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Analysis and Numerical Simulation of Deterministic Mathematical Model of Pediculosis Capitis

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Abstract: In this work, we formulated a deterministic mathematical continuous time model for the detection and elimination of a non-life threatening disease (head lice) by considering a fixed (constant) population size during the epidemic period. The formulated mathematical model was normalized for easy analysis, the model's properties were obtained, as well as the disease free equilibrium point, the local stability and the basic reproduction number. We adopted MATLAB programing language to carry out the numerical simulation of the nonlinear ordinary differential equation, as well as simulation of different model state variables and effects of different model parameters on the state variables over time. Our result shows that early detection and treatment will lead to termination of the disease.

Index Terms: Pediculosis Capitis, Disease Free Equilibrium, Local Stability, Basic Reproduction Number and Equilibrium Analysis.

1. Introduction

Pediculosis capitis is the state of having an infestation of head lice. Lice are external parasitic insects that are commonly found on the eyelashes, head and eyebrows of human as its host. Pediculosis humanus capitis, commonly known as head lice resides close to the scalp of the head to maintain its body temperature and feeds extremely on human blood to survive. There are thousands of external parasite that feeds on human blood known as lice, but only few of them namely: Pediculus humanus capitis (head lice), Pediculus humanus corpon's (body or clothing lice) and Phthirus pubis (pubic or crab lice) that infest human population [1]. Out of the three aforementioned ectoparasites that infest human populations, Pediculus humanus capitis (Head lice) are the most common species and are of public concern in most developing countries lacking the juicy world health organization (WHO) primary health care program [2]. This ectoparasite (head louse) neither moves by crawling nor flies or hop and as such it's mainly transmitted or contacted via head-to-head contact with an infested person [3]. Head lice infestation (epidemics) often occur in kindergartens and schools during classroom activities where head-to-head contact is possible, during riding the school bus and during play [4], thus infesting other members of their families at home.

Pediculus humanus capitis (head lice) go through three stages in their life cycle namely egg, nymph and adult louse. The adult female louse lays egg usually called nits. The nits are hard to see and most a times are mistaken for hair spray droplets or dandruff, located at the base of the hair shaft and usually takes (8-9) days to hatch [5]. After exiting the first stage, the nymph an immature louse moults three (3) times before becoming adult in about (7-12) days sequel to hatching. It appears like an adult head louse however it is about the size of a pinhead [5], and feeds on human blood to survive. The last stage, is the adult stage, the adult is tan to grayish white in color and is about the size of a sesame seed with 6 legs. The life span of adult lice on human head is 30 days, and feeds extremely on human blood between 3 and 10 times daily as its meal. Once it is shaken off human head, it only last few hours [6] without a blood meal.

Pediculus humanus capitis (head lice) infestation affect majorly children within age bracket of 3 and 11 years old, independent of their socio-economic status, religion, tribe, country of origin, color of skin, gender and ethnic group [7]. Its symptoms such as scratching, a tickling feeling of moving object in the hair and itching usually occurs as soon as the human host develops an allergic reaction to the saliva of the head lice, often normally (4-6)weeks including the first day of infestation. Early detection of the head lice infestation on the host scalp and hair will help the host to respond to treatment such as wet combing, oral treatment and topical application of Pediculosis [7] positively. Though pediculus humanus capitis (head louse) is not known to cause life threatening disease, for some decades now, it has remain a constant source of disturbance both to public health officials and parents of the infested children. Globally, head lice prevalence takes the range 0-59% in different regions and a prevalence of more than 5% is considered to be epidemic [8]. Over a decade, several studies carried out in some cities in Nigeria such as Ilorin in 1988, Ibadan in 2006 (amongst primary school children), Ile-Ife in 1985 and Ibadan in 1984 (among rural and urban areas) reveals prevalence rate of 0.1-3.1%, 16.7%, 12.7%, respectively [9-12]. Thus our motivation to narrow our research work to primary schools in Northern Nigeria due to high prevalence rate recorded in schools

Whereas mathematical model of some ectoparasites such as sea lice [13], ticks [14] and fleas [15] and are well established, to the best our knowledge only few mathematical stochastic model have been proposed to study the dynamics of pediculosis (head lice) infestation with non in deterministic model. [16] Introduced an epidemic model based on stochastic susceptible – infectious – susceptible (SIS). The model take into consideration a fixed population size of N, divided into two compartments namely, susceptible compartment S(t) and infectious compartment I(t) at time (t) where $t \geq 0$ and $S(t) + I(t) = N$. Also, [17] proposed a model for endemic infectious based on a stochastic SIS (Susceptible-Infectious-Susceptible) epidemic model taking into consideration addition of external source of infection. They used "the stationary distribution of the number of infected individuals, in conjunction with data from a recent study carried out in Welsh schools on the prevalence of head lice infections, and employed maximum likelihood methods to obtain estimates of the model parameters."

