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Local Stability of the Effects of Early Detection and Treatment on the Dynamics of Tuberculosis Using Lyapunov Function Method

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Authors' contributions

This work was carried out in collaboration between all authors. Author YIA designed the study, performed the statistical analysis and wrote the first draft of the manuscript. Authors JA and JAK managed the analyses of the study. All authors read and approved the final manuscript.

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ABSTRACT

This study aims to use Lyapunov function method to build a SEIR model in the analysis of early detection and treatment. The SEIR model is a system of ordinary differential equations of six dimension developed from our compartment then building a mathematical theorem which guarantees the existence of a case of TB, the disease free-equilibrium and the total eradication of the disease from its host community that is disease endemic TB. Three theorems were proved using Lyapunov function method. With these, we concluded that in this research work have given a complete stability analysis of a tuberculosis model with two differential infectivity classes of early detected infected individual and late detected infected individual. By analysing this model, we found that it is locally asymptotically stable and possesses the only locally stable equilibrium state depending on the basic reproductive ratio R_0 this steady state is either the endemic or the disease-free. The local stability of the infection-free equilibrium state implies that for an initial level of infection the disease will eventually fade out from the population when the condition for the stability, number $R_0 \leq 1$, hold. The condition $R_0 > 1$, implies that the disease will persist in a population.

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Keywords: TB disease; SEIR model; Lyapunov function; local stability; disease free and endemic.

1. INTRODUCTION

Tuberculosis (TB) is a bacterium disease caused by Mycobacterium called tuberculosis Bacilicer. It is also an infectious disease of human and animals characterised by the growth of nodules tuberculosis in tissues especially lungs chiefly caused by the bacterium of mycobacterium. There are two forms of tuberculosis, infection mycobacterium.

Man is the main host. The microbes caused pulmonary tuberculosis and spread either by a droplet of infection from individuals with active TB or in dust contaminated by infected sputum.

The second is mycobacterium Bovis: animals are the main host, the microbes are usually spread to men by untreated milk from infected cows, causing infection of the alimentary tract.

Tuberculosis (TB) is an infectious disease caused by the Mycobacterium remains one of the world's deadliest diseases [1]. According to the World Health Organization [2], about 9 million people were infected, worldwide, with TB and 1.5 million deaths from the disease were reported, 360,000 of whom were HIV-positive while 480,000 people contracted multi-drug resistant (World Health Organization, 2015). Tuberculosis is seen to be declining slowly each year and an estimated 37 million lives were saved between 2000 and 2013 through effective diagnosis and treatment (World Health Organization, 2015). On the average, TB incidence fell to about 1.5% per year, between 2000 and 2013, worldwide (World Health Organization, 2015). Globally, TB mortality rate fell by an estimated 45% between 1990 and 2013 while the prevalence rate dropped by 41% (World Health Organization, 2015).

1.1 Tuberculosis Affects Family

Although the overall number of TB deaths continues to fall across the globe, new data showed 50 percent more cases exist in India than previously thought, so the total number of cases worldwide has increased from 9.6 million to 10.4 million. Six countries - India, Indonesia, China, Nigeria, Pakistan and South Africa - account for 60 percent of the total number of people with TB.

Goosby (2016) the United Nations Special Envoy on Tuberculosis, told VOA in a Skype interview that because of better surveillance, the numbers are more accurate. But on the other hand, he said, not all cases of TB are being counted, because many countries have outdated surveillance systems.

Even with such positive results achieved within the last 14 years, it is still thought that deaths from tuberculosis are preventable; in fact, the death toll is still considered unacceptably high. Hence, efforts are geared toward accelerating programmes that will result in a reduction in the TB burden globally [within the context of the Millennium Development Goals (MDGs)], and reach the Stop TB Partnership target of a 50% reduction by 2030 (World Health Organization, 2014). More than half of the approximately 9 million individuals who are infected with tuberculosis in 2013 (56%) were in South-East Asia and Western Pacific Regions. A further one quarter of these infected individuals is in the African Region, which accounts for the highest rates of TB cases and deaths relative to population [1].

Early diagnostic and treatment services require strengthening in many settings in line with existing guidelines. Strengthening of laboratory services (for sputum-smear microscopy, culture, drug-susceptibility testing and new diagnostics) (World Health Organization, 2010) and X-ray services The Hague, (2008) is essential. Sputum smear microscopy is inexpensive and feasible in most field conditions and effectively identifies the most infectious TB cases; however, this technique has lower sensitivity to detect smear-negative, culture positive TB, especially among people living with HIV. New and better diagnostic tools with proven usefulness and affordability should be scaled up rapidly, including tools for diagnosis of MDR-TB The Hague, (2010). Further investment is needed for discovery and testing of new diagnostic tools.

