A Mathematical Modeling of Tuberculosis Dynamics with Hygiene Consciousness as a Control Strategy

Phineas Z. Mawira¹ *∗* **and David M. Malonza**²

¹*Department of Mathematics, Kenyatta University, P.O.Box 43844-00100, Nairobi, Kenya.* ²*Department of Mathematics and Actuarial Science, South Eastern Kenya University, P.O.Box 170-90200, Kitui, Kenya.*

Authors' contributions

This work was carried out in collaboration between both authors. Author DMM designed the study, and wrote the first draft of the manuscript. Both authors managed the literature searches. Author PZM carried out mathematical modelling. Both authors read and approved the final manuscript.

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Abstract

Tuberculosis, an airborne infectious disease, remains a major threat to public health in Kenya. In this study, we derived a system of non-linear ordinary differential equations from the SLICR mathematical model of TB to study the effects of hygiene consciousness as a control strategy against TB in Kenya. The effective basic reproduction number (*R*0) of the model was determined by the next generation matrix approach. We established and analyzed the equilibrium points. Using the Routh-Hurwitz criterion for local stability analysis and comparison theorem for global stability analysis, the disease-free equilibrium (DFE) was found to be locally asymptotically stable given that $R_0 < 1$.

^{}Corresponding author: E-mail: mawiraphineamaths@gmail.com;*

Also by using the Routh-Hurwitz criterion for local stability analysis and Lyapunov function and LaSalle's invariance principle for global stability analysis, the endemic equilibrium (EE) point was found to be locally asymptotically stable given that $R_0 > 1$. Using MATLAB **ode45** solver, we simulated the model numerically and the results suggest that hygiene consciousness can help in controlling TB disease if incorporated effectively.

Keywords: Tuberculosis; reproduction number; stability; numerical simulation.

2019 Mathematics Subject Classification: 53C25, 83C05, 57N16.

1 Introduction

Tuberculosis is a bacterial infection disease that is common and potentially fatal if not timely treated. It is caused by the *mycobacterium tuberculosis*. Tuberculosis usually affects the lungs; it can also affect the brain, the central nervous system, the lymphatic system, the kidneys and spine.

TB is transmitted from one person to another through the air. When a person with lung TB coughs, sneezes, speaks or spits, TB germs (bacterium) are propelled into the air. These germs enter into the respiratory system through the air inhaled. Any uninfected individual inhaling the bacteria is possibly exposed to infectious *mycobacterium tuberculosis*. An individual needs to inhale only a few of these bacteria to become infected.

Mathematical modeling is an important tool for a better understanding of the dynamics of infectious [1]. Modeling of tuberculosis was first carried out by Frost [2]. Since then, several researchers have continuously researched on how tuberculosis can be reduced using mathematical models by incorporating interventions such as treatment of tuberculosis-infected persons by using of drug chemotherapy [3], treatment of actively and latently infected persons [4], health education (see [5], [6],[7]), therapy of tuberculosis transmission [8], early treatment for latent patients and treatment of i[n](#page-10-0)fective [9] and so on. Based on the result of these studies, the [r](#page-10-1)esearchers found that tuberculosis could be controlled.

To this end, we [se](#page-10-2)ek to investigate analytically the dynamics of tubercul[os](#page-10-3)is by incorporating hygi[en](#page-10-4)e [as](#page-10-5) [a c](#page-10-6)ontrol strategy in order to eradicate th[e](#page-10-7) disease.