To study the infestation of pediculosis (Head lice), we adopt and modify the deterministic (continuous time) model proposed by [18], where the authors considered permanent immunity after recovery. In their work, total population $N(t)$ was considered constant and individuals in the population were subdivided into three (3) classes, namely, Susceptible Class $S(t)$, Infected Class $I(t)$ and Recovered Class $R(t)$. Their model was uniquely developed to consider infectious diseases such as typhoid fever, small pox and mumps where permanent immunity is acquired upon recovery. Consequently, we have formulated a continuous time deterministic mathematical model to study the dynamics of pediculosis infestation by considering a fixed or constant population and to achieve this we assume $\vartheta N = \mu \; or \; \vartheta = \mu.$ In our model, we assume that recovered individuals immediately returns to the susceptible class for possible re-infestation, since there is no any form of immunity either temporary or permanent for pediculosis (head lice) infestation.

We organize the remaining section of this research work as follows: In section two (2), we described the basic model and its properties; in section three (3), we present the model analysis and in section four (4), we discussed the numerical simulation and results of the proposed mathematical model.

2. Mathematical Model Formulation

2.1 Compartmental Model Description

First and foremost, we divide total fixed or constant population size (at time t, where $t \geq 0$) denoted by $N(t)$ into three (3) classes: Susceptible Class, $S(t)$ [Individuals who maybe infested], Infested Class, $I(t)$ [Individuals who are infested and can transfer to others] and Recovered Class, $R(t)$ [Individuals who have recovered and have no any form of immunity]. This model assumes that the population $N(t)$ is consistently mixing. It is assumed that the rate of recruitment (immigrant) ϑN into the susceptible class $S(t)$ is constant, upon contact with infested individuals, susceptible individuals get the infection and move into the infected or infested class at a rate $\frac{\beta SI}{N}$ where the rate of infective contact is β. In addition, it is assumed that infested individuals recover and move into the Recovered Class $R(t)$ at a rate δ and also at the rate η for early detection and treatment. Since, our model assumes that there is no any

form of immunity, the recovered individuals immediately return to the Susceptible Class $S(t)$ at a rate ϕ. Lastly, all classes ($S(t), I(t)$ *and* $R(t)$ experience natural death or death due to other ill health at a rate μ.

2.2 Variable and Parameter Definition

The table below (Table 1 and Table 2) describe clearly the model variables and parameters used:

Table 1. Model Variable Used

Variable	Definition
S ⇒ Susceptible	The class of individuals susceptible at time t.
I ⇒ Infested	The class of individual infested at time t.
R ⇒ Recovered	The class of individuals who recovered at time t.
N ⇒ Population	The total population at time t.

Table 2. Model Parameter Used

Parameter	Definition
μ	Natural death rate
ϑ	Recruitment rate (immigration).
β	Pediculosis transmission rate
η	Rate at which infested individuals recover after awareness and treatment (control rate).
δ	Recovery rate
ϕ	Rate at which recovered individual return to the susceptible class.

2.3 Compartmental Flow Diagram

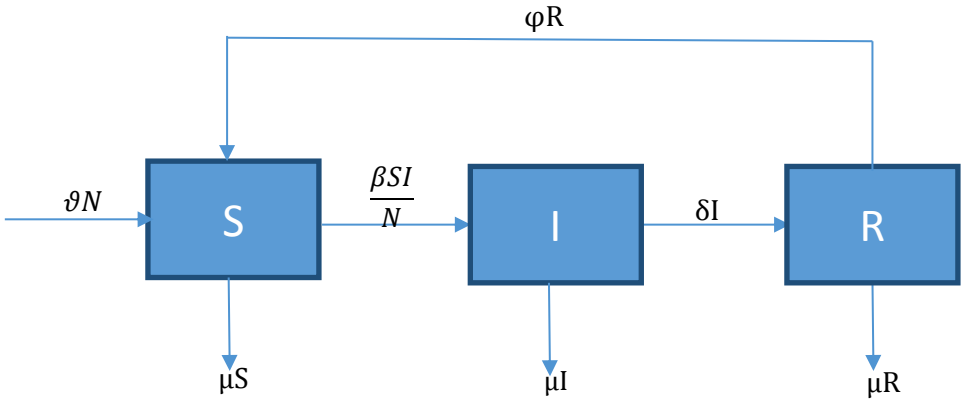

Fig. 1. Schematic Compartmental Flow Diagram of Pediculosis Capitis

2.4 Mathematical Model Equation

Taking to heart or reminiscing our model assumption in subsection (2.1) and the compartmental flow diagram in subsection (2.3) above, a deterministic (continuous time) model is formulated as follows:

$$S'(t) = \vartheta N - \frac{\beta SI}{N} + \varphi R - \mu S \tag{1}$$

$$I'(t) = \frac{\beta SI}{N} - \delta I - \eta I - \mu I \tag{2}$$

$$R'(t) = \delta I + \eta I - \varphi R - \mu R \tag{3}$$

Let (S, I, R) be any solution with positive initial conditions, that is $S(t) \geq 0, I(t) \geq 0$ *and* $R(t) \geq 0.$
Then we have that

$$S(t) + I(t) + R(t) = N(t).$$

Consequently, the time derivative of the total population $N(t)$, that is summation of equation (1-3) is given

$$S'(t) + I'(t) + R'(t) = (\vartheta N - \frac{\beta SI}{N} + \phi R - \mu S) + (\frac{\beta SI}{N} - \delta I - \eta I - \mu I) +$$
$$(\delta I + \eta I - \phi R - \mu R) \Rightarrow (S + I + R)'(t) = \vartheta N - (S + I + R)\mu$$

$$N'(t) = (\vartheta - \mu)N \tag{4}$$

2.5 Dimensionless Transformation

In order to simplify the model analysis, we carry out dimensionless transformation by adopting the approach used by [19, 20] of scaling the population of each class by the total population.
Thus,

$$s = \frac{s}{N}, i = \frac{I}{N}, \text{ and } r = \frac{R}{N}.$$

Using the state variables s, i *and* r, the normalized model system becomes:

$$s'(t) = \vartheta - \beta si + \varphi r - \mu s \tag{5}$$

$$i'(t) = \beta si - \delta i - \eta i - \mu i \tag{6}$$

$$r'(t) = \delta i + \eta i - \varphi r - \mu r \tag{7}$$

Adding equation (5-6) yields

$$s'(t) + i'(t) + r'(t) = \vartheta - \mu \tag{8}$$

where

$$s + i + r = 1$$

2.6 Positivity Solution

In this subsection, we show that all state variables in the SIR model are non-negative, since they represent human population.

Lemma 1. Let Ω be a set of non-negative region in $\Re^3; \Omega = \{(s, i, r) : s \geq 0, i \geq 0, r \geq 0\} \in \Re^3_+,$ then the solution set $\{s(t), i(t), r(t)\}$ is positive $\forall\ t \geq 0.$

Proof Considering the systems of equation in equations (5-7),
For equation (5),

$$s'(t) = \vartheta - \beta si + \varphi r - \mu s \geq -(\beta i + \mu)s$$
$$s'(t) \geq -(\beta i + \mu)s$$
$$\frac{s'(t)}{s} \geq -(\beta i + \mu)s \tag{9}$$

Solving equation (9) yields

$$s(t) \geq e^{-k[(\beta i + \mu)]t}$$
$$as\ t \to \infty, \quad s(t) \geq 0.$$

For equation (6),

$$\begin{gathered} i'(t) = \beta si - \delta i - \eta i - \mu i \geq -(\delta + \eta + \mu)i \\ i'(t) = -(\delta + \eta + \mu)i \\ \frac{i'(t)}{i} \geq -(\delta + \eta + \mu) \end{gathered} \tag{10}$$

Solving equation (10) yields

$$\begin{gathered} i(t) \geq e^{-k[(\delta+\eta+\mu)]t} \\ as\ t \to \infty, \quad i(t) \geq 0. \end{gathered}$$

Lastly, for equation (7),

$$\begin{gathered} r'(t) = \delta i + \eta i - \varphi r - \mu r \geq -(\varphi + \mu)r \\ r'(t) \geq -(\varphi + \mu)r \\ \frac{r'(t)}{r} \geq -(\varphi + \mu) \end{gathered} \tag{11}$$

Solving equation (11) yields

$$\begin{gathered} r(t) \geq e^{-k[(\varphi+\mu)]t} \\ as\ t \to \infty, \quad r(t) \geq 0 \end{gathered}$$

Thus, the proof that all state variables are positive $\forall\ t\ \geq 0$.

