Modelling Tuberculosis with Conditional Latent Period

A. Z. Loko[1*], Joseph I. K[2], Alhaji Ismaila Sulaiman[3], Lucas W. L.[1]

[1]*Mathematics Department, Nasarawa State University, Keffi, P.M.B.1022, Keffi, Nigeria*
[2]*Physics Department, Nasarawa State University, Keffi, P.M.B.1022, Keffi, Nigeria*
[3]*Statistics Department, Nasarawa State University, Keffi, P.M.B.1022, Keffi, Nigeria*
**Corresponding Author*

***Abstract*:- In this research work, we modeled Tuberculosis (TB) with conditional latent period using Delay Differential Equation. The model variables were divided into six compartments – susceptible, vaccinated latent (low and high), infected and recovered humans. We investigate the Disease Free Equilibrium (DFE) of the model and find the conditions that guarantee the asymptotic stability of corresponding steady states using stability theory of differential equations. The numerical simulations were carried out to test the effect of influence of key parameters on the spread of the TB epidemic, in particular the treatment and vaccination parameters to support the analytical solution and the stability theorem. It was shown that the introduced vaccination, high latent period and treatment parameter helps in controlling and eradication of TB epidemic in the population with respect to time. It is good to conclude that high effective treatment should be administered as well as high vaccination as this helps a long way in controlling the TB epidemic.**

Keywords: **Tuberculosis, Conditional Latent Period, Vaccination, Local Stability, Disease Free Equilibrium**

I. INTRODUCTION

Tuberculosis (TB) is a bacterial infection caused by mycobacterium tuberculosis referred to as tubercle bacilli. It most commonly affects the lungs producing pulmonary TB, and it is transmitted by blood or lymphatic system. Extra-pulmonary IB is common in children while pulmonary TB is common in adults pulmonary TB is the most common and potentially most contagious type of active TB. Usually within 2-10 weeks, the immune responses limits further multiplication and spread of the bacilli. TB is spread when an infections person coughs, sneezes, talks or signs, releasing droplets containing the bacilli into the air [1, 2].

Tuberculosis is mostly transmitted through the air by persons coughing with pulmonary tuberculosis. The probability of transmission per contact, per relevant unit of time is in general quite low [3, 4, 5]. Individuals at high risk of infection include those who are frequently exposed for long period of times to infectious individuals. Infected individuals may remain asymptomatic over their entire lives (latent TB). Active-TB (the clinical disease) can develop into pulmonary and extrapulmonary forms. Extrapulmonary TB is common in children while pulmonary TB is frequent in adults. Mycobacterium tuberculosis, the causal agent of the disease, is transmitted almost exclusively via pulmonary cases (exceptions could include laryngeal TB). The number of new cases of active TB decline almost exponentially when views as a function of age of infection [6]. In a ten year study, Styblo [6] noted that nearly 60% of the new cases arose during the first year following infection while the cumulative number of cases generated over the first five years after infection accounted for nearly 95% of the total cases observed. If this exponential decline in progression risk were to be maintained over the life time of individuals in the population then the contribution of endogenous reactivation to progression would be small, less than the 5%. However, increases in the risk of endogenous reactivation in the elderly have been observed.

Fathalla and Naim [7] investigate the qualitative analysis of delayed sir epidemic model with a saturated incidence rate. They used delayed SIR epidemic model in which the susceptibles were assumed to satisfy the logistic equation and the incidence term is of saturated form with the susceptible. In the same vein [11], did a comparative analysis of four deterministic compartmental mathematical models for controlling the spread of tuberculosis in Nigeria. The model considered high coverage of antiretroviral treatment as a means of boosting the immune system of people living with HIV and vary the contraction rate and latent TB treatments of progressors.

Despite numerous management control strategies on tuberculosis, currently TB continue to cause health effect in a number of studies over the past two decades. However, this work intends to investigate the role of the vaccination and the impact of the time delayed of the latent class to become infectious.

II. MODEL FORMULATION

We propose a simple Tuberculosis model with slow latent, $L_S(t)$ and fast latent, $L_F(t)$rate progression that incorporates vaccination using time delay differential equation. The population is divided into six (6) different classes: The susceptible Human$S(t)$, the Latent Humans with slow progression rate to infections $L_S(t)$, the Latent Humans with fast progression rate to infections $L_F(t)$, the infections Humans $I(t)$, the Vaccinated Humans $V(t)$, and the Recovered Humans $R(t)$. A susceptible is an individual that is yet to be infected, but open to infection as he or she interacts

with members of the infected class. While the latent is an individual that is infected but cannot spread the disease, an infected individual is one who has contracted the Tuberculosis and is capable of transmitting the disease, and we assumed that recruitment into the recovered-class from the infected-class depends on the effectiveness of treatment given to the infected-class, and this is done at a rateγ, the purpose of this model is to study the role of delay model and a latent period of infected persons.