A potential major obstacle for early and complete TB case detection is that 10–25% of bacteriologically-confirmed cases do not report any symptoms early in the disease course, as demonstrated in prevalence surveys China, (2010). Such cases can be identified only through screening of all people, regardless of symptoms, using chest X-ray or other highly

sensitive screening tools. These data suggest that TB screening, using investigations other than asking about symptoms, in selected risk groups is indicated in order to reach early those people who do not experience symptoms and therefore are unlikely to seek care.

A survey reported in Okuonghae and Omosigho (2010) listed some factors that can adversely affect the implementation of the directly observed treatment and early detection of TB short-course (DOTS) strategy in Nigeria (one of the high burden countries) in reducing the incidence of TB in the country. The survey revealed that most persons do not know how TB is transmitted and the signs and symptoms of tuberculosis; several individuals are not even aware of the government's health policies on tuberculosis and TB treatment. Further, the survey revealed that this lack of awareness to early detect TB can lead to delays in reporting cases for treatment (Okuonghae and Omosigho, 2010), increasing the likelihood of disease transmission.

Most of these models are of the SEIR class in which the host population is categorised by infection status as susceptible, exposed (infected but not yet infectious), infectious and recovered. One of the principal attributes of these models is that the force of infection (the rate at which susceptible leave the susceptible class and move into an infected category, i.e., become infected) is a function of the number of infectious hosts in the population at any time t and is thus a nonlinear term. Other transitions, such as the recovery of infectious individuals and death, are modelled as linear terms with constant coefficients. However, the enormous public health burden inflicted by tuberculosis necessitates the use of mathematical modelling to gain insights into it transmission dynamics and to determine effective treatment strategies. We present the use of Lyapunov stability models of the differential equation. Eradication of this disease from community occurs only when there is no more exposed and infected individual in the population. This only occurs in the absence of infectious and latently infected individual in the population.

2. MATERIALS AND METHODS

2.1 Assumption

We shall use the following assumption for the mathematical models of tuberculosis. The human population is categorised into six classes such that at time $t \geq 0$ there are S, susceptible humans, E, exposed humans to tuberculosis, I_{ED}, early-detected infected humans with active tuberculosis, I_{LD}, later detected infected humans with active tuberculosis, R, recovered humans.

i. In our model, the recruitment into the susceptible (S) human population is by births (λ). The size of the human population is further increased by the partially immune humans in (*R)* after they lose their immunity at the rate (ρ).
ii. Here we assume a homogeneous mixing of individuals in the population which means that every uninfected human have an equal likelihood of being infected when coming in contact with infected humans and that transmission of the infection occurs with a standard incidence rate.
iii. We also assume that some recruits, that is, newborns and immigrants, may possibly be exposed (E) at the time they are born or migrate into the population.
iv. Thus they will emerge in the susceptible class, (S) at a rate, λ, the exposed class at a rate (E) or the infective class at a rate (I), the treatment class at a rate (T), and the recovered class at a rate (R).
v. Infected individuals recover from the symptoms of TB after treatment (T).
vi. Infection into the group varies as from early detected I_{ED} and late detected I_{LD}.
vii. We also assume that there is no permanent immunity for TB individual.
viii. We further assume that all parameters to be used in this model are positive.

See Tables 1 and 2 for the description of the state variables and parameters respectively which will be followed by the resulting differential equations.

2.2 Variables and Parameters

Table 1. Definition of state variables

Variable	Definition
S(t)	Susceptible humans
E(t)	Exposed humans
$I_{ED}(t)$	Infected early detected humans
$I_{LD}(t)$	Infected later detected humans
T(t)	Treatment
R(t)	Recovered humans

Table 2. Description of parameters

Parameter	Description
B	Rate at which the susceptible become exposed to Mtb
H	Infection rate for early detected
Σ	Infection rate for late detected
A	Disease induced death rate
$\aleph_1$	Recovery rate due to prompt treatment
$\aleph_2$	Recovery rate due to delayed treatment
Γ	Recovery rate after treatment
P	Rate at which the recovered lose their immunity
μ	Rate of natural death
Λ	Rate of recruitment

