JOURNAL OF FUNDAMENTAL MATHEMATICS
AND APPLICATIONS (JFMA) VOL. 7 NO. 2 (NOV 2024)
Available online at www.jfma.math.fsm.undip.ac.id

ANALYSIS OF A NON LINEAR DYNAMICS MODEL FOR TRANSMISSION TUBERCULOSIS IN NIGERIA INCORPORATING TREATMENT AND VACCINATION

O.J. Fenuga[1], A. O. Yusuff[2] and S. J. Aroloye[3]

[1,2,3]*Department of Mathematics, University of Lagos, Akoka, Nigeria*
Email : [1]ofenuga@unilag.edu.ng
[*]Corresponding Author

Abstract. This work models and analyzes the transmission of tuberculosis infection with the impact of vaccination and treatment on the bacteria in Nigeria from 2010 to 2022 incorporating treatment and vaccination. The Susceptible-Vaccinated-Exposed-Infected-Recovered (SVEIR) model is used for the transmission of the bacteria in which the with immigrants are exposed to infection infectious individuals and it is assumed that there is permanent immunity and homogenous mixing against the bacteria. The constant immigration of the infected individuals into the population makes it impossible for the disease to die out and so there is no disease-free equilibrium. The fraction of chemoprophylaxis Bacillus Calmette-Guerin (BCG) was incorporated into the model equation for successful vaccination. Stability analysis shows that a disease free equilibrum is locally asymptotically stable for the basic reproduction number less than 1 and endemic equilibrum which is stable for the basic reproduction number bigger than 1 which can wipe out the whole population. Hence, treatment and vaccination are the measures that can reduce below 1 in order to control tuberculosis.

Keywords: Population, Tuberculosis, Transmission, Vaccination, Disease free equilibrum, Susceptible, Latent and infectious.

I. INTRODUCTION

Tuberculosis (TB) is a worldwide pandemic disease. According to World Health Organization (WHO), one-third of the world's population is currently infected by the TB bacillus bacteria. Being a disease of poverty, the vast majority of TB deaths are in the developing world with more than half occurring in Asia. Tuberculosis (TB) is an infectious disease caused by Mycobacterium tuberculosis bacteria. It spreads through the air like the common cold. A person is infected with Tuberculosis if the body is unable to fight the bacteria and stopping it from growing. The bacteria will become dormant and remain in the body without causing symptoms until when the immune system of the patient with then dormant bacteria is weakened when the bacteria becomes active and infect the lungs of the patient. Tuberculosis bacteria cannot be spread through handshakes, sitting on toilet seats or sharing dishes and utensils with someone who has the disease. The bacteria get into the air when someone who has a tuberculosis lung infection coughs, sneezes, shouts or spits. Although, tuberculosis is an airborne disease, it is not highly infectious and so occasional contacts with infected person rarely lead to infection.

In 1834, Johann Lukas Schoenlein was the person who first named the bacteria 'tuberculosis' and the bacteria that caused Mycobacterium tuberculosis was identified by Nobel Laureate Robert Koch in 1882 and 1900. Albert Calmetteand Camille Guerin achieved the first genuine success of immunization against tuberculosis using attenuated bovine-strain tuberculosis called Bacillus of Calmette and Guerin(BCG). It was the development of the antibiotic streptomycin in 1946 that made the effective treatment and cure of tuberculosis possible.

Vaccination is a simple, safe, and effective way of protecting people against harmful diseases, before coming into contact with the disease. Vaccination is a safe and effective way of preventing diseases and saving lives. Vaccination is safe and side effects from a vaccine are usually minor and temporary. BCG Vaccine play a protective role against Mycobacterium Tuberculosis. Tuberculosis can be cured, even in people living with HIV. Due to improved transportation, local and international human migration have increased worldwide in recent years allowing immigration from areas with high incidence of Tuberculosis to areas of low incidence of Tuberculosis. Many developed nations reduce the risk of Tuberculosis spreading by the screening the immigrants upon arrival. Mc Cluskey and van den Driessche [1] investigate a SEI Tuberculosis model with immigration that includes the infected (both latent and infectious) individuals. The model assumed constant recruitment with fixed fraction entering each class and proved that under certain restrictions on the parameters (including the treatment rates, disease transmission rate and Tuberculosis induced death rate), the disease will approach a unique endemic level. The immigration of the infected results in the disease never dies out and the usual threshold condition found in many epidemic models is not applicable. In [2], it is investigated that the impact of immigration on the transmission dynamics of tuberculosis. They incorporated the recruitment of the latent and infectious immigrants but their model regards the immigrants as a separate subpopulation from the local population. Their theoretical analysis indicated that the disease will persist in the population if there is a prevalence of Tuberculosis in the immigrants and if the disease never dies out, it becomes endemic in host areas. The usual threshold condition does not apply and a unique equilibrium exists for all parameter values. The study suggests that immigrants have a considerable influence on the overall transmission dynamics of tuberculosis. Both studies show that when there is an immigration of infected into the population, there will be no disease-free equilibrium. Most study of the model with immigration will focused on the endemic equilibrium and its stability which sometimes is supported by the numerical simulations. This is attributed to the work by McCluskey and van den Driesscheas which focused on the global stability of the model. They proved the existence of a unique endemic equilibrium using Descartes' Rule of Signs and its local stability followed by the geometric approach developed with its global stability generalizations of Bendixsons' Condition to higher dimensions.

