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FRACTIONAL MATHEMATICAL MODEL ON TRANSMISSION AND CONTROL OF THE SPREAD OF MYIASIS IN HUMAN SYSTEM USING INSECT REPELLENT

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Abstract. We proposed a fractional SEITR order model to study the transmission dynamics of Myiasis. We showed the existence of the equilibrium states. The basic reproduction number of the model was evaluated in terms of the parameters in the model using the next generation matrix approach. We provided the conditions for the stability of the disease-free and the endemic equilibrium points. Also, numerical simulations of the model were carried out using Adams-type predictor-corrector method which showed in detail the population dynamics of the disease. From these simulations, the level of impacts of the parameters of the model were demonstrated

1. Introduction

Myiasis is the infestation of live human and vertebrate animals with dipterous larvae, which, at least for a certain period, feed on the host's dead or living tissue, liquid body-substances or ingested food,[9]. It could also be expressed as a parasitic infestation of tissues and organs in living vertebrates with dipterous larvae,[1]. It is widespread in the tropics and subtropics of Africa and the America, and can occur in other parts of the world although less frequent,[2]. It is a common infestation among mammals (human and other vertebrate animals). In humans, it is seen more in rural areas where people are in more direct contact with animals,[3]. The disease occurs when the female fly lays eggs, which shortly will cause clinical manifestations that are related to the body site involved,[4]. The infestation is most often subcutaneous and produces a furunculous or boil-like lesion, but it is also known to occur in wounds and certain body cavities,[5]. Transmission of this fly larvae to human hosts differs among the many species of fly. For instance, one may have gotten an infection from accidentally ingesting larvae, from having an open wound or sore, or through your nose or ears. In tropical areas, where the infection is most likely to occur, some flies lay their eggs on drying clothes that are hung outside. Some flies attach their eggs to mosquitoes, ticks or other flies that harbor larvae and wait for mosquitoes to bite people. Their larvae then enter these

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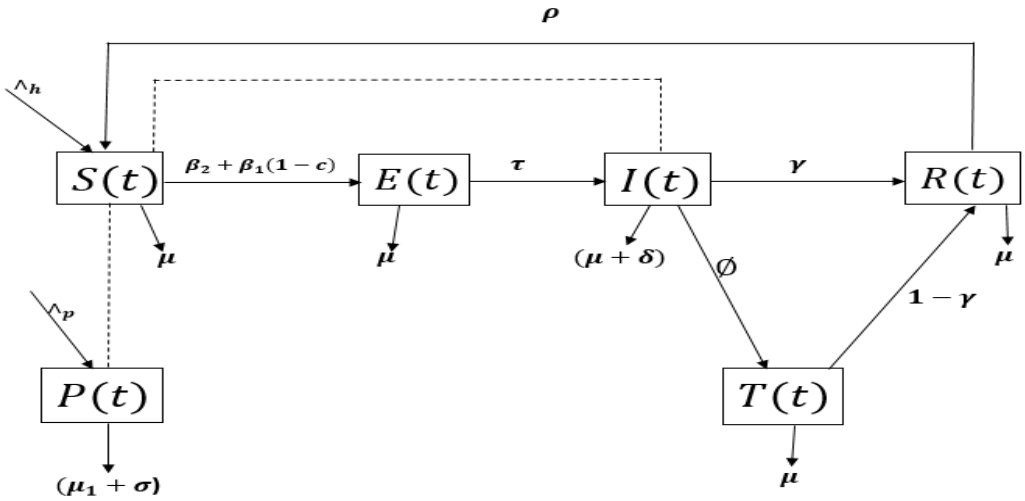
FIGURE 1. Model Flow Diagram.



bites. They can enter skin through people's bare feet when they walk through soil containing fly eggs. Some flies deposit their larvae on or near a wound or sore, depositing eggs in sloughing-off dead tissue. Most of the fly larvae are transmitted to humans through pet or domestic animals that are infested by larvae.

