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Review Article

Stability Analysis of Mathematical Modeling on the Spread of Tuberculosis Case Detection

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Abstract

In this study, a mathematical model on the transmission dynamics of tuberculosis was formulated and analyzed. The basic reproduction number (R_o) for each model is calculated and determined using the next generation method and condition for elimination (disease free equilibrium) or persistence (endemic equilibrium) in a population. Stability analysis shows that disease free equilibrium is locally asymptotically stable whenever the reproduction is less than unity. Furthermore, tuberculosis case detection continued to persist whenever the reproduction number exceeds unity. However, the models consisting of system of first order nonlinear differential equations.

Keywords: Tuberculosis, Case detection and Reproduction Number.

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1. INTRODUCTION

Tuberculosis (TB) is a chronic bacterial infectious disease caused by Mycobacterium tuberculosis which pose a major health, social and economic burden globally, especially in low and middle income countries [7]. It is the second deadliest disease due to a single infectious agent only after HIV/AIDS [10]. The surge in HIV-TB co-infection and growing emergence of multidrug-resistant TB (MDR-TB) and extensively drug resistant TB (XDR-TB) strains has further fuelled TB epidemic [19]. TB usually affects the lungs (pulmonary TB) but it can also affect other sites as well (extra-pulmonary TB). Tuberculosis is transmitted by tiny airborne droplets which are expelled into the air when a person with active pulmonary TB coughs or talks [12].

According [20], it was Estimate that TB has infected one-third of the world's population with the most infections occurring in Africa and Asia. In 2015, there were an estimated 10.4 million new TB cases (incidence) in worldwide, as well as an estimated 1.4 million TB-induced deaths, and an additional 0.4 million deaths resulting from TB disease among persons living with HIV. Furthermore, over 95% of these deaths occurred in low- and middle-income countries where the cost of diagnosis and treatment is high, and not readily accessible [21].

There was an estimated 3 million gaps between incident and notified TB cases globally, reflecting a combination of an under-diagnosis of cases and underreporting of cases that have been detected [22].

Globally, TB incidence was falling at about 2% per year and between 2015 and 2020 the cumulative reduction was 11%. This was over half way to the end TB strategy milestone of 20% reduction 2015 and 2020 [23]. People infected with TB bacteria have 5- 10% lifetime risk of failed ill with TB. Those with compromised immune systems, such as people living with HIV, malnutrition or diabetes, or people who used tobacco, have a high risk of falling ill, (2021).

Diagnosis of latent TB infections (LTBI) and prompt treatment of active cases remains an important component of effective TB control [2]. On the other hand, undetected TB infection and delay in the treatment of active TB cases leads to more severe disease conditions in th e infected person which could result in wider disease spread in the community [3]. Such delays also contribute to increased infectivity in the community [2], whereby, the infected individuals unknowingly continue to serve as a reservoir for the pathogen (M. tuberculosis). Hence, this could lead to increased risk of disease transmission in the community.

In fact, the effects of diagnosis of LTBI and delay in the treatment of active TB cases on the incidence and prevalence of TB were issues considered by some presenters at the 45th World Conference on Lung Health held in 2014. Health literacy, i.e., knowledge and education related to tuberculosis, as well as socio-cultural factors such as gender roles and status in the family has been identified as having considerable influence on undetected latent TB infection and delay in the treatment of active cases [13]. TB knowledge includes the ability to identify causes and understand the transmission path of the disease, recognize disease symptoms, and be aware of available treatment regimens such as directly observed treatment short course (DOTS); which is the TB treatment strategy recommended by the [6]. On the other hand, ignorance on the part of infected individuals about TB usually leads to postponement in seeking appropriate medical attention, and in some cases such persons will rather adopt alternative approaches, such as seeking traditional healers, before consulting DOTS facility, thereby delaying diagnosis and treatment of TB [15].

Pulmonary tuberculosis is usually diagnosed when symptomatic individuals seek care at health care facilities, and healthcare workers have a minimal role in promoting the health seeking behavior. However, some policy specialist believes the health care system could be more active in tuberculosis case detection [14]. People who are infected with Mycobacterium tuberculosis initially developed latent tuberculosis, where the infected was contained by the immune system and the person remains well [18].

