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Analysis and Modelling of Ringworm Infections in an Environment

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Abstract

Ringworm is a skin infection caused by different fungi depending on the part of the body. The fungi causing ringworms are found in the epidermis and the hair growing on the infected parts of the body. It is caused by fungal infection. Fungi causing the infections on the hair, nail bed and the skin is referred to as dermatophytes. In this study, we proposed, developed and analysed a mathematical model for ringworm that explains the mechanism of the infections. Ordinary differential equations and stability theory was used in the model's qualitative analysis. The model analysis revealed that the ringworm infections is globally asymptotically stable whenever the reproductive rate is less than unity. Sensitivity analysis was carried out to determine the contribution of each parameter on the ringworm reproduction rate. Numerical simulation of the ringworm model was conducted and the results displayed graphically.

Keywords: Ringworm infections, epidemic model, equilibrium points, stability analysis, ringworm reproductive rate.

1. INTRODUCTION

Ringworm is a skin infection which is caused by different fungi infections. The fungi causing ringworms are found in the epidermis and the hair growing on the infected parts of the body. It is caused by a fungal infection. Fungi causing the infections on the hair, nail bed, and the skin is referred to as dermatophytes.

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In a study conducted by [21], the infection can be acquired by coming into contact with a person who is infected, touching items which have been in contact with an infected person such as towels, bed linen, clothes and also from animals such as cats, dogs, guinea pigs which are infected. The study reveals that when a person gets the infection, it manifests itself as a rounded, red, inflamed patch which tends to spread outwards on the affected area of the skin. Sometimes only one patch of the infections occurs but several can occur especially if the infection is transmitted from handling an infected animal. A study conducted by [9] found out that ring worm is one of the most common and widespread ailments. The study showed that ring worm could be a temporarily disfiguring infection without accessible and successful treatment. In the early to mid-twentieth century, most of the treatments that were available tended to operate under "scorched earth" policy of treatment rather than a curative one.

A study conducted in the United States revealed that tinea capitis is predominantly a disease of per-adolescent children of ages between 5 and 10 years. It accounts up to 92.5 percent of dermatophytosis in children younger than 10 years. Tinea capitis is rare in adults but can occasionally be found in elderly patients. Internationally, tinea capitis is wide spread in some urban areas especially in children of Afro- Carribean origin, in North America, Central and South America. The disease is common in some regions of Africa and India. The infection of tinea capitis may vary with sex being 5 times more common in boys.

[6] carried out a study in South Africa where a population of 150 patients were subjected to wood light examination, microscopy and culture. The study revealed that Trichophyton Violaceum was isolated in 90 percent of positive cultures and wood light was positive in one patient with Microsporum gypseum. 50 percent of the patients presented the "black dot" type while 20 percent of the children presented with more than one clinical type simultaneously. The study concluded that the most common cause of tinea capitis in South Africa is Trichophyton Violaceum.

In a study conducted by [13], in the US and Europe showed that tinea capitis affects mainly African American children and migrant African children. The prevalence of tinea capitis varies widely in Nigeria: between 9.4 percent and 51.8 percent in the eastern part of Nigeria. In Ivory Coast, the prevalence was 11.34 percent and in Ethiopia a high prevalence similar to that of Ebonyi state in Nigeria was noted. Weger.W (2007) A study conducted in Nigeria, Kwara estate showed that a huge proportion of scalp lesions were caused by the non-dermatophyte moulds. The attitude of the general populace, especially the lower socioeconomic groups who are rural dwellers in Nigeria, is to ignore these lesions in their children since they are not life threatening and only try herbal treatment when need arises. Despite presence of florid infections, huge

percentage (85.6 percent) of the children said they did not know they had tinea infection on their scalp. There appears to be some form of tolerance to the infections because of the low percentage of children with symptomatic infections. The study revealed that the most common predisposing factors to tinea capitis included carrying objects on the scalp and sharing of hair clippers, scissors, combs, towels and fomites. Other factors reported were overcrowding and poor hygiene.

A report confirmed combs and hair trimming tools (which are often shared) as reservoirs for dermatophyte infection in families of previously infected individuals treated in a hospital. It is a common practice in the rural setting of Nigeria and Africa, to find children ferrying goods on their heads to hawk, and carry buckets which are placed in fairly unclean surroundings on their heads [1].

