity of the system.

$\therefore$ The feasible solution set of the systementers the region

$$\left\{ \begin{array}{l} \Omega = (S + E + V + I + I_T + \mathrm{I_N} + \mathrm{Q} + \mathrm{R}) \in \mathrm{R}_+^8 : S \ge 0,\ E \ge 0, V \ge 0, I \ge 0,\ I_T \ge 0, I_N \ge 0, Q \ge 0, J \ge 0, R \ge 0, \\ N = \frac{(\theta + \lambda)}{\mu} \end{array} \right\}$$

2.14**3.2 POSITIVITYOF SOLUTION**

Lemma1: We let the initial data be

$\{S(0) \geq 0, E(0) \geq 0, V(0) \geq 0, I(0) \geq 0, I_T(0) \geq 0, I_N(0) \geq 0, Q(0), J(0) \geq 0, R(0) \geq 0, \} \in R_+^9$

Then thesolution set

$\{S(t), E(t), V(t), I(t), I_T(t), I_N(t), Q(t), J(t), R(t)\}$ of the model is positive for all $t \geq 0$

Proof

Considering equation (2.1) we have the following equation

$$\frac{ds}{dt} = (\theta + \lambda) + \omega R + (1-\gamma)V - (\psi + \beta + \mu)s \geq -(\psi + \beta + \mu)s$$

$\Rightarrow$

$$\frac{ds}{dt} \geq -(\psi + \beta + \mu)s$$

$$\frac{ds}{s} \geq -(\psi + \beta + \mu)dt$$

integrating both sides now gives;

$\ln S(t) \geq -(\psi + \beta + \mu)t + c$

$S(t) \geq \ell^{-(\psi+\beta+\mu)t}.\ell^c$ *but let* $\ell^c = S(0)$

$S(t) \geq S(0)\ell^{-(\psi+\beta+\mu)t}$

where$c$ = constantof integration

Applying the initial condition at $t = 0$

$S(t) = S(0)$, it gives

$S(t) \geq S(0)\ell^{-(\psi+\beta+\mu)t} \geq 0$

Since

$\ell^{-(\psi+\beta+\mu)t} \geq 0 \text{ and } S(0) \geq 0$

$\therefore$ the solution is positive at all time $t \geq 0$

Now for equation (2.2)

$$\frac{dE}{dt} = \psi S - (\eta+\phi+\mu)E$$

$$\frac{dE}{dt} = \psi S - (\eta+\phi+\mu)E \geq -(\eta+\phi+\mu)E$$

$$\frac{dE}{dt} \geq -(\eta+\phi+\mu)E$$

$$\frac{dE}{E} \geq -(\eta+\phi+\mu)dt$$

$$\int \frac{dE}{E} \geq \int -(\eta+\phi+\mu)dt$$

$$\Rightarrow \ln E(t) \geq -(\eta+\phi+\mu)t + c$$

$$E(t) \geq \ell^{-(\eta+\phi+\mu)t}.\ell^{c}$$

$$but\, \ell^{c} = E(0)$$

$$E(t) \geq E(0)\ell^{-(\eta+\phi+\mu)t}$$

$$E(t) \geq E(0)\ell^{-(\eta+\phi+\mu)t}$$

apply the initial condition at $t = 0$

$E(t) = E(0), it\, gives$

$$E(t) \geq E(0)\ell^{-(\eta+\phi+\mu)t} \geq 0$$

since

$\ell^{-(\eta+\phi+\mu)t} \geq 0$ and $E(0) \geq 0$

$\therefore$ the solution is positve at all time $t \geq 0$

The solution is therefore positive for the remaining equations using the same method.

3.3DISEASE FREE EQUILIBRIUM (DFE)

At the disease free equilibrium, there is no infection and as such $E = I = I_T = I_N = Q = J = 0$ at the point $\frac{dE}{dt} = \frac{dI}{dt} = \frac{dI_T}{dt} = \frac{dI_N}{dt} = \frac{dQ}{dt} = \frac{dJ}{dt} = 0$ .The DFE state is represented by: $\left[S^0 \quad E^0 \quad V^0 \quad I^0 \quad I_T{}^0 \quad I_N{}^0 \quad Q^0 \quad J^0 \quad \mathrm{R}^0\right]$ which is given by:

$$DEF = \left[\left(\frac{\theta+\lambda}{\mu+\gamma}\right),\ 0,\ \frac{\beta(\theta+\lambda)}{[(\mu+\gamma)+\mu][\mu+\gamma]},\ 0,\ 0,\ 0,\ 0,\ 0,\ 0\right] \qquad (2.15)$$

3.4ENDEMIC DISEASE EQUILIBRIUM (EE)

At the endemic disease equilibrium, infection exist and as such we let

$S = S^*, \quad E = E^*, \quad V = V^*, \quad I = I^*, \quad I_T = I_T{}^*, \quad I_N = I_N{}^*, \quad Q = Q^*, \quad J = J^*, \quad R = R^*$

From equation (2.1) to (2.9) we have thus

$$S^* = \frac{(\theta+\lambda)+\omega(\delta+\rho+\mu)+(1-\gamma)I^*}{\psi(\beta+\mu+\delta)} \quad \text{.....(2.16)}$$

$$E^* = \frac{\psi(\delta+\rho+\mu)I^* + (\delta+\rho+\theta)}{(\lambda+\mu)(\sigma_1+\mu)} \quad \text{.....(2.17)}$$

$$V^* = \frac{\beta(\theta+\lambda)I^*}{\left[(1-\gamma)+\mu\right]\left[\delta+\rho+\mu\right]} \quad \text{.....(2.18)}$$

$$I^* = \frac{\psi(\theta+\lambda)}{(1+\varepsilon+\delta+\mu)(\sigma_1+\mu)} \quad \text{.....(2.19)}$$

$$I_T{}^* = \frac{(1-\sigma_2)I^* + \sigma_3(\delta+\mu)(\phi+\eta)}{(\sigma_3+\delta+\mu)(1-\sigma_2)I^*} \quad \text{.....(2.20)}$$

$$Q = \frac{\eta I^*}{(\sigma_1+\mu)(\rho+\mu)(\sigma_3+\delta+\mu)} \quad \text{.....(2.21)}$$

$$J = \frac{(\sigma_2+\sigma_1+\rho)(\delta+\rho+\mu)I^*}{(\sigma_1+\mu)(\eta+\phi+\mu)} \quad \text{.....(2.22)}$$

$$R = \frac{\rho I^* + (\delta+\rho+\mu)I^*}{(\sigma_3+\delta+\mu)(1-\sigma_2)(\rho+\mu)} \quad \text{.....(2.23)}$$

3.5THE BASIC REPRODUCTION NUMBER (R_0)

It is the expected number of secondary infection produced when one infected individual is introduce completely into a susceptible population. Deikmann and Heesterbeek (2000). The computation if R_0 involves the product of infection rate and duration of infection.

