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The Role of Polluted Air and Population Density in the Spread of Mycobacterium Tuberculosis Disease

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***Abstract*—Tuberculosis disease is an airborne disease caused by the bacterium called mycobacterium tuberculosis. In this paper we have classified human population into four compartments and designed mathematical model to describe the dynamics of tuberculosis disease. The pathogen population in the polluted air is considered as still another compartment. Formula for reproduction number is constructed and equilibrium point analysis is made. Numerical simulation study is conducted using ode45 of MATLAB. The results and interpretations are included.**

Keywords—Tuberculosis, Mathematical model, Basic reproduction number, Stability analysis, Numerical simulation

1. Introduction

Every year infectious diseases cause more than thirteen millions of deaths worldwide and hence require serious attention to fight against them. Two thirds of these diseases occur among the children under five years of age. The top infectious diseases claiming the most human lives include human immune deficiency virus (HIV), Tuberculosis (TB) and malaria [5]. Some infectious diseases like tuberculosis are air-borne while few others like cholera are water-borne. Variety types of TBs have been affecting human kind. Mycobacterium tuberculosis (MTB) is also an air-borne disease caused by the bacterium called mycobacterium. The tuberculosis disease is characterized by many symptoms including weight loss, fever, night sweats, and loss of appetite [6]. The mycobacterium tuberculosis is carried by airborne particles called droplet nuclei which are in general of 1 to 5 microns in diameter. The infectious droplet nuclei are generated and thrown into air while the persons of pulmonary or laryngeal TB disease cough, sneeze, shout, sing or even while they just talk [7]. Depending on composition of the environment these tiny particles can remain suspended in the air for several hours. Transmission occurs when a person inhales droplet nuclei containing mycobacterium tuberculosis, and the droplet nuclei traverse through the mouth or nasal passages, upper respiratory tract and bronchi to reach the alveoli of the lungs [6].

Tuberculosis most commonly affects the lungs yielding what is known as pulmonary active tuberculosis. Tuberculosis also can spread to other organs of human body such as bones etc. Tuberculosis meningitis, another type of tuberculosis, spreads to and affects human brain. Tuberculosis is a major cause of global mortality and morbidity especially in poor and developing countries where limited health care resources and weak health care systems are functioning. Over 80% of all tuberculosis patients live in 22 countries, most of them are in Sub-Saharan African and Asian continents [1]. It is therefore important that adequate attention is paid and effective control measures are initiated so that the spread of such diseases is stopped.

The purpose of the current study is to understand the dynamics of tuberculosis by (i) constructing a mathematical model, (ii) making stability analysis and (iii) performing simulation study. We will also make necessary recommendations based on the results of this study. We now list the stages of the disease. The total tuberculosis bacteria population found inside human body is divided mainly in to two categories viz., (i) intracellular bacteria which are found in lungs and (ii) extracellular bacteria which are found in other parts of human body outside the lungs. Intracellular bacteria or latent bacteria is considered as sleeping as they have been blocked by white blood cells while extracellular bacteria is considered as active as they are out of control of white blood cells.

Latent tuberculosis is one stage of tuberculosis. During this stage mycobacterium tuberculosis bacterium is already alive in the human body but it is inactive. A person of this stage does not show any symptoms of the disease or does not fall sick. With the aid of the presently available medical tests diagnose of the disease is not possible. As long as the natural immunity defense system is stronger and keeps the bacteria under control the mycobacterium tuberculosis bacteria does not spread to other parts of the body and is remains sleep in the human lungs or in the latent stage. On the other hand active tuberculosis disease can be positively diagnosed by any of the medical tests including skin test, blood test, chest x-ray, sputum smear or culture. The active tuberculosis affected person usually feels sick and develops symptoms such as coughing for 2-3 weeks or more, fever, unexplained weight loss, night sweats, fatigue, blood in the sputum or coughed up mucus, loss of appetite and chest pain[4].

We now brief the transmission factors of mycobacterium tuberculosis**.** The mycobacterium tuberculosis transmission depends on various factors that may include the following: (i) Susceptibility or immune status of a human. If an exposed individual has a weak immune system he or she will have high probability to develop active tuberculosis disease. (ii) Dens population areas. A person who lives in high population density area has more chances of getting infection than a person who lives in low population density area. (iii) Number of tubercle bacilli. Infectiousness of the person with tuberculosis disease is directly related to the number of tubercle bacilli that he or she expels into the air. Who expel many tubercle bacilli is more infectious than who expel few or no bacilli. (iv) Proximity, frequency, and duration of exposure. People who are exposed to the infection from short distances will have higher risk of transmission. People who are exposed to the infection for more number of times will have higher risk of transmission. People who are exposed to the infection for longer durations will have higher risk of transmission. (v) Environmental factors that enhance the probability that mycobacterium tuberculosis will be transmitted. These environmental factors include the following: (a) Concentration of infectious droplet nuclei. If there are more droplet nuclei in the air then there will be high probability of mycobacterium tuberculosis transmission. (b) Exposure in small enclosed spaces. If more people are confined to a small space like buses, small rooms, air ports, trains then they have got more chances of transmission. (c) No ventilation or no circulation of air. Rooms with less ventilation cause more transmission chances as infectious droplets have no way to go out. (d) Specimen handling. Improper specimen handling procedures generate infectious droplet nuclei. (e) Air flow from the room of infectious patient causes tuberculosis organisms to flow to other areas [4].