Mathematical models are capable of predicting the persistence of an infection in an environment. They explain the transmission dynamics of an infection and can as well suggest the best control measures in combating the infections. Models can also suggest the most cost effective control measure to minimise the cost in fighting an infections [8, 22, 2, 7]. Mathematical models generally contribute significantly in the fight against infectious diseases.

2. RINGWORM MODEL DESCRIPTION AND FORMULATION

The ringworm model is divided into four compartments, $S(t)$ represents the population that is at risk of being infected by ringworm, $E(t)$ represents the environment infested by dermatophytosis fungus, $I(t)$ is the infected individuals and $R(t)$ is the population which has recovered from the infection. Susceptible individuals are recruited into the population at a rate Λ.

Susceptible individuals gets infected through getting into contact with the environment with the fungus at a rate β.Those infected recover from the infection at a risk α. μrepresents the natural death rate in all the compartments. The recovered individuals can get the infection through interaction with environment at a rate of δ. The infected also introduce the fungus to the environment at a rate τ. The recovered returns to susceptible compartment at a rate γ.

The total population is given by

$$N(t) = S(t) + I(t) + R(t) \tag{1}$$

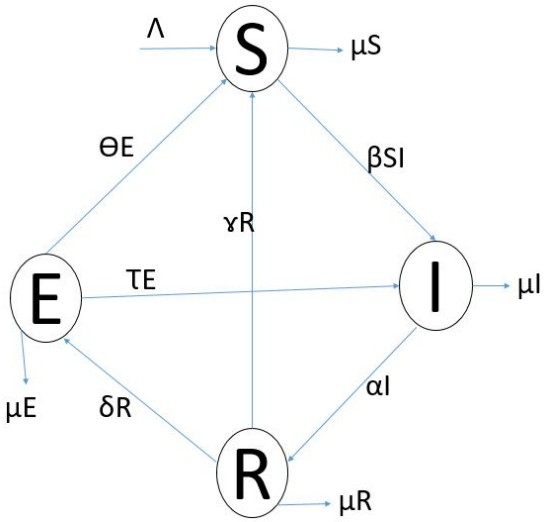

Figure 1: Ringworm schematic diagram.

The system of Ordinary differential equations are obtained as;

$$\left.\begin{aligned}
\frac{dS}{dt} &= \Lambda+\theta E+\gamma R-\beta SI-\mu S \\
\frac{dI}{dt} &= \beta SI+\tau E-(\mu+\alpha)I \\
\frac{dR}{dt} &= \alpha I-(\gamma+\delta+\mu)R \\
\frac{dE}{dt} &= \delta R-(\mu+\theta+\tau)E
\end{aligned}\right\} \tag{2}$$

3. THE POSITIVITY AND SOLUTION BOUNDEDNESS

A region in which solutions of a dynamical system is uniformly bounded in the proper subset ψCR^4_+ is said to be bounded . Consider the overall population at any given time (t).

$$N(t)=S(t)+I(t)+R(t) \tag{3}$$

$$\frac{dN}{dt}=\frac{dS}{dt}+\frac{dI}{dt}+\frac{dR}{dt} \tag{4}$$

$$\frac{dN}{dt}=\Lambda+\theta E+\gamma R-\beta SI-\mu S+\beta SI+\tau E-\mu I-\alpha I+\alpha I-\gamma R-\delta R-\mu R$$

By simplification;

$$\frac{dN}{dt} = \Lambda + \theta E + \tau E - \mu S - \mu I - \delta R - \mu R \tag{5}$$

In the absence of infection, there is no recovery.

Initially,

$N(0) = S(0)$

$$\frac{dN}{dt} = \Lambda - \mu S \tag{6}$$

Thus;

$$\frac{dN}{dt} = \Lambda - \mu N \tag{7}$$

Using the method of separation of variables,

$$\frac{dN}{\Lambda - \mu N} = dt \tag{8}$$

Solving the differential equation;

$\int \frac{dN}{\Lambda - \mu N} \leq dt$

$-\frac{1}{\mu} \ln /\Lambda - \mu N/ \leq t + C$

$\ln /\Lambda - \mu N/ \geq -\mu(t + C)$

$\Lambda - \mu N \geq e^{-\mu(t+C)} = e^{-\mu t - \mu C}$

where $e^{-\mu C} = A$

$\Lambda - \mu N \geq Ae^{-\mu t}$

at $N(0) = N_0 \Rightarrow t = 0, N = N_0$

$$\frac{\Lambda}{\mu} - N \geq \left(\frac{\Lambda - \mu N_0}{\mu}\right) e^{-\mu t} \tag{9}$$

As t$\to \infty$, $N \to \frac{\Lambda}{\mu}$.