To compute the basic reproduction number, we consider the state variable of those compartments responsible for the spread of the virus, these compartments are: $\left[E, \quad I, \quad I_T, \quad I_N, \quad Q, \quad J,\right]$

The transmission model consist of the system of equation $F_i(x) = F_i(x) - V_i(x)$ where $V_i(x) = V_i^-(x) - V_i^+(x)$.

The basic reproduction number is given by $R_0 = \rho(FV^{-1})$ where $\rho(A)$ is the spectral radius of the matrix A which is the dominant non-negative Eigen value of F and V are $M \times M$ matrix which represents the infectious classes as earlier stated.

Where $F = \frac{\partial F_i(E_0)}{\partial x_j}$ and $V = \frac{\partial V_i(E_0)}{\partial x_j}$

The infectious class are $\left[E, \quad I, \quad I_T, \quad I_N, \quad Q, \quad J,\right]$

Force of infection $\psi = \frac{S(\xi_1\alpha_1 E + \alpha_2 I + \xi_2\alpha_3 I_T + \xi_3\alpha_4 I_N + \xi_4\alpha_5 Q + \xi_5\alpha_6 J)}{N}$

$$F_i = \begin{pmatrix} \frac{\psi S}{N} \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} \quad \textit{therefore}$$

$$F = \begin{bmatrix} \xi_1\alpha_1 \frac{S^0}{N} & \alpha_2 \frac{S^0}{N} & \xi_2\alpha_3 \frac{S^0}{N} & \xi_3\alpha_4 \frac{S^0}{N} & \xi_4\alpha_5 \frac{S^0}{N} & \xi_5\alpha_6 \frac{S^0}{N} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix} \quad \text{.....(2.24)}$$

Similarly,

$$V_1 = \begin{bmatrix} (\eta+\mu)E \\ (1+\varepsilon-\delta)I \\ (\delta+\rho+\mu)I_T - (1-\sigma_2)I - \sigma_3 J \\ (\delta+\mu)I_N - \varepsilon I \\ (\sigma_1+\mu)Q - \eta E \\ (\sigma_3+\delta+\mu)J - \sigma_2 I - \sigma_1 Q \end{bmatrix} \quad \textit{therefore}:$$

$$V = \begin{bmatrix} (\eta+\mu) & 0 & 0 & 0 & 0 & 0 \\ 0 & (\delta+\varepsilon+1) & 0 & 0 & 0 & 0 \\ 0 & -(1-\sigma_2) & (\delta+\rho+\mu) & 0 & 0 & -\sigma_3 \\ 0 & 0 & 0 & (\delta+\mu) & 0 & 0 \\ 0 & -\sigma_2 & 0 & 0 & -\sigma_1 & (\sigma_3+\delta+\mu) \end{bmatrix} \quad \text{.....(2.25)}$$

where $\mathrm{a} = \frac{\xi_1\alpha_1}{\eta+\mu}$, $\mathrm{b} = \frac{\xi_2\alpha_3\sigma_3\eta}{(\eta+\mu)(\sigma_3+\delta+\mu)(\delta+\rho+\mu)}$, $\mathrm{c} = \frac{\xi_4\alpha_5\eta}{(\eta+\mu)\sigma_1}$, $\mathrm{d} = \frac{\xi_5\alpha_6\eta}{(\eta+\mu)(\sigma_3+\delta+\mu)}$

$\mathrm{e} = \frac{\alpha_2}{(\delta+\varepsilon+1)}$, $\mathrm{f} = \frac{\alpha_2\xi_5\alpha_6}{(\delta+\varepsilon+1)(\sigma_3+\delta+\mu)}$, $\mathrm{g} = \frac{(\delta\sigma_2+\mu\sigma_2-\delta-\mu-\sigma_3)(\alpha_3\xi_2)}{(\delta+\varepsilon+1)(\sigma_3+\delta+\mu)(\delta+\rho+\mu)}$

$\mathrm{h} = \frac{(\delta+\mu)(\alpha_3\xi_2)+(\xi_3\alpha_4)(\delta+\rho+\mu)}{(\delta+\rho+\mu)(\delta+\mu)}$, $\mathrm{i} = \frac{\sigma_3\xi_3\alpha_2}{(\sigma_3+\delta+\mu)(\delta+\rho+\mu)}$ $\mathrm{j} = \frac{\xi_4\alpha_5}{\sigma_1}$, $\mathrm{k} = \frac{\xi_5\alpha_6}{\sigma_3+\delta+\mu}$

$\mathrm{l} = \frac{\xi_2\alpha_3\sigma_3}{(\sigma_3+\delta+\mu)(\delta+\rho+\mu)}$, $\mathrm{m} = \frac{\xi_5\alpha_6}{\sigma_3+\delta+\mu}$, $\mathrm{h} = \frac{\alpha_3\xi_2}{\delta+\rho+\mu}$, $\mathrm{n} = \frac{\xi_3\alpha_4}{\delta+\mu}$

$$FV^{-1} = \begin{bmatrix} (\mathrm{a}+\mathrm{b}+\mathrm{c}+\mathrm{d})\frac{S^0}{N} & (\mathrm{e}+\mathrm{f}-\mathrm{g})\frac{S^0}{N} & (\mathrm{h})\frac{S^0}{N} & (\mathrm{n})\frac{S^0}{N} & (\mathrm{i}+\mathrm{j}+\mathrm{k})\frac{S^0}{N} & (\mathrm{l}+\mathrm{m})\frac{S^0}{N} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix} \quad \text{.....(2.26)}$$

The eigen value is given by $\begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ \frac{(a+b+c+d)S}{N} \end{bmatrix}$