We now list few prevention techniques of the tuberculosis disease. Tuberculosis disease can be prevented through practicing good habits and hygienic conditions: (i) Covering the mouth and noses when coughing or sneezing (ii) including adequate ventilation in the rooms (iii) improving conditions in the crowded areas and (iv) providing treatment to bring the infection under control as soon as detected. These practices are essential in reducing the spread of tuberculosis.

We here add meanings of the technical words used in this paper. (i) *Pathogen* is an agent causing disease. (ii) *Bacteria* are a group of unicellular micro organisms. Bacteria are plural while bacterium is singular. (iii) *Bacilli* are a rod shaped bacteria. Bacillus is singular while bacilli are plural. (iv) *Tubercle* is a small rounded swelling in lungs. (v) *Tuberculosis* is a disease of lung tubercles caused by the bacillus. (vi) *Tuberculin* is a serum injected to diagnose TB. (vii) *Tubercle bacillus* is a bacterium causing tuberculosis disease. (viii) *Myco* is a prefix used in combining forms. Meaning is fungi.

2. Mathematical model of tuberculosis disease

Many mathematical models have been constructed to describe the dynamics of the tuberculosis disease. These models are built based on varied assumptions. The present mathematical model of tuberculosis disease is based on the following assumptions:

(i) The total human population is divided into four compartments viz., susceptible (S), exposed (E), infected (I) and recovered (R).

(ii) The infected human after recovery may get exposed to the disease once again. Here the recovery indicates recovery from the illness but not from the disease. Thus recovery compartment has some contribution to make to the exposed compartment. That is, people have chance to migrate from recovery compartment to exposed compartment.

(iii) Treatment is given only to the people of infected compartment as they are already exposed to the disease and they show symptoms of the disease and the disease is diagnosed by medical tests.

(iv) Treatment is not given to the people of exposed compartment as they are already exposed to the disease but do not show any symptom of the disease. The infection is in sleeping condition, but not active.

(v) The effect of the environmental pathogen (P) on the spread of tuberculosis disease is considered and included in the model. The human infected with tuberculosis disease will pollute the air and in return the polluted air will infect susceptible humans.

We illustrate our model assumptions through a flow chart.

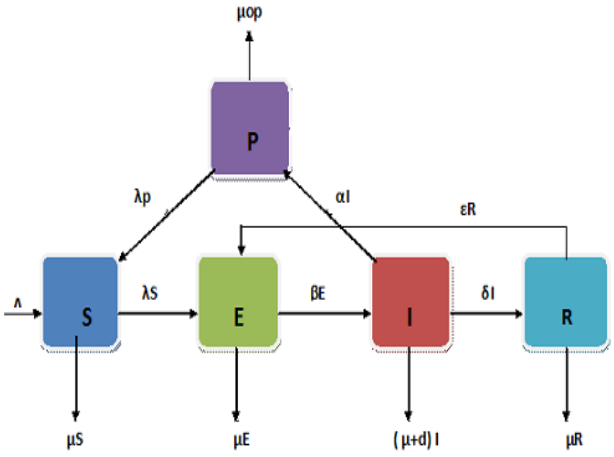

Figure 1 The flow chart of model assumptions

The human host population $N(t)$ under study is divided into four classes at any time t viz., $S(t)$, $\mathrm{E}(t)$, $\mathrm{I}(t)$ and $\mathrm{R}(t)$ denote respectively the number of individual in the susceptible, exposed, infected and temporarily recovered compartments. So that, $\mathrm{N}(t) = \mathrm{S}(t) + \mathrm{E}(t) + \mathrm{I}(t) + \mathrm{R}(t)$. We further assume that all these functions are non – negative quantities. The concentration of mycobacterium tuberculosis present in the air at any time t is denoted by $\mathrm{P}(t)$ and is called as environmental pathogen population. Thus,

$P(t)$ denotes the number of small particles called mycobacterium tuberculosis bacilli.

Susceptible human population The human population in the susceptible compartment is neither exposed nor infected by tuberculosis disease by now. But they are very sensitive, easily influenced, likely to be affected by or having the quality of receiving the disease in future. The human population of susceptible compartment increases with the recruitment of new people by both new births and immigration. The individuals are assumed to be recruited in to the susceptible compartment at a rate of Λ. The population size of susceptible compartment decreases as the humans migrate into the exposed class. The probability of susceptible people acquires TB as a result of contact with the mycobacterium tuberculosis population at a rate $\lambda = [\epsilon P/(c+P)]$. The susceptible population decreases by natural death at a rate of μ. Thus, the rate of change of susceptible population is given by $(dS/dt) = \Lambda - [(\lambda/A) + \mu]\, S$. Anyone recovered from tuberculosis is believed to be liable, unresisting or unsafe for life from the tuberculosis disease and the person may be attacked once again. Hence, the individuals from the recovered compartment are not supposed to enter into susceptible class but into the exposed class.