Bhunu et al. in [3] presented a SEIR tuberculosis model which incorporated treatment of infectious individuals and chemoprophylaxis (treatment for the latently infected). The model assumed that the latently infected individuals develop active disease as a result of endogenous re-activation, exogenous re-infection and disease relapse. The study showed that the treatment of infectious individuals is more effective in the first years of implementation as it cleared active Tuberculosis immediately and that chemoprophylaxis will do better in controlling the number of infectious due to reduced progression to active Tuberculosis. The study showed that immigration have an effect on the spread of tuberculosis in such a way that the disease persists in the host population. Zhang et al in [4] investigated dynamical aspects of a tuberculosis

transmission model incorporating vaccination and time delay by considering positivity, boundedness and local stability of the work. This work extends the work of most of the researchers' work because it include ΨR which is the rate at which actively infected individuals recover from Tuberculosis infection to the susceptible class. Many researchers have also contributed to the awareness of tuberculosis dynamics and recent studies can be assessed in [5,6,7,8,9,10,11,12] as well as some models with coinfections [13,14,15,16,17,18,19,20].

In this paper, we develop a new model for tubercolosis transmission in Nigeria with incorporating treatment and vaccination. This situation has not been considered in existing studies. Data used in this study is based on the spread of tubercolosis data in Nigeria. Analysis based on computational simulations are discussed to gain managerial insights which can be used by decision-makers/stakeholders to tackle the spread of tubercolosis in the world especially in Nigeria.

II. MATERIALS AND METHODS

This section formulates a dynamic model to investigate the current situation of Tuberculosis transmission in Nigeria. In this model, the total size of population at time *t* is divided into five subclasses: susceptible, vaccinated, exposed, infectious and recovered denoted by *S, V, E, I* and *R*, respectively. Because the vaccination strategy for Tuberculosis in Nigeria focuses on newborns, the subclass *V* is regarded as the newborns who have been vaccinated successfully. During the period of vaccination with Bacillus Calmette-Guerin(BCG), people cannot get infected even after contact with infectious individuals. But the time-efficiency of Bacillus Calmette-Guerin is very limited and the vaccinated individuals may lose immunity and become susceptible.

The susceptible class is increased at recruitment rate Λ and at the rate of *k* (from vaccinated class) by losing temporary immunity. The parameter $p \in [0, 1]$ indicates the fraction of the newborns who are vaccinated successfully. The susceptible, vaccinated, exposed, infected and recovered (SVEIR) model for the spread of Tuberculosis disease with immigration is described by the following system of differential equations.

$$\frac{dS}{dt} = \Lambda(1-P) - B(\beta)SI + KV - \mu S + \Psi R \quad (1)$$

$$\frac{dV}{dt} = \Lambda P - KV - \mu V \quad (2)$$

$$\frac{dE}{dt} = B(\beta)SI - \alpha_1 E - \delta E - \mu E \quad (3)$$

$$\frac{dI}{dt} = \delta E - \mu I - \gamma I - \alpha_2 I \quad (4)$$

$$\frac{dR}{dt} = \alpha_1 E + \alpha_2 I - (\mu + \Psi)R \quad (5)$$

where Ψ is the rate at which actively infected individuals recover from Tuberculosis infection to the susceptible class, γ is the disease induced death rate, Ψ is the rate at which actively infected individuals recover from Tuberculosis infection, α_1 is the recovery rate of the exposed class, α_2 is the **r**ecovery rate of the infected class, Λ is the recruitment rate K is the rate of Waning immunity, μ is the natural death rate, δ is the rate of progression to infectious stage from the exposed, P is the fraction of Bacillus Calmette-Guerin(BCG) vaccinated successfully, $B(\beta)$ is the transmission rate of infected population first stage (β) and second stage ($w\beta$).

2.1 Analysis of Disease Free Equilibrum

For the disease free equilibrium of the model i.e. (in the absence of Tuberculosis virus infection). Then, setting

$$\frac{dS}{dt} = \frac{dV}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0 \tag{6}$$

and assume there is no infection in the system, then we make $I = E = R = 0$ in the above equation and equation (2) gives V and S as

$$V = \frac{\Lambda P}{K+\mu} \tag{7}$$

$$S = \frac{\Lambda(K+\mu)(1-P)+K\Lambda P}{(K+\mu)\mu}. \tag{8}$$

The disease-free equilibrium point is given by

$$E_0 = \{\frac{\Lambda(K+\mu)(1-P)+K\Lambda P}{K+\mu}, \frac{\Lambda P}{K+\mu}, 0,0,0\}. \tag{9}$$