2 Model Formulation

We formulate a model with total population $N(t)$, which is divided into five classes: $S(t)$ -Susceptible individuals, L(t)-Latently Infected individuals, I(t)- Infectious individuals (Actively Infected individuals), C(t)- Hygiene conscious individuals (Infectious individuals who have become hygiene conscious) and R(t)- Recovered individuals with natural death rate μ in all classes and TB related death rate *δ* in Infectious and hygiene consciousness compartments. People are recruited to Susceptible class at the rate Λ and become Latently infected at the rate of *βI* where *β* is the effective contact rate between susceptible and infectious individuals. $\nu\beta(0 < \nu < 1)$ is the reduced effective contact rate between susceptible and infectious individuals as a result of hygiene consciousness where *ν* is the rate at which infectious individuals become hygiene conscious. Latently infected individuals progress to infectious class at σ rate and these infectious individuals recover at the rate π and others progress to hygiene consciousness class at the rate *ν*. The hygiene conscious individuals recover at ω rate. From the above description we have the following assumptions and flow diagram;

Assumptions:

- i The population birth and death rate occur at different rates.
- ii Infectious individuals observe hygiene willingly.
- iii Infectious individuals recover as result of effective treatment.
- iv There is permanent immunity after recovery.
- v All the newly born individuals join only susceptible class.
- vi Individuals in infectious and hygiene Conscious classes die naturally and as result of TB infection while those in other classes die naturally.

Fig. 1. Flow chart

From Fig. 1. we have the following equations of the model are:

$$
\frac{dS}{dt} = \Lambda - (1 - \nu)\beta IS - \mu S
$$
\n
$$
\frac{dL}{dt} = (1 - \nu)\beta IS - (\sigma + \mu)L
$$
\n
$$
\frac{dI}{dt} = \sigma L - (\nu + \pi + \delta + \mu)I
$$
\n
$$
\frac{dC}{dt} = \nu I - (\omega + \delta + \mu)C
$$
\n
$$
\frac{dR}{dt} = \omega C + \pi I - \mu R
$$
\n(2.1)

3 Model Analysis

3.1 Positivity and boundedness of solutions

The state variables of the model represent classes of human population and therefore should be non-negative for the model to be well posed. The model will make epidemiological sense where the feasible region is positively invariant. All the solutions are non-negative and bounded in the feasible $\text{region } \Omega = \{ (S, L, I, C, R) \in \mathbb{R}_+^5 : S > 0; L, I, C, R \geq 0; N \leq \frac{\Lambda}{\mu} \}$

Because the first four equations of system (2.1) are independent of variable $R(t)$, we reduce the system by to obtain;

$$
\frac{dS}{dt} = \Lambda - (1 - \nu)\beta IS - \mu S
$$
\n
$$
\frac{dL}{dt} = (1 - \nu)\beta IS - (\sigma + \mu)L
$$
\n
$$
\frac{dI}{dt} = \sigma L - (\nu + \pi + \delta + \mu)I
$$
\n
$$
\frac{dC}{dt} = \nu I - (\omega + \delta + \mu)C
$$
\n(3.1)

3.2 Existence of equilibrium points

At the equilibrium point, the right hand side of the system (3.1) is equal to zero. In that;

$$
\Lambda - (1 - \nu)\beta I S - \mu S = 0
$$

\n
$$
(1 - \nu)\beta I S - (\sigma + \mu)L = 0
$$

\n
$$
\sigma L - (\nu + \pi + \delta + \mu)I = 0
$$

\n
$$
\nu I - (\omega + \delta + \mu)C = 0
$$
\n(3.2)

For Disease Free Equilibrium point E_0 , the disease is not present. Hence $S = S_0$, $L = 0$, $I = 0$ and *C* = 0. Upon substitution of these $(S = S_0, L = 0, I = 0 \text{ and } C = 0)$ in (3.2), we obtain $S_0 = \frac{\Lambda}{\mu}$ Such that $E_0 = (\frac{\Lambda}{\mu}, 0, 0, 0)$

Next, we investigate the existence of Endemic equilibrium point $E_e(S_e, L_e, I_e, C_e)$ by substituting $S = S_e, L = L_e, I = I_e, C = C_e$ in (3.2) to get