This means that;

$0 \leq N \leq \frac{\Lambda}{\mu}$ and $N(t) \leq \frac{\Lambda}{\mu}$.

Thus,

$$\psi = \left\{ (S, I, R)\, \varepsilon R_+^3 : S + I + R \leq \frac{\Lambda}{\mu} \right\} \tag{10}$$

4. DISEASE FREE EQUILIBRIUM

At disease free equilibrium, there are no infection and recovery.

$I = R = E = 0$

$\frac{dS}{dt} = \Lambda + \theta E + \gamma R - \beta SI - \mu S$

$\Lambda + \theta E + \gamma R - \beta SI - \mu S = 0$

$$S* = \frac{\Lambda}{\mu} \tag{11}$$

Thus the disease free equilibrium,

$$(S*, 0, 0, 0) = (\frac{\Lambda}{\mu}, 0, 0, 0) \tag{12}$$

5. RINGWORM REPRODUCTIVE NUMBER

This refers to the number of ringworm infections that one infected person can produce in a completely susceptible populations. By using the concept in [4, 14, 15].

Since ringworm free equilibrium point was obtained as;

$\left(\frac{\Lambda}{\mu}, 0, 0, 0\right)$

The Jacobian matrix of the dynamical system would represented as;

Hence; $J = (S*, I*, R*, E*)$.

$$\begin{pmatrix} -\beta I - \mu & -\beta S & \gamma & \theta \\ \beta I & \beta S - (\mu + \alpha) & 0 & \tau \\ 0 & \alpha & -(\gamma + \delta + \mu) & \theta \\ 0 & 0 & \delta & -(\mu + \theta + \tau) \end{pmatrix} \tag{13}$$

Jacobian at disease free equilibrium, $\left(\frac{\Lambda}{\mu}, 0, 0, 0\right)$, becomes;

$$J(\frac{\Lambda}{\mu}, 0, 0, 0) = \begin{pmatrix} -\mu & -\beta\frac{\Lambda}{\mu} & \gamma & \theta \\ 0 & \beta\frac{\Lambda}{\mu} - (\mu + \alpha & 0 & \tau \\ 0 & \alpha & -(\gamma + \delta + \mu) & 0 \\ 0 & 0 & \delta & -(\mu + \theta + \tau) \end{pmatrix} \tag{14}$$

Hence;

$$\begin{bmatrix} -\mu & -\beta\dfrac{\Lambda}{\mu} & \gamma & \theta \\ 0 & \beta\dfrac{\Lambda}{\mu}-(\mu+\alpha) & 0 & \tau \\ 0 & \alpha & -(\alpha+\delta+\mu) & 0 \\ 0 & 0 & \delta & -(\mu+\theta+\tau) \end{bmatrix}$$

Determining the eigenvalues;

$$\begin{vmatrix} -\mu & -\beta\dfrac{\Lambda}{\mu} & \gamma & \theta \\ 0 & \beta\dfrac{\Lambda}{\mu}-(\mu+\alpha) & 0 & \tau \\ 0 & \alpha & -(\alpha+\delta+\mu) & 0 \\ 0 & 0 & \delta & -(\mu+\theta+\tau) \end{vmatrix} = 0$$

The eigenvalues are obtained as follows;

$$-\mu, \left[\beta\frac{\Lambda}{\mu}-(\mu+\alpha)\right], -(\alpha+\delta+\mu), -(\mu+\theta+\tau)$$

The dominant eigenvalue of the ringworm at disease free equilibrium would be;

$\left[\beta\dfrac{\Lambda}{\mu}-(\mu+\alpha)\right]$ if and only if $\beta\dfrac{\Lambda}{\mu} > (\mu+\alpha)$.