2.2 Analysis of Infected Steady State

Considering equations (1) to (5) for the endemic equilibrium i.e. when $\frac{dS}{dt} = \frac{dV}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0,\ S = V = E = I = R \neq 0,$ then, equations (1) to (5) be solved for $S, V, I, E \ and \ R$ as

$$v = \frac{\Lambda p}{k+\mu} \tag{10}$$

$$S = \frac{(\alpha_1+\delta+\mu)(\mu+\gamma+\alpha_2)}{B(\beta)\delta} \tag{11}$$

$$I = \frac{[\Lambda\beta\delta(K+\mu)-\mu(K+\mu)(\alpha_1+\delta+\mu)(\mu+\gamma+\alpha_2)-\mu\Lambda P\beta\delta][\delta(\mu+\Psi)]}{[\mu(\mu+\gamma+\alpha_2)(\mu+\Psi)+\delta(\mu+\Psi)(\mu+\gamma)+\mu[\alpha_1(\mu+\gamma+\alpha_2)+\delta\alpha_2]][\beta\delta(K+\mu)]} \tag{12}$$

$$R = \frac{\{\alpha_1(\mu+\gamma+\alpha_2)+\delta\alpha_2\}I}{\delta(\mu+\Psi)} \tag{13}$$

$$R = \frac{[\Lambda\beta\delta(K+\mu)-\mu(K+\mu)(\alpha_1+\delta+\mu)(\mu+\gamma+\alpha_2)-\mu\Lambda P\beta\delta][\delta(\mu+\Psi)][\alpha_1(\mu+\gamma+\alpha_2)+\delta\alpha_2]}{[\mu(\mu+\gamma+\alpha_2)(\mu+\Psi)+\delta(\mu+\Psi)(\mu+\gamma)+\mu[\alpha_1(\mu+\gamma+\alpha_2)+\delta\alpha_2]][\beta\delta(K+\mu)][\delta(\mu+\Psi)]} \tag{14}$$

$$E = \frac{(\mu+\gamma+\alpha_2)I}{\delta} \tag{15}$$

$$E = \frac{[\Lambda\beta\delta(K+\mu)-\mu(K+\mu)(\alpha_1+\delta+\mu)(\mu+\gamma+\alpha_2)-\mu\Lambda P\beta\delta][\delta(\mu+\Psi)](\mu+\gamma+\alpha_2)}{[\mu(\mu+\gamma+\alpha_2)(\mu+\Psi)+\delta(\mu+\Psi)(\mu+\gamma)+\mu[\alpha_1(\mu+\gamma+\alpha_2)+\delta\alpha_2]][\beta\delta(K+\mu)]\delta}. \tag{16}$$

2.3 The Positive Invariant Region

The total population size is

$$N = S + V + E + I + R \tag{17}$$

$$\frac{dN}{dt} = \frac{ds}{dt} + \frac{dv}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dR}{dt} \tag{18}$$

$$\frac{dN}{dt} = \Lambda - \mu S - \mu V - \mu E - \mu I - \mu R - \gamma I \tag{19}$$

The positive Invariant region can be obtained by using the following theorems.

Theorem 2.1 *The solution of the system of equations (1) - (5) are feasible for t < 0, if they enter the invariant region D.*

Proof. Let $D = (S\ V\ E\ I\ R) \in R^5$ be any solution of the system of equation (1) - (5) with non-zero initial conditions. Assuming there is no disease, equations (19) becomes

$$\frac{dN}{dt} + \mu N \leq \Lambda \tag{20}$$

solving (20) gives

$$N(t)\, e^{\mu t} \leq \frac{\Lambda}{\mu} + C_1 e^{-\mu t} \tag{21}$$

$$N(t) \leq \frac{\Lambda}{\mu} + C_1 e^{-\mu t} \tag{22}$$

using the initial condition t = 0; $N_h(0) = N_0$

$$No \leq \frac{\Lambda}{\mu} + C_1 \Rightarrow N_o - \frac{\Lambda}{\mu} \leq C_1 \tag{23}$$

$$\Rightarrow N \leq \frac{\Lambda}{\mu} + (N_o - \frac{\Lambda}{\mu}) e^{-\mu t}. \tag{24}$$

Therefore, as t tends to infinity in (**24)** the human population approaches $K = \frac{\Lambda}{\mu}$, (that is N → $k = \frac{\Lambda}{\mu}$), the parameter $k = \frac{\Lambda}{\mu}$ is called the carrying capacity. Hence all feasible solution set of the human population of the model equations (1) - (5) enter the region $D = (S, V, E, I, R) \in R^5$ for $s > 0$; $V, I \geq 0$; $R \geq 0$; $N \leq \frac{\Lambda}{\mu}$. Therefore, the region D is positively invariant, i.e the solution is positive for all time(t) and the problem (1) - (5) is mathematically well-posed in the domain D. Hence in this model, it is sufficient to consider the dynamics of flow generated by the model in addition to the existence, uniqueness and continuation of results for the system.