Exposed human population The people of exposed compartment are already infected latently, but not actively, by the mycobacterium. A human is infected latently means that the mycobacterium has already entered and present in the human body but the mycobacterium is (i) dormant (ii) lying inactive as if in sleep (iii) alive but not growing well (iv) inactive or (v) sleeping. In this model we have assumed that the latently infected individuals are not infectious as they do not to show any disease symptoms initially. Thus, the latently infected humans enter into exposed compartment. The latently infected humans are not capable of transmitting bacteria to other humans. The humans leaving from both the susceptible and recovered compartments will enter into exposed compartment. Thus, the population size of the exposed compartment increases when (i) a human of susceptible compartment gets latently infected by mycobacterium and (ii) a human recovers and comes out of the recovered compartment. The population size of the exposed compartment is decreased due to two reasons: (i) the individuals of this compartment when exposed to active tuberculosis disease will be shifted to infected compartment at a rate β and (ii) due to natural death of humans at a rate μ. Thus, the rate of change of human population in the exposed compartment can be constructed as $(dE/dt) = (\lambda/A)S + \varepsilon R - (\beta + \mu)E$. Not all exposed individuals will develop the tuberculosis disease. Only about 10% of the exposed people become infected and feel sick with tuberculosis disease after about two or more years [2, 12 – 13].

Infected human population The humans in the infected compartment are already infected by mycobacterium and are suffering from the tuberculosis disease. Further, these infected humans can influence the environment by spreading the mycobacterium. The population of infected compartment is increased by transferring humans from exposed compartment at a rate of β when they develop the symptoms of tuberculosis disease. The population of infected compartment is decreased with three reasons: (i) The infected individuals when treated and recovered from the disease are transferred to recovered compartment at a rate of δ (ii) humans die with a natural death rate of μ and (iii) humans die with induced death due to tuberculosis disease at rate of d. Thus, the rate of change of human population in the infected compartment can be constructed as $(dI/dt) = \beta E - (\delta + \mu + d)I$.

Recovered human population Here recovered means that the infected individual has recovered from the illness but not from the tuberculosis disease. Once if an individual is infected by the tuberculosis diseases he will never be completely cleared by the bacteria from their body system. Treatment is available just to (i) cure the sickness of infected individual (ii) control the spread of the bacterium inside the body of infected individuals. But there is no treatment to cure the tuberculosis disease completely. Treatment is not available to completely remove the mycobacterium from the infected human body but it converts active bacterium to latent bacterium. Thus, an infected human can be made recovered and then again exposed but never be a susceptible. The infected individuals undergo a long latency period after recovered which could last many years or even a lifetime [8]. The population size of the recovered compartment increases when the infected individuals are treated and transferred from infected compartment with a rate of δ. The population is decreased due to two reasons: (i) The recovered humans migrate to exposed class at the rate ε and (ii) the individuals die with a natural death rate of μ. Thus, the rate of change of human population in the infected compartment can be constructed as $(dR/dt) = I - (\mu + \varepsilon)R$.

Pathogen population in the polluted air Pathogen population means the population of tubercle bacilli in the air. Infected persons with tuberculosis disease will expel tubercle bacilli into the air. Infectiousness is measured in terms of the rate of tubercle bacilli are released into the air [4]. Persons who expel more tubercle bacilli are more infectious than patients who expel fewer or no bacilli. If there are more droplet nuclei of tubercle bacilli in the air then there will be more probability of mycobacterium tuberculosis transmission to susceptible humans and hence the spread of the disease is also more. In the high dens population areas like in buses, airplanes, camps, markets, schools, hospitals and homes the air pressure is positive for tuberculosis disease transmission. The tuberculosis organisms flow with air to other areas and spread the disease to susceptible humans. The infected individuals contribute to the population size of tubercle bacilli in the air with

excretion at a rate of α. The population size of tubercle bacilli in the polluted air is died out naturally at the rate of μ_0. Thus, the rate of change of pathogen population in the air can be constructed as $(dP/dt) = \alpha I - \mu_0 P$.

Thus the system of non linear differential equations describing the dynamics of mycobacterium tuberculosis disease transmission model is given by

$$(dS/dt) = \Lambda - [(\lambda/A) + \mu]\, S \quad 1(a)$$

$$(dE/dt) = (\lambda/A)S + \varepsilon R - (\beta + \mu)E \quad 1(b)$$

$$(dI/dt) = \beta E - (\delta + \mu + d)I \quad 1(c)$$

$$(dR/dt) = I - (\mu + \varepsilon)R \quad 1(d)$$

$$(dP/dt) = \alpha I - \mu_0 P \quad 1(e)$$

Table 1 Notations and description of the model variables

Symbol	Description
S(t)	Human population size of susceptible compartment at time t
$E(t)$	Human population size of exposed compartment at time t
$I(t)$	Human population size of infected compartment at time t
$R(t)$	Human population size of recovered compartment at time t
$P(t)$	Mycobacterium population size in the air at time t
$N_H(t)$	Total human population size at time t. It is a constant.

Table 2 Description of the model parameters

Symbol	Description
Λ	Recruitment rate of susceptible humans through natural birth and immigration
β	Progression rate of humans from exposed compartment to infected compartment
ϵ	Exposure rate of human population to polluted air
δ	Progression rate of humans from infected compartment to recovered compartment
ε	Progression rate of humans from recovered compartment to exposed compartment.
c	Concentration of mycobacterium tuberculosis in air that yields 50% of chances of catching the tuberculosis disease
A	Area in square meters occupied by human population
μ	Natural death rate of human population
α	Contribution of each infected human to the population of mycobacterium tuberculosis in the air
d	Death rate of humans due to tuberculosis disease
μ_0	Natural death rate of mycobacterium tuberculosis that presents in the air
λ	The probability of catching tuberculosis due to polluted air