2.7 Feasible Region

Since the population in consideration is human population, all state variables in the SIR model remains positive all the time. Thus, the model system equations (5-7) in the region Ω is restricted to a non-negative condition.

$$\Omega = \{(s, i, r) : s > 0, i > 0, r > 0\} \in \Re_+^3,$$

where $s + i + r = 1.$

Our model equation (5-7) is biologically meaningful where the feasible region is positively invariant otherwise it is not.

3. Model Analysis

3.1 Equilibrium Analysis

Here we carried out stability analysis to determine the disease free equilibrium point (DFE) of the model. To determine the disease free equilibrium point (DFE), we equate each equation in equation (5-7) to zero, that is $s'(t) = 0, i'(t) = 0\ and\ r'(t) = 0,$ thus yielding

$$\vartheta - \beta si + \varphi r - \mu s = 0 \tag{12}$$

$$\beta si - \delta i - \eta i - \mu i = 0 \tag{13}$$

$$\delta i + \eta i - \varphi r - \mu r = 0 \tag{14}$$

From equation (12 - 14) above, we can obtain the equilibrium points $s, i\ and\ r$.

3.2 Disease Free Equilibrium (DFE)

The model's disease free equilibrium is obtained by equating $i = 0$ that is a condition where there is no spread of pediculosis.

From equation (14) above,

$$\delta i + \eta i - \varphi r - \mu \mathrm{r} = 0$$
$$-(\mu + \varphi) r = 0$$
$$r = 0$$

From equation (12) above,

$$\vartheta - \beta s i + \varphi r - \mu \mathrm{s} = 0$$
$$\vartheta - \mu \mathrm{s} = 0$$
$$s = \frac{\vartheta}{\mu}$$

Therefore, the disease free equilibrium points for pediculosis are:

$$\mathrm{D}^0 = (s^0, i^0, r^0) = (\frac{\phi}{\mu}, 0, 0)$$

3.3 Local Stability

The local stability of the disease free equilibrium is computed using Jacobian matrix of equation (5-7) at the disease free equilibrium point

$$\mathrm{D}^0 = (s^0, i^0, r^0) = (\frac{\phi}{\mu}, 0, 0) \cdot$$

We obtain the Jacobian matrix of equation (5-7) as follows

$$\xi(g_k, k = 1,2,3) = \begin{bmatrix} \frac{\partial g_1}{\partial s} & \frac{\partial g_1}{\partial i} & \frac{\partial g_1}{\partial r} \\ \frac{\partial g_2}{\partial s} & \frac{\partial g_2}{\partial i} & \frac{\partial g_2}{\partial r} \\ \frac{\partial g_3}{\partial s} & \frac{\partial g_3}{\partial i} & \frac{\partial g_3}{\partial r} \end{bmatrix} \tag{15}$$

then

$$\xi(g_k, k = 1,2,3) = \begin{bmatrix} -(\beta i + \mu) & -\beta s & \phi \\ \beta i & \beta s - \delta - \eta - \mu & 0 \\ 0 & \delta + \eta & -(\varphi + \mu) \end{bmatrix} \tag{16}$$

At the disease free equation

$$\mathrm{D}^0 = (s^0, i^0, r^0) = (\frac{\varphi}{\mu}, 0, 0)$$
$$\xi(\mathrm{D}^0) = \begin{bmatrix} -\mu & \frac{-\beta\varphi}{\mu} & \phi \\ 0 & \frac{\beta\varphi}{\mu} - \delta - \eta - \mu & 0 \\ 0 & \delta + \eta & -(\varphi + \mu) \end{bmatrix} \tag{17}$$

Now, we compute the eigenvalues of equation (17). Outcome of the eigenvalues will determine whether the disease free equation ($\mathcal{D}^0$) is locally asymptotic stable or not.

Let

$$\left|\xi(\mathrm{D}^0) - \rho I\right| = 0,$$

be a characteristics equation.