Theorem 2.2 (Positivity of Solution) *Let the initial data be S (0), V (0), I (0), E (0), R (0) ≥ 0. Then, the solution set (S, V, E, I, R) (t) of the system of equations (1) - (5) is positive for all t >0. In this case, by considering only the natural death rate of each class.*

Proof. From equation (1)

$$\frac{dS}{dt} = \Lambda(1 - P) - B(\beta)SI + KV - \mu S + \Psi R$$

since only the natural death rate of the susceptible class is considered, $\frac{dS}{dt} \geq -\mu S$. Solving (25) gives $S(t) \geq Pe^{-\mu t}$ where $P = e^{C_2}$. Using the initial condition $t = 0, S(0) \geq K$ gives $S(t) \geq K e^{-\mu t} \geq 0$. From (2), $\frac{dV}{dt} = \Lambda P - KV - \mu V \geq -(K + \mu)V$. This means that $\frac{dV}{V} \geq -(K + \mu)\, dt$ with the solution $V(t) \geq \eta\, e^{-(k+\mu)t}$ where $\eta = e^{C_3}$. Using initial condition t=0 , $V(0) \geq K$, then $V(t) \geq K e^{-\mu t} \geq 0$. From (3)

$$\frac{dE}{dt} = \beta SI - \alpha_1 E - \delta E - \mu E \geq -(\alpha_1 + \delta + \mu)E$$

$$\frac{dE}{dt} \geq -(\alpha_1 + \delta + \mu)E$$

$$E(t) \geq Qe^{-\mu t}, \quad \text{where } Q = e^{C_4}$$

Using initial condition $t=0, E(0) \geq K$, then $E(t) \geq Ke^{-(\alpha_1+\delta+\mu)}$. From (4), $\frac{dI}{dt} = \delta E - \mu I - \gamma I - \alpha_2 I \geq -(\mu + \gamma + \alpha_2) I$. Whose solution is $I(t) \geq \beta e^{-(\mu+\gamma+\alpha_2)t}$, where $\beta = e^{C_5}$. Using the initial condition $I(0) \geq K$, then $I(t) \geq Ke^{-(\mu+\gamma+\alpha_2)t} \geq 0$. From (5), $\frac{dR}{dt} = \alpha_1 E + \alpha_2 I - (\mu+\Psi)R \geq -(\mu+\Psi)R$ and $\frac{dR}{dt} \geq -(\mu+\Psi)R$. Whose solution is $R(t) \geq \alpha\, e^{-(\mu+\Psi)t}$, where $\alpha = e^{C_6}$ using the initial conditions $R(0) \geq K$, then $R(t) \geq K e^{-(\mu+\Psi)t} \geq 0$. In a similar manner, it can be verified that the equations (1) to (5) are positive for all t > 0 and for all $W \in R$.

2.3 Basic Reproduction Number

For any epidemic model, the basic reproduction number is the average number of secondary infectious cases produced by a single infection in total susceptible population. The basic reproduction number is calculated by $R_0 = \boldsymbol{\rho}(FV^{-1})$,where $\boldsymbol{\rho}$ is the spectral radius of the matrix FV^{-1} while F and V are the matrices of new infection terms and the remaining transmission terms respectively. At disease free equilibrium we get

$$S = \Lambda(1-P) + \frac{K\Lambda P}{K+\mu}$$

$$S = \frac{\Lambda(K+\mu)(1-P) + K\Lambda P}{K+\mu}$$

$$R_0 = \frac{B\Lambda\beta\delta(K+\mu)(1-P) + K\Lambda P}{(K+\mu)(\delta+\mu+\alpha_1)(\mu+\gamma+\alpha_2)}.$$

2.4 Local Stability of Disease Free Equilibrium

Local stability of equilibrium points of the model can be investigated by the theorem below:

Theorem 2.3 *Let* $\frac{dx}{dt} = P(x,y), \frac{dy}{dt} = Q(x,y), X = \begin{pmatrix} x \\ y \end{pmatrix}$ *and suppose* $X_i = \begin{pmatrix} x_i \\ y_i \end{pmatrix}$ *is an equilibrium point of the plane ,* $X_i = g(X) = \begin{pmatrix} P(x,y) \\ Q(x,y) \end{pmatrix}$, *where autonomous systems* $P(x,y)$ *and* $Q(x,y)$ *have continuous first partial derivatives in a neighborhood* X_i *.Then we have the followin: If the eigenvalues of* $A = g_i(X_i)$ *have negative real parts, then* X_i *is a locally asymptotically stable equilibrium point. If* $A = g_i(X_i)$ *has an eigenvalue with positive real root, then* X_I *is an unstable equilibrium point.*

Proof. The Jacobian matrix of the above model at disease free is given by

$$J = \begin{pmatrix} -\mu & k & 0 & -B\beta S & \Psi \\ 0 & -(k+\mu) & 0 & 0 & 0 \\ B\beta I & 0 & -(\delta+\Psi+\alpha_1) & 0 & 0 \\ 0 & 0 & \delta & -(\mu+\eta+\alpha_2) & 0 \\ 0 & 0 & \alpha_1 & \alpha_2 & -(\mu+\Psi) \end{pmatrix}$$

The characteristics equation $|J - \lambda I| = 0$ where I is a 5×5 identity matrix gives the following $\lambda_1 = -\mu,\ \lambda_2 = -(k+\mu),\ \lambda_3 = -(\delta+\mu+\alpha_1), \lambda_4 = -(\mu+\eta+\alpha_2)$ *and* $\lambda_5 = -(\mu+\Psi)$. Since all the eigenvalues have negative real part, then, it follows by Theorem 2.3 that the disease free equilibrium P is locally asymptotically stable.