2.3 Model Building

2.3.1 Model description

The size of the human population is decreased by natural deaths (μ) and exposure to Mtb. exposed susceptibles to Mtb move to the exposed classes *E* with the force of infection being β*I* resulting in an increase in the exposed class. The exposed class is further decreased by natural deaths (μ) and the proportion that move to the infected class (η) and (σ) after developing active tuberculosis. The early detected infected class I_{ED} is reduced by natural deaths (μ) and those who undergo treatment (r_1), while those in the late detected infected class I_{LD} is also reduced by natural deaths (μ), disease induced death (α), and those who undergo treatment (r_2). Those who suffered treatment are reduced by natural death μ, disease induced deaths (α), and those who recover (γ) from the diseases thread. Thus the treatment class increase by two infected class (*I*) who gain partial immunity at the rates (r_1) and (r_2) respectively thus moving to the recovered class *R* thus reducing their respective classes by (γ) and also increasing the recovered class while the recovered class is reduced by natural deaths (μ) and those who lose their partial immunity at the rate ρ. The standard SEIRS mathematical model was extended for the transmission of tuberculosis which will demonstrate the transmission of the Mycobacterium tuberculosis in human hosts taking into account the multidrug-resistant (MDR) tuberculosis [3]. Most classical models developed for studying tuberculosis dynamics often ignore the resistant class and early/late detection and if they include them, they end up with very complicated models. Side et al. [3] seek to present a simple model that can easily be analysed so as to properly understand the dynamics and stability of this disease.

Humans can contract Mtb tuberculosis through contact with individuals who are infected with the disease. After which they enter the exposed (latent) phase where a proportion of this class develop active tuberculosis thus moving into the two infectious classes. If treatment is administered promptly, those who recover from the disease will move to the recovered class and those who delay treatment as a result of late detection and develop MDR tuberculosis will move to the resistant class, die or recovered eventually. Those who recover from MDR tuberculosis will move to the recovered cla. Given that there is no permanent immunity to tuberculosis, the recovered can lose their immunity and become susceptible again [4].

From our assumptions, parameters and variables in section 2.1 and 2.2 we obtain the below flow diagram and it has been constructed with these assumptions: recruitment is by births only, a variable population, a constant mortality rate, no permanent immunity to tuberculosis, no immediate infectivity.

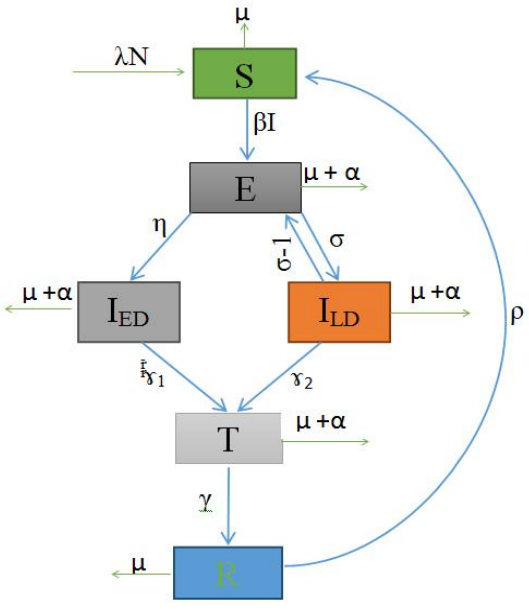

Fig. 1. Schematics of the compartmental model. State variables: S, susceptible human, E, exposed human with Mtb, I_{ED} and I_{LD}, Infected human with active tuberculosis, R, recovered humans from both active tuberculosis and MDR tuberculosis

Equations of the Model: Applying the assumptions and the flow diagram in Fig. 1, we obtained the following differential equations:

$$\begin{aligned}
\frac{ds}{dt} &= \lambda N - \beta SI - \mu S + \rho R \\
\frac{dE}{dt} &= \beta SI - (\mu + \eta + \sigma)E + \sigma(1 - r_2)I_{LD} \\
\frac{dI_{ED}}{dt} &= \eta E - (\mu + \alpha + r_1)I_{ED} \\
\frac{dI_{LD}}{dt} &= \sigma E - (\mu + \alpha + r_2)I_{LD} - \sigma(1 - r_2)I_{LD} \\
\frac{dT}{dt} &= r_1 I_{ED} + r_2 I_{LD} - (\mu + \alpha + \gamma)T \\
\frac{dR}{dt} &= \gamma T - \mu R - \rho R
\end{aligned} \tag{1}$$

Thus the size of the human population is given as $N = S + E + I_{ED} + I_{LD} + T + R$.