Table 1: Model Variables and Parameters

Variable	Description
$S(t)$	Number of susceptible human at time t
$L_S(t)$	Number of humans in latent period at slow rate of incubation at time t
$L_H(t)$	Number of humans in latent period at high rate of incubation at time t
$I(t)$	Number of infected humans at time t
$R(t)$	Number of recovered humans at time t
$V(t)$	Number of vaccinated humans at time t

Parameters	Description
ρ	human birth rate
β	incidence rate per susceptible
θ	proportion of infections instantaneous incidence with slow progression rate
ε	recovery rate of $L_S(t)$ to susceptible
ψ	recovery rate of $L_F(t)$ to susceptible
?	natural death rate
δ_1	progression from $L_S(t)$ to $I(t)$
δ_2	progression from $L_F(t)$ to $I(t)$
α	vaccination rate
δ	disease induced death rate
η	rate at which individual from $R(t)$ return to $S(t)$ class
π	progression rate of $L_S(t)$ to $L_F(t)$

The following diagram describes the dynamics of infection, and will be useful in the formulation of model equations.

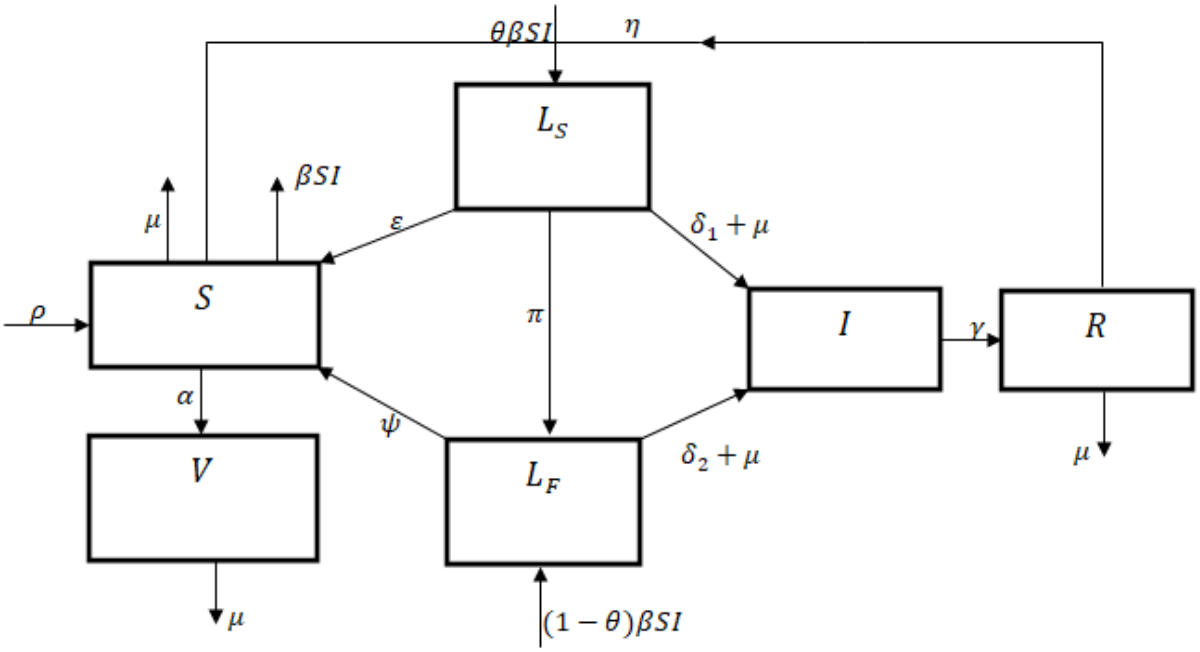

Figure 1: The flow chart of the model

III. ASSUMPTION OF THE MODEL

1. In the model, we assumed that the latent-class is divided into two (2) subgroup called the latent-class with slow progression rate to infection-class and latent-class with fast progression rate to the infection-class, denoted by $L_S(t)$ and $L_F(t)$respectively.
2. We also assumed that both the $L_S(t)\ and\ L_F(t)$ progress to infection-class at different rate $\delta_1\ and\ \delta_2$
3. We assumed that the susceptible receive vaccine and therefore are prevented from the disease at the rateα.
4. We also assumed that the $L_S(t)$ and$L_F(t)$ returns back to the susceptible-class at the rate $\varepsilon\ and\ \psi$ respectively when treated early.
5. We assumed that the Recovered-class returned back to the susceptible at the rate η.
6. We assumed that some individuals in the infected-class died as a result of the Tuberculosis at the rateδ.