Hence, by imposing the condition that; $\beta\dfrac{\Lambda}{\mu} > (\mu+\alpha)$;

Ringworm reproductive number is given by;

$$R_r = \beta\frac{\Lambda}{\mu}-(\mu+\alpha) \tag{15}$$

5.1 Stability of the disease free equilibrium

The ringworm free equilibrium of the model was obtained as $\left(\dfrac{\Lambda}{\mu}, 0, 0, 0\right)$

Computing the Jacobian matrix at disease free equilibrium;

$$\begin{pmatrix} -\beta I-\mu & -\beta S & \gamma & \theta \\ \beta I & \beta S-(\mu+\alpha) & 0 & \tau \\ 0 & \alpha & -(\gamma+\delta+\mu) & \theta \\ 0 & 0 & \delta & -(\mu+\theta+\tau) \end{pmatrix} \tag{16}$$

Jacobian matrix at ringworm free equilibrium becomes

$$\begin{pmatrix} -\mu & -\beta\dfrac{\Lambda}{\mu} & \gamma & \theta \\ 0 & \beta\dfrac{\Lambda}{\mu}-(\mu+\alpha & 0 & \tau \\ 0 & \alpha & -(\gamma+\delta+\mu) & 0 \\ 0 & 0 & \delta & -(\mu+\theta+\tau) \end{pmatrix} \tag{17}$$

Hence;

$$\begin{bmatrix} -\mu & -\beta\dfrac{\Lambda}{\mu} & \gamma & \theta \\ 0 & \beta\dfrac{\Lambda}{\mu}-(\mu+\alpha) & 0 & \tau \\ 0 & \alpha & -(\alpha+\delta+\mu) & 0 \\ 0 & 0 & \delta & -(\mu+\theta+\tau) \end{bmatrix}$$

Eigenvalues at ringworm free equilibrium;

$$\begin{vmatrix} -\mu & -\beta\dfrac{\Lambda}{\mu} & \gamma & \theta \\ 0 & \beta\dfrac{\Lambda}{\mu}-(\mu+\alpha) & 0 & \tau \\ 0 & \alpha & -(\alpha+\delta+\mu) & 0 \\ 0 & 0 & \delta & -(\mu+\theta+\tau) \end{vmatrix} = 0$$

The eigen values are obtained as follows;

$$-\mu, -\left[(\mu+\alpha)-\beta\frac{\Lambda}{\mu}\right], -(\alpha+\delta+\mu), -(\mu+\theta+\tau)$$

The ringworm free equilibrium is locally asymptotically stable whenever the eigenvalues of the Jacobian matrix are negative. Hence, ringworm free equilibrium point is locally asymptotically stable [16, 11, 19].

6. ENDEMIC EQUILIBRIUM

This is evaluated by setting the system of the differential equations to zero.

$$\frac{dE}{dt} = \delta R - (\mu+\theta+\tau)E = 0$$

$$R* = \frac{(\mu+\theta+\tau)E*}{\delta} \tag{18}$$

$$\frac{dS}{dt} = \Lambda + \theta E + \gamma R - (\beta I + \mu)S = 0$$

$$S* = \frac{\Lambda + \theta E* + \gamma R*}{\beta I* + \mu} \tag{19}$$

$$\frac{dI}{dt} = \{\beta S - (\mu + \alpha)\} I + \tau E = 0$$

$$E* = \frac{(\mu + \alpha - \beta S*) I*}{\tau} \tag{20}$$

$$\frac{dR}{dt} = \alpha I - (\gamma + \delta + \mu) R = 0$$

$$I* = \frac{(\gamma + \delta + \mu) R*}{\alpha} \tag{21}$$

Thus, the ringworm endemic equilibrium $(E*, S*, I*, R*)$;

$$\left(\frac{(\mu + \alpha - \beta S*) I}{\tau}, \frac{\Lambda + \theta E* + \gamma R*}{\beta I* + \mu}, \frac{(\gamma + \delta + \mu) R*}{\alpha}, \frac{(\mu + \theta + \tau) E*}{\delta} \right) \tag{22}$$

6.1 Stability of the endemic equilibrium

Theorem: If $R_0 > 1$.then the endemic equilibrium is globally asymptotically stable.

