Available online at http://scik.org
J. Math. Comput. Sci. 10 (2020), No. 6, 2808-2819
https://doi.org/10.28919/jmcs/4949
ISSN: 1927-5307

MATHEMATICAL MODEL ON THE IMPACT OF PROTECTION AGAINST TUNGIASIS TRANSMISSION DYNAMICS

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Abstract. In this study, a mathematical model based on a system of ordinary differential equations is formulated to describe the dynamics of tungiasis infection incorporating protection as a control strategy against infection. The basic reproduction number is computed using the next generation matrix approach. The existence of the steady states of the model are determined and the stability analysis of the model carried out. By Routh-Hurwitz criterion the disease free (DFE) and the endemic equilibrium (EE) points are found to be locally asymptotically stable. Numerical simulation of the model carried out showed that a high protection rate leads to a low tungiasis prevalence in a given population.

Keywords: tungiasis; basic reproduction number; stability.

2010 AMS Subject Classification: 93A30.

1. INTRODUCTION

Tungiasis is a parasitical infestation caused by a female ectoparasite called *Tunga penetrans* commonly known as the jigger flea. *Tunga penetrans* is the smallest known flea at only 1mm in length and is usually recognizable in its parasitic phase. However, when attached beneath the

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Received August 15, 2020

skin, it can reach up to 1cm in length [1]. Epidemiologically, human-to-human transmission is not possible. The jigger must go through phases of its life cycle in sandy or dusty soil before becoming infective to another human. Animals, mostly mammals, can also be infected with jiggers and serve as reservoir hosts thus continuing the cycle and contamination of the environment [2].

The first evidence of the infection is a tiny black lesion on the skin at the point penetration. The area around the embedded flea becomes very itchy and inflamed leading to ulcerations and accumulation of pus. The spread of HIV/AIDS can be passed from one person to anther due to sharing of pins and needles while extracting the flea [3]. Other possible infections include Tetanus, Lumphangitis, Gangrene and Bacterium [7]. The disease is endemic in developing countries along the tropics particularly where poverty and poor standards of basic hygiene exist like in the resource poor communities of South America, the Carribean and Sub-Saharan Africa [10]. Possible treatment may be natural extrusion of the flea and/or egg sac with a sterile pin, followed by an antiseptic dressing [3]. Benzyl benzoate emulsion and potassium permanganate are commonly used to treat the infection. If it is possible to locate the area of the soil where the flea originates, it could be burnt off or sprayed with a suitable insecticide in an effort to kill the fleas. It is worth noting that thorough fumigation of homes is necessary for complete eradication of the infection. Tungiasis can be prevented by observing high standards of cleanliness, encouraging wearing of shoes and use of flea repellants on the skin [2].

In order to control, prevent and treat Tungiasis effectively, it is vital to understand its transmission dynamics. Nthiiri [4] formulated an SIR model for the transmission dynamics of jigger infection incorporating treatment as a control strategy and found out that effective treatment of jigger infection prevents rapid progression of the disease. Kahuru *et al* [9], carried out a research on an optimal control technique in a mathematical model for the dynamics of Tungiasis in a community. The authors found out that controlling of infected soils and animal reservoirs with insecticides, environmental hygiene may serve as possible approach to control Tungiasis. In this study, we look into the impact of protection on the transmission dynamics of Tungiasis.

2. MODEL FORMULATION

In the model we divided the total population (N) into three epidemiological classes; susceptible individuals (S), individuals who are protected against tungiasis (P) and infectious individuals (I) and schematically described the dynamics of the model in the Figure 1;

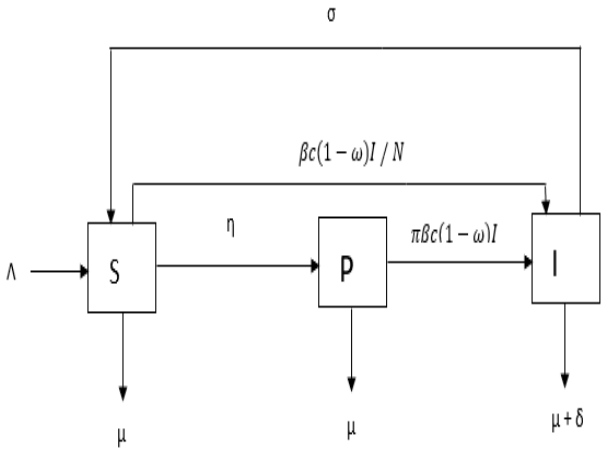

FIGURE 1. Flow chart

From the Figure 1, the model can be presented as the system ODEs;