2.1 Invariant regions of the model

On differentiating the system of equations (1) with respect to time and on summing up we get $N_H{}'(t) = S'(t) + E'(t) + I'(t) + R'(t)$ or equivalently $N_H{}'(t) = \Lambda - \mu N_H - dI$. In the present paper over head prime represents the derivative with respect to time. The death rate of humans due to tuberculosis disease is very small and even closer to zero $d \approx 0$. Thus, we have $N_H{}'(t) \leq \Lambda - \mu N_H$. On integrating this inequality using Birkhoff and Role's theorems we obtain the form $N_H \leq \{(\Lambda/\mu) - [(\Lambda/\mu) - N_0]\, e^{-\mu t}\}$. Here we have used the condition that the initial human population size is given by N_0, that is $N_H(0) = N_0$ at $t = 0$. It can be observed that as $t \to \infty$ the human population size N_H approaches (Λ/μ). Therefore, the feasible solution of human population enters the region $\Omega_H = \{(S, E, I, R) \in \mathbb{R}_+^4, N_H \leq (\Lambda/\mu)\}$.

We now integrate the differential equation $(dP/dt) = \alpha I - \mu_0 P$ given in (1e) to obtain temporal function describing pathogen population. (i) It sounds well to represent the pathogen population size of by N_P rather than by P and (ii) it is straight forward to verify that $I \leq N_H \leq (\Lambda/\mu)$. In view of these two observations we can rewrite (1e) as $(dN_P/dt) = \alpha(\Lambda/\mu) - \mu_0 N_P$ which is a first order ordinary linear differential equation having the particular solution $N_P(t) \leq (\alpha\Lambda/\mu\,\mu_0) - [(\alpha\Lambda/\mu\,\mu_0) - N_0]e^{-\mu_0 t}$. Here we have assumed that the initial pathogen population is given by $N_P(0) = N_0$. Further it can be observed that as $t \to \infty$ the pathogen population size N_P satisfies the relation $0 \leq N_P \leq (\alpha\Lambda/\mu\mu_0)$. Therefore, the feasible region of the pathogen population can be represented by the set $\Omega_P = \{N_P \in \mathbb{R}_+ : N_P \leq (\alpha\Lambda/\mu\mu_0)\}$.

Combining the feasible regions of both human population Ω_H and pathogen population Ω_P we obtain the feasible region of whole population considered in the present model Ω as $\Omega = \{(S, E, I, R, P) \in \mathbb{R}_+^5 : (S, E, I, R, P) \geq 0, N_H \leq (\Lambda/\mu)\ and\ N_P \leq (\alpha\Lambda/\mu\mu_0)\}$. Recall that $N_H = (S + E + I + R)$ and $N_P = P$ represent the population sizes of humans and pathogens respectively. Further, it can be verified that Ω is positively invariable set induced by the system of equations (1). Hence the system (1) is biologically meaningful and mathematically well-posed with in the domain given by the set Ω. Thus, it is feasible to consider the dynamics and flow of human and pathogen populations as described by the model (1) with in the set Ω.

3. Stability analysis of the model

In this section, we analyzed the model (1) in order to obtain (i) conditions for the existence and uniqueness of non-trivial equilibrium points (ii) the threshold condition for the asymptotic stability of equilibrium points and (iii) formula for the basic reproduction number represented by R_0. At equilibrium points the first order derivatives of the variables vanish and the tangents are horizontal or parallel to time axis. Thus, the equilibrium points of

the present model are obtained by setting the right-hand sides of the system (1) to zero $(dS/dt) = (dE/dt) = (dI/dt) = (dR/dt) = (dP/dt) = 0$. The foregoing condition is a requirement for existence of equilibrium points.

The Disease free Equilibrium Point Let $x_0 = (S^*, E^*, I^*, R^*, P^*)$ represents the disease free equilibrium point of the present model given by system (1). Disease free equilibrium points are steady state solutions of a mathematical model indicating that there is no disease. The compartmental classification of humans reveals that the "diseased" human population is distributed only in exposed and infected compartments. Hence, the absence of infection leads to emptiness of infected, exposed and pathogen compartments i.e., $I^* = 0, E^* = 0$ and $P^* = 0$. Also $P^* = 0$ leads to $\lambda = 0$ since the formula for λ is given by $\lambda = [\epsilon P/(c + P)]$. On substituting these requirements for disease free equilibrium point in the system of equations (1) we obtain $R^* = 0$ and $S^* = (\Lambda/\mu)$. Therefore the mycobacterium tuberculosis disease free equilibrium point is given by $x_0 = (S^*, E^*, I^*, R^*, P^*) = ((\Lambda/\mu), 0, 0, 0, 0)$.

The Basic Reproduction Number A human, infectious with tuberculosis disease, is supposed to propagate the disease to susceptible individuals. These are called secondary infection cases. The basic reproduction number is denoted by R_0 and is defined as the average number of secondary infection cases caused by infectious human during his or her entire period of infectiousness. The formula for reproduction number R_0 can be constructed using the next generation operator method as described in [10]. It is an important parameter in epidemiology as it sets the threshold of a disease. The reproduction number is used for predicting the speed of propagation of the disease and to identify the control strategies. Presence or absence of the disease in a community depends on the size of the reproduction number.

Suppose that there are *n* disease compartments and *m* non disease compartments, and let $x \in \mathbb{R}^n$ and $y \in \mathbb{R}^m$ be the subpopulations of these compartments. Let (i) $f_i(x)$ denotes the rate of appearance of new infection cases in i^{th} compartment (ii) v_i^+ denotes the transfer rate of individuals into i^{th} compartment by all means (iii) v_i^- denotes the transfer rate of individuals out from i^{th} compartment by all means and (iv) we also denote $v_i = v_i^- - v_i^+$. Further, it is assumed that each of the three functions $f_i(x)$, v_i^+ and v_i^- is continuously differentiable at least for two times with respect to their arguments.

