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Mathematical Analysis Of A Tuberculosis Epidemic Model

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ABSTRACT *In this paper, we analyze a mathematical model of tuberculosis (TB) epidemics for stability with respect to the basic reproduction number R_o. The basic reproduction number R_o is determined. We give criteria for stability of the disease – free equilibrium (DFE) and the endemic equilibrium*
***Keywords**: tuberculosis, mathematical model, stability, disease – free equilibrium, endemic equilibrium, basic reproduction number.*

I Introduction

Infection with mycobacterium tuberculosis leads to tuberculosis (TB) disease which causes more adult deaths than any other infections diseases [1]. Primary progression after a recent infection, re-activation of a latent infection and exogenous re-infection of previously infected individual are three possible routes of tuberculosis infection [2]. The global burden of tuberculosis (TB) has increased over the past two decades despite widespread implementation of control measures including BCG vaccination and the World Health Organization's DOTS strategy which focuses on case finding and short-course chemotherapy [3,4,5,6,7,8]. This is due to the emergence of drug-resistant TB strains and the convergence of human immunodeficiency virus (HIV) and TB epidemics [4, 10]. The rise in TB incidence has led to a growing consensus among public health policy makers that new strategies will be needed to achieve TB control especially in sub-Saharan Africa, Asia and Eastern Europe where the disease is predominant [4,5,11]. Proposed approaches include active case finding, isoniazid preventative therapy (IPT), anti-retroviral therapy among HIV-infected and improved detection and treatment of patients with multidrug-resistant TB [2,3,8,9 10,11,12,13,14]. Over the years, researchers have formulated and developed a large number of mathematical models in order to gain insights into the transmission dynamics of TB epidemics (see [1,2,3,4,7,15,16,17,18,19,20,21,22, 23]) and the references therein. In this paper we are interested in the model of Blower et al. [3]. We analyze the dynamics of this model by a threshold quantity called the basic reproduction number (denoted by R_o) which measures the number of new TB cases an infected individual will generate in a completely susceptible population. We formulate theorems on stability of disease-free equilibrium point and endemic equilibrium point and establish the proof of the theorems.

II Mathematical Formulation and Stability Analysis

We consider the model presented by Blower et al. [3].

$$
\begin{aligned}
S^1 &= \Pi - \beta IS - \mu S \\
L^1 &= (1-\rho)\beta IS - (v+\mu)L \qquad (1) \\
I^1 &= \rho\beta IS + VL - (\mu + \mu_T)L
\end{aligned}
$$

We present in table 1 below the detailed descriptions of the parameters of the model.

Table 1: Description of variables and parameters for the model

Variables	Description
S	Susceptible individuals
L	Latently infected individuals
I	Infections individuals

Parameters	Description	Range	Reference
Π	Recruitment rate of susceptible individuals	0.60,080	[3,7]
μ	Natural death rate	0.01425	[7]
μ_T	Death rate due to TB infection	0.0042, 0.0068	[3,7]
v	Rate of slow progression	0004, 0.570	[3,7]
ρ	Rate of fast progression	0.004, 0.0088	[3,7]
β	Transmission rate of active TB	0.0238,0.0856	[3,7]

2.1 The basic reproduction number R_o

Using the formulation of R_o presented by Diekmann and Heesterbeek [24], the basic reproduction number for model (1) is

$$R_o = \frac{\rho\beta\Pi}{\mu(\mu+\rho)} \tag{2}$$

2.2 The Critical (Equilibrium) Points

The critical points of model (1) is

$$P_O = \left(\frac{\Pi}{\mu}, o, o\right) \quad and\ P^* = \left(\frac{\Pi}{\mu}, \frac{(1-\rho)\beta}{\mu(\mu+\upsilon)}, \frac{\mu+\mu_T-\upsilon}{\mu(\mu+\upsilon)}\right)$$

where P_O is the disease – free equilibrium point and P^* is endemic equilibrium.

2.3 Stability Theorems

We shall need the theorems below in order to determine the nature of the critical points

Theorem 1 [25,26]

Let $\frac{dx}{dt}$ = P(x,y), $\frac{dy}{dt}$ = Q (x,y) and X = $\begin{pmatrix} x \\ y \end{pmatrix}$

Let $x_1 = \begin{pmatrix} x_1 \\ y_1 \end{pmatrix}$ be a critical point of the plane autonomous system

$X_1 = g(x) = \begin{pmatrix} P(x,y)_1 \\ Q(x,y) \end{pmatrix}$, where P(x,y) and Q(x,y) have continuous first partial derivatives in a neighbourhood of X_1

(a) If the eigenvalues of A = $g^1(X_1)$ have negative real part then X_1 is an asymptotically stable critical point

(b) If A = $g^1(X_1)$ has an eigenvalue with positive real part, then X_1 is an unstable critical point.