7. We also assumed that the saturated contact is given by the Holling type functional response term $\frac{\beta SI}{1+\delta S}$

IV. MODEL EQUATIONS

In view of the above flow chart the spread of the disease will be governed by thefollowing system of differential equations:

$$\frac{dS}{dt} = \rho - \beta SI + \varepsilon L_s + \eta R + L_F\psi - (\alpha + \mu)S \quad (1)$$

$$\frac{dL_S}{dt} = \theta\beta SI - (\mu + \pi + \delta_1 + \varepsilon)L_S \quad (2)$$

$$\frac{dL_F}{dt} = (1-\theta)\beta SI + \pi L_s - (\mu + \delta_2 + \psi)L_F \quad (3)$$

$$\frac{dI}{dt} = \delta_1 L_F + \delta_2 L_S - (\mu + \delta + \gamma)I \quad (4)$$

$$\frac{dR}{dt} = \gamma I - (\eta + \mu)R \quad (5)$$

$$\frac{dV}{dt} = \alpha S - \mu V \quad (6)$$

If a disease is not of short duration, then several changes need to be made to the model above [8].Saturating contact rate of individual contacts is very important in an epidemiology model, for more convenience and a practical point of view the bilinear transmission incidence rate βSI is replaced by Holling type functional response term, which is saturated with the susceptible[8], the model (1) – (6) takes the form:

$$\frac{dS}{dt} = \rho - \frac{\beta SI}{1+\sigma S} + \varepsilon L_S + L_F\psi - \eta R - (\alpha + \mu)S \quad (7)$$

$$\frac{dL_S}{dt} = \frac{\theta\beta SI}{1+\sigma S} + (\mu + \pi + \delta_1 + \epsilon)L_S \quad (8)$$

$$\frac{dL_F}{dt} = \frac{(1-\theta)\beta SI}{1+\sigma S} + \pi L_S - (\mu + \delta_2 + \psi)L_F \quad (9)$$

$$\frac{dI}{dt} = \delta_1 L_F + \delta_2 L_S - (\mu + \delta + \psi)I \quad (10)$$

$$\frac{dR}{dt} = \gamma I - (\eta + \mu)R \quad (11)$$

$$\frac{dV}{dt} = \alpha S - \mu V \quad (12)$$

whereδ is the saturation factor that measure the inhibitory effect.

The model in (7) – (12) representing the transfer rate between exposed and infected-classes can be rigorously be derived using an age-structured modeling approach as in [10]. Let $I(t,\alpha)$ denotes the density of cells at time t that were infected τ times units before t (i.e. cells of disease ageτ).

Because$I(t,\tau)$ is the rate at which cells move from exposed to the infection-class, since it takes τ time units for an infection to "mature" in a given cell. Since per-capital death rate for exposed class is constant(μ), it is appropriate to assume that $I(t,\tau)$ satisfies the MC Kendoick Von-Foerster age-structured model, and $I(t,\tau)$ is given explicitly as [10].

$$I(t,\tau) = \frac{\beta e^{-\mu\tau} S(t-\tau)I(t-\tau)}{1+\sigma S(t-\tau)} \quad (13)$$

Hence, our model equations (7) – (12) now takes the new form.

$$\frac{dS}{dt} = \rho - \frac{\beta e^{-\mu\tau} S(t-\tau)I(t-\tau)}{1+\sigma S(t-\tau)} + \varepsilon L_S + L_F\psi - \eta R - (\alpha + \mu)S \quad (14)$$

$$\frac{dL_S}{dt} = \theta\beta e^{-\mu\tau} S(t-\tau)I(t-\tau) - (\varepsilon + \pi + \mu + \delta_1)L_s \quad (15)$$

$$\frac{dL_F}{dt} = \frac{(1-\theta)\beta e^{-\mu\tau} S(t-\tau)I(t-\tau)}{1+\sigma S(t-\tau)} + \pi L_S - (\mu + \delta_2 + \psi)L_F \quad (16)$$

$$\frac{dI}{dt} = \delta_1 L_F + \delta_2 L_S - (\mu + \delta + \psi)I \quad (17)$$

$$\frac{dV}{dt} = \alpha S - \mu V \quad (18)$$

$$\frac{dR}{dt} = \gamma I - (\eta + \mu)R \quad (19)$$

V. MATHEMATICAL ANALYSIS OF THE MODEL

Disease Free Equilibrium (DFE) - E_τ

The model (14) – (19) has non negative equilibrium points, which is the Disease Free Equilibrium (E_τ). At equilibrium point $\frac{dS}{dt} = \frac{dL_S}{dt} = \frac{dL_F}{dt} = \frac{dI}{dt} = \frac{dV}{dt} = \frac{dR}{dt} = 0$

Hence the systems of equations (14) – (19) is solved simultaneously for equilibrium points to get