Let us now define two matrices F and V by $F = \left[\partial f_i(x_0)/\partial x_j\right]$ and $V = \left[\partial v_i(x_0)/\partial x_j\right]$ respectively. Here the number of compartments denoted by i satisfies the condition $i \geq 1$ while the number of infected compartments denoted by j satisfies the condition $1 \leq j \leq n$. Then the matrix FV^{-1} is referred to as the next generation matrix for the system of model equations at the disease free equilibrium point. Also the reproduction number is defined in terms of next generation matrix as $R_0 = \rho(FV^{-1})$. Here $\rho(A)$ denotes the spectral radius or magnitude of the largest eigenvalue of matrix *A*. Also, (i) the matrix of the new infection terms denoted by F and (ii) the non singular matrix of the remaining transfer terms denoted by V are $n \times n$ matrices, where n is the number of infected compartments. The elements of the matrix F are non-negative. The (i, j) element of the matrix F represents the rate at which infected individuals transfer from j^{th} compartment to i^{th} compartment.

Consider that an infected individual is transferred into a disease free compartment k. and the (j, k) entry of V^{-1} is the average time an infected individual spends in compartment *j* during its lifetime, assuming that the population remains near the DFE and barring re-infection. Hence, the (i, k) entry of the product FV^{-1} is the expected number of new infections in compartment *i* produced by the infected individual originally introduced into compartment *k*. Using the next-generation approach and taking the infected compartments to be E, I and P from system of equations (1) gives $(dE/dt) = \lambda S + \varepsilon R - (\beta + \mu)E$, $(dI/dt) = \beta E - (\delta + \mu + d)I$ and $(dP/dt) = \alpha I - \mu_0 P$. From these we define f_i and v_i as

$$f_i = \begin{bmatrix} \frac{\epsilon PS}{A(c+P)} \\ 0 \\ 0 \end{bmatrix} \quad v_i = \begin{bmatrix} (\beta+\mu)E \\ (\delta+\mu+d)I \\ \mu_0 P - \alpha I \end{bmatrix}$$

Finding the partial differentiation with respect to E, I, P and evaluating at the disease free point gives the Jacobian matrices

$$F = \begin{bmatrix} 0 & 0 & \epsilon\Lambda/cA\mu \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, V = \begin{bmatrix} \beta+\mu & 0 & 0 \\ -\beta & \delta+\mu+d & 0 \\ 0 & -\alpha & \mu_0 \end{bmatrix}$$

Then we have to find the inverse of the Jacobian matrix of V, which is given by

$$V^{-1} = \begin{bmatrix} \frac{1}{\beta+\mu} & 0 & 0 \\ \frac{\beta}{(\delta+\mu+d)(\beta+\mu)} & \frac{1}{(\delta+\mu+d)} & 0 \\ \frac{\alpha\beta}{(\delta+\mu+d)(\beta+\mu)\mu_0} & \frac{\alpha}{(\delta+\mu+d)\mu_0} & \frac{1}{\mu_0} \end{bmatrix}$$

We now compute the product of both matrices *F* and V^{-1} which gives

$$FV^{-1} = \begin{bmatrix} \frac{\alpha\epsilon\beta\Lambda}{cA\mu(\delta+\mu+d)(\beta+\mu)\mu_0} & \frac{\alpha\epsilon\Lambda}{cA\mu(\delta+\mu+d)\mu_0} & \frac{\epsilon\Lambda}{cA\mu\mu_0} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

The eigenvalues of FV^{-1} for the equation *G* = $|FV^{-1} - I\lambda|$ = 0 (where I is identity matrix) are given by.

$$\lambda_1 = \frac{\alpha\epsilon\Lambda\beta}{Ac\mu(\beta+\mu)(\delta+\mu+d)\mu_0} \text{ and } \lambda_2 = \lambda_3 = 0$$

The dominant eigenvalue is the basic reproduction number, R_0 of the model. In this case, it is clearly seen to be λ_1. Thus, the reproduction number is given by

$$R_0 = \frac{\alpha\epsilon\Lambda\beta}{Ac\mu(\beta+\mu)(\delta+\mu+d)\mu_0}$$

The ratio $\frac{\beta}{(\beta+\mu)}$ is the fraction individuals that progress from E to I

Local Stability of the Disease-free Equilibrium Point The local stability of the disease-free equilibrium point $x_0 = (\Lambda/\mu, 0, 0, 0, 0)$ can be discussed by examining the linearization form of the system (1) at the given steady state x_0. This is done by computing the Jacobian matrix of the model (1). The Jacobian matrix is computed by differentiating each equation in the system with respect to the state variables S, E, I, R and P. The system is then re-defined as

$$f_1(S,E,I,R,P) = \Lambda - (\frac{\lambda}{A} + \mu)S$$
$$f_2(S,E,I,R,P) = \lambda S + \varepsilon R - (\beta + \mu)E$$
$$f_3(S,E,I,R,P) = \beta E - (\delta + \mu + d)I$$
$$f_4(S,E,I,R,P) = \delta I - (\mu + \varepsilon)R$$
$$f_5(S,E,I,R,P) = \alpha I - \mu_0 P$$