Proof: By use of the concept of Lyapunov function defined by;

$L(E*, S*, I*, R*)$

$$\left.\begin{aligned} L(E*, S*, I*, R*) &= \left(E - E* - E* \ln\left(\frac{E*}{E}\right)\right) + \left(S - S* - S* \ln\left(\frac{S*}{S}\right)\right) \\ &= + \left(I - I* - I* \ln\left(\frac{I*}{I}\right)\right) + \left(R - R* - R* \ln\left(\frac{R*}{R}\right)\right) \end{aligned}\right\}$$

By computing the derivative of the L along the solution of the system equations directly

$$\frac{dL}{dt} = \left(\frac{E - E*}{E}\right)\frac{dE}{dt} + \left(\frac{S - S*}{S}\right)\frac{dS}{dt} + \left(\frac{I - I*}{I}\right)\frac{dI}{dt} + \left(\frac{R - R*}{R}\right)\frac{dR}{dt}$$

Hence;

$$\left.\begin{aligned} \frac{dL}{dt} &= \left(\frac{E - E*}{E}\right)\delta R - (\mu + \theta + \tau) E + \left(\frac{S - S*}{S}\right)\Lambda + \theta E + \gamma R - (\beta I + \mu) S \\ &= + \left(\frac{I - I*}{I}\right)\beta S I + \tau E - (\mu + \alpha) I + \left(\frac{R - R*}{R}\right)\alpha I - (\gamma + \delta + \mu) R \end{aligned}\right\}$$

By expansion;

$$\left.\begin{aligned}\frac{dL}{dt} &= \delta R-(\mu+\theta+\tau)E-\delta R\frac{E*}{E}+(\mu+\theta+\tau)E*+\Lambda+\theta E+\gamma R-(\beta I+\mu)S-\\ &= (\Lambda+\theta E+\gamma R)\frac{S*}{S}+(\beta I+\mu)S*+\beta SI+\tau E-(\mu+\alpha)I-\beta SI*-\tau E\frac{I*}{I}\\ &= +(\mu+\alpha)I*+\alpha I-(\gamma+\delta+\mu)R-\alpha I\frac{R*}{R}+(\gamma+\delta+\mu)R*\end{aligned}\right\}$$

Let,

$$\frac{dL}{dt}=P-Q \tag{23}$$

where P are the positive terms and Q are the negative terms

$$P=\Lambda+(\delta+\gamma)R+(\mu+\theta+\tau)E*+\mu S*+(\mu+\alpha)I* \tag{24}$$

$$Q=\mu S+(\Lambda+\theta E+\gamma R)\frac{S*}{S}+(\mu+\alpha)I+\tau E\frac{I*}{I} \tag{25}$$

If $P<Q$, then $\frac{dL}{dt}\leq o$

$\frac{dL}{dt}=0$, if and only if $S=S*, I=I*, E=E*, R=R*$

The largest compact set in $\left\{S,E,I,R\varepsilon\psi : \frac{dL}{dt}=0\right\}$is a singleton $H*$,where $H*$is the endemic equilibrium.

Therefore the endemic equilibrium is globally asymptotically stable in the invariant ψ if $P<Q$ by [8, 10, 20, 17].

7. SENSITIVITY ANALYSIS

Sensitivity analysis is the study of how the uncertainty in the output of a mathematical model or system (numeric or otherwise) can be divided and allocated to different sources of uncertainty in its inputs. The importance of the contribution of each parameter to the basic reproduction numbers to determine the effectiveness of each parameter value to the persistence of the disease in the environment [18, 12, 5, 3].

Sensitivity analysis is performed in order to determine the level of contribution of each parameter to the basic reproduction number, R_r

$$R_r=\frac{\beta\Lambda-\mu(\mu+\alpha)}{\mu} \tag{26}$$

The sensitivity index of a variable P is given by,

$$S_P^{R_r} = \frac{\partial R_r}{\partial P} * \frac{P}{R_r} \tag{27}$$

For β,

$S_\beta^{R_r} = \frac{\partial R_r}{\partial \beta} * \frac{\beta}{R_r}$

$S_\beta^{R_r} = \frac{\Lambda}{\mu} * \frac{\beta\mu}{\beta\Lambda - \mu(\mu+\alpha)}$

$$S_\beta^{R_r} = \frac{\beta\Lambda}{\beta\Lambda - \mu(\mu+\alpha)} \tag{28}$$

For Λ,

$$S_\Lambda^{R_r} = \frac{\partial R_r}{\partial \Lambda} * \frac{\Lambda}{R_r} \tag{29}$$