The Disease Free Equilibrium (E_τ)

$$E_\tau = \left(\frac{\rho}{\alpha+\mu}, 0,0,0,0,0\right) \quad (20)$$

VI. COMPUTATION OF THE BASIC REPRODUCTION NUMBER, R_0

The basic reproduction number $R_0$is defined as the effective number of secondaryinfectious caused by typical infected individual during his interred periods of infectiousness [8, 9]. It is obtaining by taking the largest (dominant) Eigen value (spectral radius) of

$$R_0 = \left[\frac{\partial \mathcal{F}_i}{\partial x_j}(E_\tau)\right] \times \left[\frac{\partial \mathcal{V}_i}{\partial x_j}(E_\tau)\right]^{-1}$$

where, $F = \left[\frac{\partial \mathcal{F}_i}{\partial x_j}(E_\tau)\right]$and $V = \left[\frac{\partial \mathcal{V}_i}{\partial x_j}(E_\tau)\right]$,with $1 \le i,j \le m$and m is the number of infected classes.In particular $m = 3$, we have

$$FV^{-1} = \begin{pmatrix} 0 & 0 & 0 \\ \frac{(1-\theta)\beta e^{-\mu\tau}}{H_4H_1} & \frac{(1-\theta)\beta e^{-\mu\tau}\delta_1}{H_1H_2H_4} & \frac{\beta(1-\theta)e^{-\mu\tau}(H_2\delta_2+\pi\delta_1)}{H_1H_2H_3H_4} \\ \frac{\theta\beta e^{-\mu\tau}}{H_1H_4} & \frac{\theta\beta e^{-\mu\tau}\delta_1}{H_1H_2H_4} & \frac{\theta\beta e^{-\mu\tau}(H_2\delta_2+\pi\delta_1)}{H_1H_2H_3H_4} \end{pmatrix} \quad (21)$$

Then eigenvalues of equation (21) is given explicitly as

$$\lambda_1 = \lambda_2 = 0$$

$$\lambda_3 = \frac{\beta e^{-\mu\tau}\delta_1 H_3\theta + H_2\theta\beta e^{-\mu\tau}\delta_2 + \beta e^{-\mu\tau}\delta_1 H_3 + \theta\beta e^{-\mu\tau}\pi\delta_1}{H_1H_2H_3H_4}$$

Hence the Basic Reproduction Number, R_0 being the most positive eigenvalues is given by λ_3 which is

$$R_0 = \frac{\beta e^{-\mu\tau}(\delta_1 H_3\theta + H_2\theta\delta_2 + \delta_1 H_3 + \theta\pi\delta_1)}{H_1H_2H_3H_4} \quad (22)$$

From the solution, it is noted that with an increase in R_0which can be viewed as a function of *c*. Thus in order to keep the spread of the disease at minimum, the number of saturated incidence rate should be restricted.

VII. LOCAL STABILITY OF THE EQUILIBRIA

Theorem 1: *The disease free equilibrium of the system is locally asymptotically stable if*

$R_0 < 1$*and unstable* $R_0 > 1$

Proof: Now to determine the local stability of $E_\tau$the following variational matrix is computed corresponding to equilibrium point E_τ:

$$M_1 = \rho - \frac{\beta e^{-\mu\tau}S(t-\tau)I(t-\tau)}{1+\sigma S(t-\tau)} + \varepsilon L_S + L_F\psi - \eta R - (\alpha+\mu)S \quad (23)$$

$$M_2 = \theta\beta e^{-\mu\tau}S(t-\tau)I(t-\tau) - (\varepsilon+\pi+\mu+\delta_1)L_s \quad (24)$$

$$M_3 = \frac{(1-\theta)\beta e^{-\mu\tau}S(t-\tau)I(t-\tau)}{1+\sigma S(t-\tau)} + \pi L_S - (\mu+\delta_2+\psi)L_F \quad (25)$$

$$M_4 = \delta_1 L_F + \delta_2 L_S - (\mu+\delta+\psi)I \quad (26)$$

$$M_5 = \alpha S - \mu V \quad (27)$$

$$M_7 = \gamma I - (\eta+\mu)R \quad (28)$$

The Jacobian(J_{E_τ}) is given by

$$(J_{E_\tau}) = \begin{pmatrix} \frac{\beta e^{-\mu\tau}}{(\mu+\alpha+\delta\rho)} - (\mu+\alpha) & \varepsilon & \psi & \frac{\beta e^{-\mu\tau}}{(\mu+\alpha+\delta\rho)} & 0 & \eta \\ \frac{\theta\beta e^{-\mu\tau}}{(\mu+\alpha+\delta\rho)} & -(\varepsilon+\pi+\mu+\delta_1) & 0 & \frac{\theta\beta e^{-\mu\tau}}{(\mu+\alpha+\delta\rho)} & 0 & 0 \\ \frac{(1-\theta)\beta e^{-\mu\tau}}{(\mu+\alpha+\delta\rho)} & \pi & -(\mu+\delta_2+\psi) & \frac{(1-\theta)\beta e^{-\mu\tau}}{(\mu+\alpha+\delta\rho)} & 0 & 0 \\ 0 & \delta_2 & \delta_1 & -(\mu+\delta+\psi) & 0 & 0 \\ \alpha & 0 & 0 & \phi & -\mu & 0 \\ 0 & 0 & \alpha_3 & 0 & 0 & -(\eta+\mu) \end{pmatrix} \quad (29)$$