2. Model Formulation

We assume that the disease cannot kill once an individual is treated; individuals become exposed once they interact with the pathogen. One can recovered in the treatment and may die if not treated due to the disease and the study is carried out in the tropical region. Let S(t), E(t), I(t), T(t), R(t) and P(t) denote the number of susceptible, exposed, infected, recovered and pathogen class. The schematic diagram of the disease on which we base our model is as follow:

2.1. **Model Equation.**

$$\begin{cases} D_t^\alpha S(t) = \Lambda_h + \rho R - \frac{\beta_1(1-c)SP - \beta_2 SI}{N} - \mu S \\ D_t^\alpha E(t) = \frac{\beta_1(1-c)SP + \beta_2 SI}{N} - E(\mu + \tau) \\ D_t^\alpha I(t) = \tau E - I(\gamma + \phi + \mu + \delta) \\ D_t^\alpha T(t) = \phi I - T[\mu + (1-\gamma)] \\ D_t^\alpha R(t) = \gamma I + (1-\gamma)T - R(\mu + \rho) \\ D_t^\alpha P(t) = \Lambda_p - P(\mu_1 + \sigma) \end{cases} \tag{1}$$

2.2. **Invariant Region.**

Lemma 3. The closed set $\Omega = \{(S, E, I, T, R) \in R_+^5 : S + E + I + T + R = N_h\}$ is positively invariant with respect to model (1).

TABLE 1. Parameters of the model and their Description

Parameters	Description
Λ_h	Recruitment rate of human
β_1	The rate at which the susceptible individuals interact with the pathogen
β_2	The rate at which the susceptible individuals interact with the infected individuals
μ	The natural death rate of human
μ_1	The natural death rate of pathogen
δ	The disease induced death rate
τ	The rate at which the exposed individuals progressed to be infected
γ	The rate at which the infected individuals recover from the disease without treatment
ρ	The rate at which recovered individuals becomes susceptible
ϕ	The rate at which the infected individuals are for treatment
σ	The rate at the pathogens die as a result of the predator feeding on them
Λ_p	Recruitment rate of the pathogens
c	Rate of adherent to the use of insect repellent

Proof

The fractional derivative of the total human population, obtained by adding all the human equations of model (1), is given by

$$D_t^{\alpha} N_h = \Lambda_h - \mu N_h \tag{2}$$

The Laplace transform of (2) gives:

$$S^{\alpha} N(s) - S^{\alpha-1} N(0) = \frac{\Lambda}{s} - \mu N(s) \tag{3}$$

$$\Rightarrow N(s) = \frac{\Lambda}{S(S^{\alpha} + \mu)} + \frac{S^{\alpha-1}}{s^{\alpha} + \mu} N(0) \tag{4}$$

Taking the inverse Laplace transform of (4), we have:

$$N(t) = E_{\alpha,1}(-\mu t^{\alpha}) + \Lambda t^{\alpha} E_{\alpha,\alpha+1}(-\mu t^{\alpha}) \tag{5}$$

where $E_{\alpha,\beta}$ is the Mittag-Leffler function. But the fact that the Mittag-Leffler functions has an asymptotic behavior, it follows that:

$$E_{\alpha,1} N(t) = \sum_{k=0}^{\infty} \frac{N^K(t)}{\Gamma(\alpha k + 1)}, \alpha > 0 \tag{6}$$

$$E_{\alpha,\alpha+1} N(t) = \sum_{k=0}^{\infty} \frac{N^K(t)}{\Gamma(\alpha k + \alpha + 1)}, \alpha > 0 \tag{7}$$

Expanding (6), we have

$$E_{\alpha,1} N(t) = \frac{1}{\Gamma(1} + \frac{N(t)}{\Gamma(\alpha + 1)} + \frac{N^2(t)}{\Gamma(2\alpha + 1)} + ... \tag{8a}$$

Expanding (7), we have

$$E_{\alpha,\alpha+1} N(t) = \frac{1}{\Gamma(\alpha + 1)} + \frac{N(t)}{\Gamma(2\alpha + 1)} + \frac{N^2(t)}{\Gamma(3\alpha + 1)} + ... \tag{8b}$$

Since Mittag-Leffler function has an asymptotic property, we have

$$N_h(t) = 1 + O(N) \tag{9}$$

Taking limit as k$\longrightarrow \infty$, we have

$$N_h(t) \approx 1 \tag{10}$$

Then, it is clear that Ω is a positive invariant set.Therefore, all solutions of the model with initial conditions in Ω remain in Ω for all t > 0. Then , $\Omega = N_h(t) > 0$ implies that it is feasible with respect to model (1).