$$
\Lambda - (1 - \nu)\beta I_e S_e - \mu S_e = 0
$$

$$
(1 - \nu)\beta I_e S_e - (\sigma + \mu)L_e = 0
$$

$$
\sigma L_e - (\nu + \pi + \delta + \mu)I_e = 0
$$

$$
\nu I_e - (\omega + \delta + \mu)C_e = 0
$$

then by solving for S_e , L_e , I_e and C_e we obtain

$$
E_e \begin{pmatrix} S_e \\ I_e \\ C_e \end{pmatrix} = E_e \begin{pmatrix} \frac{2(1-\nu)\beta\sigma\Lambda - \mu(\sigma+\mu)(\nu+\pi+\delta+\mu)}{(1-\nu)\mu\sigma\beta} \\ \frac{\mu(\sigma+\mu)(\nu+\pi+\delta+\mu)-(1-\nu)\beta\sigma\Lambda}{\sigma(1-\nu)\beta(\sigma+\mu)} \\ \frac{\mu(\sigma+\mu)(\nu+\pi+\delta+\mu)-(1-\nu)\beta\sigma\Lambda}{(1-\nu)\beta(\sigma+\mu)(\nu+\pi+\delta+\mu)} \\ \frac{\nu(\sigma+\mu)(\nu+\pi+\delta+\mu)-(1-\nu)\nu\beta\sigma\Lambda}{(1-\nu)\beta(\sigma+\mu)(\nu+\pi+\delta+\mu)(\omega+\delta+\mu)} \end{pmatrix}
$$

3.3 The basic reproduction number (R_0)

The basic reproduction number *R*⁰ is the average number of secondary infection cases arising from a typical primary infection case in an entirely susceptible population. Using the next generation matrix approach by Van den Driessche and Watmough [10], *R*⁰ is given by the spectral radius

1 \mathbf{I}

 $\rho(\text{F}V^{-1})$ where **F** is the Jacobian matrix of f_j at E_0 , where f_j contains terms that directly lead to new infections entering each compartment j and **V** is the Jacobian matrix of v_j at E_0 , where v_j is the rate of transfer of individuals into and out of compartment j. The disease compartments are L, I and C. Their equations are;

$$
\frac{dL}{dt} = (1 - \nu)\beta IS - (\sigma + \mu)L
$$
\n
$$
\frac{dI}{dt} = \sigma L - (\nu + \pi + \delta + \mu)I
$$
\n
$$
\frac{dC}{dt} = \nu I - (\omega + \delta + \mu)C
$$

(3.4)

System (3.4) can be expressed as;

$$
\begin{bmatrix}\n\frac{dL}{dt} \\
\frac{dL}{dt} \\
\frac{dL}{dt}\n\end{bmatrix} = f_j - v_j = \begin{bmatrix}\n(1 - \nu)\beta IS \\
0\n\end{bmatrix} - \begin{bmatrix}\n(\sigma + \mu)L \\
(\nu + \pi + \delta + \mu)I - \sigma L \\
(\omega + \delta + \mu)C - \nu I\n\end{bmatrix}
$$
\nWe can see;
\n
$$
f_i = \begin{bmatrix}\n(1 - \nu)\beta IS \\
0 \\
0\n\end{bmatrix} \text{ and } v_i = \begin{bmatrix}\n(\sigma + \mu)L \\
(\nu + \pi + \delta + \mu)I - \sigma L \\
(\omega + \delta + \mu)C - \nu I\n\end{bmatrix}
$$
\nThe jacobian of f_j at E_0 is
\n
$$
\mathbf{F} = \begin{bmatrix}\n0 & \frac{(1 - \nu)\beta\Lambda}{\beta} & 0 \\
0 & 0 & 0 \\
0 & 0 & 0\n\end{bmatrix}
$$
\nand that of v_j at E_0 is
\n
$$
\mathbf{V} = \begin{bmatrix}\n(\sigma + \mu) & 0 & 0 \\
-\sigma & (\nu + \pi + \delta + \mu) & 0 \\
0 & -\nu & (\omega + \delta + \mu)\n\end{bmatrix}
$$
\nThe determinant of \mathbf{V} is given by;
\n
$$
det(V_0) = (\sigma + \mu)(\nu + \pi + \delta + \mu)(\omega + \delta + \mu)
$$