3. RESULTS AND DISCUSSION

Invariant Region:

Note that $$\frac{dN}{dt} \geq \lambda N - \mu N \tag{2}$$

We now apply Birkhoff and Rota's theorem on differential inequality (4.4). By separation of variables of differential inequality (4.4), we get

$$\frac{dN}{\lambda N - \mu N} \geq dt \tag{3}$$

Integrating (4.5) on both side we have

$$\int \frac{dN}{\lambda N - \mu N} \geq \int dt = \frac{-1}{\mu}\ln(\lambda N - N) + k$$

$$\ln(\lambda N - N) \geq -\mu(t + k)$$

Therefore, taking the ln of both side we have

$$\lambda N - N \geq D\ell^{-\mu t} \tag{4}$$

where Δ is a constant. Now, applying the initial condition N(0) = N0 in (4), we get

$$D = \lambda N - \mu N_0 \tag{5}$$

Substituting for D in (4) gives

$$\lambda N - \mu N \geq \lambda N - \mu N_0 \ell^{-\mu t} \tag{6}$$

Making N the subject formula in (6) we have,

$$N \leq \frac{\lambda N}{\mu} - \left[\frac{\lambda N - \mu N_0}{\mu}\right]\ell^{-\mu t} \tag{7}$$

As $\tau \to \infty$ in (7) above, the population size N, approaches

$$0 \leq N \leq \frac{\lambda N}{\mu} \Rightarrow N \to \frac{\lambda N}{\mu} \tag{8}$$

Note that when there is no disease in the population, $N = \frac{\lambda N}{\mu}$, and it is naturally expected that the spread of the disease in the population will reduce $N\left(i.e, N < \frac{\lambda N}{\mu}\right)$ the feasible region of the model system (1) is

$$\Omega_\varepsilon = \left\{S(t), E(t), I_{ED}(t), I_{LD}(t), T(t), R(t) \in R^6_{,+} : N \leq \frac{\lambda N}{\mu}\right\}$$

In this case, whenever $N > \frac{\lambda N_0}{\mu}$, then $\frac{dN}{dt} < 0$ which means that $N \to \frac{\lambda N}{\mu}$ on the other hand, whenever $N \leq \frac{\lambda N_0}{\mu}$, every solution with the initial condition in R^6_+ remains in that region for $\tau > 0$. Thus, the region Ω_ε is positively-invariant. Where ε is a positive constant with respect to model system (1)

Positivity of solutions for our model: Since the system (1) is dealing with a population of TB, all the variables and parameters of the model are non-negative. It was claimed the following:

Lemma 4.2.1. Let the initial data be $\{(S_0, E_0, I_{ED0}, I_{LD0}, T_0, R_0) \geq 0\} \in \Omega_\varepsilon$. then, the solution set $\{S(t), E(t), I_{ED}(t), I_{LD}(t), T(t), R(t)\}$ of system (4.1) is positive for all *t>0*

Proof: Let $\lambda = \beta I$. From the first equation of model system (1),

That is, $$\frac{dS}{dt} = \lambda N - \lambda S - \mu S + \rho R \geq -(\lambda + \mu)S \quad (9)$$

$$\frac{dS}{dt} \leq -(\lambda + \mu)S.$$

Integrating (9) by separation of variables gives

$$\int \frac{dS}{S} \geq -\int (\lambda + \mu)dt.$$

Therefore,

$$S(t) \geq S(0)e^{-\int (\lambda+\mu)dt} > 0.$$

This proves that $\Sigma(\tau) > 0$ for all $\tau \geq 0$. Similarly, it can be shown that the remaining variables of system (1) are also positive $\forall \tau > 0$.

Remarks for $e^k > 0 \text{ for all } k \in \mathbb{R}.$

Theorem (1) Let $\{S(t) \geq 0, E(t) \geq 0, I_{ED}(t) \geq 0, I_{LD}(t) \geq 0, T(t) \geq 0, R(t) \geq 0\}$ completion of the system (1) with the initial condition or state $S_0, E_0, I_{ED0}, I_{LD0}, T_0, R_0$ compact set in e.g. (10)

$$D = \{S(t), E(t), I_{ED}(t), I_{LD}(t), T(t), R(t) \in R_+^6 L \leq N\}$$

To model the system (1), this positively invariant seen that covers all settlement in R^6_+.