$$
\begin{aligned}
\frac{dS}{dt} &= \Lambda - \left[\frac{(1-\omega)\beta cI}{N} + \eta + \mu\right] S + \sigma I \\
\frac{dP}{dt} &= \eta S - \left[\frac{(1-\omega)\pi\beta cI}{N} + \mu\right] P \qquad (1) \\
\frac{dI}{dt} &= \frac{(1-\omega)\beta cI}{N} S + \frac{(1-\omega)\pi\beta cI}{N} P - (\sigma + \delta + \mu) I
\end{aligned}
$$

where; Λ represents recruitment rate of the susceptibles, μ represents the natural mortality rate, β is the probability of an individual getting infected by an infectious individual , c is the per capita contact rate, π is a modification parameter, $0 \leq \omega \leq 1$ is the measure of protection efficacy, η is the rate at which susceptibles progress to protected class, σ represent recovery rate without immunity, and finally δ is tungiasis disease induced death rate.

3. ANALYSIS OF THE MODEL

Since the system (1) describes human population, all the solutions of state variable with non-negative initial conditions are non-negative $\forall\ t > 0$ and they are bounded in the feasible region $\Gamma = \{(S,P,I) \in \mathbb{R}^3_+; S > 0, P, I \geq 0; N \leq \frac{\Lambda}{\mu}\}$

3.1. Disease Free Equilibrium (DFE). The disease-free equilibrium denoted by E_0 a point where the disease is not present in the population. The DFE of the system (1) is given by

$$E_0 \begin{pmatrix} S_0 \\ P_0 \\ I_0 \end{pmatrix} = E_0 \begin{pmatrix} \frac{\Lambda}{(\eta+\mu)} \\ \frac{\eta\Lambda}{(\eta+\mu)\mu} \\ 0 \end{pmatrix}$$

3.2. The Protection Reproduction Number. The protection reproduction number $(\mathscr{R}_P)$ is the expected number of secondary infections produced in a completely susceptible population by a typical infected individual during his/her infectious lifetime in the presence of protection. By using the next generation matrix approach [6], $(\mathscr{R}_P)$ is given by the spectral radius of the matrix FV^{-1}.

the matrices F and V are given by

$$F = \begin{bmatrix} 0 & 0 \\ 0 & \frac{(1-\omega)\beta c\Lambda}{N(\eta+\mu)} + \frac{(1-\omega)\pi\beta c\eta\Lambda}{N(\eta+\mu)\mu} \end{bmatrix}$$

$$V = \begin{bmatrix} [\eta+\mu] & \frac{(1-\omega)\beta c}{N}\frac{\Lambda}{(\eta+\mu)} - \sigma \\ 0 & (\sigma+\delta+\mu) \end{bmatrix}$$

and it follows that

$$FV^{-1} = \begin{bmatrix} 0 & 0 \\ 0 & \frac{(1-\omega)\beta c\Lambda}{(\sigma+\delta+\mu)}\left(\frac{\mu+\pi\eta}{N(\eta+\mu)\mu}\right) \end{bmatrix}$$

Thus

$$\mathscr{R}_P = \frac{\beta c}{(\sigma+\delta+\mu)}\left(\frac{(1-\omega)\Lambda(\mu+\pi\eta)}{N(\eta+\mu)\mu}\right)$$

3.3. Relation of the protection reproduction number to the basic reproduction number. The basic reproduction number or the reproduction number without protection is given by

$$\mathscr{R}_0 = \frac{\beta c}{(\sigma+\delta+\mu)}$$

Thus

$\mathscr{R}_P = \mathscr{R}_0 \left(\frac{(1-\omega)\Lambda(\mu+\pi\eta)}{N(\eta+\mu)\mu} \right)$

Since

$(1-\omega)\Lambda(\mu+\pi\eta) < N(\eta+\mu)\mu,$

$\mathscr{R}_P = \mathscr{R}_0 \left(\frac{(1-\omega)\Lambda(\mu+\pi\eta)}{N(\eta+\mu)\mu} \right) < \mathscr{R}_0$ for $0 < \eta < 1$

and $\mathscr{R}_P = \mathscr{R}_0$ for $\eta = 0$.

Thus the protection will always reduce the basic reproduction number of tungiasis.