Then,

$$\left|\xi(\mathrm{D}^0)-\rho I\right|=\left|\begin{bmatrix} -\mu & \dfrac{-\beta\varphi}{\mu} & \phi \\ 0 & \dfrac{\beta\varphi}{\mu}-\delta-\eta-\mu & 0 \\ 0 & \delta+\eta & -(\varphi+\mu) \end{bmatrix}-\begin{pmatrix} \rho & 0 & 0 \\ 0 & \rho & 0 \\ 0 & 0 & \rho \end{pmatrix}\right|=0 \tag{18}$$

$$\begin{vmatrix} -\mu-\rho & \dfrac{-\beta\varphi}{\mu} & \phi \\ 0 & \dfrac{\beta\varphi}{\mu}-\delta-\eta-\mu-\rho & 0 \\ 0 & \delta+\eta & -(\varphi+\mu)-\rho \end{vmatrix}=0 \tag{19}$$

Evaluating equation (19) abrove yields

$$-(\mu+\rho)\left[\left(\frac{\beta\varphi}{\mu}+\delta+\eta+\mu+\rho\right)(\varphi+\mu+\rho)\right]=0$$

$$-(\mu+\rho)=0,\ \frac{\beta\varphi}{\mu}+\delta+\eta+\mu+\rho=0,\quad \varphi+\mu+\rho=0$$

$$\rho_1=-\mu,\quad \rho_2=-(\delta+\eta+\mu+\frac{\beta\varphi}{\mu}),\quad \rho_3=-(\varphi+\mu).$$

Since all the eigenvalues of $\mathcal{J}(\mathcal{D}^0)$ <1 that is $\rho_1=\rho_2=\rho_3<1$, therefore the disease free equilibrium is locally asymptotically stable (LAS).

3.4 Basic Reproduction Number (R_0)

In this subsection, we obtain or compute the basic reproduction number (R_0) which is defined as the number of secondary infection produced by a single infective individual when introduced into a completely susceptible population. In order to obtain the basic reproduction number (R_0), we adopt the same approach used in [20] where the authors used two matrices F *and* V, with F representing the new infection terms and V representing the remaining terms of transfer.

Our model has only three (3) compartments with only one (1) infective compartment.

From equation (6),

$$i'(t)=\beta si-\delta-\eta i-\mu i$$

We have that

$$F=[\beta si],$$

and

$$V=[(\delta+\eta+\mu)i].$$

We obtain the matrices F *and* V by taking the Jacobian of $\mathcal{F}$ *and* $\mathcal{V}$ respectively at the disease free equilibrium, $\mathcal{D}^0$.

$$F=DF(x)=\frac{\partial F_j(x)}{\partial x_k}$$

$$F=\left[\frac{\partial F_1}{\partial i}\right]=\left[\frac{\partial(\beta si)}{\partial i}\right]$$

$$F=[\beta s].$$

At disease free equilibrium, the matrix F becomes

$$F = \left[\frac{\beta\phi}{\mu} \right].$$

Also,

$$V = DV(x) = \frac{\partial V_j(x)}{\partial x_k}$$
$$V = \left[\frac{\partial V_1}{\partial i} \right] = \left[\frac{\partial(\delta + \eta + \mu)i}{\partial i} \right]$$
$$V = (\delta + \eta + \mu).$$

At disease free equilibrium, the matrix V becomes

$$V = (\delta + \eta + \mu)$$

The inverse of matrix V becomes

$$V^{-1} = \frac{1}{(\delta + \eta + \mu)}.$$

Let

$$M = FV^{-1}.$$

Then

$$M = \left[\frac{\beta\varphi}{\mu} \right] \left[\frac{1}{(\delta + \eta + \mu)} \right],$$
$$M = \frac{\beta\varphi}{\mu(\delta + \eta + \mu)} \tag{20}$$

Using the characteristics equation $\left| FV^{-1} - \rho I \right| = 0$ we compute the eigenvalues of equation (20) as follows

$$\left| FV^{-1} - \rho I \right| = 0 \Rightarrow \left| M - \rho I \right| = 0,$$

Where I is an identity matrix (in this case, it is 1).

$$\left| M - \rho I \right| = 0,$$
$$\left| \frac{\beta\varphi}{\mu(\delta + \eta + \mu)} - \rho \right| = 0,$$
$$\frac{\beta\varphi}{\mu(\delta + \eta + \mu)} - \rho = 0,$$
$$\rho = \frac{\beta\varphi}{\mu(\delta + \eta + \mu)}.$$

Therefore, the basic reproduction number (R_0) is given as

$$R_0 = \frac{\beta\phi}{\mu(\delta + \eta + \mu)} < 1.$$

4. Simulation and Discussion of Result

4.1 Simulation

To simulate the formulated mathematical model in equation (5-9), we adopted MATLAB programming language using parameter values presented in Table C below. Due to unavailability of some of these parameter values, we obtain them from different sources, estimated some and assumed some of the parameter values.