Proof: consider the Lyapunov function candidate for the following:

$$L(t) = S + E + I_{ED} + I_{LD} + T + R$$

Derivation of the function with respect to time satisfied in (10)

$$\frac{dL}{dt} = S^1 + E^1 + I_{ED}^1 + I_{LD}^1 + T^1 + R^1$$

$$\lambda N - \beta SI - \mu S + \rho R + \beta SI - (\mu + \eta + \sigma)E + \sigma(1 - r_2)I_{LD} + \eta E - (\mu + \alpha + r_1)I_{ED}$$

$$+ \sigma E - (\mu + \alpha + r_2)I_{LD} - \sigma(1 - r_2)I_{LD} + r_1 I_{ED} + r_2 I_{LD} - (\mu + \alpha + \gamma)T$$

$$+ \gamma T - \mu R - \rho R$$

$$\frac{dL}{dt} = \lambda N - \mu N - \alpha N \Rightarrow \frac{dL}{dt} = \lambda N - \mu L(t) - \alpha L(t)$$

Not difficult to prove that equation (2) below

$$\frac{dL}{dt} = \lambda N - \mu L(t) - \alpha L(t) \leq 0 \, for L \geq N \quad (10)$$

Then, from the above equation, it is known as $\frac{dL}{dt} \leq 0$, that means D is a set of position invariant. Conversely, by completing the system (1) is obtained that, $0 \leq L(t) \leq N + L(0)e^{-\mu t}$ which $L(0)$ is the limited condition of $L(t)$.

Therefore as $t \to \infty, 0 \leq L(t) \leq N$ and conclude that D is a set of positive invariant and cover all of the settlement in R_+^6. This proves the theorem, this theorem guarantees the existence of TB disease in an area that was initially not found a virus carrier TB bacteria, then changed after the discovery at the population suspected but not yet infected, S(t) > 0, exposed TB E(t) >0 early infected TB I_{ED} (t) >0 late detected infected TB I_{LD}(t) >0, treated TB T(t) >0 Recovered TB, R(t) >0.

This theorem also gives the conclusion that further investigation of TB cases on this stage can identify the disease spread endemic to the stage using our model.

Local Stability of Disease – Free Equilibrium for Our Model: System (1) always has a disease-free equilibrium $P^* = \left(S^*_{,} E^*_{,} I^*_{,} T^*_{,} R^*_{,}\right) = \left(\frac{\lambda N}{\mu}, 0,0,0,0,0\right)$ which means the disease will disappear. This section will examine the behaviour of the local balance of disease free for the system.

Theorem 3: if $R_o \leq 1$, the disease free equilibrium P* for our model is stable asymptotic local stage in D.

Proof: suppose candidate lyapunov function is in equation. (12)

W (t) = (S – S* in S) + E + I_{ED} + I_{LD} + T + R (12)

By differentiating function of time obtained by the following equation

$$\dot{W}(t) = S^1\left(1 - \frac{S^*}{S}\right) + E + I^1_{ED} + I^1_{LD} + T^1 + R^1$$

$$W(t) = \lambda \mathrm{N} - \beta SI - \mu S + \rho R\left(1 - \frac{S^*}{S}\right) + \beta SI - \left(\mu + \eta + \sigma\right)\mathrm{E} + \sigma(1 - \gamma_2) I_{ED} + \eta \mathrm{E} - \left(\mu + \alpha + \gamma_1\right) I_{ED}$$
$$+ \sigma E - \left(\mu + \alpha + r_2\right) I_{LD} - \sigma\left(1 - r_2\right) I_{LD} + r_1 I_{ED} + r_2 I_{LD} - \left(\mu + \alpha + \gamma\right) T + \gamma T - \rho R$$

$$W(t) = \lambda \mathrm{N} - \beta \mathrm{SI} - \mu S + \rho R - \lambda \mathrm{N}\frac{S^*}{S} + \beta \mathrm{I} S^* + \mu S^* - \rho R\frac{S^*}{S} + \beta \mathrm{SI} - \mu \mathrm{E} - \eta \mathrm{E} - \sigma \mathrm{E} + \sigma\left(1 - \gamma_2\right)\mathrm{I}_{LD} + \eta \mathrm{E}$$
$$- \mu \mathrm{I}_{ED} - \alpha \mathrm{I}_{ED} - r_1 \mathrm{I}_{ED} + \sigma \mathrm{E} - \mu \mathrm{I}_{LD} - \alpha \mathrm{I}_{LD} - r_2 - \sigma\left(1 - \gamma_2\right)\mathrm{I}_{LD} + r_1 \mathrm{I}_{ED} + r_2 \mathrm{I}_{LD} - \mu T - \alpha T - \gamma T + \gamma T$$
$$- \mu R - \rho R$$

$$W(t) = \lambda \mathrm{N}\left(1 - \frac{S^*}{S}\right) - \mu\left(1 - \frac{S^*}{S}\right) + \rho R\left(1 - \frac{S^*}{S}\right) - \mu\left(\mathrm{E} + I_{ED} + I_{LD} + T + R\right) - \alpha\left(I_{ED} + I_{LD} + T\right)$$

$$W(t) = \lambda \mathrm{N}\left(1 - \frac{S^*}{S}\right) - \mu\left(1 - \frac{S^*}{S}\right) + \rho R\left(1 - \frac{S}{S^*}\right) - \mu \mathrm{N} - \alpha \mathrm{N}$$