$S_\Lambda^{R_r} = \frac{\beta}{\mu} * \frac{\Lambda\mu}{\beta\Lambda - \mu(\mu+\alpha)}$

$$S_\Lambda^{R_r} = \frac{\beta\Lambda}{\beta\Lambda - \mu(\mu+\alpha)} \tag{30}$$

For μ,

$$S_\mu^{R_r} = \frac{\partial R_r}{\partial \mu} * \frac{\mu}{R_r} \tag{31}$$

$S_\mu^{R_r} = \frac{-\beta\Lambda - \mu^2}{\mu^2} * \frac{\mu^2}{\beta\Lambda - \mu(\mu+\alpha)}$

$$S_\mu^{R_r} = \frac{-(\beta\Lambda + \mu^2)}{\beta\Lambda - \mu(\mu+\alpha)} \tag{32}$$

For α,

$$S_\alpha^{R_r} = \frac{\partial R_r}{\partial \alpha} * \frac{\alpha}{R_r} \tag{33}$$

$$S_\alpha^{R_r} = \frac{-\mu}{\beta\Lambda - \mu(\mu+\alpha)} \tag{34}$$

From table 1, the sign of the sensitivity index helps to indicate the contribution of each parameter to the basic reproduction number. If the human recruitment is increase the basic reproduction number increases. Also , decrease in human recruitment rate will

reduce the basic reproduction number. Hence, both the contact and recruitment rates increases the reproductive rate.

Table 1: Sensitivity indices of ringworm model parameters.

Parameter	Sensitivity Ind ext
Λ	$+ve$
β	$+ve$
μ	$-ve$
α	$-ve$

8. NUMERICAL SOLUTIONS

Numerical simulation was done on the ringworm model parameters to find out the dynamics of the population of the susceptible, infectious and recovered persons. This was done to determine how the population of the susceptible, infectious and recovered population change with time as shown in 2, 3, 4 and 5.

8.1 Total population dynamics

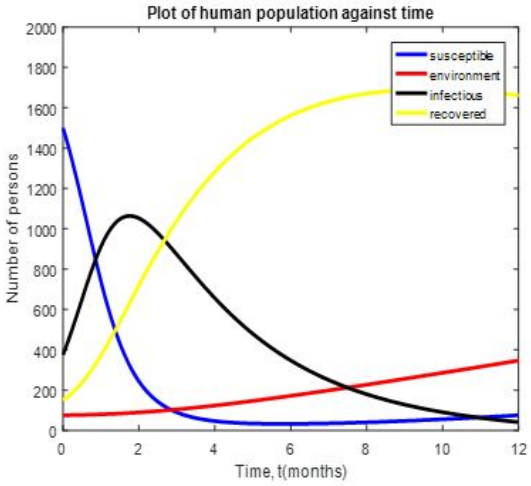

Figure 2: Ringworm population dynamics.

8.2 Susceptible population

From figure 3, the number of susceptible individuals with time as the move from the susceptible compartment to infectious compartment. This could be as a result of getting

into contact with the infection from the environment. By sharing bedding, combs and clothes.

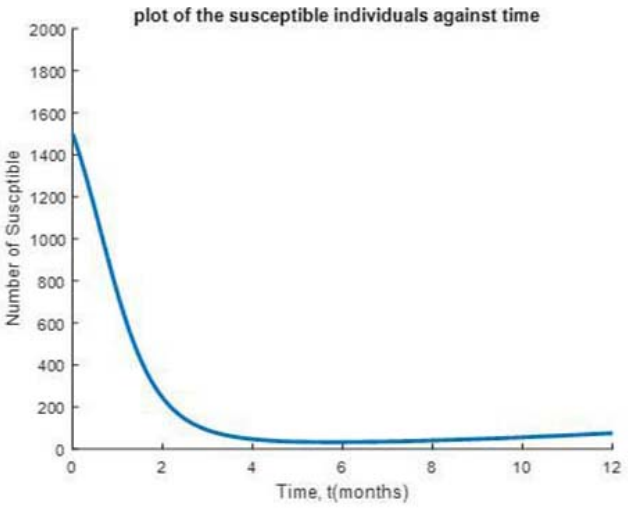

Figure 3: Population of susceptible individuals.