4. Model Analysis

4.1. **The Basic Reproduction Number, R_0.** The Disease free equilibrium point is evaluated as:

$$(S^0, E^0, I^0, T^0, R^0, P^0) = (\frac{\Lambda_h}{\mu}, 0, 0, 0, 0, 0) \tag{11}$$

We use Next Generation Matrix which comprises of two parts: F and V^1,

$$R_0 = \rho(FV^{-1}) \tag{12}$$

Where

$$F = \left| \frac{\partial f_i x_{(0)}}{\partial x_j} \right| \ , \qquad V = \left| \frac{\partial v_i x_{(0)}}{\partial x_j} \right|$$

$\rho =$ spectral value (highest eigenvalue)
On the estimation,We used the following disease compartments:

$$\begin{cases} D_t^\alpha E(t) = \frac{\beta_1(1-c)SP+\beta_2 SI}{N} - E(\mu+\tau) \\ D_t^\alpha I(t) = \tau E - I(\gamma+\phi+\mu+\delta) \\ D_t^\alpha T(t) = \phi I - T[\mu+(1-\gamma)] \\ D_t^\alpha P(t) = \Lambda_p - P(\mu_1+\sigma) \end{cases} \tag{13}$$

Define

$$f_i = \begin{pmatrix} \frac{\beta_1(1-c)SP+\beta_2 SI}{N} \\ 0 \\ 0 \\ 0 \end{pmatrix} \tag{14}$$

$$v_i = \begin{pmatrix} \mu\mathrm{E}+\tau\mathrm{E} \\ \mathrm{I}(\gamma+\phi+\mu+\delta) - \tau\mathrm{E} \\ \mathrm{T}[\mu+(1-\gamma)] - \phi\mathrm{I} \\ \mathrm{P}(\mu_1+\sigma) - \Lambda_p \end{pmatrix} \tag{15}$$

Differentiating (14) with respect to the outlined variables in (13), we have:

$$F = \begin{pmatrix} 0 & \frac{\beta_2 S}{N} & 0 & \frac{\beta_1(1-c)S}{N} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \tag{16}$$

and, performing the usual derivative operation on (15), we have

$$V = \begin{pmatrix} \mu+\tau & 0 & 0 & 0 \\ -\tau & \gamma+\phi+\mu+\delta & 0 & 0 \\ 0 & -\phi & \mu+1-\gamma & 0 \\ 0 & 0 & 0 & \mu_1+\sigma \end{pmatrix} \tag{17}$$

$$V^{-1} = \begin{pmatrix} (\mu+\tau)^{-1} & 0 & 0 & 0 \\ \frac{\tau}{(\mu+\tau)(\gamma+\phi+\mu+\delta)} & (\gamma+\phi+\mu+\delta)^{-1} & 0 & 0 \\ -\frac{\phi\,\tau}{(\mu+\tau)(\gamma+\phi+\mu+\delta)(-\mu-1+\gamma)} & -\frac{\phi}{(\gamma+\phi+\mu+\delta)(-\mu-1+\gamma)} & -(-\mu-1+\gamma)^{-1} & 0 \\ 0 & 0 & 0 & (\mu_1+\sigma)^{-1} \end{pmatrix} \tag{18}$$

$$FV^{-1} = \begin{pmatrix} \frac{\beta_2 S\tau}{N(\mu+\tau)(\gamma+\phi+\mu+\delta)} & \frac{\beta_2 S}{N(\gamma+\phi+\mu+\delta)} & 0 & \frac{\beta_1(1-c)S}{N(\mu_1+\sigma)} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \tag{19}$$

Therefore, the eigenvalue of equation (19) is

$$Eigenvalue(FV^{-1}) = \begin{pmatrix} 0 \\ 0 \\ 0 \\ \frac{\beta_2 S\tau}{N(\delta\,\mu+\delta\,\tau+\gamma\,\mu+\gamma\,\tau+\mu^2+\mu\,\phi+\mu\,\tau+\phi\,\tau)} \end{pmatrix} \tag{20}$$

we have the dominant eigenvalue as follows:

$$R_0 = \frac{\tau\beta_2 S}{N(\delta\mu+\delta\tau+\gamma\mu+\gamma\tau+\mu^2+\mu\phi+\mu\tau+\phi\tau)} \tag{21}$$

At Disease Free Equilibrium,

$$R_0 = \frac{\tau\beta_2}{(\mu+\tau)(\delta+\gamma+\phi+\mu)} \tag{22}$$

4.2. **Sensitive Indices of the Parameters of Myiasis Infection.** . Recall the obtained reproduction number with respect to model(1) evaluated as equation (22) above.