By substituting now, we have that the dominant eigen value as

$$R_0 = \left[\frac{\xi_1 \alpha_1}{\eta+\mu} + \frac{\xi_2 \alpha_3 \sigma_3 \eta}{(\eta+\mu)(\sigma_3+\delta+\mu)(\delta+\rho+\mu)} + \frac{\xi_4 \alpha_5 \eta}{(\eta+\mu)\sigma_1} + \frac{\xi_5 \alpha_6 \eta}{(\eta+\mu)(\sigma_3+\delta+\mu)} \right] \frac{S^0}{N}$$

where $S^0 = \frac{(\theta+\lambda)}{\mu+\gamma}$, $N = \frac{\theta+\lambda}{\mu}$ $\therefore \frac{S^0}{N} = \frac{(\theta+\lambda)\mu}{(\mu+\gamma)}$

$$\Rightarrow R_0 = \left[\frac{\xi_1 \alpha_1}{\eta+\mu} + \frac{\xi_2 \alpha_3 \sigma_3 \eta}{(\eta+\mu)(\sigma_3+\delta+\mu)(\delta+\rho+\mu)} + \frac{\xi_4 \alpha_5 \eta}{(\eta+\mu)\sigma_1} + \frac{\xi_5 \alpha_6 \eta}{(\eta+\mu)(\sigma_3+\delta+\mu)} \right] \frac{(\theta+\lambda)\mu}{(\mu+\gamma)} \quad \text{.....(2.27)}$$

3.6LOCAL STABILITY ANALYSIS OF THE DISEASE FREE EQUILIBRIUM

We investigate the local stability of the disease free equilibrium point of the model, first we linearize the model by computing its Jacobian matrix (J) at the disease free equilibrium point.

$$\left[S^0 \quad E^0 \quad V^0 \quad I^0 \quad I_T{}^0 \quad I_N{}^0 \quad Q^0 \quad J^0 \quad R^0\right] = \left[\frac{(\theta+\lambda)\mu}{(\mu+\gamma)}, \ 0, \ \frac{\beta(\theta+\lambda)}{\left[(1-\gamma)+\mu\right]\left[\mu+\gamma\right]}, 0, 0, 0, 0, 0, 0 \right]$$

..... (2.28)

at disease free equilibrium

$E = 0,\ I = 0,\ I_T = 0,\ I_N = 0,\ Q = 0,\ J = 0,\ and\ R = 0$

The eigen value using Maple softwardare $D_1 = -(\psi+\beta+\mu),\ D_2 = -(\eta+\theta+\mu),\ D_3 = -(1+\gamma+\mu)$

$D_4 = -(1+\varepsilon+\delta),\ D_5 = -(\delta+\rho+\mu),\ D_6 = -(\delta+\mu),\ D_7 = -(\delta+\mu),\ D_8 = -(\sigma_3+\delta+\mu)$

$D_9 = -(\omega+\mu)$

where $D_1 - D_9$ are the eigen values

Using Routh-Hurwitz theorem which stated that an equilibrium state will be asymptotically stable if and only if all the eigen values of the characteristics equation have negative real parts.
We can therefore conclude that, the disease free equilibrium state of our model is locally asymptotically stable.

3.7LOCAL STABILITY ANALYSIS OF THE ENDEMIC EQUILIBRIUM POINT

Using the method developed by Nthiri-et-al (2016), they stated that "The endemic equilibrium is locally asymptotically stable provided the determinant (J^*) is greater than zero and the trace of (J^*) is less than zero". Trace of (J^*) is defined as the sum of the major diagonal element of the Jacobian points.

$$Trace = -[\beta+\delta\mu+\eta+\theta+\alpha\delta+\rho+\sigma_1+\sigma_3+\omega-\psi-\gamma-\varepsilon] \quad \text{.....(2.29)}$$

$$\Rightarrow Trace(J^*) < 0$$

To find the determinant of J_0, using Maple 2015, we let

$A = -(\psi+\beta+\mu),\ B = (1-\gamma),\ C = \omega,\ D = \beta,\ E = -(\eta+\phi+\mu),\ F = \beta,\ I = \phi$

$G = (1+\varepsilon+\delta), K = (1-\sigma_2),\ L = -(\delta+\rho+\mu),\ M = \sigma_3,\ N = \varepsilon,\ O = -(\delta+\mu),\ P = \eta$

$Q = -(\sigma_1+\mu),\ R = \sigma_2,\ S = \sigma_1,\ Y = -(\sigma_3+\delta+\mu),\ X = \gamma,\ Z = -(\omega+\mu)$

The determinant (J^*) =

$[-(\psi+\beta+\mu)(1+\varepsilon+\delta)(-(\omega+\mu)) - (1-\gamma)(\beta)(-(\omega+\mu)) + (1+\varepsilon+\delta)(\beta)(\gamma)]$

$[-(\sigma_3+\delta+\mu)(1+\varepsilon+\delta)(-(\sigma_1+\mu))(-(\sigma_3+\delta+\mu))(-(\delta+\rho+\mu))(-(\delta+\mu))]$

$\therefore$ The determinant (J^*) =

$\Rightarrow [(\psi+\beta+\mu)(1+\varepsilon+\delta)(\omega+\mu) + (1-\gamma)(\beta)(\omega+\mu) + (1+\varepsilon+\delta)\beta\gamma]$

$[(\sigma_3+\delta+\mu)(1+\varepsilon+\delta)(\sigma_1+\mu)(\sigma_3+\delta+\mu)(\delta+\rho+\mu)(\delta+\mu)]$ .....(2.30)

The determinant is greater than zero(0). We can therefore concluded that,
the endemic disease equilibrium is asymptotically stable.

VI. Simulations

The model is now simulated using the values of the variables and parameters we got from Ika Christian Hospital, Ankpa, Kogi State(unless otherwise stated). The values are tabulated in table (3.0)& (4.0) respectively below:

Table (3.0): Nominal values of Variables .**Source**: Ika Christian Hospital, Ankpa. 2018

S/N	VARIABLES	NORMINAL VALUE	REFERENCES
1	S	5000	Ika General Hospital
2	E	1000	Ika General Hospital
3	I	171	Ika General Hospital
4	V	2000	Ika General Hospital
5	Q	560	Ika General Hospital
6	J	153	Ika General Hospital
7	I_T	136	Ika General Hospital
8	I_N	18	Ika General Hospital
9	R	10	Ika General Hospital

Table (4.0): Nominal values of Parameters .**Source**: Ika Christian Hospital, Ankpa. 2018