Theorem 2 [27] (DESCARTES' RULE OF SIGNS)

The number of positive zeros (negative zeros) of polynomials with real coefficients is either equal to the number of change in sign of the polynomial or less than this by an even number (by counting down by two's).

Theorem 3

The critical point of the disease-free equilibrium is asymptotically stable if $R_o < 1$ and if $\mu > 0, v > 0, \mu_T > 0$

Proof

Linearizing our system (1) about the DFE, the Jacobian matrix of the DFE at P_O is

$$J(P_O) = \begin{pmatrix} -\mu & 0 & 0 \\ 0 & v+\mu & 0 \\ 0 & 0 & \mu+\mu_T \end{pmatrix} \qquad (3)$$

The eigenvalues are given by

$$(-\lambda-\mu)(-\lambda-(v+\mu))(-\lambda-(\mu+\mu_T)) = 0 \qquad (4)$$

Hence, $\lambda_1 = -\mu, \lambda_2 = -(v+\mu), \lambda_3 = -(\mu+\mu_T)$

If $\mu > 0, v > 0, \mu_T > 0$ $R_o < 1$ in equation (4), then there are no change in signs which implies that there are no positive solutions of equation (4). If λ is replaced by $-\lambda$ in equation (4), then there are 3 sign changes so that equation (4) has exactly 3 negative roots. This implies that all the eigenvalues $\lambda_1, \lambda_2, \lambda_3$ are negative. Hence, the disease-free equilibrium point P_O is asymptotically table.

Remark

Using the data [3,7], R_o = 0.5716 <1. It shows that the disease-free equilibrium P_O is asymptotically stable.

Theorem 4

The critical point P^* of the endemic equilibrium is unstable if R_o >1 and $\mu > 0, v > 0, \rho > 0, \beta > 0, \Pi > 0, \mu_T > 0$

Proof

The Jacobian matrix of equation (1) at P^* is

$$J(P^*) = \begin{pmatrix} \frac{(1-\rho)\beta\Pi}{\mu(\mu+v)} & 0 & \frac{-\mu}{1-\rho} \\ 0 & \frac{-\rho\beta}{\mu(\mu+v)} & \mu+\upsilon \\ \frac{-v}{\mu(\mu+v)} & -v & \frac{-(\mu+\mu_T-v)}{\mu(\mu+v)} \end{pmatrix} \qquad (5)$$

The characteristics equation of (5) is

$$\left(\frac{(1-\rho)\beta\Pi}{\mu(\mu+v)}-\lambda\right)\left[\left(\frac{-\rho\beta}{\mu(\mu+v)}-\lambda\right)\left(\frac{-(\mu+\mu_T-v)}{\mu(\mu+v)}-\lambda\right)\right]-\left(\frac{\mu}{1-\rho}\right)\left[\left(\frac{-v}{\mu(\mu+v)}\right)\left(\frac{-\rho\beta}{\mu(\mu+v)}-\lambda\right)\right]=0 \qquad (6)$$

Expanding and manipulating the algebra, we have

$$-\lambda^3+\left(\frac{v\beta\Pi}{\mu(\mu+v)}\right)\lambda^2-\left(\frac{1-\rho)\beta\Pi}{\mu(\mu+v)}\right)\lambda+\left(\frac{(\mu+\mu_T-v)(1-\rho)}{\mu(\mu+v)}\right)=0 \qquad (7)$$

If we let $\rho > 0, \mu > 0, v > 0, \mu_T > 0, \beta > 0, v > 0,$ *and* $R_o > 1$ in equation (7), it follows then that there is only 1 sign change which implies that there is exactly I positive root. If λ is replaced by - λ is equation (7) and by the conditions of Theorem 4, equation (7) yields 2 sign changes and there are exactly 2 negative roots or zero root. Hence, there is exactly 1 positive root and 2 negative roots of equation (7). It follows that the endemic equilibrium point P^* is unstable.

Remark

By using the model parameter values in Table 1, R_o = 1.1894 >1. Hence, the critical point of the endemic equilibrium is unstable.

III Conclusion

From the stability analysis results, we have shown that the disease-free equilibrium point is asymptotically stable while the endemic equilibrium point is unstable. In addition, the basic reproduction number R_o is determined and shown as a threshold value of the disease dynamics. In particular, it is shown that the disease-free equilibrium is asymptotically stable if $R_o<1$ while the endemic equilibrium is unstable if $R_o>1$.