Where $\lambda = \frac{\epsilon P}{(c+P)}$ The Jacobian of the system is then given by

$$J = \begin{bmatrix} -(\epsilon P/A(c+P)+\mu) & 0 & 0 & 0 & -\epsilon P/A(c+P) + \epsilon PS/A(c+P)^2 \\ \epsilon P/A(c+P) & -(\beta+\mu) & 0 & \varepsilon & \epsilon P/A(c+P) + \epsilon PS/A(c+P)^2 \\ 0 & \beta & -(\delta+\mu+d) & 0 & 0 \\ 0 & 0 & \delta & -(\mu+\varepsilon) & 0 \\ 0 & 0 & \alpha & 0 & -\mu_0 \end{bmatrix} \quad (2)$$

Evaluating equation (2) at the DFE, when $S = \frac{\Lambda}{\mu}, E = 0, I = 0, R = 0$ and $P = 0$ we have

$$J(x_0) = \begin{bmatrix} -\mu & 0 & 0 & 0 & -\epsilon\Lambda/cA\mu \\ 0 & -(\beta+\mu) & 0 & \varepsilon & \epsilon\Lambda/cA\mu \\ 0 & \beta & -(\delta+\mu+d) & 0 & 0 \\ 0 & 0 & \delta & -(\mu+\varepsilon) & 0 \\ 0 & 0 & \alpha & 0 & -\mu_0 \end{bmatrix} \quad (3)$$

The disease-free equilibrium point, x_0, is said to be locally asymptotically stable if the real parts of the found eigenvalues are all negative, otherwise it is said to be unstable. Consider the matrix (3) and let *k* be the eigenvalue. Then we have

$|J(x_0) - k\,I| = 0$, where I is a 5×5 identity matrix. Thus, we have

$$|J(x_0)| = \begin{vmatrix} -\mu-\kappa & 0 & 0 & 0 & -\epsilon\Lambda/cA\mu \\ 0 & -(\beta+\mu)-\kappa & 0 & \varepsilon & \epsilon\Lambda/cA\mu \\ 0 & \beta & -(\delta+\mu+d)-\kappa & 0 & 0 \\ 0 & 0 & \delta & -(\mu+\varepsilon)-\kappa & 0 \\ 0 & 0 & \alpha & 0 & -\mu_0-\kappa \end{vmatrix} \quad (4)$$

As the first column corresponding to the total human populations contain only the diagonal term, these diagonal term form one eigenvalues of the Jacobian $(-\mu - k) = 0$ and that implies $\kappa_1 = -\mu$. The other four eigenvalues are the roots of the characteristic equation of the matrix formed by excluding the first row and first column of the Jacobian (4), we obtain the matrix

$$|J^*(x_0)| = \begin{vmatrix} -(\beta+\mu+\kappa) & 0 & \varepsilon & \epsilon\Lambda/cA\mu \\ \beta & -(\delta+\mu+d+\kappa) & 0 & 0 \\ 0 & \delta & -(\mu+\varepsilon+\kappa) & 0 \\ 0 & \alpha & 0 & -(\mu_0+\kappa) \end{vmatrix} \quad (5)$$

The corresponding characteristic equation is

$$(\beta + \mu + \kappa)(\delta + \mu + d + \kappa)(\mu + \varepsilon + \kappa)(\mu_0 + \kappa) - \varepsilon\delta\beta(\mu_0 + \kappa) - \frac{\epsilon\alpha\beta\Lambda}{Ac\mu}(\mu + \varepsilon + \kappa) = 0$$

Or equivalently,

$$A_1\kappa^4 + B_1\kappa^3 + C_1\kappa^2 + D_1\kappa + N_1 = 0 \quad (6)$$

Here in (6) we have used the notations $A_1 = 1$, $B_1 = 3\mu + \varepsilon + \mu_0 + \beta + \delta + d$, $C_1 = \mu_0(3\mu + \varepsilon + \mu_0 + \beta + \delta + d) + (\mu + \varepsilon)(\beta + 2\mu + \delta + d) + (\beta + \mu)(\mu + \delta + d$, $D_1 = (\mu + \varepsilon)[\mu_0(\beta + 2\mu + \delta + d) + (\beta + \mu)(\mu + \delta + d)] + (\beta + \mu)(\mu + \delta + d)\mu_0 - \varepsilon\delta\beta - \epsilon\alpha\beta\Lambda/cA\mu$ and $N_1 = (\mu + \varepsilon)[(\beta + 2\mu + \delta + d)\mu_0 - \epsilon\alpha\beta\Lambda/cA\mu] - \varepsilon\delta\beta\mu_0$. Due to the complexity in determining the signs of the remaining eigenvalues, we employ Routh-Hurwitz conditions for stability. The Routh-Hurwitz

conditions to ensure that all roots of (6) have negative real parts are $A_1 > 0, C_1 > 0, D_1 > 0$ and $A_1B_1C_1 > C_1^2 + A_1D_1$ clearly A_1, B_1 and C_1 are positive. For D_1 to be positive, set $(\mu+\varepsilon)[\mu_0(\beta+2\mu+\delta+d)+(\beta+\mu)(\mu+\delta+d)]+(\beta+\mu)(\mu+\delta+d)\mu_0-\varepsilon\delta\beta-\epsilon\alpha\beta\Lambda/cA\mu>0$. This shows that $R_0<0$ and $\varepsilon\delta\beta/(\beta+\mu)(\mu+\delta+d)\mu_0<0$.