The sensitivity index of $'\beta'$ with respect to R_0 , is given as:

$$W_{\beta}^{R_0} = \frac{\partial R_0}{\partial \beta} \times \frac{\beta}{R_0} = 1 \tag{23a}$$

The sensitivity index of $'\Lambda_h'$ with respect to R_0 , is given as:

$$W_{\Lambda_h}^{R_0} = \frac{\partial R_0}{\partial \Lambda_h} \times \frac{\Lambda_h}{R_0} = 1 \tag{23b}$$

The sensitivity index of $'\mu'$ with respect to R_0 , is given as:

$$W_{\mu}^{R_0} = \frac{\partial R_0}{\partial \mu} \times \frac{\mu}{R_0} < 0 \tag{23c}$$

The sensitivity index of $'\mu_1'$ with respect to R_0 , is given as:

$$W_{\mu_1}^{R_0} = \frac{\partial R_0}{\partial \mu_1} \times \frac{\mu_1}{R_0} < 0 \tag{23d}$$

The sensitivity index of $'\sigma'$ with respect to R_0 , is given as:

$$W_{\sigma}^{R_0} = \frac{\partial R_0}{\partial \sigma} \times \frac{\sigma}{R_0} < 0 \tag{23e}$$

The sensitivity index of $'\tau'$ with respect to R_0 , is given as:

$$W_{\tau}^{R_0} = \frac{\partial R_0}{\partial \tau} \times \frac{\tau}{R_0} = 0 \tag{23f}$$

Where $'W'$ = Sensitive Index
Every other parameter that are not reflected in the reproduction number of the model (1) has its sensitivity index as zero. The result show that some of the parameters have positive signs and others have negative signs while some parameters are zero. This implies that some of the parameters have positive effect on the basic reproduction number (R_0); some have negative effect on it while some have zero effect.

4.3. **Stability Analysis.** Here, we studied the stability at *Disease free equilibrium*(DFE) and *Endemic state equilibrium point*(EEP).

4.3.1. *Local Stability at DFE.* We evaluated the Disease free equilibrium points as:

$$(S^0, E^0, I^0, T^0, R^0, P^0) = \left(\frac{\Lambda_h}{\mu}, 0, 0, 0, 0, 0\right) \tag{24}$$

The Jacobian matrix at Disease free equilibrium is given as:

$$J^0 = \begin{pmatrix} -\mu & 0 & \frac{-\beta_2 S}{N} & 0 & \rho & \frac{-\beta_1(1-c)S}{N} \\ 0 & -(\mu+\tau) & \frac{\beta_2 S}{N} & 0 & 0 & \frac{\beta_1(1-c)S}{N} \\ 0 & \tau & -(\gamma+\phi+\mu+\delta) & 0 & 0 & 0 \\ 0 & 0 & \phi & -[\mu+(1-\gamma)] & 0 & 0 \\ 0 & 0 & \gamma & 1-\gamma & -(\mu+\rho) & 0 \\ 0 & 0 & 0 & 0 & 0 & -(\mu_1+\sigma) \end{pmatrix} \tag{25}$$

By characteristic equation

$$|J^0 - I\lambda| = 0 \tag{26}$$

matrix (25) become:

$$\begin{bmatrix} -\mathrm{a}-\lambda & 0 & \frac{-\beta_2 S}{N} & 0 & \rho & \frac{-\beta_1(1-c)S}{N} \\ 0 & -\mathrm{b}-\lambda & \frac{\beta_2 S}{N} & 0 & 0 & \frac{\beta_1(1-c)S}{N} \\ 0 & \tau & -\mathrm{c}-\lambda & 0 & 0 & 0 \\ 0 & 0 & \phi & -\mathrm{d}-\lambda & 0 & 0 \\ 0 & 0 & \gamma & 1-\gamma & -\mathrm{h}-\lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & -\mathrm{f}-\lambda \end{bmatrix} = 0 \tag{27}$$

Thus, we have

$$(a-\lambda)(-b-\lambda)(-c-\lambda)(-d-\lambda)(-h-\lambda)(-f-\lambda) = 0 \tag{28}$$

Where
$a = \mu$, $b = \mu + \tau$, $c = (\gamma + \phi + \mu + \delta)$, $d = [\mu + (1-\gamma)]$, $h = \mu + \rho$, $f = \mu_1 + \sigma$

Then from (28), the respective eigenvalues are:

$$\lambda_1 = -a = -\mu \tag{29a}$$
$$\lambda_2 = -b = -(\mu + \tau) \tag{29b}$$
$$\lambda_3 = -c = -(\gamma + \phi + \mu + \delta) \tag{29c}$$
$$\lambda_4 = -d = -[\mu + (1 - \gamma)] \tag{29d}$$
$$\lambda_5 = -h = -(\mu + \rho) \tag{29e}$$
$$\lambda_6 = -f = -(\mu_1 + \sigma) \tag{29f}$$

Since all the above eigenvalue are negative, the equilibrium point (24) is **locally asymptotically stable**.