Treatment of drug sensitive pulmonary tuberculosis requires patients to take a combination of medicines for six to nine months [20], while drug resistant forms typically require much longer courses. Guideline in some high burden counties advises health workers to consider pulmonary tuberculosis in all people with cough lasting more than two weeks [20]. Moreover, most people diagnosed with tuberculosis have been coughing for much longer than the time they were tested [11].

Maximizing early case detection and prompt treatment of notified cases is very useful in tuberculosis control especially in high burden countries. The tracking of TB contacts provides a good platform for early diagnosis, educating the household on TB disease and infection control as well as breaking the chain of transmission [16].

Early tuberculosis case detection is important for early commencement of treatment to improve treatment outcomes and also to prevent community spread of the disease. Moreover, there is paucity of data in Ghana on the efficiency of the Symptom – Based Screening tool (SBS tool) to detect Mycobacterium tuberculosis in the communities [8].

Since a number of studies have been carried - out to analyze the roles of mathematical model in tuberculosis as mentioned above and ways to control the spread of the disease in the communities. This work extract the works of [1, 17, 4, 9] by incorporating $\left[I_R\right]$ and $\left[I_P\right]$ for the rich infectious individuals and poor infectious individuals respectively. It further extracts the work of [5] by incorporating $\left[I_D\right]$ for detection infectious individual.

2. Model formulation

The total homogeneously-mixing population at time t, denoted by N(t), is sub-divided into mutually-exclusive compartments of susceptible (S(t)), exposed/latent (E(t)), Rich Infectious Individuals ($I_R(t)$), Poor Infectious Individuals ($I_P(t)$), Detected Infectious Individuals ($I_D(t)$), Failed Treated individuals ($T(t)$, Effectively Treated Individuals ($J(t)$), and together with Recovery Individuals ($R(t)$), so that:

$$N(t) = S(t) + E(t) + I_R(t) + I_P(t) + I_D(t) + T(t) + J(t) + R(t) \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots (1)$$

The susceptible population is increased by the recruitment of people (either by birth or immigration) into the population (all recruited individuals are assumed to be susceptible), at a rate λ. This population is decreased by infection, which can be acquired following effective contact with infectious individuals in the Rich Infectious Individuals ($I_R(t)$), Poor Infectious Individuals ($I_P(t)$), Detected Infectious Individuals ($I_D(t)$), Failed Treated individuals ($T(t)$ categories, at a rate Γ given as:

$$\Gamma = \beta\left(\frac{I_P + \eta_1 I_R + \eta_2 I_D + \eta_3 T}{N}\right) \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots (2)$$

Description of parameters

μ	Natural death rate
θ	Progression rate from exposed compartment to infectious compartment
q	Proportion of poor infectious individual
δ_1	TB induced death rate for rich infectious individual
δ_2	TB induced death rate for poor infectious individual
δ_3	TB induced death rate for detected infectious individual
δ_4	TB induced death rate for failed treated individual
ϕ_1	Detection rate for infectious rich individuals
ϕ_2	Detection rate for infectious poor individuals
σ_1	Recovery rate for infectious rich individuals
σ_2	Recovery rate for infectious poor individuals
σ_3	Recovery rate for infectious detected individuals
σ_4	Recovery rate for infectious failed treated individuals
τ	Treatment rate of detected individuals
w	Proportion of detected individuals whose treatment failed (was not successful)
β	Effective contact rate for susceptible individuals with infectious poor individuals
η_1	Modification parameter associated to infectious rich individuals
η_2	Modification parameter associated to detected individuals
η_3	Modification parameter associated to unsuccessful treated individuals
λ	Recruitment rate

3. Equilibrium State and Stability Analysis of the Model

Model Equation

$$\frac{dS}{dt} = \lambda - \Gamma S - \mu S$$

$$\frac{dE}{dt} = \Gamma S - (\theta + \mu)E$$

$$\frac{dI_R}{dt} = (1-q)\theta E - (\mu + \delta_1 + \phi_1 + \sigma_1) I_R$$

$$\frac{dI_P}{dt} = q\theta E - (\mu + \delta_2 + \phi_2 + \sigma_2) I_P$$

$$\frac{dI_D}{dt} = \phi_1 I_R + \phi_2 I_P - (\mu + \delta_3 + \sigma_3 + \tau) I_D \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (3)$$

$$\frac{dT}{dt} = \omega\tau I_D - (\mu + \delta_4 + \sigma_4) T$$

$$\frac{dJ}{dt} = (1-\omega)\tau I_D - \mu J$$

$$\frac{dR}{dt} = \sigma_1 I_R + \sigma_2 I_P + \sigma_3 I_D + \sigma_4 - \mu R$$