S/N	PARAMETERS	NOMINAL VALUE	REFERENCES
1	θ	0.984	Ika General Hospital
2	λ	0.016	Ika General Hospital
4	ϕ	0.0337	Ika General Hospital
5	β	0.394	Ika General Hospital
7	γ	0.0098	Ika General Hospital
8	η	0.110	Ika General Hospital
9	σ_1	0.020	Ika General Hospital
10	σ_2	0.030	Ika General Hospital
11	σ_3	0.0268	Ika General Hospital
13	ρ	0.0268	Ika General Hospital
14	ε	0.0035	Ika General Hospital
15	μ	0.01425	Ika General Hospital
16	δ	0.0028	Ika General Hospital
17	ω	0.009	Ika General Hospital
19	α_1	0.0005	Assumed
20	α_2	0.0004	Assumed
21	α_3	0.0002	Assumed
22	α_4	0.0006	Assumed
23	α_5	0.0001	Assumed
24	α_6	0.0003	Assumed
25	ζ_1	0.0001	Assumed
26	ζ_2	0.0001	Assumed
27	ζ_3	0.0001	Assumed
28	ζ_4	0.0001	Assumed
29	ζ_5	0.0001	Assumed

(4.1) NUMERICAL VALUE OF THE BASIC REPRODUCTION NUMBER

$$R_0 = \left[\frac{\xi_1 \alpha_1}{\eta + \mu} + \frac{\xi_2 \alpha_3 \sigma_3 \eta}{(\eta + \mu)(\sigma_3 + \delta + \mu)(\delta + \rho + \mu)} + \frac{\xi_4 \alpha_5 \eta}{(\eta + \mu)\sigma_1} + \frac{\xi_5 \alpha_6 \eta}{(\eta + \mu)(\sigma_3 + \delta + \mu)} \right] \frac{(\theta + \lambda)\mu}{(\mu + \gamma)}$$

After substituting the above numerical values into the expression for the basic reproduction number we have that

$$R_0 = [8.450704855 \times 10^{-7}][5.925155925 \times 10^{-1}] \text{..... ....} (2.31)$$

$$R_0 = 5.007174394 \times 10^{-7}$$

……(2.32)