For N_1 to be positive, set $(\mu+\varepsilon)[(\beta+2\mu+\delta+d)\mu_0-\epsilon\alpha\beta\Lambda/cA\mu]-\varepsilon\delta\beta\mu_0>0$. This simplifies to

$$1=\alpha\epsilon\beta\Lambda\ /\ cA\mu\ (\beta+\mu)(\delta+\mu+d)\ \mu\_(0\ )-(\varepsilon\delta\beta\mu\_0)\ /\ ((\mu+\varepsilon)(\beta+\mu)(\delta+\mu+d)\ \mu\_0\ )>0$$

This leads to $1-R_0-\varepsilon\delta\beta/(\mu+\varepsilon)(\beta+\mu)(\delta+\mu+d)>0$. This implies both $R_0<0$ and $\varepsilon\delta\beta/(\mu+\varepsilon)(\beta+\mu)(\delta+\mu+d)<0$ which is true. Hence, by Routh – Hurwitz criterion, all the eigenvalues have negative real parts, if $R_0<0$, thereby making x_0 locally asymptotically stable and x_0 is unstable for $R_0>0$ and hence $R_0<0$.

It is expected that, if $\mathrm{R}_0<1$, then no TB epidemic can develop in the population, and if $\mathrm{R}_0>1$, a TB epidemic can develop and become endemic in the population.

4. Numerical Simulations

Numerical Simulations of the dynamic model were carried out by MATLAB function ode45, using the Runge-Kutta of order four. The set of parameter values in table we were used to investigate the effect of habitat area in the control of the spread of TB. This parameter values whose sources are mainly from literature as well as assumptions. Five hypothetical cases were considered and in each case, the probability that individuals who are exposed to the diseases will progress to infectious class depends on the level of immunity individual has. It is prominent to note here that when TB patient are separated from non-infected people and kept in a wider area, it is assumed that they will have herd immunity i.e. the level of immunity in a population which prevents epidemics. The parameter values are listed in a tabular form. The following initial conditions have been considered and used S[0]= 1600 ; E[0]= 1500; I[0]= 150 ; R[0]= 140 ; P[0]= 100 at time $t_0=0$ and $t_f=10$.

Table 3: The parameter values

Parameter	Case 1	Case 2	Case 3	Case 4	Case 5	Reference
Λ	200	200	200	200	200	estimated
μ	0.02	0.02	0.02	0.02	0.02	calculated
β	0.352	0.352	0.352	0.3523	0.352	estimated
δ	1.24	1.24	1.24	1.24	1.24	calculated
ε	0.98	0.98	0.98	0.98	0.98	estimated
d	0.365	0.365	0.365	0.365	0.365	[2]
α	0.4	0.4	0.4	0.4	0.4	estimated
ϵ	0.2	0.2	0.2	0.2	0.2	estimated
μ_0	0.98	0.98	0.98	0.98	0.98	estimated
c	20	20	20	20	20	estimated
A	0.20	0.90	2	20	200	[2]

The numerical results have been done to show the dynamics of the disease in the population when there are no interventions.

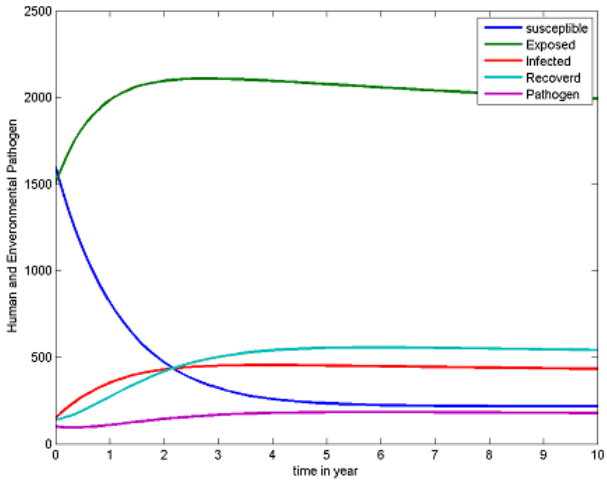

Figure 2 Reproduction ratio R_0 = 118.4610 and force of contact rate λ = 0.1798 at area = 0.2 square kilometer

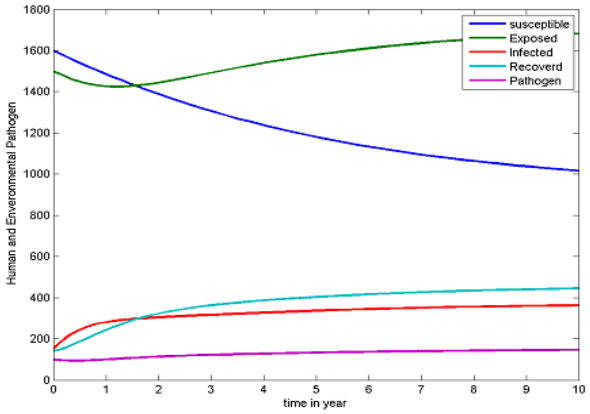

Figure 3 The Reproduction ratio R_0 = 26.4082 and force of contact rate λ = 0.760 at area = 0.9 square kilometer