III. RESULTS AND DISCUSSION

A model for the transmission of tuberculosis have been analyzed. The numerical analysis for the transmission and vaccination of tuberculosis is considered as in system (1) - (5) under different conditions. The results showed that the model has a disease free equilibrium which is locally asymptotically stable for $R_0 < 1$ and endemic equilibrium which is stable for $R_0 > 1$.The stability of endemic equilibrium suggests a wiping out of the population. Hence, vaccination or treatment measures aimed at reducing R_0 below 1 should be intensified in controlling the disease. The parameter values for Table1 and the initial conditions are chosen as $S(0) = 100000, V(0) = 2500, E(0) = 600.5, I(0) = 1, R(0) = 0$

Table 1. Parameter values and symbols

Symbol	Parameter	Value
ʌ	Recruitment rate	1.6 x 10^7
μ	Natural death rate for first stage second stage	0.0143 $year^{-1}$ and 0.0139 $year^{-1}$
B(β)	Transmission rate of infected population First stage(β) Second stage(wβ)	 2.5338 x 10^{-10} $year^{-1}$ 5.055410^{-10} $year^{-1}$
ϰ	Disease-induced death rate	0.006 $year^{-1}$
K	Rate of waning immunity	0.25 $year^{-1}$
α_1	Recovery rate of the exposed	0.6683 $year^{-1}$
α_2	Recovery rate of the infectious First stage Second stage	 0.0634$year^{-1}$ 0.3972$year^{-1}$
P	The fraction of Bacillus Calmette-Guerin(BCG)	0.6
Δ	Rate of progression to infectious stage from the exposed.	6$year^{-1}$

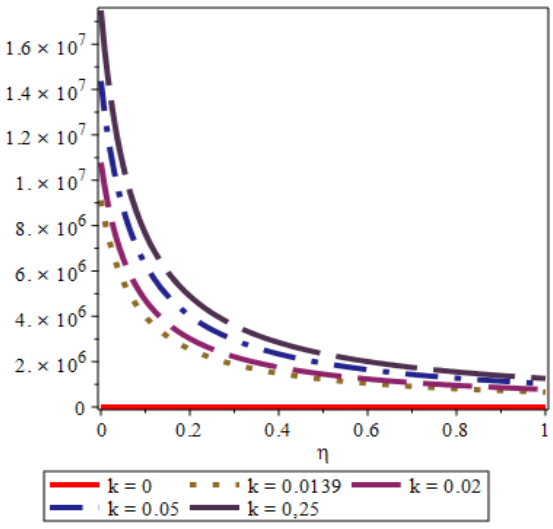

Fig. 1. Effect of P on R_0 when K is varied

Fig. 1 shows that increasing the fraction of successful vaccinated infants helps to reduce Tuberculosis.

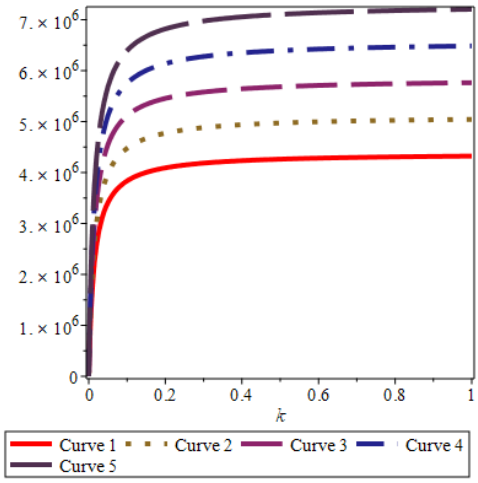

Fig. 3.2. Effect of K on R_0 when P is varied

Fig. 2 shows how the basic reproduction number R_0 depends on the various weights of K with different P as K decreases, the basic reproduction number decreases, that is increasing the protection period can do active effect on Tuberculosis control.

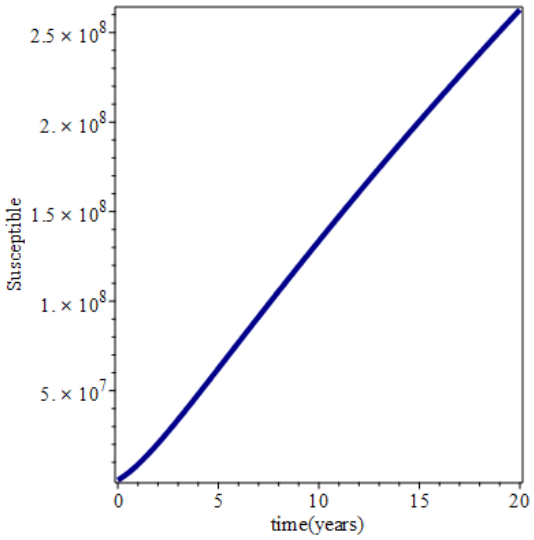

Fig. 3. Graph of susceptible individual in the absence of treatment against time (t)

Fig. 3 shows that the susceptible class are not dependent on the treatment, once infected, one will not return to susceptible class even after being treated as there is no permanent cure for Tuberculosis, the susceptible class increases due to increase in recovery rate.