Therefore

$$
\mathbf{V}^{-1} = \begin{bmatrix} \frac{1}{(\sigma+\mu)} & 0 & 0\\ \frac{\sigma}{(\sigma+\mu)(\nu+\pi+\delta+\mu)} & \frac{1}{(\nu+\pi+\delta+\mu)} & 0\\ \frac{\sigma\nu}{(\sigma+\mu)(\nu+\pi+\delta+\mu)(\omega+\delta+\mu)} & \frac{\nu}{(\nu+\pi+\delta+\mu)(\omega+\delta+\mu)} & \frac{1}{(\omega+\delta+\mu)} \end{bmatrix}
$$

By definition, $R_0 = \rho (\mathbf{F} \mathbf{V}^{-1})$.

Where

$$
\mathbf{F} \mathbf{V}^{-1} = \begin{bmatrix} \frac{(1-\nu)\beta\sigma\Lambda}{\mu(\sigma+\mu)(\nu+\pi+\delta+\mu)} & \frac{(1-\nu)\beta\Lambda}{\mu(\nu+\pi+\delta+\mu)} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}
$$

Therefore

 $R_0 = \frac{(1-\nu)\beta\sigma\Lambda}{\mu(\sigma+\mu)(\nu+\pi+\delta+\mu)}$

4 Stability Analysis

In this section we will analyse local and global stability of the disease-free equilibrium and endemic equilibrium of the system (3.1).

4.1 Local stability of the disease free equilibrium point

In this section we study lo[cal](#page-3-1) stability of the disease-free equilibrium of the system (3.1).

Theorem 1. *The disease-free equilibrium of the system (3.1) is locally asymptotically stable when the reproduction number* $R_0 < 1$ *.*

Proof. Evaluating the jacobian matrix of system (3.1) at E_0 , we have;

$$
J(E_0) = \begin{bmatrix} -\mu & 0 & -\frac{(1-\nu)\beta\Lambda}{\mu} & 0\\ 0 & -(\sigma+\mu) & \frac{(1-\nu)\beta\Lambda}{\mu} & 0\\ 0 & \sigma & -(\nu+\pi+\delta+\mu) & 0\\ 0 & 0 & \nu & -(\omega+\delta+\mu) \end{bmatrix}
$$

From $J(E_0)$ the determinant is given by

 $Det J(E_0) = -\mu [-(\sigma + \mu)(\nu + \pi + \delta + \mu)(\omega + \delta + \mu) + \sigma(\omega + \delta + \mu)\frac{(1-\nu)\beta\Lambda}{\mu}]$

and the trace is given by $tr(J(E_0)=-\mu-(\sigma+\mu)-(\nu+\pi+\delta+\mu)-(\omega+\delta+\mu)$

Since all the parameters are positive, it can be seen that the $tr(J(E_0) < 0$ and $Det(J(E_0) > 0$ when $R_0 < 1$ i.e $(\sigma + \mu)(\nu + \pi + \delta + \mu) > \frac{(1-\nu)\beta\sigma\Lambda}{\mu}$

Applying Routh-Hurwitz criterion for necessary and sufficient conditions as in Enagi et al. [11], the characteristic polynomial has all roots with negative real part since $tr(J(E_0)) < 0$ and $Det(J(E_0)) >$ 0 when $R_0 < 1$.

Thus, by Routh-Hurwitz criteria, the disease-free equilibrium is locally asymptotically stable when $R_0 < 1$ П

4.2 Global stability of the disease free equilibrium point

Now we use comparison theorem as in [12], to prove the global stability of DFE

Theorem 2. *The disease-free equilibrium of the system (3.1) is glocally asymptotically stable when the reproduction number* $R_0 < 1$ *.*