Where

$$\Gamma = \beta\left(\frac{I_P + \eta_1 I_R + \eta_2 I_D + \eta_3 T}{N}\right) \quad \text{..... (x)}$$

$$N = S + E + I_p + I_R + I_D + T + J + R \quad \text{..... (xx)}$$

EQUILIBRIUM POINT

At equilibrium Point, $\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI_P}{dt} = \frac{dI_R}{dt} = \frac{dI_D}{dt} = \frac{dT}{dt} = \frac{dJ}{dt} = \frac{dR}{dt} = \frac{dN}{dt} = 0$

So equations (3) becomes

$$\lambda - \Gamma S - \mu S = 0$$
$$\Gamma S - k_1 E = 0$$
$$L_1 E - k_2 I_p = 0$$
$$L_2 E - K_3 I_R = 0$$
$$\phi_1 I_R + \phi_2 I_P - K_4 I_D = 0 \quad \text{..... (4)}$$
$$G_1 I_D - K_5 T = 0$$
$$G_2 I_D - \mu J = 0$$
$$\sigma_1 I_R + \sigma_2 I_p + \sigma_3 I_D + \sigma_4 T - \mu R = 0$$

Where,

$L_1 = q\theta$, $L_2 = (1-q)\theta$, $G_1 = \omega\tau$ an $G_2 = (1-\omega)\tau$, $K_1 = \theta + \mu$, $K_2 = (\mu + \delta_2 + \phi_2 + \sigma_2)$, $K_3 = \mu + \delta_1 + \phi_1 + \sigma_1$, $K_4 = \mu + \delta_3 + \sigma_3 + \tau$, $K_5 = \mu + \delta_4 + \sigma_4$

Disease Free Equilibrium

Let P_0 denote the disease free equilibrium (i.e. the equilibrium point in the absence of infection). Thus, by definition, $\Gamma = 0$ and from (4), we respectively obtained

$$S = \frac{\lambda}{\mu} \text{ , } E = 0 \text{ , } I_P = 0 \text{ , } I_R = 0 \text{ , } I_R = 0 \text{ , } I_D = 0 \text{ , } T = 0 \text{ , } J = 0 \text{ , } R = 0 \quad \text{..... (5)}$$

Thus,

$$P_0 = \left(S^0, E^0, I_p^0, I_R^0, I_D^0, T^0, J^0, R^0\right) = \left(\frac{\lambda}{\mu}, 0, 0, 0, 0, 0, 0, 0\right)$$

Computation of Basic Reproductive Number (R_v)

$$F = \begin{pmatrix} 0 & \beta & \beta\eta_1 & \beta\eta_2 & \beta\eta_3 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

$$V=\begin{pmatrix} k_1 & 0 & 0 & 0 & 0 & 0 & 0 \\ -L_1 & k_2 & 0 & 0 & 0 & 0 & 0 \\ -L_2 & 0 & k_3 & 0 & 0 & 0 & 0 \\ 0 & -\phi_2 & -\phi_1 & k_4 & 0 & 0 & 0 \\ 0 & 0 & 0 & -G_1 & k_5 & 0 & 0 \\ 0 & 0 & 0 & -G_2 & 0 & -\mu & 0 \\ 0 & -\sigma_2 & -\sigma_1 & \sigma_3 & \sigma_4 & 0 & -\mu \end{pmatrix}$$

$$R_v=\beta\left[\frac{k_4k_5(k_3L_1+k_2L_2\eta_1)+(k_5\eta_2+G_1\eta_3)A_1}{k_1k_2k_3k_4k_5}\right] \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots \quad (6)$$