(4.2) MODEL GRAPHS

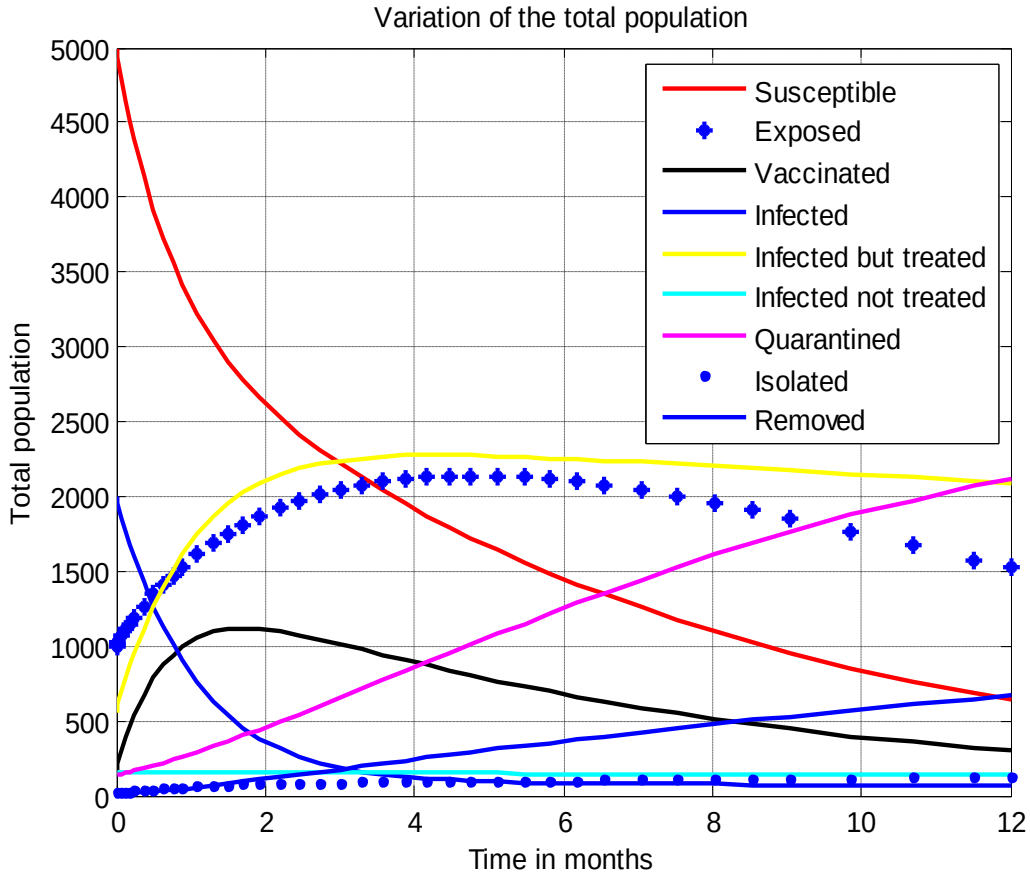


Figure: 2.0 Graph showing variation of the total population

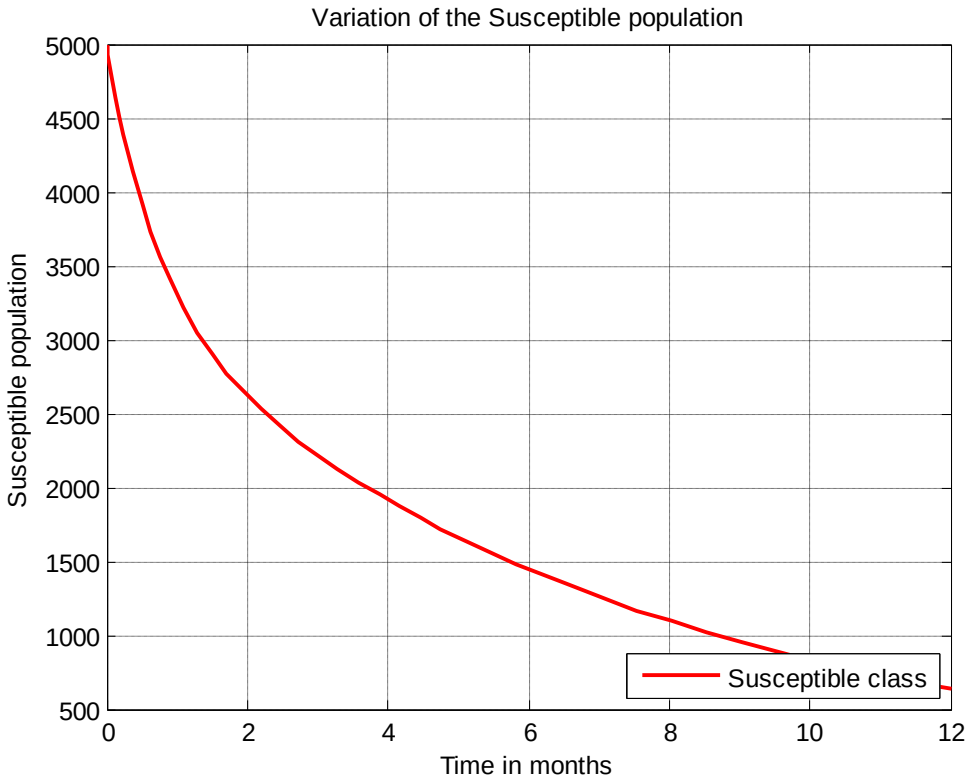


Figure: 3.0 Graph showing variation of the susceptible population: The population of the susceptible class reduces because of the control measures.

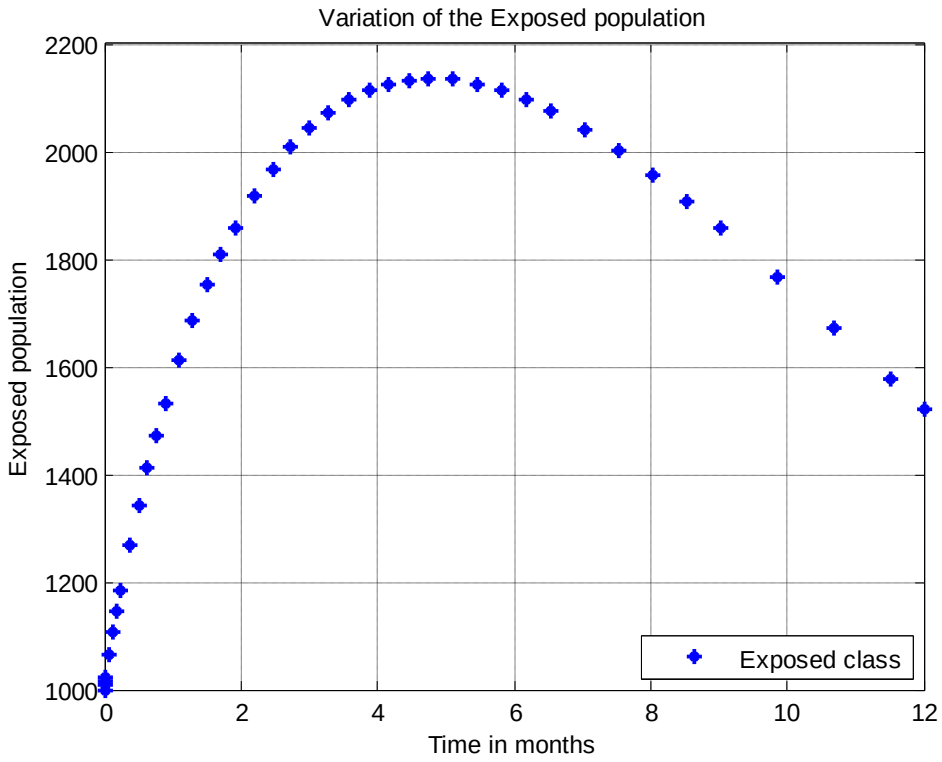


Figure: 4.0 Graph showing variation of the Exposed population

The Exposed class rises significantly and drop drastically, it is due to the reduction of contacts that exist between the Exposed and the Infected.

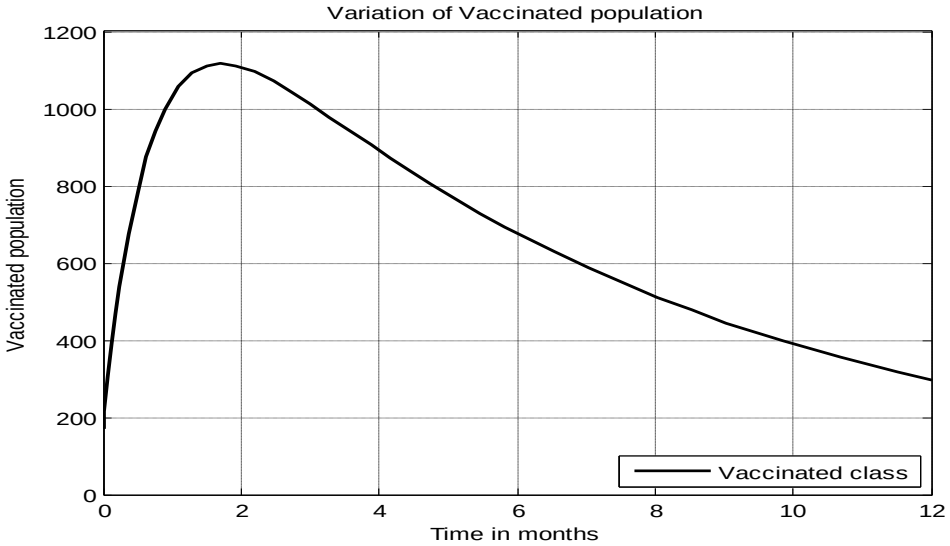


Figure: 5.0 Graph showing variation of the vaccinated population.

The Exposed class rises significantly and drop drastically, the increment was due to the acceptance of the population of the Susceptible to go for vaccination. The seriousness of the Susceptible to go for vaccination reduces the class after sometimes.