Proof. Using the comparison theorem i[n \[](#page-10-8)12] we rewrite the disease compartments as

$$
\begin{bmatrix}\n\frac{dL}{dt} \\
\frac{dL}{dt} \\
\frac{dE}{dt}\n\end{bmatrix} = (\mathbf{F} \cdot \mathbf{V}) \begin{bmatrix}\nL \\
I \\
C\n\end{bmatrix} - \begin{bmatrix}\n(1 - \nu)\beta I(S_0 - S) \\
0 \\
0\n\end{bmatrix}
$$
\nWhere \mathbf{F} and \mathbf{V} are defined in section (3.2.4)\n
\nSince $S \leq S_0 = \frac{\Lambda}{\mu} \forall t > 0$, it follows that\n
$$
\begin{bmatrix}\n\frac{dL}{dt} \\
\frac{dL}{dt} \\
\frac{dL}{dt}\n\end{bmatrix} \leq (\mathbf{F} \cdot \mathbf{V}) \begin{bmatrix}\nL \\
I \\
C\n\end{bmatrix}
$$
\nWhere $\mathbf{F} - \mathbf{V} = \begin{bmatrix}\n-(\sigma + \mu) & \frac{(1 - \nu)\beta \Lambda}{\mu} & 0 \\
\sigma & -(\nu + \pi + \delta + \mu) & 0 \\
0 & -\nu & -(\omega + \delta + \mu)\n\end{bmatrix}$ \nThe characteristic equation is given by

 $-((\sigma+\mu)+\lambda)((\nu+\pi+\delta+\mu)+\lambda)((\omega+\delta+\mu)+\lambda)+\sigma((\omega+\delta+\mu)+\lambda)\frac{(1-\nu)\beta\Lambda}{\mu}=0$

Which upon expansion gives

$$
((\omega + \delta + \mu) + \lambda)[\lambda^2 + a_1\lambda + a_2] = 0
$$
\n(4.1)

where

 $a_1 = (\sigma + \mu) + (\nu + \pi + \delta + \mu)$ $a_2 = (\sigma + \mu)(\nu + \pi + \delta + \mu) - \frac{(1-\nu)\beta\sigma\Lambda}{\mu}$

From equation (4.1) it is clear that one of the eigenvalues is $-(\omega + \delta + \mu)$ and other eigenvalues are given by the roots of

$$
\lambda^2 + a_1 \lambda + a_2 = 0 \tag{4.2}
$$

By using Routh Hurwitz criterion, equation (4.2) has roots with negative real part if $a_1a_2 > 0$.

We c[a](#page-6-0)n see that $a_1 > 0$, this implies that for $a_1 a_2 > 0$ to be satisfied, $a_2 > 0$.

Clearly $a_2 > 0$ when $R_0 < 1$ i.e $(\sigma + \mu)(\nu + \pi + \delta + \mu) > \frac{(1-\nu)\beta\sigma\Lambda}{\mu}$

Since we have shown that **F***−***V** has negative e[igen](#page-6-1)values when $R_0 < 1$, it means that $(L(t), I(t), C(t))$ \rightarrow (0*,* 0*,* 0*)* as $t \rightarrow \infty$. Using comparison theorem, it follows that $(L(t), I(t), C(t)) \rightarrow (0, 0, 0)$ and $S(t) \longrightarrow \frac{\Lambda}{\mu}$ as $t \longrightarrow \infty$ $(S(t), L(t), I(t), C(t)) \longrightarrow E_0$

Therefore the disease free equilibrium is globally asymptotically stable when $R_0 < 1$. \Box

4.3 Local stability of the endemic equilibrium point

Theorem 3. *The endemic equilibrium point* (*Ee*) *of system (3.1) is locally asymptotically stable when* $R_0 > 1$

Proof. We prove the theorem by determining if there exist negative eigenvalues when $R_0 > 1$. We start by evaluating the Jacobian matrix of system (3.1) at the [end](#page-3-1)emic equilibrium point. Thus

$$
J(E_e) = \begin{bmatrix} -(1-\nu)\beta I_e - \mu & 0 & -(1-\nu)\beta S_e & 0\\ (1-\nu)\beta I_e & -(\sigma+\mu) & (1-\nu)\beta S_e & 0\\ 0 & \sigma & -(\nu+\pi+\delta+\mu) & 0\\ 0 & 0 & \nu & -(\omega+\delta+\mu) \end{bmatrix}
$$