Where

$$A_1=K_2L_2\phi_1+K_3L_1\phi_2$$

Endemic Equilibrium Point

Let P_1 denote the endemic equilibrium of the model (that is, the equilibrium where at least one of the infected components of the model is non zero). Thus from (4), we have the following:

$$S=\frac{\lambda}{\Gamma+\mu}\ ,\ E=\frac{\Gamma S}{k}\ ,\ I_P=\frac{L_1\Gamma S}{k_1k_2}\ ,\ I_R=\frac{L_2\Gamma S}{k_1k_3}\ ,\ I_D=\frac{\Gamma SA_1}{k_1k_2k_3k_4}\ ,\ T=\frac{\Gamma SG_1A_1}{k_1k_2k_3k_4k_5}\ ,\ J=\frac{\Gamma SA_1G_2}{k_1k_2k_3k_4\mu} \text{ and}$$

$$R=\frac{\Gamma SA_2}{\mu k_1k_2k_3k_4k_5} \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots \quad (7)$$

$$A_2=k_4k_5\left[k_2L_2\sigma_1+L_1k_3\sigma_2\right]+\left(\sigma_3k_5+\sigma_4G_1\right)A_1$$

Substituting (7) in to (xx) to have

$$N=S\left(1+\frac{\Gamma}{k_1}+\frac{L_1\Gamma}{k_1k_2}+\frac{L_2\Gamma}{k_1k_3}+\frac{\Gamma A_1}{k_1k_2k_3k_4}+\frac{A_1G_1\Gamma}{k_1k_2k_3k_4k_5}+\frac{\Gamma A_1G_2}{k_1k_2k_3k_4\mu}+\frac{\Gamma A_2}{\mu}\right)\ N=S\left(1+\Gamma A_3\right) \quad \ldots.. \quad (8)$$

Where

$$A_3=k_4k_5\mu\frac{\left[k_2L_2+k_3\left(k_2+L_1\right)\right]+A_1\left[\mu\left(k_5+G_1\right)+G_2k_5\right]+A_2}{\mu k_1k_2k_3k_4k_5}$$

Substitute (7) in to (x) to obtain

$$\Gamma=\frac{\beta\left(I_p+\eta_1I_R+\eta_2I_D+\eta_3T\right)}{N}$$

$$\Gamma N=\beta\Gamma S\left(\frac{L_1}{k_2k_2}+\frac{\eta_1L_2}{k_1k_3}+\frac{\eta_3A_1}{k_1k_2k_3k_4}+\frac{\eta_3G_1A_1}{k_1k_2k_3k_4k_5}\right)$$

$$\Gamma N=\frac{\beta\Gamma S\left(k_4k_5\left(k_3L_1+\eta_2L_2K_2\right)+\eta_3\left(k_5+G_1A_1\right)\right)}{k_1k_2k_3k_4k_5} \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots \quad (9)$$

Simplifying (9) with the aid of (6) to get

$$\Gamma N=\Gamma SR_v$$

$$\therefore\frac{S}{N}=\frac{1}{R_v} \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots \quad (10)$$

Substitute (8) in to (10) to have

$$\Gamma = \frac{R_v - 1}{A_3} \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (11)$$

Expressing (7) in term of R_v respectively as:

$$S = \frac{A_3\lambda}{(R_v - 1) + \mu A_3}$$

$$E = \frac{\lambda(R_v - 1)}{K_1}$$

$$I_p = \frac{L_1\lambda(R_v - 1)}{K_1K_2}$$

$$I_R = \frac{L_2\lambda(R_v - 1)}{K_1K_3}$$

$$I_D = \frac{A_1\lambda(R_v - 1)}{K_1K_2K_3K_4} \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (12)$$

$$T = \frac{G_1A_1\lambda(R_v - 1)}{K_1K_2K_3K_4K_5}$$

$$J = \frac{G_2\lambda(R_v - 1)}{\mu K_1K_2K_3K_4}$$

$$R = \frac{A_2\lambda(R_v - 1)}{\mu K_1K_2K_3K_4K_5}$$

Thus the components of $P_1 = (S^+, E^+, I_P^+, I_R^+, I_D^+, T^+, J^+, R^+)$ and expressed by (12) respectively.

LOCAL STABILITY at DISEASE FREE EQUILIBRIUM (DFE)

Theorem1: The disease free equilibrium P_o of the model is locally asymptotically stable for $R_v < 1$ and unstable for $R_v > 1$.