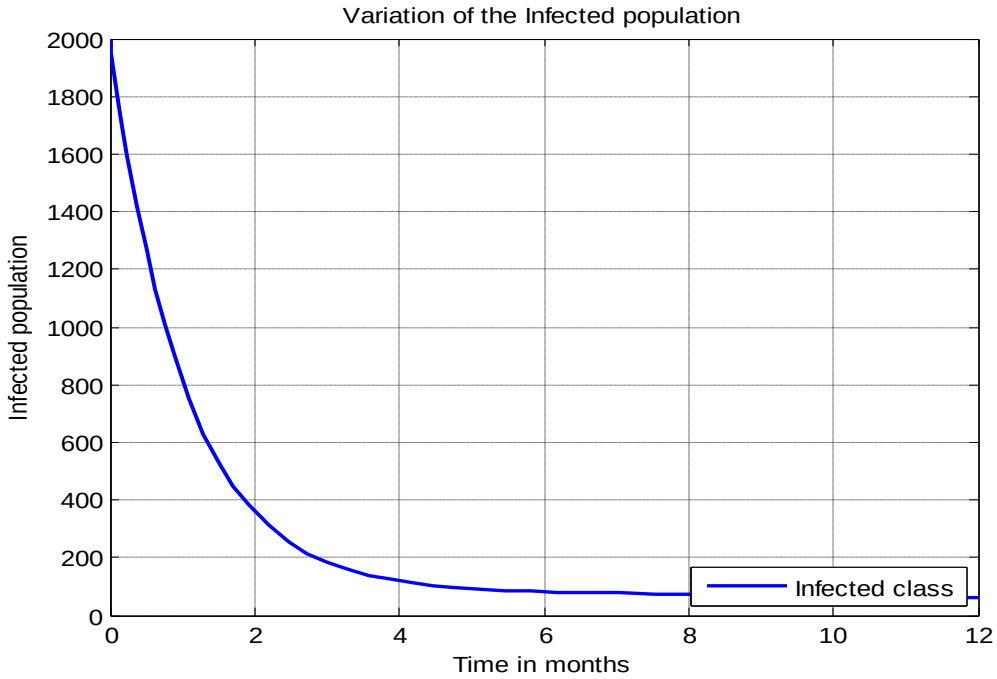


Figure: 6.0 Graph showing variation of the infected population: The population of the infected class reduces because of the acceptance of the infected to go for treatment.

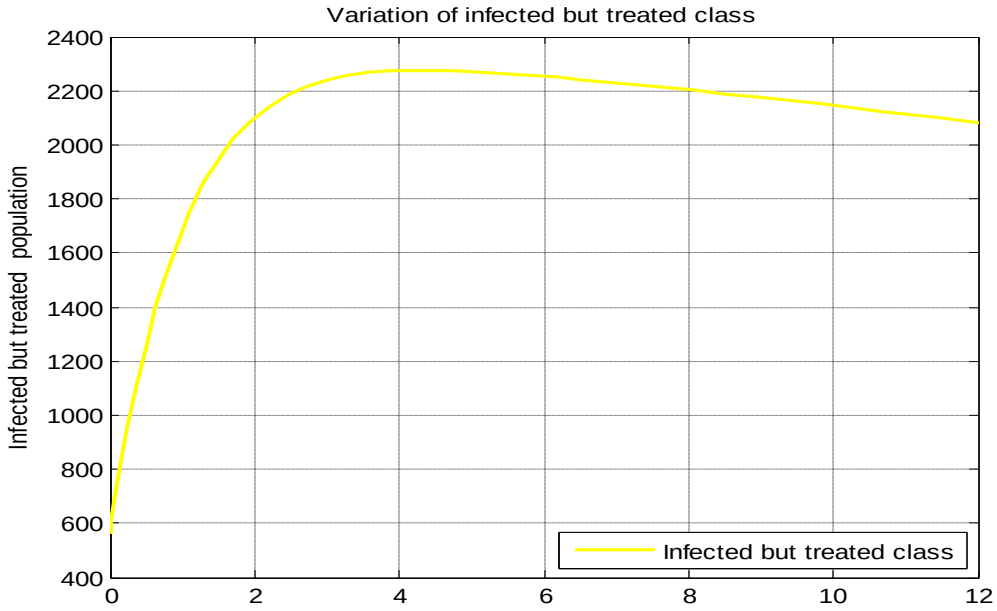


Figure: 7.0 Graph showing variation of the Infected but treated population:

The Infected but treated class rises significantly and drop drastically, the increment was due to the acceptance of the population of the Infected to go for treatment.

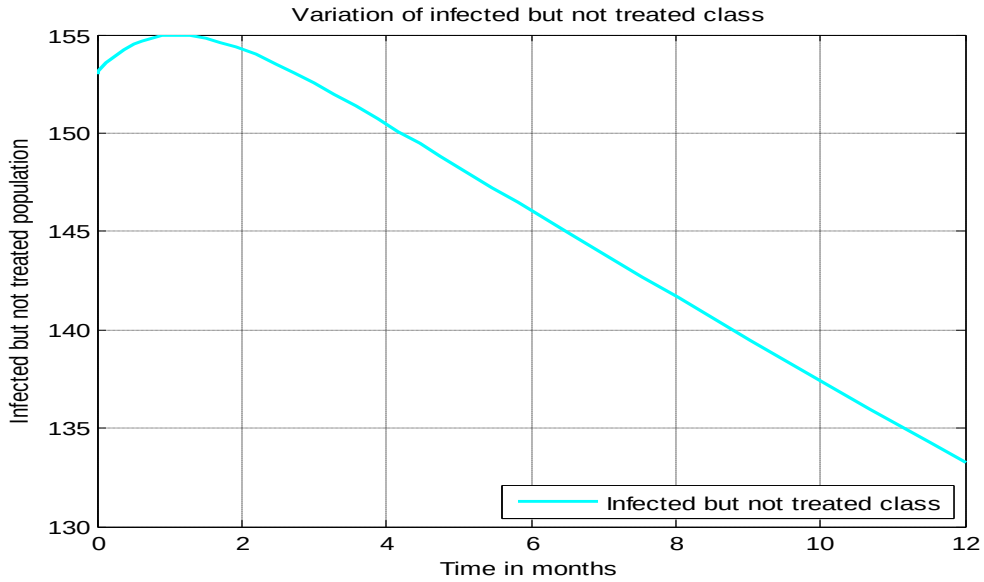


Figure: 8.0 Graph showing variation of the Infected but not treated population

The population reduces due to the fact that many accepted to go for treatment and as such reduces the population of the infected but not treated.