We see that $-(\omega + \delta + \mu)$ is one of [the](#page-3-1) eigenvalues, the rest can be determined by reducing $J(E_e)$ to

$$
J_1(E_e) = \begin{bmatrix} -[(1-\nu)\beta I_e + \mu] & 0 & -(1-\nu)\beta S_e \\ (1-\nu)\beta I_e & -(\sigma+\mu) & (1-\nu)\beta S_e \\ 0 & \sigma & -(\nu+\pi+\delta+\mu) \end{bmatrix}
$$

From $J_1(E_e)$,

 $tr(J_1(E_e)) = -((1 - \nu)\beta I_e + 3\mu + \sigma + \nu + \pi + \delta)$

 $Det(J_1(E_e)) = - [(1-\nu)\beta I_e + \mu] \left\{ \frac{2\mu(\sigma+\mu)(\nu+\pi+\delta+\mu)-2(1-\nu)\beta\sigma\Lambda}{\mu} \right\}$

 $\text{Clearly } Det(J_1(E_e)) > 0 \text{ for } R_0 > 1 \text{ i.e } (1 - \nu)\beta\sigma\Lambda > \mu(\sigma + \mu)(\nu + \pi + \delta + \mu)$

Applying Routh-Hurwitz criterion for necessary and sufficient conditions as in Enagi et al. [11], $tr(J_1(E_e)) < 0$ and $Det(J_1(E_e)) > 0$ when $R_0 > 1$. Implying that all eigenvalues of $J_1(E_e)$, have negative real part when $R_0 > 1$. Thus E_e is locally asymptotically stable when $R_0 > 1$. \Box

4.4 Global stability of the endemic equilibrium point

We study the global asymptotic stability of the endemic equilibrium using LaSalles invariance principle [13].

1 \mathbf{I} $\overline{1}$ $\overline{1}$ **Theorem 4.** *The Endemic Equilibrium Point E^e of the system (3.1) is globally asymptotically stable if* $R_0 > 1$ *.*

Proof. We apply [13] approach to prove global stability of *Ee*. Consider the following Lyapunov function

 $G(S,L,I,C)=(S-S_eln\frac{S}{S_e})+M(L-L_eln\frac{L}{L_e})+P(I-L_eln\frac{I}{I_e})+Q(C-C_eln\frac{C}{C_e})$ The derivative of G is ;

 $\frac{dG}{dt} = (1 - \frac{S_e}{S})\frac{dS}{dt} + M(1 - \frac{L_e}{L})\frac{dL}{dt} + P(1 - \frac{I_e}{I})\frac{dI}{dt} + Q(1 - \frac{C_e}{C})\frac{dC}{dt}$ $\frac{dG}{dt} = (1 - \frac{S_e}{S})\frac{dS}{dt} + M(1 - \frac{L_e}{L})\frac{dL}{dt} + P(1 - \frac{I_e}{I})\frac{dI}{dt} + Q(1 - \frac{C_e}{C})\frac{dC}{dt}$ $\frac{dG}{dt} = (1 - \frac{S_e}{S})\frac{dS}{dt} + M(1 - \frac{L_e}{L})\frac{dL}{dt} + P(1 - \frac{I_e}{I})\frac{dI}{dt} + Q(1 - \frac{C_e}{C})\frac{dC}{dt}$ Next, we substitute $\frac{dS}{dt}$, $\frac{dL}{dt}$, $\frac{dI}{dt}$ and $\frac{dC}{dt}$ from system (3.1), to have;

$$
\frac{dW}{dt} = \left(1 - \frac{S_e}{S}\right)(\Lambda - (1 - \nu)\beta IS - \mu S) + M\left(1 - \frac{L_e}{L}\right)((1 - \nu)\beta IS - (\sigma + \mu)L) +
$$
\n
$$
P\left(1 - \frac{L_e}{I}\right)(\sigma L - (\nu + \pi + \delta + \mu)I) + Q\left(1 - \frac{C_e}{C}\right)(\nu I - (\omega + \delta + \mu)C) \tag{4.3}
$$