Proof

The Jacobian matrix of the model evaluated at P_o is obtained as

$$J(P_o) = \begin{bmatrix} -\mu & 0 & -\beta & -\beta\eta_1 & -\beta\eta_3 & 0 & 0 \\ 0 & X & X & X & X & X & X \\ 0 & X & X & X & X & X & X \\ 0 & X & X & X & X & X & X \\ 0 & X & X & X & X & X & X \\ 0 & X & X & X & X & X & X \\ 0 & X & X & X & X & X & X \end{bmatrix} \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (13)$$

Where

$$X=\begin{bmatrix} -K_1 & \beta & \beta\eta_1 & \beta\eta_2 & \beta\eta_3 & 0 & 0 \\ L_1 & -K_2 & 0 & 0 & 0 & 0 & 0 \\ L_2 & 0 & -K_3 & 0 & 0 & 0 & 0 \\ 0 & \phi_2 & \phi_1 & -K_4 & 0 & 0 & 0 \\ 0 & 0 & 0 & G_1 & -K_5 & 0 & 0 \\ 0 & 0 & 0 & G_2 & 0 & -\mu & 0 \\ 0 & \sigma_2 & \sigma_1 & \sigma_3 & \sigma_4 & 0 & -\mu \end{bmatrix} \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots (14)$$

Clearly, $-X=\left[x_{ij}\right]$ is a 7x7 matrix with $x_{ij}\le 0$, for $i\neq j$, $i,j=1,...,7$ and $x_{ii}>0$ for $i=1,...,7.$
Subsequently, we have the positive vector d defined as:

$$d=\left[\frac{L_1}{K_2},\frac{L_2}{K_3},\frac{A_1}{K_2K_3K_4},\frac{A_1G_1}{K_2K_3K_4K_5},\frac{A_1G_2}{\mu K_2K_3K_4},\frac{A_2}{\mu K_2K_3K_4K_5}\right]^T \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots (15)$$

If $R_v<1, -Xd=\left[K_1(1-R_v),0,0,0,0,0,\right]^T\ge 0$

$$\left|-X\right|=\mu^2\left(K_1K_2K_4K_5-\beta\left[K_4K_5\left(K_3L_1+K_2L_2\eta_1\right)+(K_5\eta_2+G_1\eta_3)A_1\right]\right) \quad \ldots\ldots\ldots\ldots\ldots (16)$$

Simplifying (16) using (6) to have

$$\left|-X\right|=\mu^2\left(K_1K_2K_3K_4K_5\right)\left(1-R_v\right) \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (17)$$

Thus, for $R_v<1$, $\left|-X\right|>0$ and $-X$ has non zero eigenvalue.
Finally, if $R_v<1$, $-X$ is an irreducible matrix with $x_{ii}>0$ and $x_{ij}\le 0\ \left(i\neq j\right)$, there exist a positive vector d such that $-Xd\ge 0$. Hence the real part of each non zero eigenvalue of $-X$ is positive according M- matrix theory , that is , each eigenvalue of X has negative real part. Thus concluding the proof

LOCAL STABILITY OF ENDEMIC EQUILIBRIUM POINT (EEP)

The locally asymptotically stability of the (EEP) is explained for a special case, where the disease – induced death rate for individuals in I_P, I_R, I_D, and T compartment are assumed to be negligible and is set to zero. Thus, under this setting (with $\delta_1=\delta_2=\delta_3=\delta_4=0$), the rate of change of the total population of the model becomes:

$$\frac{dN}{dt}=\lambda-\mu N$$

Thus $N(t)\to\frac{\lambda}{\mu}=N^*$ as $t\to\infty$ and $S=N^*-I_P-E-I_R-I_D-T-J-R$ ……. (18x)

Substituting $N=N^*$ in to (x) gives

$$\Gamma=\frac{\beta\left(I_P+\eta_1I_R+\eta_2I_D+\eta_3T\right)}{N^*} \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (19)$$

Thus (6) becomes

$$R_v^*=\frac{\beta\left[K_4'K_5'\left(K_3'L_1+K_2'L_2\eta_1\right)+\left(K_5'\eta_2+G_1\eta_3\right)A_1'\right]}{K_1'K_2'K_3'K_4'K_5'} \quad \ldots\ldots\ldots\ldots\ldots (20)$$

Where

$K_1^{'} = \mu + \phi_2 + \sigma_2$, $K_3^{'} = \mu + \phi_1 + \delta_1$, $K_4^{'} = \mu + \delta_3 + \tau$, $K_5^{'} = \mu + \delta_4$ and $A_1^{'} = K_2^{'} L_2 \phi_1 + K_3^{'} L_1 \phi_2$