Table 3. Model Parameter Values Used and Their Sources

Model Parameter	Model Parameter Value	Sources of Model Parameter Value
μ	0.0500	Assumed
ϑ	0.0500	Assumed
β	0.0100	[14]
η	[0.0000,0.2000,0.4000,0.6000,0.8000]	Assumed
δ	0.2000	Estimated
ϕ	0.2000	Estimated

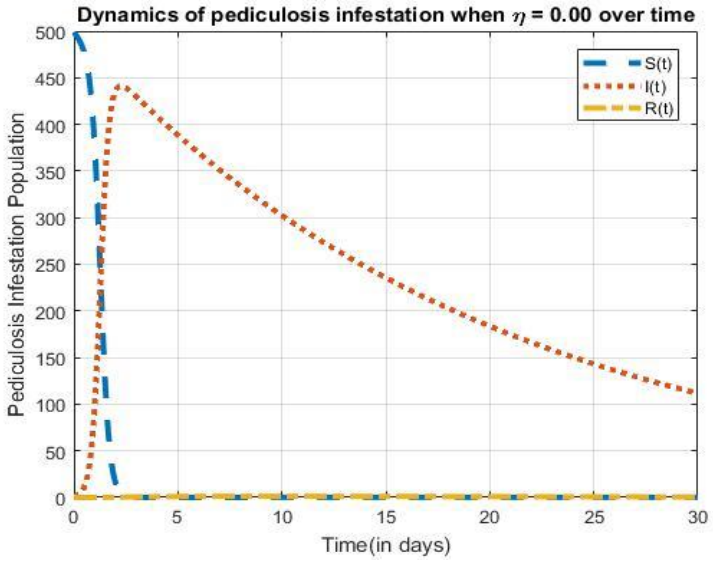

Fig. 2. Dynamics of pediculosis infestation when $\eta = 0.00$.