3.4. Local stability of disease free equilibrium point. In this section we analyze the stability of the disease free equilibrium point. This is carried out to predict the long term behaviour of the solutions of the model. The Jacobian Matrix of System (1) is given as:

$$J = \begin{bmatrix} -\left[\frac{(1-\omega)\beta cI}{N} + \eta + \mu\right] & 0 & -\frac{(1-\omega)\beta c}{N}S + \sigma \\ \eta & -\left[\frac{(1-\omega)\pi\beta cI}{N} + \mu\right] & -\frac{(1-\omega)\pi\beta c}{N}P \\ \frac{(1-\omega)\beta cI}{N} & \frac{(1-\omega)\pi\beta cI}{N} & \frac{(1-\omega)\beta c}{N}S + \frac{(1-\omega)\pi\beta c}{N}P - (\sigma+\delta+\mu) \end{bmatrix}$$

Theorem 3.1. *The disease free equilibrium (E_0) is locally asymptotically stable for $\mathscr{R}_P < 1$, otherwise unstable.*

Proof. E_0 of system (1) is locally asymptotically stable if $Re(\lambda) < 0$ where λ can be evaluated from the relation $|\lambda I - J(E_0)| = 0$, where $J(E_0)$ is the Jacobian matrix of system (1) at E_0

$J(E_0)$ is given by

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

By using the relation $|\lambda I - J(E_0)| = 0$, we obtain $Re(\lambda)$ as

$\lambda_1 = -(\eta+\mu)$

$\lambda_2 = -\mu$

$\lambda_3 = \frac{(1-\omega)\beta c\Lambda\mu + (1-\omega)\pi\beta c\eta\Lambda}{N(\eta+\mu)\mu} - (\sigma+\delta+\mu)$

Clearly, $\lambda_1, \lambda_2 < 0$ and $\lambda_3 < 0$ for$\mathscr{R}_P < 1$ and $\lambda_3 > 0$ for$\mathscr{R}_P > 1$

Thus E_0 is locally asymptotically stable for $\mathscr{R}_P < 1$ and unstable for $\mathscr{R}_P > 1$ □

This implies that for a small perturbation of the DFE, the solutions of system (1) will eventually converge to the DFE whenever $\mathscr{R}_P < 1$. Epidemiologically, it implies that if a few infectious individuals are introduced into a fully susceptible population, the disease would die out whenever $\mathscr{R}_P < 1$, otherwise the disease would spread.

3.5. Global Stability of the Disease Free Equilibrium Point. Now we use comparison theorem as in Lakshmikantham et al. [5] , to prove the global stability of DFE.

Theorem 3.2. *The disease-free equilibrium of the system (1) is globally asymptotically stable when the reproduction number $\mathscr{R}_P < 1$ and unstable when $\mathscr{R}_P > 1$.*

Proof. Using the comparison theorem in Lakshmikantham et al. [5] we rewrite the disease compartments as

$$\begin{bmatrix} \frac{dS}{dt} \\ \frac{dI}{dt} \end{bmatrix} = (F-V)\begin{bmatrix} S \\ I \end{bmatrix} - \begin{bmatrix} -\frac{\beta cI}{N}(S_0 - S) \\ \frac{(1-\omega)\beta cI}{N}(S_0 - S) + \frac{(1-\omega)\pi\beta cI}{N}(P_0 - P) \end{bmatrix}$$

Where F and V are defined in section (3.2)

Since $S \leq S_0 = \frac{\Lambda}{(\eta+\mu)}$ and $P \leq P_0 = \frac{\eta\Lambda}{(\eta+\mu)\mu}$ $\forall\, t > 0$, it follows that

$$\begin{bmatrix} \frac{dS}{dt} \\ \frac{dI}{dt} \end{bmatrix} \leq (F-V)\begin{bmatrix} S \\ I \end{bmatrix}$$

Where $F - V = \begin{bmatrix} -[\eta+\mu] & -\frac{(1-\omega)\beta c\Lambda}{N(\eta+\mu)} + \sigma \\ 0 & \frac{(1-\omega)\beta c\Lambda}{N(\eta+\mu)} + \frac{(1-\omega)\pi\beta c\eta\Lambda}{N(\eta+\mu)\mu} - (\sigma+\delta+\mu) \end{bmatrix}$