□

4.3.2. *Global Stability at DFE.* Here, we study the global stability with construction of a Lyapunov function of the system(1).

Theorem 4.1. The disease-free equilibrium $\mathcal{E}_0$, given by (24), of the model (1) is globally asymptotically stable if $R_0 < 1$.

Proof

Observing the conditions in [10], from system(1) we select the infected classes $(E(t), I(t))$ of the human population to construct a Lyapunov function $G(t)$ such that

$$G(t) = x_1 E(t) + x_2 I(t) \tag{30}$$

It is good to note that for $G(t)$ to be a Justifiable Lyapunov function, the coefficients of (30) will be chosen such that $x_1 > 0$ and $x_2 > 0$ (non-negotiable). The derivative of equation (30) becomes

$$\dot{G}(t) = x_1 \dot{E}(t) + x_2 \dot{I}(t) \tag{31}$$
$$= x_1[aS - E(\mu + \tau)] + x_2[\tau E - I(\delta + \gamma + \phi + \mu)] \tag{32}$$

we recall:

$$a = \frac{\beta_1(1 - c)P + \beta_2 I}{N}, \quad S(t) = S, I(t) = I, P(t) = P, N(t) = N$$

rearranging equation(32), thus gives

$$\dot{G}(t) = x_1 aS - E[x_1(\mu + \tau) - x_2\tau] - I[x_2(\delta + \gamma + \phi + \mu)] \tag{33}$$

The following steps will be observed in forming the Lyapunov function:

- set the coefficient of aS to the numerator of R_0 (excluding β_2).
- set the coefficient of I to the denominator of R_0
- set the coefficient of E to zero.

Then, we have:

$$\begin{cases} x_1 = \tau, \\ x_2(\delta + \gamma + \phi + \mu) = (\mu + \tau)(\delta + \gamma + \phi + \mu) \\ x_1(\mu + \tau) - x_2\tau = 0 \end{cases} \tag{34}$$

Solving, we have

$$\begin{cases} x_1 = \tau, \\ x_2 = \mu + \tau \end{cases} \tag{35}$$

substituting for x_1 and x_2 in (33), we have

$$\dot{G}(t) = x_1 aS - I(\mu+\tau)(\delta+\gamma+\phi+\mu) \quad (36)$$

$$= \tau\left[\frac{\beta_1(1-c)P+\beta_2 I}{N}\right]S - I(\mu+\tau)(\delta+\gamma+\phi+\mu) \quad (37)$$

But at DFE $S = N$ and $P = 0$, therefore

$$\dot{G}(t) = \tau\beta_2 I - I(\mu+\tau)(\delta+\gamma+\phi+\mu) \quad (38)$$

$$= I\left[\frac{\tau\beta_2}{(\mu+\tau)(\delta+\gamma+\phi+\mu)} - 1\right](\mu+\tau)(\delta+\gamma+\phi+\mu) \quad (39)$$

$$= I[R_o - 1](\mu+\tau)(\delta+\gamma+\phi+\mu) \quad (40)$$

Therefore, $\dot{G}(t) \leq 0$ for $R_0 < 1$. By Lyapunov-LaSalle's invariant principle [12], (24) is globally asymptotically stable.

□

4.3.3. *Local Stability at EEP.* Again, we study the stability of the Endemic equilibrium points.

$$E^* = (S^*, E^*, I^*, T^*, R^*, P^*) = (I^*K_5, I^*K_2, I^*K_3, I^*K_4, I^*K_1, K_1) \quad (41)$$

Where
$K_1 = \frac{\Lambda_p}{\mu_1+\sigma}$, $K_2 = \frac{(\gamma+\phi+\mu+\delta)}{\tau}$, $K_3 = \frac{\phi}{\mu+(1-\gamma)}$, $K_4 = \frac{1}{\mu+\rho}(\gamma + \phi\frac{(1-\gamma)}{\mu+(1-\gamma)})$, $K_5 = \frac{(\gamma+\phi+\mu+\delta)(\mu+\tau)(\mu_1+\sigma)}{\tau\beta\Lambda_p(1-c))}$.
The Jacobian matrix of Equation (1) is given as:

$$J^E = \begin{pmatrix} \frac{-\beta_1 P(1-c)-\beta_2 I}{N} - \mu & 0 & \frac{-\beta_2 S}{N} & 0 & \rho & \frac{-\beta_1(1-c)S}{N} \\ \frac{\beta_1 P(1-c)+\beta_2 I}{N} & -(\mu+\tau) & \frac{\beta_2 S}{N} & 0 & 0 & \frac{\beta_1(1-c)S}{N} \\ 0 & \tau & -(\gamma+\phi+\mu+\delta) & 0 & 0 & 0 \\ 0 & 0 & \phi & -[\mu+(1-\gamma)] & 0 & 0 \\ 0 & 0 & \gamma & 1-\gamma & -(\mu+\rho) & 0 \\ 0 & 0 & 0 & 0 & 0 & -(\mu_1+\sigma) \end{pmatrix} \quad (42)$$

Then we compute $|J^E - \lambda I|$, where I is an (6×6) identity matrix.

$$|J^E - \lambda I| = \begin{vmatrix} -\left(\frac{\beta_1 P(1-c)+\beta_2 I}{N} + \mu\right) - \lambda & 0 & \frac{-\beta_2 S}{N} & 0 & \rho & \frac{-\beta_1(1-c)S}{N} \\ \frac{\beta_1 P(1-c)+\beta_2 I}{N} & -(\mu+\tau) - \lambda & \frac{\beta_2 S}{N} & 0 & 0 & \frac{\beta_1(1-c)S}{N} \\ 0 & \tau & -(\gamma+\phi+\mu+\delta) - \lambda & 0 & 0 & 0 \\ 0 & 0 & \phi & -[\mu+(1-\gamma)] - \lambda & 0 & 0 \\ 0 & 0 & \gamma & 1-\gamma & -(\mu+\rho) - \lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & -(\mu_1+\sigma) - \lambda \end{vmatrix} \quad (43)$$

By characteristic equation, we have:

$$-[(\mu_1+\sigma)+\lambda]\begin{vmatrix} -\left(\frac{\beta_1 P(1-c)+\beta_2 I}{N} + \mu\right) - \lambda & 0 & \frac{-\beta_2 S}{N} & 0 & \rho \\ \frac{\beta_1 P(1-c)+\beta_2 I}{N} & -(\mu+\tau) - \lambda & \frac{\beta_2 S}{N} & 0 & 0 \\ 0 & \tau & -(\gamma+\phi+\mu+\delta) - \lambda & 0 & 0 \\ 0 & 0 & \phi & -[\mu+(1-\gamma)] - \lambda & 0 \\ 0 & 0 & \gamma & 1-\gamma & -(\mu+\rho) - \lambda \end{vmatrix} = 0 \quad (44)$$

Assuming $\rho \approx 0$, (44) now becomes

$$[-(\mu_1+\sigma)-\lambda]\cdot[-(\mu+\rho)-\lambda]\cdot[-(\mu+1-\gamma)-\lambda]\cdot\begin{vmatrix} -\left(\frac{\beta_1 P(1-c)+\beta_2 I}{N} + \mu\right) - \lambda & 0 & \frac{-\beta_2 S}{N} \\ \frac{\beta_1 P(1-c)+\beta_2 I}{N} & -(\mu+\tau) - \lambda & \frac{\beta_2 S}{N} \\ 0 & \tau & -(\gamma+\phi+\mu+\delta) - \lambda \end{vmatrix} = 0 \quad (45)$$

Considering the remaining (3×3) matrix, we have

$$\begin{vmatrix} -(a+\mu)-\lambda & 0 & -b \\ a & -d-\lambda & b \\ 0 & \tau & -e-\lambda \end{vmatrix} = 0 \tag{46}$$

where

$$a = \frac{\beta_1 P(1-c)+\beta_2 I}{N},\ b = \frac{-\beta_2 S}{N},\ d = (\mu+\tau),\ e = (\gamma+\phi+\mu+\delta)$$

The resulting characteristics equation of (35) becomes

$$\lambda^3+(d+e+a+\mu)\lambda^2+(de+ad+ae+\mu d+\mu e-\tau b)\lambda+(ade+\mu de-\tau\mu b) = 0 \tag{47}$$

By **Descartes' Rule of Signs** [11], if the coefficient of λ and the constant term in (36) are greater than zero, then, the eigenvalues of (32) are certain to be all negative. As such, asymptotical stability of (30). Otherwise, unstable.

5. Numerical Simulation

we used MATLAB to get the numerical solution of our model by applying Adams-type predictor-corrector method. This method is well known for numerical solutions of first-order problems,[6].