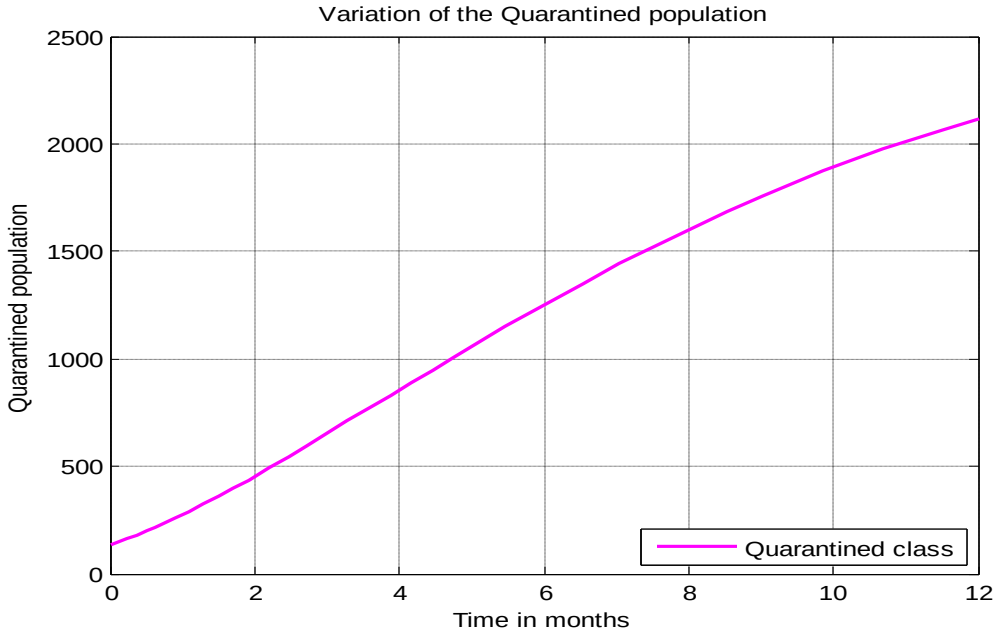


Figure: 9.0 Graph showing variation of the Quarantined population: The population increases because many suspected cases were discovered.

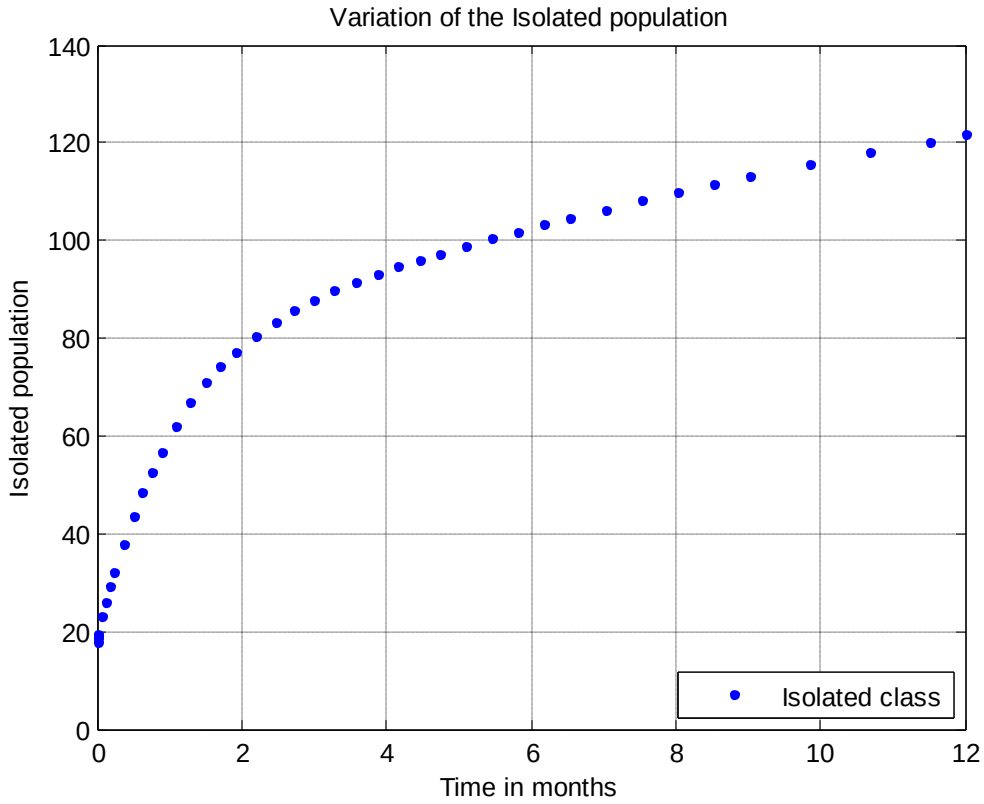


Figure: 10 Graph showing variation of the isolated population: The population increases because many confirmed cases were discovered.

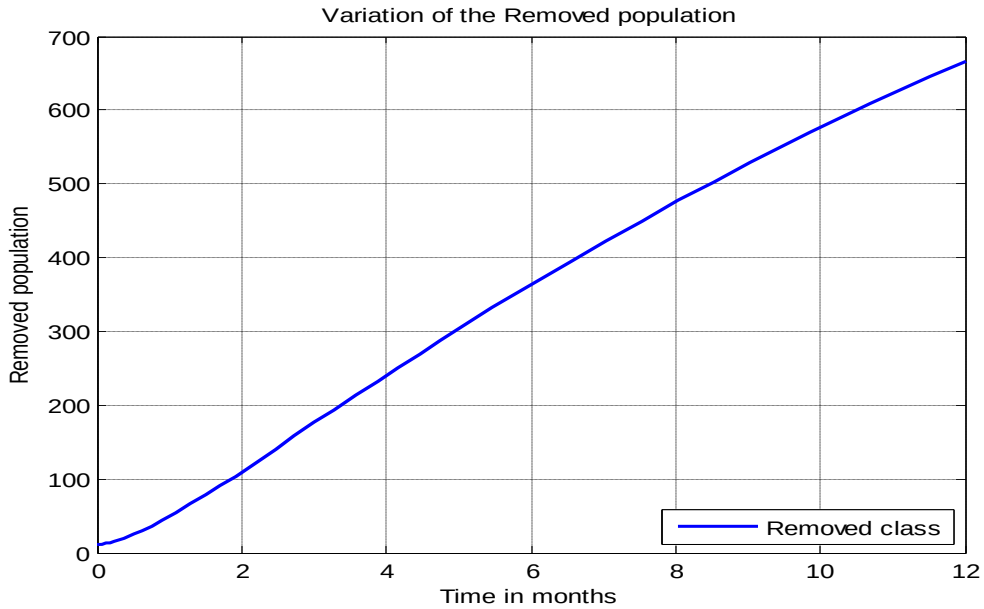


Figure: 11 Graph showing variation of the Removed population: The population increases because many recovered due to treatment, those that are successfully vaccinated are removed.