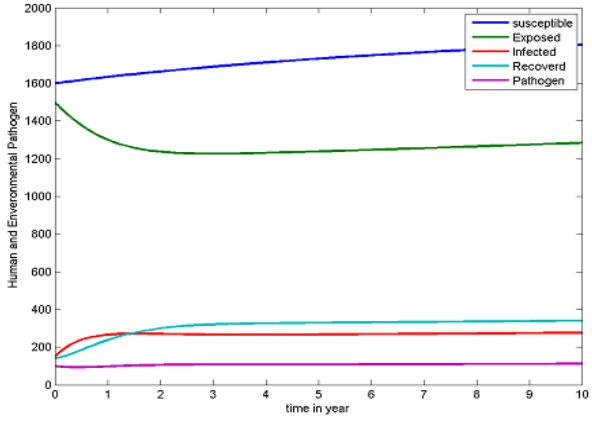

Figure 4 Reproduction ratio R_0= 11.8839 and force of contact rate λ = 0.1697 at area of = 2 square kilometer

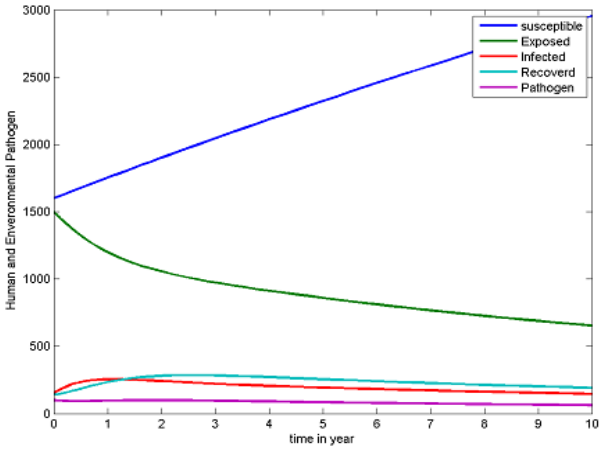

Figure 5 Reproduction ratio R_0= 1.1884 and force of contact rate λ = 0.1519 at area of = 20 square kilometer.

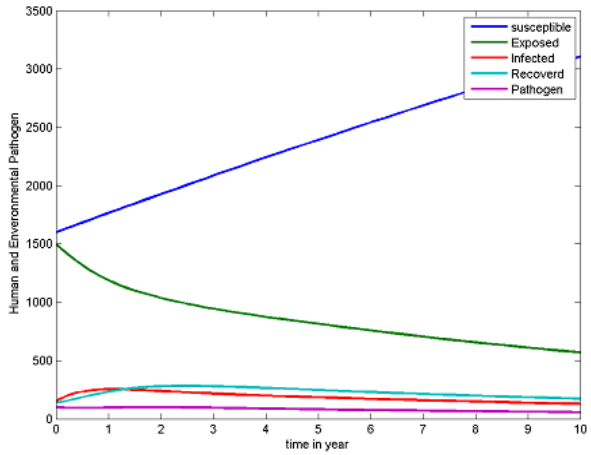

Figure 6 Reproduction ratio *R* 0 = 0.1188 and force of contact rate λ = 0.1479 at area of = 200 square kilometer.

The effect of variation of the size of the area occupied, A on the different epidemiological classes: This effect is studied using A = 0.2 square kilometers, 0.9 square kilometers, 2 square kilometers, 20 square kilometers, 200 square kilometers. We observe in Fig.2-6, that when the size of the area occupied, A is reduced from 2 square kilometers to 0.2 square kilometers thereby increasing the population density, the population will first increase because of the recruitment rate (through birth and immigration). It will there after decline because of the increased disease incidence. There are more infections resulting from the close contact due to high population density. Figure 2-4 shows that for a smaller area size, A = 0.2, A = 0.9 and A = 2 square kilometers hence higher population density, there is a higher rate of infection and increase in population size of the exposed individuals shall b e faster than in all the other cases. This is as a result of increased infection rate due to a higher contact rate of the susceptible with the infectious individuals. It means that the likelihood of new infections is high and this may lead to a wiping out of the total susceptible population. When area is increased to say 20 square kilometers or 200 square kilometers, there is a slight deviation in the population sizes in the two cases, implying that there is a threshold area size. However, in both cases, we observe that the number of susceptible will increase because of the reduced disease incidence due to lower population density (when the area is bigger s ay, A = 20 square kilometers or 200 square kilometers), the number of exposed individuals declining, though slowly and infected individuals approaches to the zero.

5. Conclusions

In our model we considered to gain more insight into the effect of Habitat area on dynamic spread of TB. This Habitat area plays a crucial role in the control of spread of TB virus in the environment. It is observed from the results above that the higher the Habitat area, the lower will b e spread of this TB virus and the higher will b e the recovery rate. From the stability analysis results, we have shown that the disease-free equilibrium point is asymptotically stable while the endemic equilibrium point is unstable. Whether the disease becomes persistent or dies out depends on the magnitude of the basic reproductive number R_0. We found that if the basic reproduction ratio $R_0 \leq 1$ then each solution limits to the disease-free equilibrium. In other words, every infectious individual will cause less than one secondary infection and hence the disease will die. If $R_0 > 1$, then there exists a unique endemic equilibrium which is globally asymptotically stable among all states for which the disease is present; Tuberculosis infection and re-infection are always existent in a community due to respiratory contact between the susceptible individuals and the recovered. Numerical experiments suggest that a country must detect and treat TB over a long course because TB transients can be very long. However, if keep TB infected individuals separately from the community, and giving treatment in hospitals, this could significantly reduce TB mortality and incidence and thus lowers R_0. Adequate ventilation, infected people cover their mouth and nose when coughing or sneezing and improving crowded conditions are essential in decreasing the spread of tuberculosis.

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