Equation (29) can rewritten as

$$(J_{E_\tau}) = \begin{pmatrix} \frac{\beta e^{-\mu\tau}}{H_4} - H_5 & \varepsilon & \psi & \frac{\beta e^{-\mu\tau}}{H_4} & 0 & \eta \\ \frac{\theta\beta e^{-\mu\tau}}{H_4} & -H_3 & 0 & \frac{\theta\beta e^{-\mu\tau}}{H_4} & 0 & 0 \\ \frac{(1-\theta)\beta e^{-\mu\tau}}{H_4} & \pi & -H_2 & \frac{(1-\theta)\beta e^{-\mu\tau}}{H_4} & 0 & 0 \\ 0 & \delta_2 & \delta_1 & -H_1 & 0 & 0 \\ \alpha & 0 & 0 & \phi & -\mu & 0 \\ 0 & 0 & \alpha_3 & 0 & 0 & -H_6 \end{pmatrix} \quad (30)$$

where,

$$\begin{cases} H_1 = (\mu+\delta+\psi), H_2 = (\mu+\delta_2+\psi), \\ H_3 = (\varepsilon+\pi+\mu+\delta_1), H_4 = (\mu+\alpha+\delta\rho), H_5 = (\mu+\alpha), H_6 = (\mu+\eta) \end{cases} \quad (31)$$

Hence the determinant and trace of the equation (30) is given as

$$Det(J_{E_\tau}) = -\frac{1}{H_4}\{\mu(H_6\rho\beta e^{-\mu\tau}\phi H_3H_5\delta_1 + H_6\pi\beta e^{-\mu\tau}\theta H_1\delta_1\psi - H1H2H3H4H5H6+H1H2H3H6\beta e-\mu\tau+H1H2H6\beta e-\mu\tau\varepsilon+H1\theta\eta\pi\beta e-\mu\tau\gamma+H1H3\phi\eta\beta e-\mu\tau\gamma+H5H6\theta\pi\rho\beta e-\mu\tau\delta1+H2H5H6\theta\rho\beta e-\mu\tau\delta2+H1H3H6\phi\psi\beta e-\mu\tau \quad (32)$$

$$Trace(J_{E_\tau}) = \frac{\beta e^{-\mu\tau}}{H_4} - H_5 - H_3 - H_2 - H_1 - \mu - H_6 \quad (33)$$

Expressing H_1 in terms of R_0, we get

$$H_1 = \frac{\beta e^{-\mu\tau}(\delta_1 H_3\theta + H_2\theta\delta_2 + \delta_1 H_3 + \theta\pi\delta_1)}{H_2H_3H_4R_0} \quad (34)$$

Substituting equation (34) into equation (32) we get

$$Det(J_{E_\tau}) = -\frac{1}{H_4}\{\mu(H_6\rho\beta e^{-\mu\tau}\phi H_3H_5\delta_1 + H6\pi\theta\beta e-\mu\tau2\varpi H2H3H4R0\delta1\psi-\beta e-\mu\tau\varpi R0H5H6+\beta e-\mu\tau2\varpi H3H4R0H3H6+\beta e-\mu\tau2\varpi H3H4R0H6\varepsilon+\beta e-\mu\tau2\varpi H2H3H4R0\theta\eta\pi\gamma+\beta e-\mu\tau2\varpi H2H4R0\phi\eta\gamma+H5H6\theta\pi\rho\beta e-\mu\tau\delta1+H2H5H6\theta\rho\beta e-\mu\tau\delta2+\beta e-\mu\tau2\varpi H2H4R0H6\phi\psi > 0, \quad (35)$$

If and only if $H_4 < 0$. Otherwise $Det(J_{E_\tau}) < 0$.

where, $\varpi = (\delta_1 H_3\theta + H_2\theta\delta_2 + \delta_1 H_3 + \theta\pi\delta_1)$

Similarly,

$$Trace(J_{E_\tau}) = \frac{\beta e^{-\mu\tau}}{H_4} - H_5 - H_3 - H_2 - H_1 - \mu - H_6 < 0, \text{since} e^{-\mu\tau} < 0 \quad (36)$$

provided that $H_4 > 0$.

VIII. RESULTS

Numerical Experiments of the Model

The age-structured deterministic model (3.1)–(3.6) was solved numerically using Runge-Kutta-Fehllberg 4-5th order method and implemented using Maple 17 Software (Maplesoft, Waterloo Maple Inc, 2012). Parameters were chosen in consonance with the R_0 values obtained in the stability analysis of the disease free equilibrium state of the model. We used the estimated values of the parameters by [7, 11] in the numerical experiments.