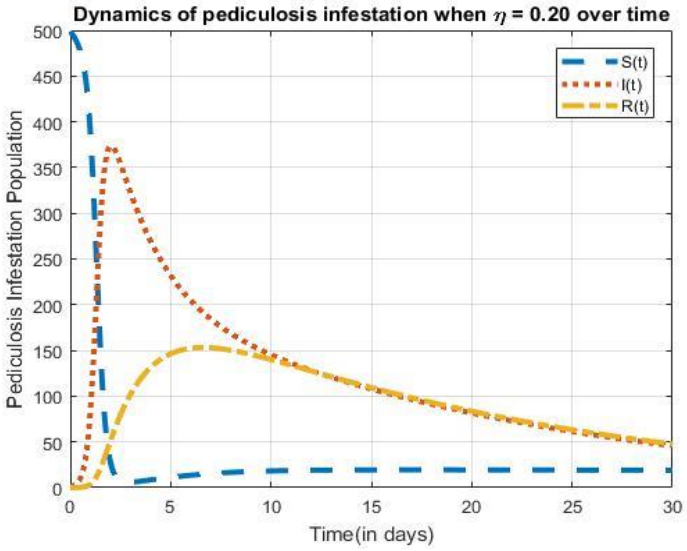

Fig. 3. Dynamics of pediculosis infestation when $\eta = 0.20$

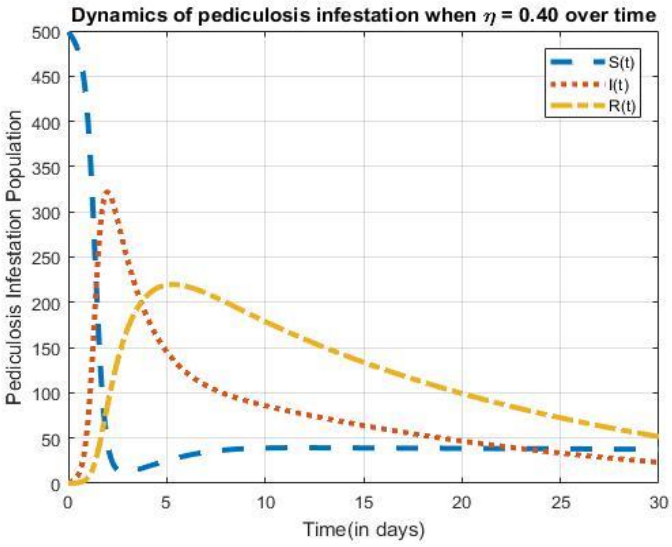

Fig. 4. Dynamics of pediculosis infestation when η = 0.40

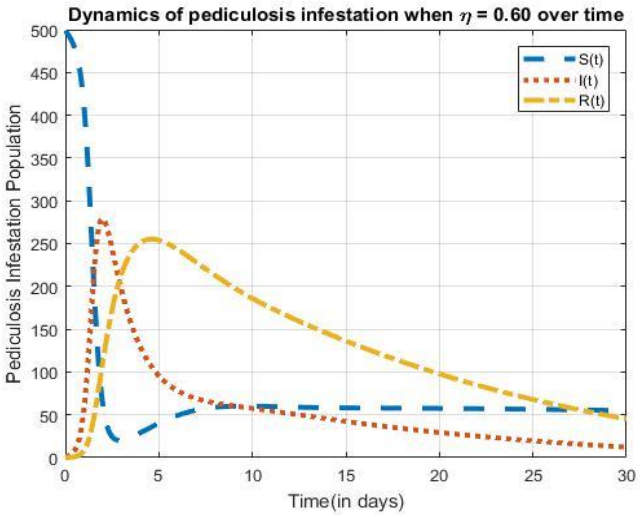

Fig. 5. Dynamics of pediculosis infestation when η = 0.60.

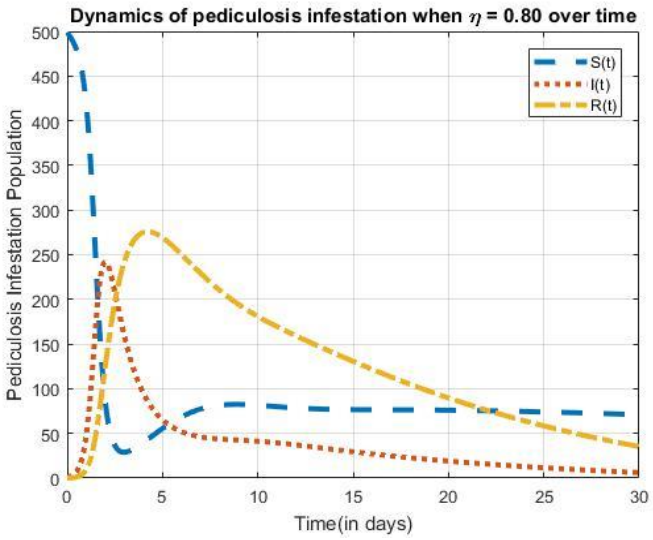

Fig. 6. Dynamics of pediculosis infestation when η = 0.80.