Table 2. Estimated initial conditions and the parameters with values and their sources

Parameter	**Value**	**Source**
Λ_h	$2,000$	[7]
ρ	0.5120	[8]
β_1	0.5172	[8]
β_2	0.4828	[8]
μ	1.9249	[8]
τ	0.5013	[8]
γ	0.4885	[8]
ϕ	1.4422	[8]
δ	0.1570	[8]
$1-\gamma$	0.5115	[8]
Λ_p	1,000	Estimated
μ_1	0.9407	[8]
σ	1.0345	[8]
c	$0 < c < 1$	[8]

Variable at initial condition	Value	Source
$S(0)$	20,000	Estimated
$E(0)$	5,000	Estimated
$I(0)$	2,000	Estimated
$T(0)$	1,000	Estimated
$R(0)$	18000	Estimated
$P(0)$	9,000	Estimated

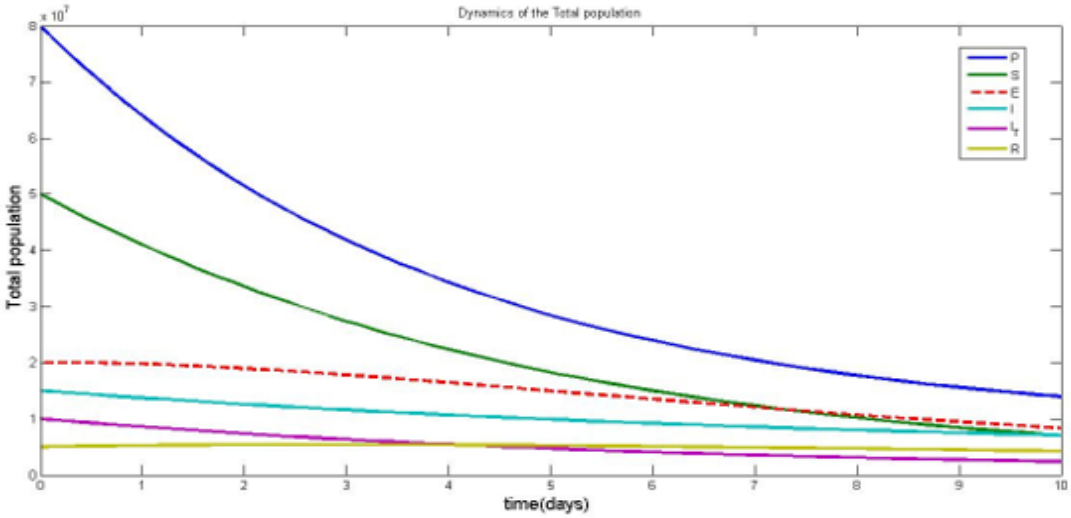


FIGURE 2. Dynamics of the Diseased Population.

5.1. **Graphical Simulation.** From the figure above, the infestation will be high at the earlier stage due to the lack of awareness of its endemic, therefore the susceptible population will be decreasing once the interaction with the pathogen is in action. This implies that the disease will invade the population with time.

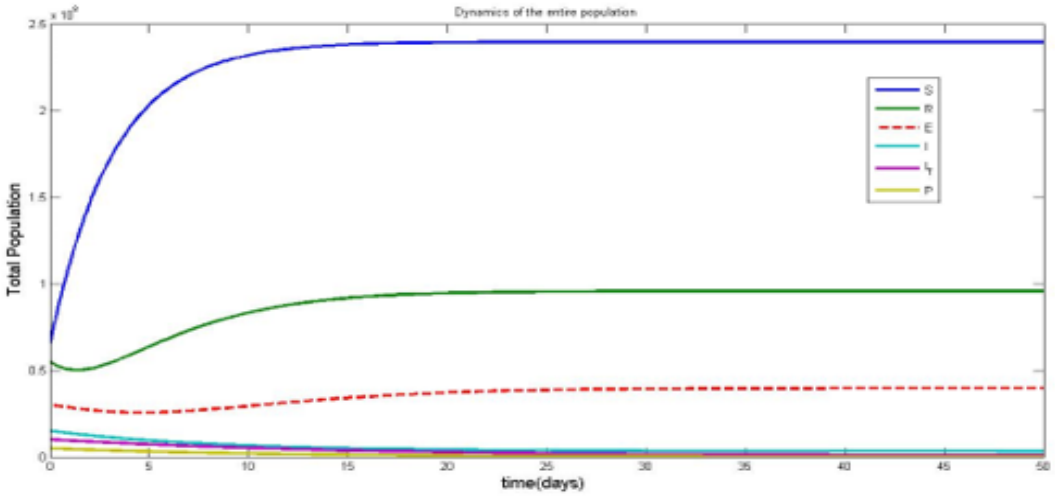


FIGURE 3. Effect of Insect Repellent on Diseased Population.