Let μ and ρR be equal to λN so that our model becomes

$$= \lambda N\left(1 - \frac{S^*}{S}\right) + \lambda N\left(1 - \frac{S^*}{S}\right) + \lambda N\left(1 - \frac{S}{S^*}\right) - \mu\left(E + I_{ED} + I_{LD} + T + R\right)$$

$$= \lambda N\left(3 - \frac{\mathrm{S}^*}{\mathrm{S}} - \frac{\mathrm{S}^*}{\mathrm{S}} - \frac{\mathrm{S}}{\mathrm{S}^*}\right) + \mu\left(E + I_{ED} + I_{LD} + T + R\right)$$

$$= \lambda N\left(\frac{S - S^*}{S}\right)\left(\frac{S - S^*}{S}\right)\left(\frac{S - S}{S^*}\right) \tag{13}$$

$$= \lambda N\left(\frac{3S^2 S^* + S^{*3} - 3SS^{*2} - S^3}{SS^*}\right) + \mu\left(E + I_{ED} + I_{LD} + T + R\right)$$

$$= \lambda N\left(\frac{S - S^*}{SS^*}\right)^3 + \mu\left(E + I_{ED} + I_{LD} + T + R\right)$$

Therefore $\dot{W}(t) \leq 0$ and by using advance Lassel (1979) on lyapunov theorem, final set of defined each settlement in contained in the layest in variant set $S = S^*$, then the reproduction ratio $R_0 = 0$ is a singleton {P*}. This means that the disease-free equilibrium P* is the local stage is a stable asymptotic in D. this concludes the proof.

Local stability theorem for this model describes the state of the existence of TB cases as described in theorem (1). This step explains that if an individual is infected with TB but $R_6 \leq 1$, it means that it will not cause other individuals to be infected it also means that in the region TB disease can still be controlled and overcome since at that stage is not alarming as a result of it been early detected and treated immediately.

Local Stability of the Endemic Equilibrium for Our Model: Simplify our model in the system of eqn (1) we obtained the following equations.

$$\begin{aligned}
&\lambda N - \beta SI - \mu S + \rho R = 0 \\
&\beta SI - (\mu + \eta + \sigma)E + \sigma(1 - r_2)I_{LD} = 0 \\
&\eta E - (\mu + \alpha + r_1)I_{ED} = 0 \\
&\sigma E - (\mu + \alpha + r_2)I_{LD} - \sigma(1 - r_2)I_{LD} = 0
\end{aligned} \tag{14}$$

System (6) has an equilibrium point $P^{**} = \left(S^{**}, E^{**}, I_{ED}^{**}, I_{LD}^{**}\right) \in D$ known as endemic equilibrium and satisfied $S^{**} > 0, E^{**} > 0, I_{ED}^{**} > 0, I_{LD}^{**} > 0$ with

$$S^{**} = \frac{\lambda N + \rho R}{\beta I + \mu}$$

$$E^{**} = \frac{\beta SI + \sigma(1 - r_2)I_{ED}^{**}}{(\mu + \eta + \sigma)}$$

$$I_{ED}^{**} = \frac{\eta E^{**}}{(\mu + \alpha + r_1)}$$

$$I_{LD}^{**} = \frac{\sigma E^{**}}{(\mu + \alpha + r_2) + \sigma(1 - r_2)}$$

The following theorem will provide a global explanation of endemic equilibrium in (14)

Theorem 6 if $R_o > 1$, then the equilibrium state of endemic system exist and asymptotic local stage is stable on D, with assumption that in (15)

$$\begin{aligned}
&(\mu + \alpha + r_1) = \frac{\eta E^{**}}{I_{ED}^{**}} \\
&S = S^{**} \\
&E = E^{**} \\
&(\mu + \alpha + r_2) + \sigma(1 - r_2) = \frac{\sigma E^{**}}{I_{LD}^{**}}
\end{aligned} \tag{15}$$

With $(\mu + \alpha + r_1)$ are the rate of infected but early detected to recovered individual and the death rate by the disease/natural and also $(\mu + \alpha + r_2) + \sigma(1 - r_2)$ are the rate of the infected individual but late detected to recovered humans as well as the rate at which the recovered humans lust their immunity and died by the disease/natural from the existing population.