List of Numerical Experiments

(1) The graph of Susceptible Humans when the instantaneous contact rate is low with high ψ, ε *and* η.

(2) The graph of Infectious Humans when ψ *and* ε is constant with low treatment rates
(3) The graph of Infectious Humans when ψ *and* ε is high with low treatment rates.

(4) The graph of Infectious Humans when ψ *and* ε is high with high treatment rates.
(5) The graph of Recovered Humans when $\gamma = 0$ is high with rate returning back to susceptible humans.
(6) The graph of Recovered Humans when γ is low and high with low rate of returning to susceptible humans.

Experiment 1: The graph of Susceptible Humans when the instantaneous contact rate is low with high ψ, ε *and* η.

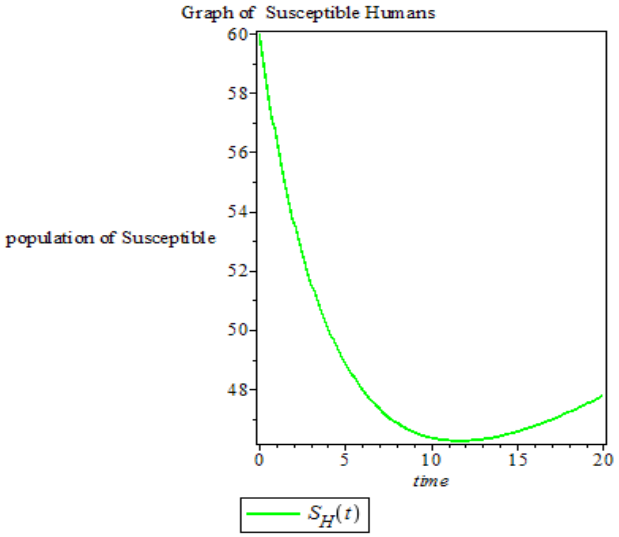

Figure 1: Showing the effect of when the instantaneous on susceptible humans when contact rate is low with high ($\psi = 0.6, \varepsilon = 0.5$ *and* $\eta = 0.8$)

Experiment 2: The graph of Infectious Humans when ψ *and* ε is constantwith low treatment rates.

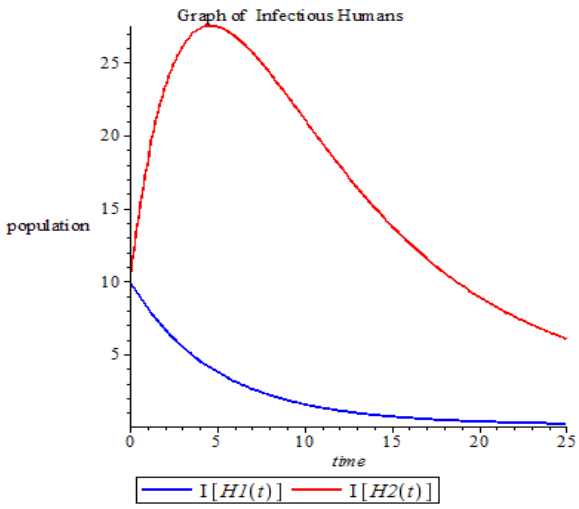

Figure 2: Showing the effect of low treatment rates when ψ *and* ε is constant.

Experiment 3: The graph of Infectious Humans when ψ *and* ε is high with low treatment rates.

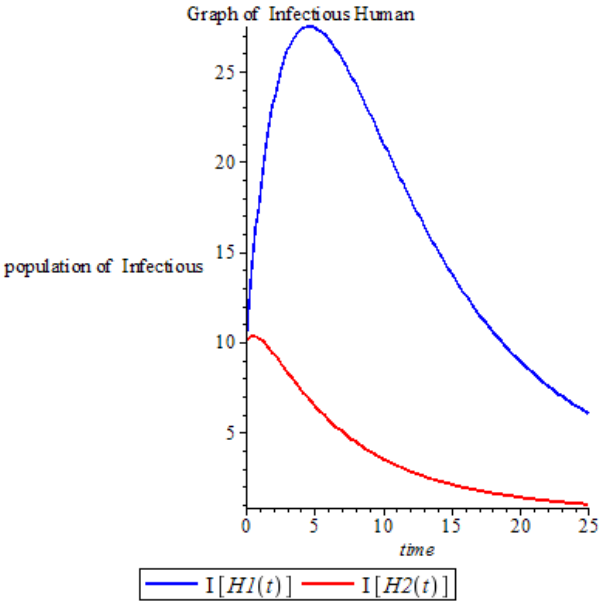

Figure 3: Showing the effect of low treatment rates with high ($\psi = 0.6, \varepsilon =$ *0.5 and* γ*=0.8*

Experiment 4: The graph of Infectious Humans when ψ *and* ε is high with high treatment rates.