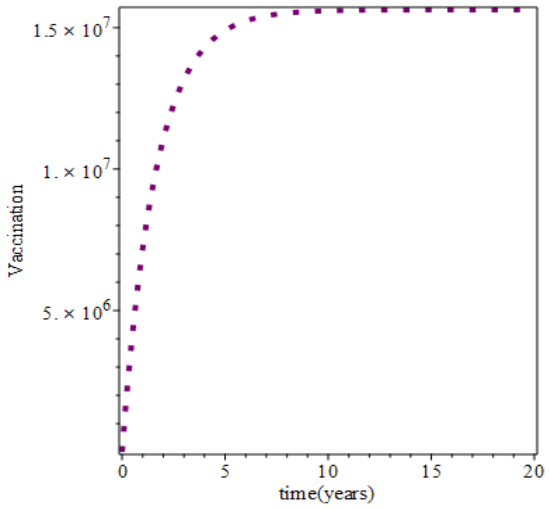

Fig. 4. Effect of vaccination

Fig. 4 shows that increase in vaccination rates lead to a decline in transmission and death rate and increase in the protection period against the virus.

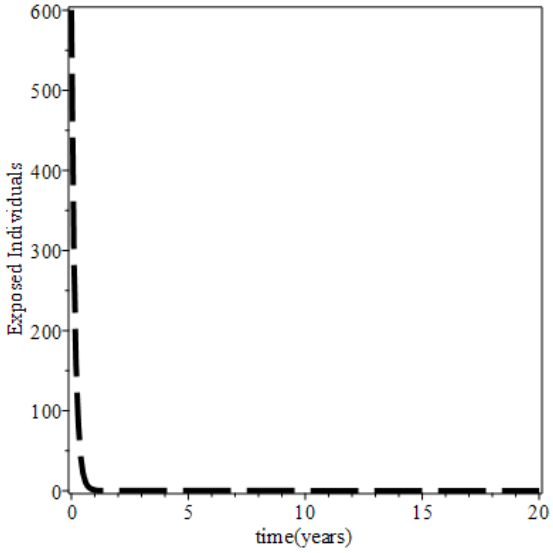

Fig. 5. Graph of exposed individuals in the presence of treatment against time(t)

Fig. 5 shows that the exposed individual reduces because the vaccine is effective, we can see how treatment affects the latently infected individuals by lowering the graph from its peak.

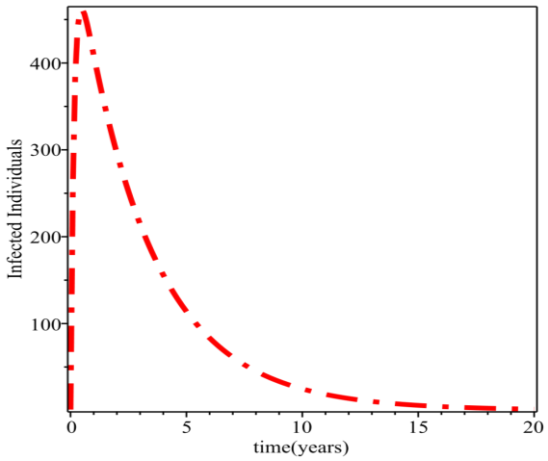

Fig. 6. Graph of infected individuals in the presence of treatment against time(t)

Fig. 6 shows that the infected individual reduces because the vaccine is effective, we can also see from the graph that the total population will eventually die out in about 20 years' time.

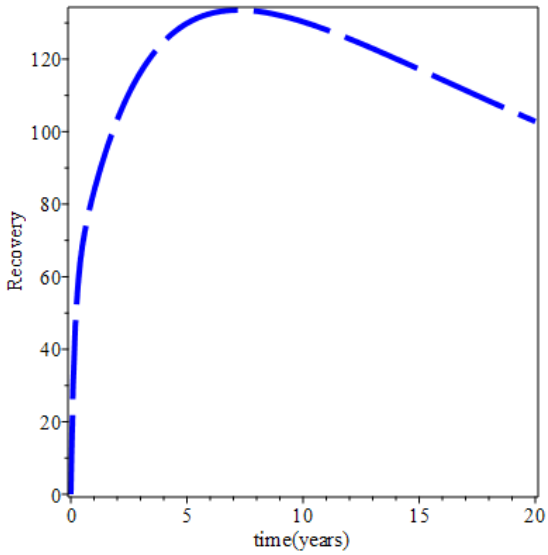

Fig. 7. Graph of recovered individual in the presence of treatment against time(t)

Fig. 7 shows a big difference to the recovered population when treatment is given. The graph sky rocketed to its peak before it gradually declined as there is a decrease in the number of individuals in the exposed and infectious classes.