Similarly, the components of the endemic equilibrium of the model under these setting $(\delta_1 = \delta_2 = \delta_3 = \delta_4 = 0)$ denoted by P_2 are giving by

$$S^* = \frac{A_3^{'}\lambda}{R_v^* + \mu A_3^{'}}, \quad E^* = \frac{\lambda\left(R_v^* - 1\right)}{K_1^{'}}, \quad I_P^* = \frac{L_1\lambda\left(R_v^* - 1\right)}{K_1^{'}K_2^{'}}, \quad I_R^* = \frac{L_2\lambda\left(R_v^* - 1\right)}{K_1^{'}K_3^{'}}, \quad I_D^* = \frac{A_1^{'}\lambda\left(R_v^* - 1\right)}{K_1^{'}K_2^{'}K_3^{'}K_4^{'}},$$

$$T^* = \frac{G_1 A_1^{'}\lambda\left(R_v^* - 1\right)}{K_1^{'}K_2^{'}K_3^{'}K_4^{'}K_5^{'}}, \quad J^* = \frac{G_2\lambda\left(R_v^* - 1\right)}{\mu K_1 K_2^{'}K_3^{'}K_4^{'}} \text{ and } R^* = \frac{A_2^{'}\lambda\left(R_v^* - 1\right)}{\mu K_1 K_2^{'}K_3^{'}K_4^{'}K_5^{'}} \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (21)$$

Where

$$A_2^{'} = K_4^{'}K_5^{'}\left[K_2^{'}L_2\delta_1 + L_1K_3^{'}\delta_2\right] + \left(\delta_3 K_5^{'} + \delta_4 G_1\right)A_1^{'}$$

Assuming (18x), the model is reduced to

$$\begin{aligned}
\frac{dE}{dt} &= \Gamma\left(N^* - E - I_P - I_R - I_D - T - J - R\right) - K_1 E \\
\frac{dI_P}{dt} &= L_1 E - K_2 I_P \\
\frac{dI_R}{dt} &= L_2 E - K_3 I_R \\
\frac{dI_D}{dt} &= \phi_1 I_R + \phi_2 I_P - K_4 I_D \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (22) \\
\frac{dT}{dt} &= G_1 I_D - K_5 T \\
\frac{dJ}{dt} &= G_2 I_D - \mu J \\
\frac{dR}{dt} &= \sigma_1 I_R + \sigma_2 I_P + \sigma_3 I_D + \sigma_4 T - \mu R
\end{aligned}$$

Theorem : The endemic equilibrium P_2 of (22) is locally asymptotically stable if $R_v^* > 1$ and unstable otherwise.

Proof

The proof of the above theorem is based on the kransnoselskii .By linear Zing (22) at P_2 gives

$$\begin{aligned}
\frac{d\mathrm{E}}{dt} &= \left(-a_1 - K_1\right)E + \left(a_2 - a_1\right)I_P + \left(a_2\eta_1 - a_1\right)I_R + \left(a_2\eta_2 - a_1\right)I_D + \left(a_2\eta_3 - a_1\right)T - a_1 J - a_1 R \\
\frac{dI_P}{dt} &= L_1 E - K_2 I_P \\
\frac{dI_R}{dt} &= L_2 E - K_3 I_R \\
\frac{dI_D}{dt} &= \phi_1 I_R + \phi_2 I_P - K_4 I_D \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (23) \\
\frac{dT}{dt} &= G_1 I_D - K_5 T
\end{aligned}$$

$$\frac{dJ}{dt} = G_2 I_D - \mu J$$

$$\frac{dR}{dt} = \sigma_1 I_R + \sigma_2 I_P + \sigma_3 I_D + \sigma_4 T - \mu R$$

Where

$$a_1 = \frac{\beta\left(I_P + \eta_1 I_R + \eta_2 I_D + \eta_3 T\right)}{N^*} \text{ and } a_2 = \frac{\beta S^*}{N^*}$$