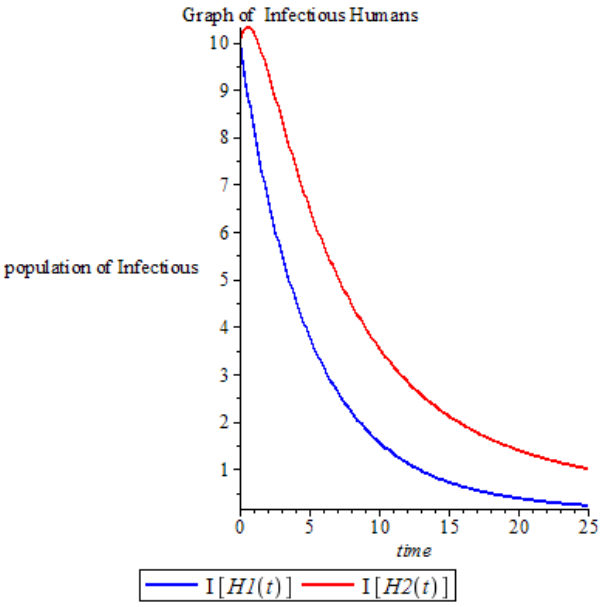

Figure 4: Showing the effect of high treatment rates when the contact rate high ($\psi = 0.6, \varepsilon = 0.5$ *and* $\eta = 0.8$)

Experiment 5: The graph of Recovered Humans when $\gamma = 0.8$ is high with rate returning back to susceptible humans.

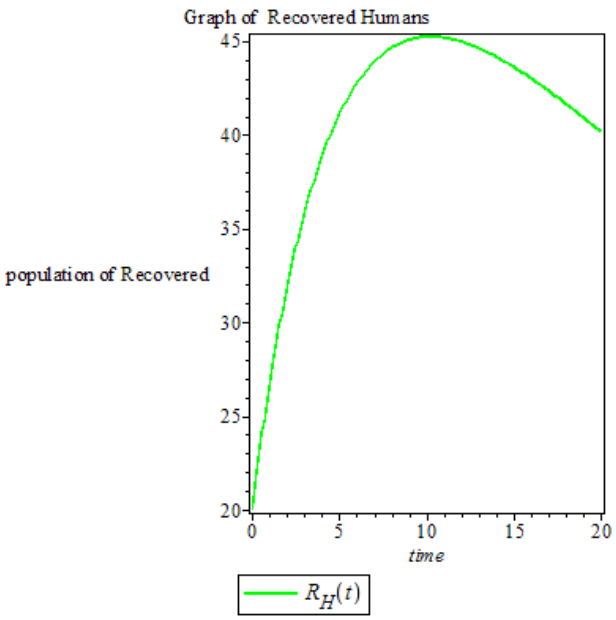

Figure 5: Showing the effect of hightreatment rates when the contact rate high ($\eta = 0.8$)

Experiment 6: The graph of Recovered Humans when γ is low and high with low rate of returning to susceptible humans.

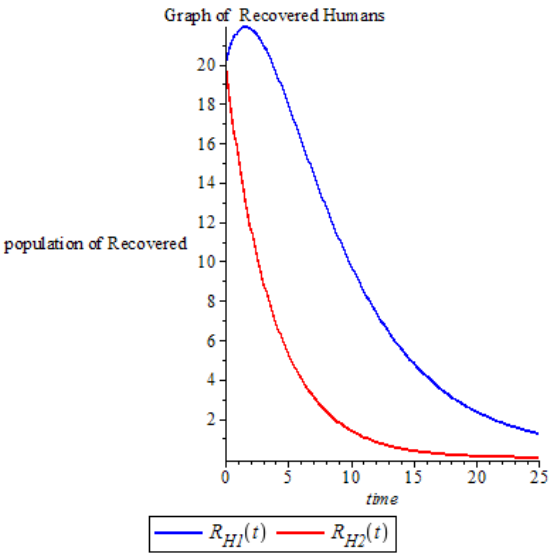

Figure 6: Showing the effect of low treatment rates on the recovered humans when γ low and high

IX. DISCUSSION OF RESULTS

Figure 1 shows the graphical representation of susceptible humans when the instantaneous constant rate $\beta = 0.01$with $(\psi = 0.5, \varepsilon = 0.3\ and\ \eta = 0.8)$. It was observed that there is a drastic full in the population of the susceptible due to the number of individuals leaving the population and at $t = 20$, the population would begin raise due to an increase in $\psi, \varepsilon\ and\ \eta$).