Proof: suppose a lyapunov function in equation (16) below

$$\begin{aligned}
W(t) &= \left(S - S^{**} \ln S\right) + \left(E - E^{**} \ln E\right) + \left(I_{ED} - I_{ED}^{**} \ln I_{ED}\right) + \left(I_{LD} - I_{LD}^{**} \ln I_{LD}\right) \\
&= \left(S^1 - S^{**} \ln S\right) + \left(E^1 - E^{**} \ln E\right) + \left(I_{ED}^1 - I_{ED}^{**} \ln I_{ED}\right) + \left(I_{LD}^1 - I_{LD}^{**} \ln I_{LD}\right) \\
&= S^1\left(1 - \frac{S^{**}}{S}\right) + E^1\left(1 - \frac{E^{**}}{E}\right) + I_{ED}^1\left(1 - \frac{I_{ED}^{**}}{I_{ED}}\right) + I_{LD}^1\left(1 - \frac{I_{LD}^{**}}{I_D}\right) \\
&= \lambda N - \beta SI - \mu S + \rho R\left(1 - \frac{S^{**}}{S}\right) + \beta SI - (\mu + \eta + \sigma)E + \sigma(1 - r_2)I_{ED}\left(1 - \frac{E^{**}}{E}\right) \\
&+ \eta E - (\mu + \alpha + r_1)I_{ED}\left(1 - \frac{I_{ED}^{**}}{I_{ED}}\right) + \sigma E - (\mu + \alpha + r_2)I_{LD} - \sigma(1 - r_2)I_{LD}\left(1 - \frac{I_{LD}^{**}}{I_{LD}}\right) \\
&= \lambda N - \beta SI - \mu S + \rho R - \lambda N\frac{S^{**}}{S} + \beta IS^{**} + \mu S^{**} + \rho R\frac{S^{**}}{S} + \beta SI - (\mu + \eta + \sigma)E + \sigma(1 - r_2)I_{ED} \\
&- \beta SI\frac{E^{**}}{E} + (\mu + \eta + \sigma)E^{**} - \sigma(1 - r_2)I_{ED}\frac{E^{**}}{E} + \eta E - (\mu + \alpha + r_1)I_{ED} - \eta E\frac{I_{ED}^{**}}{I_{ED}} + (\mu + \alpha + r_1)I_{ED}^{**} \\
&+ \sigma E - (\mu + \alpha + r_2)I_{LD} - \sigma(1 - r_2)I_{LD} - \sigma E\frac{I_{LD}^{**}}{I_{LD}} + (\mu + \alpha + r_2)I_{LD}^{**} + \sigma(1 - r_2)I_{LD}^{**}
\end{aligned} \tag{16}$$

Then we have

$$= \lambda N\left(1-\frac{S^{**}}{S}\right)+\beta I\left(1-\frac{S}{S^{**}}\right)+\mu\left(1-\frac{S}{S^{**}}\right)+\rho R\left(1-\frac{S^{**}}{S}\right)+\beta I\left(1-\frac{E^{**}}{E}\right)+\mu\left(1-\frac{E}{E^{**}}\right)+\eta\left(1-\frac{E}{E^{**}}\right)$$
$$+\sigma\left(1-\frac{E}{E^{**}}\right)+\sigma(1-r_2)I_{ED}\left(1-\frac{E^{**}}{E}\right)+\eta E\left(1-\frac{I_{ED}^{**}}{I_{ED}}\right)+\mu\left(1-\frac{I_{ED}}{I_{ED}^{**}}\right)+\alpha\left(1-\frac{I_{ED}}{I_{ED}^{**}}\right)+r_1\left(1-\frac{I_{ED}}{I_{ED}^{**}}\right)$$
$$+\sigma E\left(1-\frac{I_{LD}^{**}}{I_{LD}}\right)+\mu\left(1-\frac{I_{LD}}{I_{LD}^{**}}\right)+\alpha\left(1-\frac{I_{LD}}{I_{LD}^{**}}\right)+r_2\left(1-\frac{I_{LD}}{I_{LD}^{**}}\right)+\sigma(1-r_2)\left(1-\frac{I_{LD}}{I_{LD}^{**}}\right)$$