Thus the Jacobian matrix of (23) gives

$$J\left(P_2\right) = \begin{bmatrix} \left(-a_1 - K_1\right) & a_2 - a_1 & a_2\eta_1 - a_1 & a_2\eta_2 - a_1 & a_2\eta_3 - a_1 & -a_1 & -a_1 \\ L_1 & -K_2 & 0 & 0 & 0 & 0 & 0 \\ L_2 & 0 & -K_3 & 0 & 0 & 0 & 0 \\ 0 & \phi_2 & \phi_1 & -K_4 & 0 & 0 & 0 \\ 0 & 0 & 0 & G_1 & -K_5 & 0 & 0 \\ 0 & 0 & 0 & G_2 & 0 & -\mu & 0 \\ 0 & \sigma_2 & \sigma_1 & \sigma_3 & \sigma_4 & 0 & -\mu \end{bmatrix} \quad \ldots\ldots\ldots\ldots\ldots\ldots (24)$$

Now, the linearized system (23) at P_2 is shown not to solution of the form

$$\overline{Z}\left(t\right) = \overline{Z_o} e^{gt} \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (25)$$

With $\overline{Z_o} = \left(Z_1, Z_2, ..., Z_7\right)$, $Z_i \in C, g \in C$ and $R_e g \geq 0$

(This implies that the eigenvalue of the characteristic equation of (24) will have negative real part, thus P_2 is locally asymptotically stable). Substituting the solution of the form (25) into system (23) gives:

$$\begin{aligned} gZ_1 &= \left(-a_1 - K_1\right)Z_1 + \left(a_2 - a_1\right)Z_2 + \left(a_2\eta_1 - a_1\right)Z_3 + \left(a_2\eta_2 - a_1\right)Z_4 + \left(a_2\eta_3 - a_1\right)Z_5 - a_1 Z_6 - a_1 Z_7 \\ gZ_2 &= L_1 Z_1 - K_2 Z_2 \\ gZ_3 &= L_2 Z_1 - K_3 Z_3 \\ gZ_4 &= \phi_1 Z_3 + \phi_2 Z_2 - K_4 Z_4 \\ gZ_5 &= G_1 Z_4 - K_5 Z_5 \\ gZ_6 &= G_2 Z_4 + \mu Z_6 \\ gZ_7 &= \sigma_1 Z_3 + \sigma_2 Z_2 + \sigma_3 Z_4 + \sigma_4 Z_5 - \mu Z_7 \end{aligned} \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (26)$$

Simplify (26) to get

$$\begin{aligned} \left[1 + F_1\left(g\right)\right]Z_1 &= \left(MZ\right)_1 \\ \left[1 + F_2\left(g\right)\right]Z_2 &= \left(MZ\right)_2 \\ \left[1 + F_3\left(g\right)\right]Z_3 &= \left(MZ\right)_3 \\ \left[1 + F_4\left(g\right)\right]Z_4 &= \left(MZ\right)_4 \\ \left[1 + F_5\left(g\right)\right]Z_5 &= \left(MZ\right)_5 \\ \left[1 + F_6\left(g\right)\right]Z_6 &= \left(MZ\right)_6 \end{aligned} \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (27)$$

$$\left[1+F_7(g)\right]Z_7=(MZ)_7$$

Where

$$F_1(g)=\frac{g+a_1\left(1+Q_1+Q_2+Q_3+Q_4+Q_5+Q_6\right)}{K_1},\ F_2(g)=\frac{g}{K_2},\ F_3(g)=\frac{g}{K_3},\ F_4(g)=\frac{g}{K_4},$$

$$F_5(g)=\frac{g}{K_5},\ F_6(g)=\frac{g}{\mu},\ F_7(g)=\frac{g}{\mu},\ Q_1=\frac{L_1}{g+K_2},\ Q_2=\frac{L_2}{g+K_3},\ Q_3=\frac{\phi_1Q_2+\phi_2Q_1}{K_4+g}$$

$$Q_4=\frac{G_1Q_3}{K_5+g},\ Q_5=\frac{G_2Q_3}{g+\mu},\ Q_6=\frac{\sigma_1Q_2+\sigma_2Q_1+\sigma_3Q_3+\sigma_4Q4}{\mu+g}$$