REFERENCES

[1] Dye, C. (2006). Global epidemiology of tuberculosis, Lancet, 367, 938-940.
[2] Cohen, T., Colijn, C., Finklea, B., and Murray, M., (2007). Exogenous re-infection and the dynamics of tuberculosis epidemics: local effects in a network model of transmission. J.R. Soc. Interface 4(14), 523 – 531.
[3] Blower, S.M., McLean, A.R., Porco, T.C., Small, P.M., Hopewell, P.C., Sanchez, M.A. and Moss, A.R., (1995). The intrinsic transmission dynamics of tuberculosis epidemics, Natl. Med. 1(8), 815-821.
[4] Egbetade, S.A. and Ibrahim, M.O. (2012). Stability analysis of equilibrium states of an SEIR Tuberculosis model. Journal of NAMP 20, 119 – 124.
[5] Corbett, E.L., Matson, B., Churchyard, G.J., DeCock, K.M. (2006). Tuberculosis in sub-Saharan African: Opportunities, Challenges and change in the era of antiretroviral treatment. Lancet 367, 926 – 937.
[6] Gomes, M.G.., Franco, A.O., Gomes, M.C. and Medley, G.F. (2004). The reinfection threshold promotes variability in tuberculosis epidemiology and vaccine efficacy. Proc. R. Soc. B., 271, 617-623.
[7] Colijn, C., Cohen, T., and Murray M, (2006). Mathematical models of tuberculosis: accomplishments and challenges. Proc. Natl. Acad. Sci. USA, 103(11): 1-28.
[8] Dye, C., Garnett, G.P., Sleeman K. and Williams, B.G. (1998). Prospects for Worldwide tuberculosis control under the World Health Organization DOTS strategy. Lancet, 352, 1886-1891.
[9] World Health Organization (2010). Tuberculosis fact sheet. webpage: www.who.org
[10] Porco, T.C., Small, P.M. and Blower, S.M., (2001). Amplification dynamics: predicting the effect of HIV on tuberculosis outbreaks. J. Acquire. Immune Defic. Syndr., 28(5), 437-444.
[11] Harries, A.D. and Dye, C. (2006). Tuberculosis Ann. Trop. Med. Parasitol 100, 415 – 431.
[12] Alland, D., Kalkut, G.E., Moss, A.R., McAdam, R.A., Hann, J.A., Bosworth, W., Drucker, E. and Bloom, B.R. (1994). Transmission of tuberculosis in New York city. An analysis by DNA finger printing and conventional epidemiologic methods. N.Engl. J. Med.330, 1710 – 1716.
[13] Pourbohloul, B., Meyers, L.A., Skowronski, D.M., Krajden, M., Patrick, D.M. and Brunham, R.C. (2005). Modelling Control Strategies of respiratory pathogens. Emerg. Infect. Dis. 11, 1249 – 1256.
[14] Ferguson, N.M., Keeling, M.J., Edmunds, W.T., Gani, R., Grenfell B.T., Anderson, R.M. and Leach, S. (2007). Planning for small pox outbreaks. Nature 425, 681 – 685.
[15] Enarson, D. and Rouillon, A. (1994). The epidemiological basis of tuberculosis control Chapman and Hail, London, U.K.
[16] Chiang, C.Y. and Riley, L.W. (2005). Exogenous re-infection in tuberculosis. Lancet Infect. Dis. 5, 629 – 636.
[17] Keeling, M.J. and Eames, K.T.D. (2005). Networks and epidemic models. J.R. Soc. Interface 2, 295 – 307.
[18] Keeling, M.T. (1999). The effects of local spatial structure on epidemiological invasions. Proc. R. Soc. B. 266, 859 – 867.
[19] Song, B., Castillo – Chavez, C. and Aparicio,, J.P. (2002). Tuberculosis models with fast and slow dynamics: the role of close and casual contacts. Math. Biosci. 180, 187 – 205.
[20] Vynnycky, E. and Fine, P.E. (1997). The natural history of tuberculosis: the implications of age – dependent risks of disease and role of reinfection. Epidemiol. Infect 119, 183 – 201.
[21] Watts, D.J. and Strogatz, S.H. (1998). Collective dynamics of 'small world' networks Nature 393, 440 – 442.
[22] Cohen, T. and Murray, M. (2004). Modelling epidemics of multidrug- resistant Mycobaterium Tuberculosis of heterogenous fitness. Natl. Med. 10, 1117 – 1221.
[23] Murray, C.J. and Salomon, J.A. (1998). Modelling the impact of global tuberculosis control strategies. Proc. Natl. Acad. Sci. USA. 95(13), 881 – 886.
[24] Diekmann, O., and Heesterbeek ,J.A, (2000). Mathematical Epidemiology of Infectious Disease, John-Wiley & Sons, Chichester.
[25] Dennis, G. Zill., and Micheal, R., C.(2005). Differential equations with boundary value problems. Addison – Wesley publishing Company, Inc., 395 - 417.
[26] Adeniyi, M.O. and Kolawole, M.K. (2012). Mathematical analysis of the global dynamics of a power law model for HIV infection of CD4+ T cells. J.of NAMP 20, 125 – 130.
[27] Anderson, B., Jackson, J. and Sithram, M. (1998). "Descartes' Rule of signs Revisited". Amer. Math. Monthly 105, 447 – 451.