Fig. 8 is a simulation showing the trend of Exposed (E) or latently infected individuals. The Infected individuals (I) and the recovered individuals(R) over 20 years' time period. The most distinct characteristics is that the number of infected individuals increased to maximum almost immediately at the beginning before gradually decreasing and eventually dies out due to the high rate of re-infection for the exposed class. Even though treatment is administered to both latently and actively infected individuals, the number of recovered individual is very small. The result of the treatment can only be seen in the first year of implementation represented by slight bump on the graph. This can be explained by high rate of re-infection and relapse cases

for recovered individuals since the disease mortality rate is considerably high, the whole total population eventually will die out in about 20 years' time.

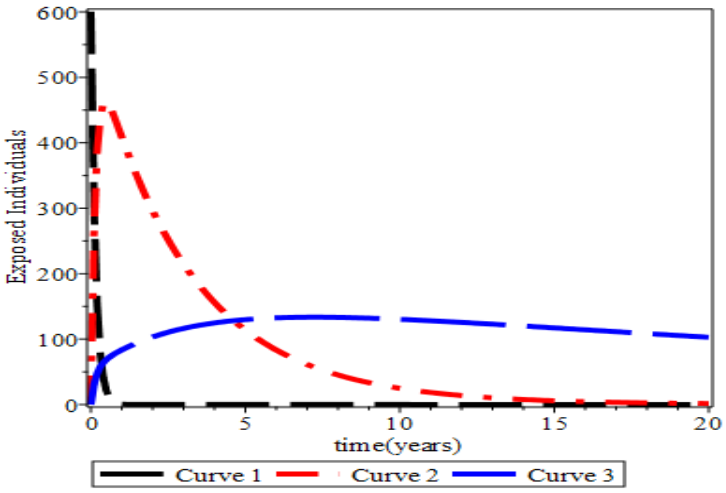

Fig. 8. The latently infected and the infectious population with and without treatment

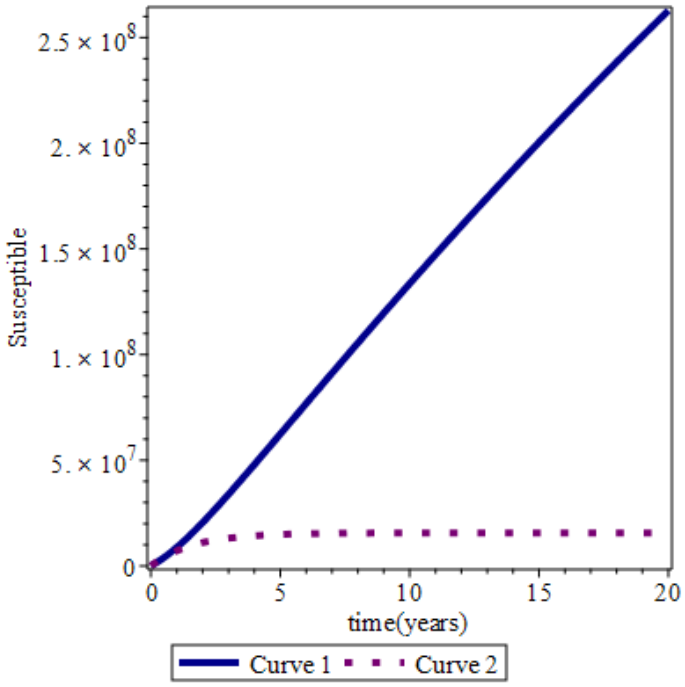

Fig. 9. Effect of vaccination to susceptible individuals only

Fig. 9 shows that vaccination and treatment in the model can increase the recovery rate thereby increasing the susceptible class since there is no permanent cure for Tuberculosis i.e. after recovery, the recovered individual will still have to return to the susceptible class. Hence, increase in recovery rate due to the vaccine will increase the numbers of susceptible individuals.

IV. CONCLUSION

This work modelled and analyzed the dynamics of Tuberculosis with the role vaccination and treatment play in the dynamics of Tuberculosis in Nigeria. The result of the stability analysis of the disease free equilibrium state shows that it is possible to effectively control or even completely eradicate tuberculosis in any population if effort is made to ensure that the product of total contraction and total breakdown of the susceptible class must be greater than the total removal rate from both the latent and the infectious classes. This can be achieved through more effort at immunizing newborn babies through vaccination and treatment of both the latently infected and actively infected individuals.

The result from the graph indicates that the rate of infected individual decreased due to treatment and vaccination parameters. The existing model was modified by adding ΨR i.e. the rate at which actively infected individuals recover from Tuberculosis infection to the susceptible class. Exploring the objective functional value, it was discovered that early treatment of Tuberculosis is optimal and Optimal treatment can be actualized when treatment is given within a specific number of days. Vaccination and treatment strategies are effective in reducing the transmission of Tuberculosis..