The characteristic equation is given by

$$-((\eta+\mu)+\lambda)((\tfrac{(1-\omega)\beta c\Lambda}{N(\eta+\mu)} + \tfrac{(1-\omega)\pi\beta c\eta\Lambda}{N(\eta+\mu)\mu} - (\sigma+\delta+\mu)) - \lambda) = 0$$

$$\lambda_1 = -(\eta+\mu)$$

$$\lambda_2 = \tfrac{(1-\omega)\beta c\Lambda}{N(\eta+\mu)} + \tfrac{(1-\omega)\pi\beta c\eta\Lambda}{N(\eta+\mu)\mu} - (\sigma+\delta+\mu)$$

Clearly $\lambda_1 < 0$ and $\lambda_2 < 0$ when $\mathscr{R}_P < 1$. Since all the eigenvalues of the matrix $F - V$ have negative real parts, then system (1) is stable whenever $\mathscr{R}_P < 1$. Therefore, $(S,I) \to (\frac{\Lambda}{\eta+\mu}, 0)$ as $t \to \infty$. By the comparison theorem, it follows that $(S,I) \to (\frac{\Lambda}{\eta+\mu}, 0)$ and $P \to \frac{\eta\Lambda}{(\eta+\mu)\mu}$ Then $(S,P,I) \to E_0$ as $t \to \infty$. Thus, E_0 is globally asymptotically stable for $\mathscr{R}_P < 1$. □

This implies that given a large perturbation of the DFE, the solutions of system (1) will eventually converge to the DFE whenever $\mathscr{R}_P < 1$. Epidemiologically, it implies that if a large number

of infectious individuals are introduced in to a fully susceptible population the disease would die out whenever $\mathscr{R}_P < 1$, otherwise, the disease would spread.

3.6. Existence of endemic equilibrium point. Let $E^*(S^*, P^*, I^*)$ denote the endemic equilibrium point of system (1).

Theorem 3.3. *There exists a unique endemic equilibrium of system (1) when $\mathscr{R}_P > 1$.*

Proof. Equating the right hand side of system (1) to zero and substituting $E^*(S^*, P^*, S^*)$ for S, P and I , we obtain

$$\begin{aligned} 0 &= \Lambda - \left[\frac{(1-\omega)\beta c I^*}{N} + \eta + \mu\right] S^* + \sigma I^* \\ 0 &= \eta S^* - \left[\frac{(1-\omega)\pi\beta c I^*}{N} + \mu\right] P^* \\ 0 &= \frac{(1-\omega)\beta c I^*}{N} S^* + \frac{(1-\omega)\pi\beta c I^*}{N} P^* - (\sigma + \delta + \mu) I^* \end{aligned}$$

(2)

From the first and second equations of system (2) we have

$$S^* = \frac{N(\Lambda + \sigma I^*)}{((1-\omega)\beta c I^* + N(\eta + \mu))} \tag{3}$$

$$P^* = \frac{N^2 \eta (\Lambda + \sigma I^*)}{((1-\omega)\beta c I^* + N(\eta + \mu))\,((1-\omega)\pi\beta c I^* + N\mu)} \tag{4}$$

To solve for I^*, we substitute equations (3)and(4) in the last equation of system (2)and simplify to get

$$AI^{2*} + BI^* + C = 0 \tag{5}$$

where

$A = (\delta + \mu)\frac{(1-\omega)^2\pi(\beta c)^2}{N}$

$B = (\eta + \mu)(\sigma + \delta + \mu)(1-\omega)\pi\beta c + \mu(\delta + \mu)(1-\omega)\beta c - \frac{(1-\omega)^2\pi(\beta c)^2\Lambda}{N} - (1-\omega)\pi\beta c\eta\sigma$

$C = -(1-\omega)\beta c\Lambda(\mu + \pi\eta) + (\sigma + \delta + \mu)N(\eta + \mu)\mu$

Clearly $C < 0$ when $\mathscr{R}_P > 1$ and $A > 0$. This implies that irrespective of the sign of B, equation (5) has two real roots: positive root and negative root. Hence there exist a positive unique endemic equilibrium. □

3.7. Local stability of endemic equilibrium point.

Theorem 3.4. *The endemic equilibrium of system (1) is locally asymptotically stable whenever* $\mathscr{R}_P > 1$

Proof. We use the trace and the determinant to investigate endemic equilibrium's stability. The The Jacobian matrix at $E^*(S^*, P^*, S^*)$ is given by