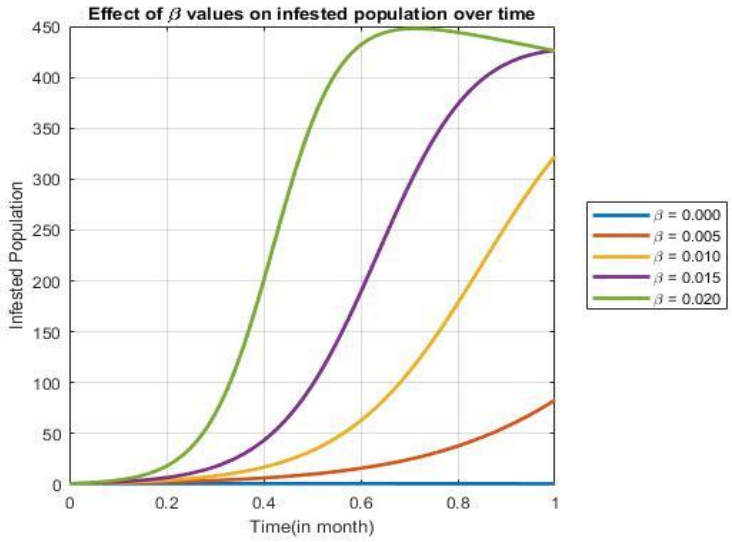

Fig. 7. Effect of different β values on infested population over time

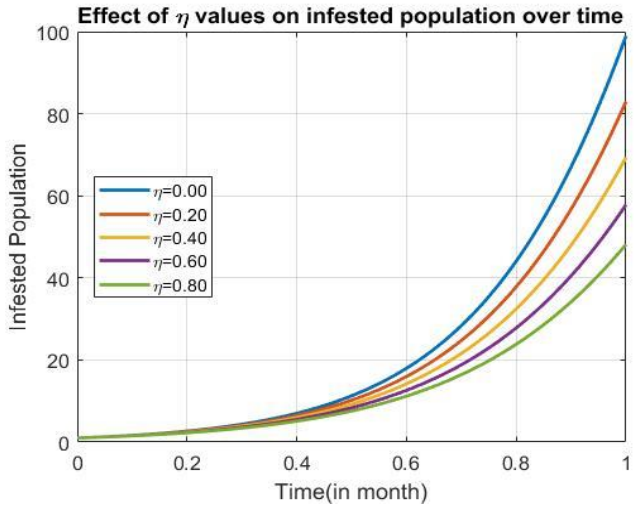

Fig. 8. Effect of different η values on infested population over time

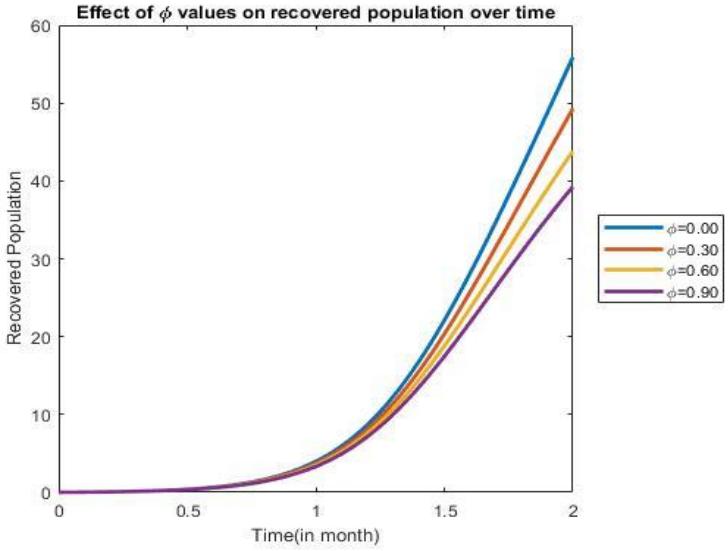

Fig. 9. Effect of different ϕ values on recovered population over time.

4.2 Discussion of Results

In order to have clear insight of the dynamics of pediculosis capitis, we displayed nine (9) figures for parameter analysis.

Figure (2-6) above shows the dynamics of pediculosis infestation for different values of η. We observed that as the value of η increases, the population of the infested individuals reduces thus leading to the increase in the population of the recovered individual over time. We also observed that as the η values increase, it takes shorter time for the infested population to reduce. Biologically, it implies that pediculosis capitis is curable over time within the population provided there is public enlightenment and treatment. The more the public are sensitized and treated, the more diminishing the disease becomes in the population.

Also, in figure 7, a simulation to visualize the effect of β on infested population was carried out by varying the values of β. We held other parameter values constant as in Table 3 above and use different values of β that is $\beta = [0.000, 0.005, 0.010, 0.015, 0.020]$. We observed that increase in β values leads to increase in the infested population which totally agrees with our proposed model and gives us a linear relationship between β and the infested population.

Furthermore, in figure 8 and 9, the outcome of the simulation for different values of η that is $\eta = [0.00, 0.20, 0.40, 0.60, 0.80]$, holding other parameter values constant as in Table 3, shows that η has an important effects on both the infested population and the recovered population over time. We observed from Figure 8 that increase in η value leads to decrease in the infested population thereby giving us an inverse relationship between η and infested population. Also, on the contrary, we observed from Figure 9 that increase in η values leads to increase in the recovered population thus giving us a linear relationship between η and the recovered population. From these relationships, we deduce that η play a significant role in controlling the dynamics of pediculosis.

Lastly, in figure 10, we observed that increase in ϕ values that is $\phi = [0.00, 0.30, 0.60, 0.90]$ leads to decrease in recovered population, thus giving us an inverse relationship between ϕ and the recovered population. This is in agreement with our model as the recovered individual's returns to the susceptible class for possible infestation due to absence of any form of immunity either temporary or permanent.

5. Conclusion

A Mathematical model for transmission dynamics of pediculosis infestation is formulated and analyzed. The model has only one equilibrium namely disease free equilibrium point (DFE). Since the model's basic reproduction number (R_0) is less than one (1), that is $R_0 < 1$, the disease free equilibrium point (DEF) is locally asymptotically stable (LAS). Also, numerical simulation is carried out and it was observed that the parameter η plays a significant role in controlling the dynamics of pediculosis infestation. This work, however did not address the asymptomatic population. This work can be extended to incorporate the asymptomatic compartment to further investigate the dynamics of pediculosis capitis in a population.

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