8.3 Population infected with ringworm

Figure 4 represents the relationship between the infectious population with time. The infectious population increases with time as the individuals move from the susceptible compartment. The reduction in the number of infectious population with time could be attributed to the rise in the number of recovered population as shown in 5.

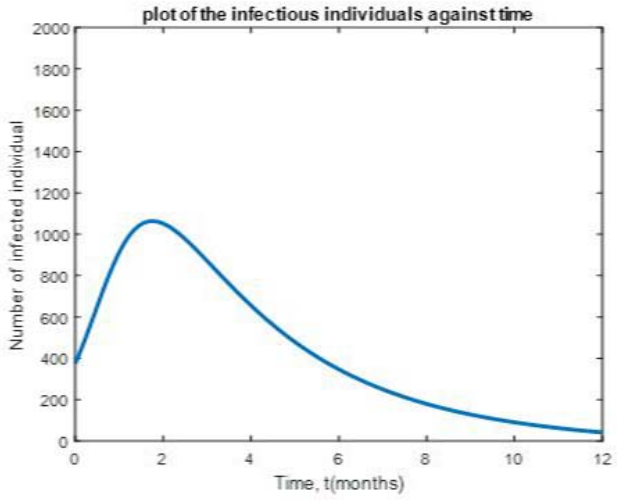

Figure 4: Population of infected individuals.

8.4 Population recovered from ringworm infections

Figure 5 shows the recovers individuals over time. The number of the recovered individuals is increasing indicating that the rate of recovery is high. This could be as a result of medication, maintaining personal hygiene and avoiding contact with the infected individuals and environment.

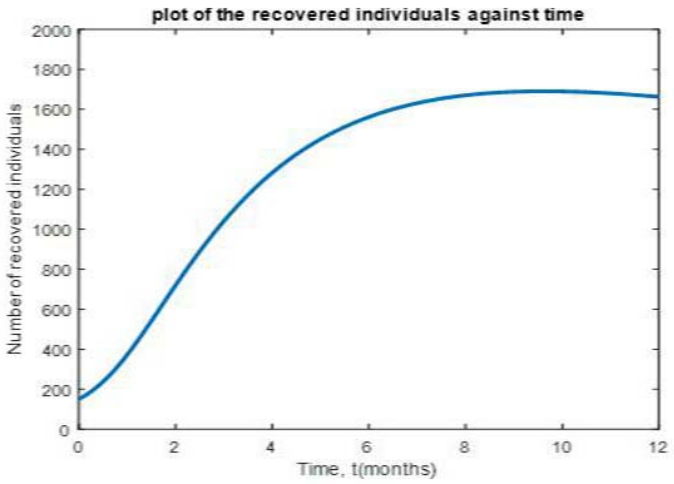

Figure 5: Population of recovered individuals.

9. CONCLUSION

A ringworm epidemic model was developed to analyse the transmission mechanism of ringworm infection in an environment. The model was analysed both quantitatively and qualitatively. Analysis of the basic reproduction number, equilibrium points and local and global stability was performed. Sensitivity analysis of the basic reproduction number was performed for each parameter to determine the contribution of each parameter to the basic reproduction number. The model's qualitative analysis revealed that the ringworm infections is globally asymptotically stable whenever the reproductive rate is less than unity.

This analysis revealed that both contact and recruitment rates causes an increase in the ringworm reproductive number. If there is any increase in the recruitment rate, it means that the basic reproduction number also increases and the disease would persist in the environment if the basic reproduction number is greater than one. However, an increase in the contact rate increases the value of the reproduction number as well.

From the numerical simulations conducted, the susceptible population reduces with time as the infectious population increases. therefore the relationship between the susceptible population varies inversely. Total number of recovered individuals increases

with time. This could be as a result of medication and maintaining personal hygiene. Increase in the contact rate with infected persons causes a rise of the infectious population.

Data availability

The data used in the analysis of the ringworm model were from previously published articles and reported studies which have been duly cited accordingly. Some of the parameter values are assumed and others are taken from published articles.

Conflicts of interest

Authors declare that there is no conflict of interest regarding the publication of this manuscript.

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