Figure 2 depicts the graphical representation of infectious humans when$(\psi = 0.02\ and\ \varepsilon = 0.01)$ is low. The figure shows raising in the population of infectious humans due to low treatment rate $(\gamma = 0.0125)$, but as a result of natural death and induced death due to the TB disease the population falls drastically. This further suggests that when the treatment rate is high the population of infectious humans would totally be eradication of TB.

Figure 3 describes the graphical representation infectious humans when the treatment rate is low with high $(\psi = 0.5, \varepsilon = 0.3\ and\ \eta = 0.8)$. The graph is almost similar with figure 2 above, only difference is that more individuals would leave the infectious population which lead to the eradication of the people with TB.

Figure 4 shows the graphical representation of infectious humans when the high as well as high $(\psi = 0.5, \varepsilon = 0.3\ and\ \eta = 0.8)$. It is observed that the TB disease will wipe out entirely in show time compare to experiments 2 and 3 above. This is as a result of high treatment and more individuals leaving the population.

Figure 5 shows the pictorial representation of recovered humans when $(\gamma = 0.8)$ and $(\eta = 0.8)$ is high. It is observe that because the treatment is high the recovered population increased drastically but would later begin to full as a result high η and natural death rate. This is why vaccination is very important to individual that recovered completely from the TB disease will not return back to the susceptible to contact the disease the again.

Figure 6 shows the graphical representation of recovered humans when $\gamma = 0$ and $\gamma = 0.0125$ is zero and low with low η. The graph shows decrease in the population of recovered due to no treatment as well as high η.

The results suggest that when the treatment and vaccination rate is high the population of infectious humans would totally be eradicated. The result further suggests that at low latent rate the TB epidemic persist, while high latent rate indicates eradication of the disease. This result corroborate with [7, 11] which suggest that model with immunization and infectious TB treatment) would lead to eradication of TB epidemic.

X. CONCLUSION

In this article, we developed a new mathematical model which incorporated some essential parameters that plays crucial role in the eradication of TB epidemic. These factors include low and high latent parameters,TB vaccination and treatment parameters. The introduced vaccination, high latent and treatment parameters help in controlling and eradication of TB epidemic with respect to time. We also computed the basic reproduction numbers, R_0. Our analysis shows that the disease can be control if the basic reproduction number, $R_0$is less than one regardless of the initial population profile. Thus, every effort must be put in place by all concerned to prevent the epidemic by reducing R_0strictly less than unity. Finally, there is need for further research work on the effects of various

control strategy such as quarantine, vaccination, personal hygiene dynamics of TB epidemic as well as stability analysis for endemic equilibrium.

REFERENCES

[1]. International Council of Nurses (2008). Tb Guidelines for Nurses in the Care and Control of Tuberculosis and Multi-drug Resistant Tuberculosis. Retrieved from: https://www.icn.ch/sites/default/files/inline-files/tb_mdrtb_guideline.pdf

[2]. Alimuddin, Z. Mario, R., Richard, H., and Fordham von Reyn, C. (2013). Current Concepts: Tuberculosis. *The New England Journal of Medicine, 745-755*

[3]. Baxter, T. (1993). Low infectivity of tuberculosis, The Lancet, 342 (1993), 371

[4]. Centers for Disease Control and Prevention(1992-1995). Exposure of passengers and flight crew to Mycobacterium tuberculosis on commercial aircraft, MMWR, 44 (1995), 137–140.

[5]. McFarland, J. W., Hickman, C., Osterholm, M. T. and MacDonald, K. L. (1993). Exposure to Mycobacterium tuberculosis during air travel, The Lancet, 342, 112–113.

[6]. Styblo, K (1991). "Epidemiology of Tuberculosis," Selected Papers, 24, Royal Netherlands Tuberculosis Association, The Hague, 1991.

[7]. Fathalla, A. R. and Naim M. (2012). Qualitative Analysis of Delayed SIR Epidemic Model with a Saturated Incidence Rate. *International Journal of Differential Equations Volume 2012, Article ID 408637, 13 pages doi:10.1155/2012/408637*

[8]. Van den Driessche, P. and Watmough, J. (2002). Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. Math.Biosci. 180, 29–48, 2002 doi:10.1016/S0025-5564(02)00108-6

[9]. Diekmann, O., Heesterbeek, J. A. P. and Metz, J. A. P. (1990)."On the definition and computation of the basic reproduction ratio R_0 in the model of infectious disease in heterogeneous populations*", Journal Math. Biol. 2(1), pp. 265-382.*

[10]. Stephen A. G., Yang, K. & John, D. N. (2008). Dynamics of a delay differential equation model of hepatitis B virus infection. *Journal of Biological Dynamics, 2:2,140-153, DOI: 10.1080/17513750701769873*

[11]. Abdullah, I. E. (2013). A Comparative Analysis of Four Deterministic Compartmental Mathematical Models for Controlling the Spread of Tuberculosis in Nigeria. *The Pacific Journal of Science and Technology14(2), 182-193*