At endemic equilibrium, system (3.1) become;

$$
\Lambda = (1 - \nu)\beta I_e S_e + \mu S_e
$$

\n
$$
(\sigma + \mu) = \frac{(1 - \nu)\beta I_e S_e}{L_e}
$$

\n
$$
(\nu + \pi + \delta + \mu) = \frac{\sigma L_e}{I_e}
$$

\n
$$
(\omega + \delta + \mu) = \frac{\nu I_e}{C_e}
$$
\n(4.4)

Substituting (4.4)in (4.3), we get;

$$
\frac{dG}{dt} = \left(1 - \frac{S_e}{S}\right) \left[(1 - \nu)\beta I_e S_e + \mu S_e - (1 - \nu)\beta I S - \mu S\right] \n+ M\left(1 - \frac{L_e}{L}\right) \left[(1 - \nu)\beta I S - \frac{(1 - \nu)\beta I_e S_e L}{L_e}\right] \n+ P\left(1 - \frac{I_e}{I}\right) \left[\sigma L - \frac{\sigma L_e I}{I_e}\right] + Q\left(1 - \frac{C_e}{C}\right) \left[\nu I - \frac{\nu I_e C}{C_e}\right]
$$
\n(4.5)

Equation (4.5) can be written as

$$
\frac{dG}{dt} = -\mu \frac{(S - S_e)^2}{S} + \left(1 - \frac{1}{w}\right)(1 - \nu)\beta I_e S_e - \left(1 - \frac{1}{w}\right)(1 - \nu)wy\beta I_e S_e
$$

$$
+ M(1 - \nu)\beta I_e S_e \left(1 - \frac{1}{x}\right)(wy - x)
$$

$$
+ P\sigma L_e \left(1 - \frac{1}{y}\right)(x - y) + Q\nu I_e \left(1 - \frac{1}{z}\right)(y - z)
$$
\n(4.6)

where $w = \frac{S}{S_e}$, $x = \frac{L}{L_e}$, $y = \frac{I}{I_e}$ and $z = \frac{C}{C_e}$ S_e ^{*,u*} = L_e ^{*,y*} = I_e

Further, equation (4.6) can be written as $\frac{dG}{dt} = -\mu \frac{(S - S_e)^2}{S} + f(w, x, y, z)$ Where

$$
f(w, x, y, z) = (1 - \frac{1}{w})(1 - \nu)\beta I_e S_e - (1 - \frac{1}{w})(1 - \nu)wy\beta I_e S_e + M(1 - \nu)\beta I_e S_e (1 + wy - \frac{wy}{x} - x) + P\sigma L_e (1 + x - \frac{x}{y} - y) + Q\nu I_e (1 + y - \frac{y}{z} - z)
$$
(4.7)

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To determine M, P and Q, set the coefficients of wy, y and z of equation (4.7) equal to zero. Thus we obtain,

$$
M(1 - \nu)\beta I_e S_e = 0
$$

$$
-P\sigma L_e = 0
$$

$$
-Q\nu I_e = 0
$$

let M=1, solving for P and Q, we have;

$$
P = \frac{(1-\nu)\beta I_e S_e}{\sigma L_e}, Q = \frac{(1-\nu)\beta S_e}{\nu}
$$

Substituting for M, P and Q in equation (4.7) , we obtain

$$
f(w, x, y, z) = \left(1 - \frac{1}{w}\right)(1 - \nu)\beta I_e S_e - \left(1 - \frac{1}{w}\right)(1 - \nu)wy\beta I_e S_e + (1 - \nu)\beta I_e S_e(1 + wy - \frac{wy}{x} - x) + (1 - \nu)\beta I_e S_e(1 + x - \frac{x}{y} - y) + (1 - \nu)\beta I_e S_e(1 + y - \frac{y}{z} - z)
$$

Which upon simplification gives

$$
f(w, x, y, z) = (1 - \nu)\beta I_e S_e (4 + y - \frac{y}{z} - z - \frac{x}{y} - \frac{1}{w} - \frac{wy}{x})
$$