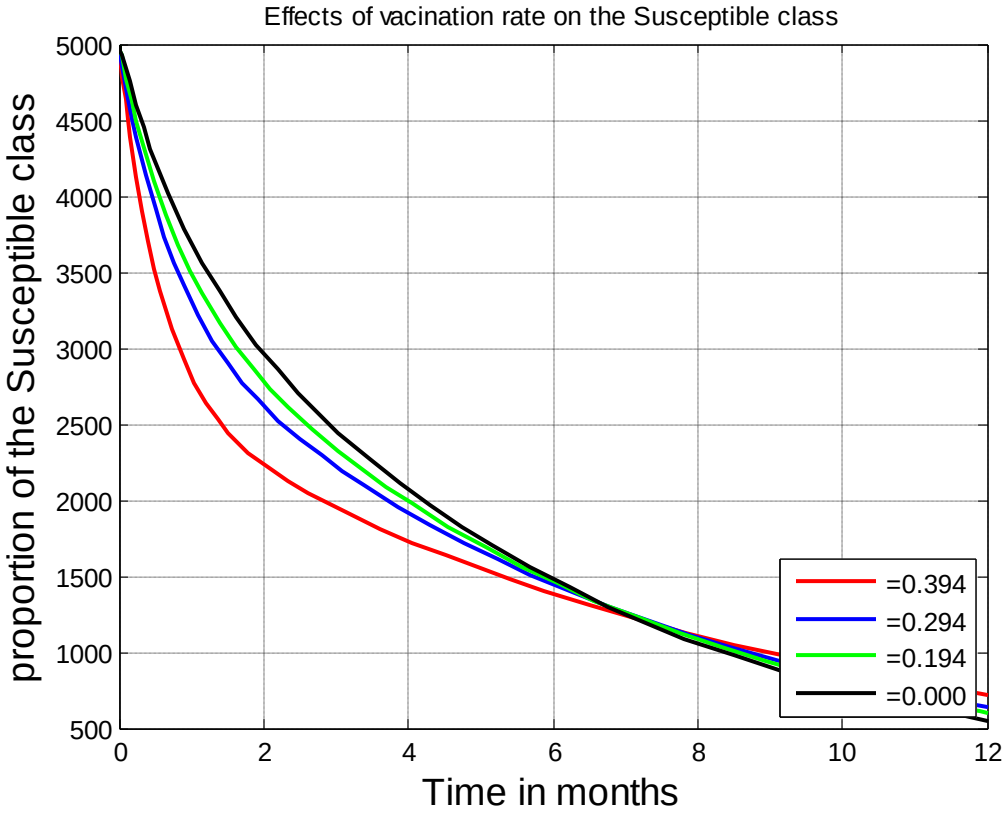


Figure: 12 Graph showing effects of vaccination rate on the susceptible class: The graph shows that, as the vaccination rate increases, it reduces the population of the susceptible class.

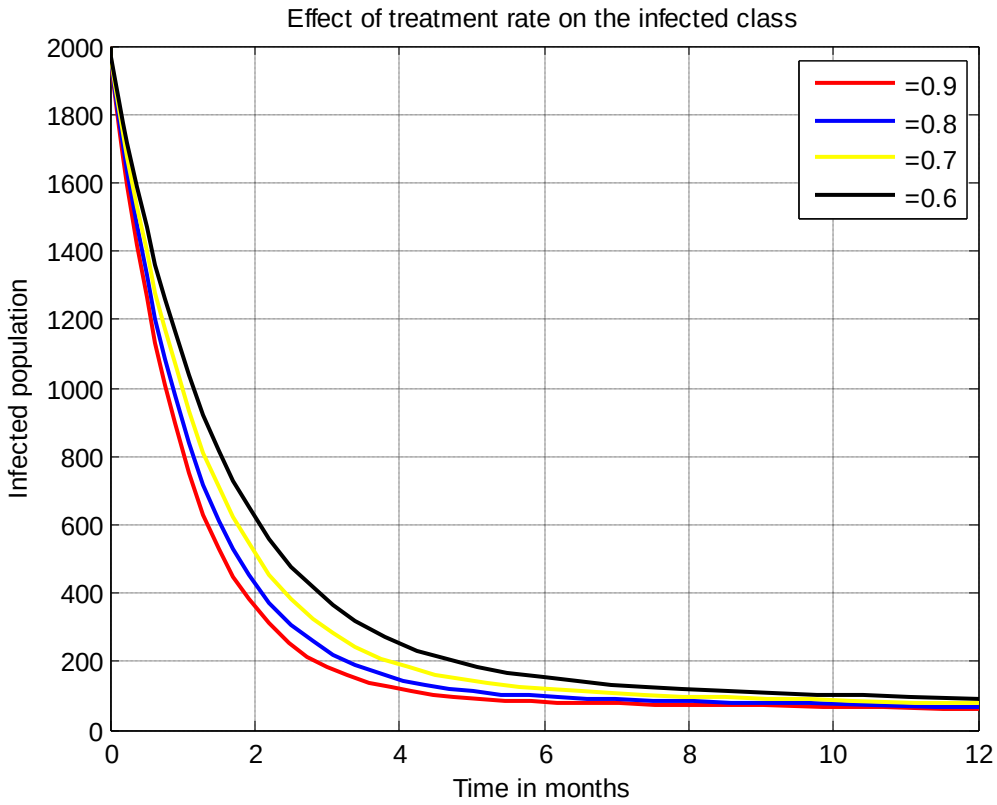


Figure: 13 Graph showing effects of treatment rate on the infected class: The graph shows as the treatment rate increases, it reduces the population of the infected class.

V. Discussion & Conclusion

In this paper, we developed a deterministic model for the transmission dynamics of Tuberculosis and its control, a case study of Ika Christian Hospital, Ankpa L.G.A, Kogi State, Nigeria. The model which adopts a standard incidence formulation incorporates treatment and vaccination as control strategies.

Some of the key findings of the study are as follows:

(1) The Disease Free Equilibrium state of the model is shown to be locally asymptotically stable.
(2) Local stability analysis of the Endemic Equilibrium state of the model was shown to be asymptotically stable using the trace and determinant method.
(3) The Basic Reproduction Number of the model was less than one (1), which shows that the disease can be wiped out from the population with the control measures.
(4) Increasing the vaccination rate decreases the population of the Susceptible population
(5) Increasing the treatment rate reduces the population of the infected population

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