$$= \lambda N\left(1-\frac{S^{**}}{S}\right)+\rho R\left(1-\frac{S^{**}}{S}\right)+\beta I\left(1-\frac{S}{S^{**}}\frac{E^{**}}{E}\right)+\mu\left(1-\frac{S}{S^{**}}\frac{E}{E^{**}}\frac{I_{ED}}{I_{ED}^{**}}\frac{I_{LD}}{I_{LD}^{**}}\right)+\eta\left(1-\frac{E}{E^{**}}\right)+\sigma\left(1-\frac{E}{E^{**}}\right)$$
$$+\sigma(1-r_2)I_{ED}\left(1-\frac{E^{**}}{E}\right)+\eta E\left(1-\frac{I_{ED}^{**}}{I_{ED}}\right)+\alpha\left(1-\frac{I_{ED}}{I_{ED}^{**}}\frac{I_{LD}}{I_{LD}^{**}}\right)+r_1\left(1-\frac{I_{ED}}{I_{ED}^{**}}\right)+\sigma E\left(1-\frac{I_{LD}^{**}}{I_{LD}}\right)+r_2\left(1-\frac{I_{LD}}{I_{LD}^{**}}\right)$$
$$+\sigma(1-r_2)\left(1-\frac{I_{LD}}{I_{LD}^{**}}\right)$$

Substituting equation (7) above for which S* = S** E* = E** we have

$$= \mu\left(1-\frac{I_{ED}}{I_{ED}^{**}}\frac{I_{LD}}{I_{LD}^{**}}\right)+\eta E\left(1-\frac{I_{ED}^{**}}{I_{ED}}\right)+\alpha\left(1-\frac{I_{ED}}{I_{ED}^{**}}\frac{I_{LD}}{I_{LD}^{**}}\right)+r_1\left(1-\frac{I_{ED}}{I_{ED}^{**}}\right)+\sigma E\left(1-\frac{I_{LD}^{**}}{I_{LD}}\right)+r_2\left(1-\frac{I_{LD}}{I_{LD}^{**}}\right) \quad (17)$$
$$+\sigma(1-r_2)\left(1-\frac{I_{LD}}{I_{LD}^{**}}\right)=0 \Rightarrow W(t)\le 0$$

The above equation ensures that w(t) ≤ 0 for all S(t), E(t), $I_{ED}(t)$, $I_{LD}(t)$ and W(t) = 0 fulfilled if and only if S = S^{**} is only positive invariant set of system of our assumption which is contained entirely within N = S(t), E(t), $I_{ED}(t)$, $I_{LD}(t)$, S = S^{**}, E = E**, I_{ED} = I^{**}_{ED}, I_{LD} = I^{**}_{LD}} and subsequently by asymptotic stability theorem Lasalle J. P. [5] endemic positive balance. P^{**} is endemic asymptotic global stage is stable is in this proves the theorem.

Local stability theorem for SEIR model at this stage to explain that is an individual is infected with TB disease R_0>1, that if the individual is not early detected, then that individual will transmit the virus to other individuals. This means that at this stage of TB disease is endemic because it no longer can be controlled and is at an alarming stage, thus becoming a threat to the human population in the region. On the other hand if I_{LD} = I_{LD}** then R_0<1 that means an individual is early detected so that he cannot transmit the virus to other individuals and can no longer be spread therefore is not endemic because this stage of TB cannot be a threat to human population in the region Korobeinikov [6] and Korobeinikov and Maini [7] have used the Lyapunov function method for SEIR and SEIS epidemic models. Syafruddin and Noorani [8] and Tewa et al. [9] have used the Lyapunov function method for SIR an SEIS model dengue fever disease. Side [10] has made mathematical modelling SIR for tuberculosis disease but has not discussed the SEIR model, global stability and the Lyapunov function method. In this study, Lyapunov function method is used to SIR an SEIR model for tuberculosis disease.

4. CONCLUSION

This research project presents a comprehensive, continuous and more realistic Lyapunov functions in the dynamics of tuberculosis in the effect of early detection with treatment. In contrast to many TB models in the literature, we have included two infectious classes emanating from diagnosed and late diagnosed infectious. The late diagnosed subclass is of particular importance in modelling TB in developing countries like sub-Saharan Africa where public health is underdeveloped. In particular the proportion of individuals that present themselves to medical facilities, *h* is worth nothing. This parameter can be used to measure successes of

educational campaigns that encourage individuals to go for TB screening. It can also be a measure of the level of awareness of the implication of not having TB diagnosis. We further conclude that it is essential that the poorest and most vulnerable groups have access to quality assured diagnosis, treatment, care and support. Poor and vulnerable populations which are not early detected are those most likely to contract the infection, develop the disease, have poor treatment outcomes, and experience severe social and economic hardship from the disease. Specific action is therefore required to ensure equity linked to broader effort to strengthen health systems, especially at the community level.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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