REFERENCES

[1] McCluskey, C.C. and van den Driessche, P. (2004): Global Analysis of Two Tuberculosis Models. *Journal of Dynamics and Differential Equations*, Vol.16, 139-166.

[2] Jia Z.W, Tang G.Y, Zhen Jin, Dye C., Vlas S.J. and Li X.W. (2008): Modeling the Impact of Immigration on the Epidemiology of Tuberculosis. *Theoretical Population Biology*, 73, 437–448.

[3] Bhunu, C.P., Garira, W., Mukandavire, Z. and Zimba, M. (2008): Tuberculosis Transmission Model with Chemoprophylaxis and Treatment. *Bulletin of Mathematical Biology*, 70, 1163-1191.

[4] Z. Zhang, W. Zhang, K.S. Nisar, N. Gul, A. Zeb and V. Vijayakumar (2023): Dynamical aspects of a tuberculosis transmission model incorporating vaccination and time delay. *Alexadria Engineering Journal, Alexandria University, Elsevier*; Vol. 66, 287-300.

[5] J. Gichuki, D. Mategula, Characterisation of tuberculosis mortality in informal settlements in Nairobi, Kenya: Analysis of data between 2002 and 2016. *BMC Infect. Dis.* 21 (2021), 718.

[6] R. Aggarwal, Y. A. Raj, A fractional order HIV-TB co-infection model in the presence of exogenous reinfection and recurrent TB. *Nonlinear. Dynam,* 104(4)(2021), 4701-25.

[7] S. Kasereka Kabunga, E. F. Doungmo Goufo, V. Ho Tuong, Analysis and simulation of a mathematical model of tuberculosis transmission in Democratic Republic of the Congo. *Adv. Differ. Equ.* 2020(2020), 642.

[8] N. P. Mnyambwa, D. Philbert, G. Kimaro, S. Wandiga, B. Kirenga, B. T. Mmbaga, E. Ngadaya, Gaps related to screening and diagnosis of tuberculosis in care cascade in selected health facilities in East Africa countries: A retrospective study. *J. Clin. Tuberc. Other Mycobact. Dis*. 25(2021), 100278.

[9] S. Lee, H.-Y Park, H. Ryu, J.-W. Kwon, Age-specific mathematical model for tuberculosis transmission dynamics in South Korea. *Mathematics*, 9(2021), 804.

[10] M. A. Kuddus, M. T. Meehan, M. A. Sayem, E. S. McBryde, Scenario analysis for programmatic tuberculosis control in Bangladesh: A mathematical modelling study. *Sci. Rep.* 11(2021), 4354. https:// doi. org/ 10. 1038/ s41598- 021- 83768-y.
[11] M. A. Kuddus, M. T. Meehan, L. J. White, E. S. McBryde, A. I. Adekunle, Modeling drug-resistant tuberculosis amplification rates and intervention strategies in Bangladesh. *PLoS ONE*, 15(2020), e0236112.
[12] NABTi, Abderrazak. "Dynamical analysis of an age-structured SEIR model with relapse." Zeitschrift für angewandte Mathematik und Physik 75.3 (2024): 84.
[13] Zahli, Rizlane, Khadija Zeroual, and Nadia Idrissi Fatmi. "Tuberculosis and HIV/AIDS co-dynamics: a mathematical model and sensitivity analysis." *Commun. Math. Biol. Neurosci*. 2024 (2024): Article-ID.
[14] Ansori, Fandi. "Mathematical Modeling and Sensitivity Analysis of COVID-19 and Tuberculosis Coinfection with Vaccination." *Mathematical Modelling of Engineering Problems* 11.1 (2024).
[15] Jain, Kshama, Anuradha Bhattacharjee, and Srikumar Krishnamurhty. "Mathematical analysis of COVID-19 and TB co-infection dynamics with optimal control." *Modeling Earth Systems and Environment* 11.1 (2025): 1-22.
[16] Rashid, Saima, et al. "Theoretical and mathematical codynamics of nonlinear tuberculosis and COVID-19 model pertaining to fractional calculus and probabilistic approach." *Scientific Reports* 14.1 (2024): 8827.
[17] Akanni, J. O., S. Ajao, and S. F. Abimbade. "Dynamical analysis of COVID-19 and tuberculosis co-infection using mathematical modelling approach." *Mathematical Modelling and Control* 4.2 (2024): 208-229.
[18] Shah, Kamil, et al. "Modeling the dynamics of co-infection between COVID-19 and tuberculosis with quarantine strategies: A mathematical approach." *AIP Advances* 14.7 (2024).
[19] Zahli, Rizlane, Khadija Zeroual, and Nadia Idrissi Fatmi. "Tuberculosis and HIV/AIDS co-dynamics: a mathematical model and sensitivity analysis." *Commun. Math. Biol. Neurosci.* 2024 (2024): Article-ID.
[20] Ayele, Tigabu Kasie, Emile Franc Doungmo Goufo, and Stella Mugisha. "Co-infection mathematical model for HIV/AIDS and tuberculosis with optimal control in Ethiopia." *PloS one* 19.12 (2024): e0312539.