Using geometric mean inequality , we obtain

$$
\frac{y}{z} + z + \frac{x}{y} + \frac{1}{w} + \frac{wy}{x} - y > 4
$$

Thus $f(w, x, y, z) \leq 0$. Hence $\frac{dG}{dt} \leq 0$ in Ω The equality $\frac{dG}{dt} = 0$ iff w=x=y=z=1 and $S = S_e$, $L = L_e, I = I_e, C = C_e$. Therefore, using LaSalle's invariance principle, the endemic equilibrium point of the system (3.1) is globally asymptotically stable. □

5 Numerical Simulation

We carry out nume[rica](#page-3-1)l simulations of the model (3.1), using MATLAB **ode45** solver. The parameter values used are presented in Table 1. Simulation results are presented in Figures (a) to (d).

Parameter symbol	Value	Source
	8.7×10^{-3} /day	CIA(2014)
μ	7.0×10^{-3} /day	CIA(2014)
	0.0011/day	Estimate
π	10^6 8.0 \times $10^{-1}/day$	Bhunu and Garira (2009)
δ	$3.95 \times 10^{-1}/day$	Roeger et al. (2009)
ω	$8.7 \times 10^{-1}/day$	Estimate
σ	$5.0 \times 10^{-1}/day$	KAIS (2012)
$\boldsymbol{\nu}$	$0 < \nu < 1$	Assumed

Table 1. Parameter val[ues](#page-3-1) of the model

Fig. 2(a) is graphical representation indicating the trends of population classes in the absence of hygiene consciousness. It can be seen that susceptibles fall to zero because they are infected by Infectious individuals whereas latently Infected and Infectious individuals increase rapidly to approximately 9900 and 3400 respectively then start declining and eventually converge to zero. Fig. 2(b) to 2(d) demonstrate the trends of population classes in the presence hygiene consciousness. In

Fig. 2. Simulation results demonstrating the trends of (a) S(t), L(t) and I(t) when there is no hygiene consciousness, (b) all population classes when $\nu = 0.3$, (c) all **population classes when** $\nu = 0.6$ **and** (d) all population classes when $\nu = 0.9$

Fig. 2(b) it is observed that for $\nu = 0.3$ the number of latently Infected and Infectious individuals increases to a certain point and then start declining, implying that $\nu = 0.3$ is not effective in controlling tuberculosis. From Fig. 2(c) it can be observed that $\nu = 0.6$, number of latently Infected and Infectious individuals increases to a certain point and then start declining but the peaks of the two graphs in Fig. 2(c) are a bit lower as compared to those in Fig. 2(b). Fig. 2(d) shows that for $\nu = 0.9$, the numbers of all population classes fall off and eventually converge to zero. This suggest that effective hygiene consciousness is sufficient in controlling TB epidemic. The infectious individuals can observe hygiene through cough etiquette, ensuring that windows are open while in congested area and not spitting everywhere. Also, hygiene can be observed through proper cleaning and sterilization of hospital equipments.

6 Conclusion

A mathematical model of TB which captures the effects of hygiene consciousness is formulated. We proved the positivity of the solutions and determined the equilibrium points. We carried out stability analysis of the model and it showed that the disease-free equilibrium point is both locally and globally asymptotically stable given that $R_0 < 1$, implying that TB can be eliminated from the population. Whereas the endemic equilibrium point is both locally and globally asymptotically stable given that $R_0 > 1$, implying that TB will eventually be able to invade the population but its transmission levels can be kept at manageable levels in the presence of hygiene consciousness. The obtained numerical simulations results suggest that in the absence of hygiene consciousness TB disease could invade the population while in the presence of effective hygiene consciousness, the development of TB disease is slowed down. Hence this implies that there is a need to embrace hygiene consciousness as a control measure of TB.

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Competing Interests

Authors have declared that no competing interests exist.

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 $\mathcal{L}=\{1,2,3,4\}$, we can consider the constant of the con *⃝*c *2020 Mawira and Malonza; This is an Open Access article distributed under the terms of the Creative Commons Attribution License http://creativecommons.org/licenses/by/4.0, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.*

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