(6)
$$J(E^*) = \begin{bmatrix} -\left(\frac{(1-\omega)\beta cI^*}{N} + \eta + \mu\right) & 0 & -\frac{(1-\omega)\beta cS^*}{N} + \sigma \\ \eta & -\left(\frac{(1-\omega)\pi\beta cI^*}{N} + \mu\right) & -\frac{(1-\omega)\pi\beta cP^*}{N} \\ \frac{(1-\omega)\beta cI^*}{N} & \frac{(1-\omega)\pi\beta cI^*}{N} & \frac{(1-\omega)\beta cS^*}{N} + \frac{(1-\omega)\pi\beta cP^*}{N} - (\sigma+\delta+\mu) \end{bmatrix}$$

In view of the third equation of system (2) we have

(7)
$$\frac{(1-\omega)\beta c}{N}S^* + \frac{(1-\omega)\pi\beta c}{N}P^* = (\sigma+\delta+\mu)$$

Substituting equation (7) in the Jacobian matrix (6) we obtain

(8)
$$J(E^*) = \begin{bmatrix} -\left(\frac{(1-\omega)\beta cI^*}{N} + \eta + \mu\right) & 0 & -\frac{(1-\omega)\beta cS^*}{N} + \sigma \\ \eta & -\left(\frac{(1-\omega)\pi\beta cI^*}{N} + \mu\right) & -\frac{(1-\omega)\pi\beta cP^*}{N} \\ \frac{(1-\omega)\beta cI^*}{N} & \frac{(1-\omega)\pi\beta cI^*}{N} & 0 \end{bmatrix}$$

From the Jacobian matrix (8), the trace($tr(J(E^*))$) and the determinant ($Det(J(E^*))$) are given by

$$tr(J(E^*)) = -\left(\frac{(1-\omega)\beta cI^*}{N} + \frac{(1-\omega)\pi\beta cI^*}{N} + 2\mu + \eta\right)$$

$$\begin{aligned}
Det(J(E^*)) = &\left(\frac{(1-\omega)\beta cI^*}{N}\right)\left(\frac{(1-\omega)\beta cS^*}{N}\right)\left(\frac{(1-\omega)\beta cI^*}{N}\right)\\
&+ (\sigma+\delta+\mu)\left(\frac{(1-\omega)\beta cI^*}{N}\right)\eta + (\sigma+\delta+\mu)\left(\frac{(1-\omega)\beta cI^*}{N}\right)\mu\\
&+ \sigma\left(\frac{(1-\omega)\pi\beta cI^*}{N}\right)\eta + \sigma\left(\frac{(1-\omega)\pi\beta cI^*}{N}\right)\left(\frac{(1-\omega)\beta cI^*}{N}\right)\\
&+ \sigma\left(\frac{(1-\omega)\beta cI^*}{N}\right)\mu - \left(\frac{(1-\omega)\beta cI^*}{N}\right)(\sigma+\delta+\mu)\left(\frac{(1-\omega)\beta cI^*}{N}\right)\\
&- \left(\frac{(1-\omega)\beta cS^*}{N}\right)\left(\frac{(1-\omega)\beta cI^*}{N}\right)\eta - \left(\frac{(1-\omega)\beta cS^*}{N}\right)\left(\frac{(1-\omega)\beta cI^*}{N}\right)\mu\\
&- \left(\frac{(1-\omega)\beta cS^*}{N}\right)\left(\frac{(1-\omega)\pi\beta cI^*}{N}\right)\eta\\
&- \left(\frac{(1-\omega)\beta cS^*}{N}\right)\left(\frac{(1-\omega)\pi\beta cI^*}{N}\right)\left(\frac{(1-\omega)\beta cI^*}{N}\right)\\
&- \left(\frac{(1-\omega)\beta cS^*}{N}\right)\left(\frac{(1-\omega)\beta cI^*}{N}\right)\mu
\end{aligned}$$