With

$$M=\begin{bmatrix}
0 & \frac{\beta S}{NK_1} & \frac{\beta S\eta_1}{NK_1} & \frac{\beta S\eta_2}{NK_1} & \frac{\beta S\eta_3}{NK_1} & 0 & 0\\
\frac{L_1}{K_2} & 0 & 0 & 0 & 0 & 0 & 0\\
\frac{L_2}{K} & 0 & 0 & 0 & 0 & 0 & 0\\
0 & \frac{\phi_2}{K_4} & \frac{\phi_1}{K_4} & 0 & 0 & 0 & 0\\
0 & 0 & 0 & \frac{G_1}{K_5} & 0 & 0 & 0\\
0 & 0 & 0 & \frac{G_2}{\mu} & 0 & 0 & 0\\
0 & \frac{\sigma_2}{\mu} & \frac{\sigma_1}{\mu} & \frac{\sigma_3}{\mu} & \frac{\sigma_4}{\mu} & 0 & 0
\end{bmatrix} \quad \text{..... (28)}$$

The equilibrium $P_2=\left(E^*,I_P^*,I_R^*,I_D^*,T^*,J^*,R^*\right)$ satisfies $P_2=MP_2$ and $\left((MZ)_i\,C_i=1,...,7\right)$ denotes the i^{th} coordinate of the vector MZ, and the matrix M has non-negative entries. If Z is a solution of (27) , then it is possible to find a minimal positive real number q such that

$$\|Z\|\le qP_2 \quad \text{..... (29)}$$

Where $\|Z\|=\left(\|Z_1\|,\|Z_2\|,\|Z_3\|,\|Z_4\|,\|Z_5\|,\|Z_6\|,\|Z_7\|\right)$ with lexicographic order, and $\|.\|$ is a norm C. The aim is to establish that $\mathrm{R}_e(g)<0.$ we assume by contradiction that $R_e(g)\ge 0$, thus two cases shall be considered.

Case 1: g=0

This case implies that (26) is homogeneous linear system in the variable $Z_i\,(i=1,...,7)$. The determinant of this system gives

$$\Delta=-\mu\left[a_1A_3+\mu K_1K_2K_3K_4K_5\left(1-\frac{S^*}{N^*}R_v^*\right)\right] \quad \text{..... (30)}$$

Where

$$A_3 = \mu\left[G_1A_1 + K_5\left[K_3L_1\left(\phi_2 + K_4\right) + K_2K_4\left(K_3 + L_2\right)\right]\right] + \left(A_2 + G_2K_5A_1\right)$$

Evaluating (10) at P_2 gives $\frac{S^*}{N^*} = \frac{1}{R_v^*}$ …… (31)

Substituting (30) into (31) to get

$\Delta = -\mu a_1$, $A_3 < 0$ …….. (32)

It follows that, system (32) has a unique solution given by $Z = 0$ (which corresponds to DFE ($\left(P_0\right)$).

Case 2: $g \neq 0$

Since $R_e\left(g\right) > 0$, then $\left|1 + F_i\left(g\right)\right| > 1$ for all $i = 1, 2, ..., 7$. Define $F\left(g\right) = \min\left|1 + F_i\right|$. Then, $F\left(g\right) > 1$ and $\frac{q}{F\left(g\right)} < g$. Furthermore, since q is a minimal positive real number such that $\|Z\| \leq qP_2$, it follows then that

$$\|Z\| > \frac{q}{F\left(g\right)}P_2 \text{ …….. (33)}$$

On the other hand, by taking the norm of both sides of the second equation in (74), it follows that:

$$F\left(g\right)\|Z_2\| \leq \left|1 + F_2\left(g\right)\right|, \|Z_2\| = \|(MZ)_2\| \leq M\|Z_2\| \leq qM(P_2)_2 = q(P_2)_2 = qI_P^* \text{ …….. (34)}$$

Thus from (34),

$\|Z_2\| \leq \frac{q}{F(g)}I_P^*$, which contradict (33). Hence $\mathrm{R}_e\left(g\right) < 0$. Thus, all eigenvalues of the characteristic equation associated with the linearized system (23s) will have negative real part, so that P_2 is locally asymptotically stable whenever $R_v > 1$.

4. CONCLUSIONS

In this study, a non linear mathematical model was formulated and analyzed to study the transmission dynamic of tuberculosis case detection in population varying with time. The model formulated consists of system of first order non linear differential equations. The equilibrium points of the model were obtained in term of basic reproduction number. The disease free equilibrium was shown to be locally asymptotically stable whenever the reproduction number is less than unity. This implies that the menace of tuberculosis case detection can be eradicated in population (the disease will die out). Furthermore, tuberculosis case detection will continue to persist whenever the reproduction numbers exceed unity. This means that the disease will remain in population.

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