Clearly $tr(J(E^*)) < 0$ and $Det(J(E^*)) > 0$ if

(9)
$$\begin{aligned}
&\left(\frac{(1-\omega)\beta cI^*}{N}\right)\left(\frac{(1-\omega)\beta cS^*}{N}\right)\left(\frac{(1-\omega)\beta cI^*}{N}\right) + (\sigma+\delta+\mu)\left(\frac{(1-\omega)\beta cI^*}{N}\right)\eta\\
&+ (\sigma+\delta+\mu)\left(\frac{(1-\omega)\beta cI^*}{N}\right)\mu + \sigma\left(\frac{(1-\omega)\pi\beta cI^*}{N}\right)\eta\\
&+ \sigma\left(\frac{(1-\omega)\pi\beta cI^*}{N}\right)\left(\frac{(1-\omega)\beta cI^*}{N}\right) + \sigma\left(\frac{(1-\omega)\beta cI^*}{N}\right)\mu\\
&> \left(\frac{(1-\omega)\beta cI^*}{N}\right)(\sigma+\delta+\mu)\left(\frac{(1-\omega)\beta cI^*}{N}\right) + \left(\frac{(1-\omega)\beta cS^*}{N}\right)\left(\frac{(1-\omega)\beta cI^*}{N}\right)\eta\\
&\quad + \left(\frac{(1-\omega)\beta cS^*}{N}\right)\left(\frac{(1-\omega)\beta cI^*}{N}\right)\mu + \left(\frac{(1-\omega)\beta cS^*}{N}\right)\left(\frac{(1-\omega)\pi\beta cI^*}{N}\right)\eta\\
&\quad + \left(\frac{(1-\omega)\beta cS^*}{N}\right)\left(\frac{(1-\omega)\pi\beta cI^*}{N}\right)\left(\frac{(1-\omega)\beta cI^*}{N}\right)\\
&\quad + \left(\frac{(1-\omega)\beta cS^*}{N}\right)\left(\frac{(1-\omega)\beta cI^*}{N}\right)\mu
\end{aligned}$$

From Theorem 3.3, a unique endemic equilibrium $E^*(S^*,P^*,I^*)$ of system (1) exists when $\mathscr{R}_P > 1$. Since $tr(J(E^*))$ and $Det(J(E^*))$ are functions of $E^*(S^*,P^*,S^*)$ and $tr(J(E^*)) < 0$ and $Det(J(E^*)) > 0$ provided that inequality (9) is satisfied, we conclude that the endemic equilibrium of system (1) is locally asymptotically stable whenever $\mathscr{R}_P > 1$ □

This implies that for a small perturbation of the EE, the solutions of system (1) will always converge to the EE whenever $\mathscr{R}_P > 1$. Epidemiologically, it implies that if a few infectious individuals are introduced in a fully susceptible population with $\mathscr{R}_P > 1$, then the disease would persist in the population.

4. Numerical Simulation

We carry out numerical simulations of the model (1), using MATLAB **ode45** solver. The parameter values used are presented in Table 1. Simulation results are presented in Figure 2.

TABLE 1. Parameter values of the model

Parameter symbol	Value	Source
Λ	4.4×10^{-3}/day	[12]
μ	1.6×10^{-2}/day	[12]
β	1.4989×10^{-2}/day	[13]
π	0.2-0.990	Assumed
δ	5.0×10^{-2}/day	[13]
ν	1.431×10^{-2}/day	Assumed
σ	4.27×10^{-1}/day	[13]
ω	$0 < \omega < 1$	Assumed

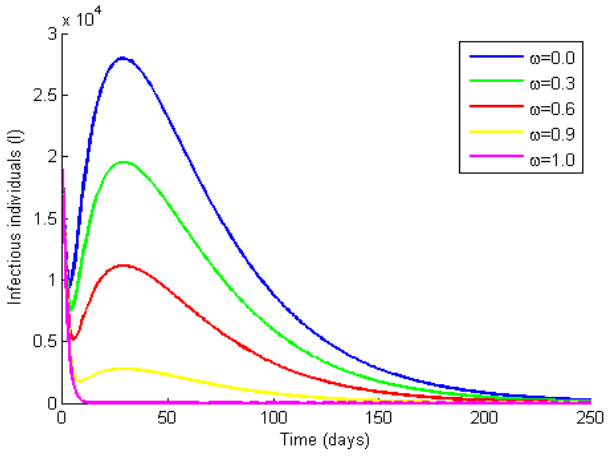

FIGURE 2. The impact of protection on infectious individuals

Figure 2, shows how protection can reduce the infected individuals. As the protection efficacy increases, the infected individuals reduce. This implies that people need to enhance protection measures in order eradicate tungiasis infection. This can be achieved through public awareness campaign on protective measures .

5. Conclusion

We conclude that effective protection of tungiasis infection prevents its rapid progression in the population. The government should also aim on improving the life standards of its citizens by mobilizing observation proper sanitation standards, wearing of shoes for all schooling children, provision of proper food to the venerable individuals and watering dusty floors in all public places. This would drastically reduce new infections and re-infection of tungiasis.

Conflict of Interests

The authors declare that there is no conflict of interests.

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