From the figure above, the graph showed us that the disease infection started responding to the insect repellent containing diethyltoluamide once the population became aware of the disease endemic and adhered to the use of it. Therefore, as time goes on, the use of insect repellent will become more effective, as so fizzle out the disease as the curve does not collapse to zero but will consequently increase the population class with time and hence gives us a locally asymptotically stability

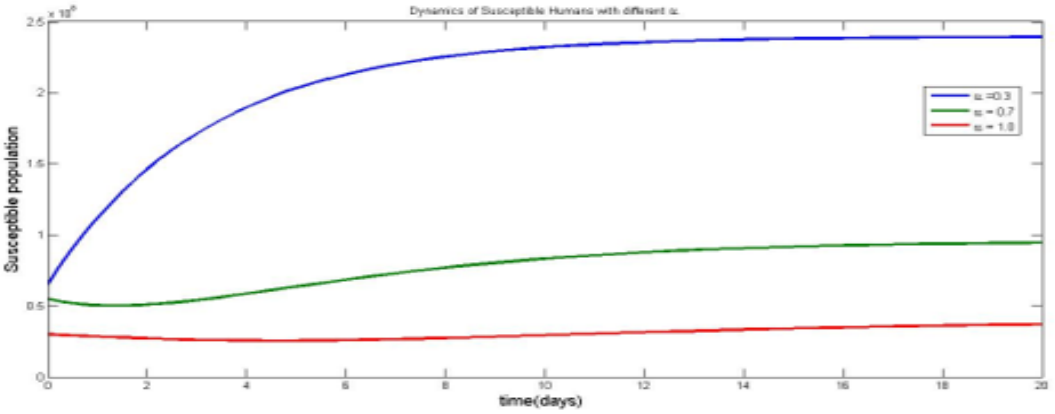


FIGURE 4. Variation of Susceptible Class with Different α.

The above graph is from the controlled population. Here, we could be able to analyze the authenticity between fractional and the integer approach and we found

out that the fractional approach is proved to be better than those at the integer case. As it is seen from the dialogue box, $\alpha = 1.0$ is undoubtedly a good one but as the population is under control, it has given the fractional approach a better result at $\alpha = 0.3$ since the susceptible class have gained more individuals. Therefore, the graph $\alpha = 0.3$ shows how the susceptible population increases with time and stabilizes at some point and that means that the system is locally asymptotically stable at that point.

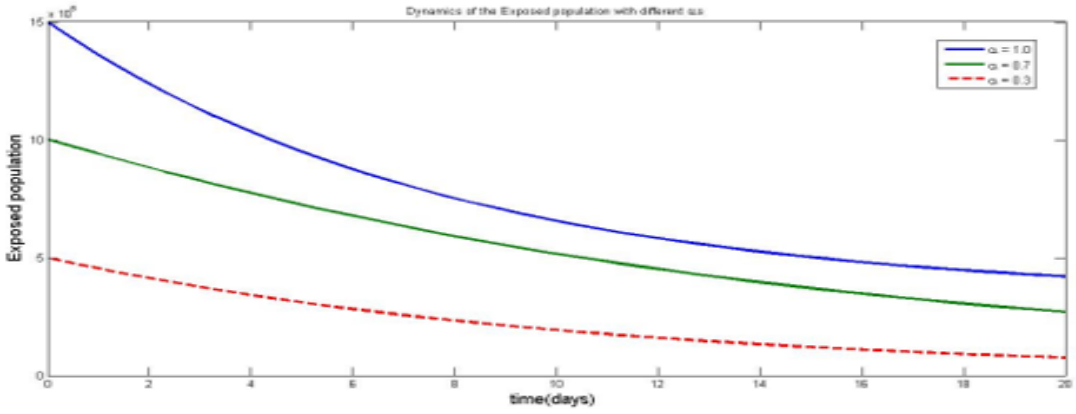


FIGURE 5. Variation of Exposed Class with Different α

From the above graph, it captures a decrease in the exposed population with time and clearly shows that the exposed population will vanish at some point since the graph is from controlled population. Therefore, $\alpha = 0.3$ is a better result compared to $\alpha = 1.0$ and $\alpha = 0.7$ Therefore, since the graph captures a decrease in the exposed population with time and clearly shows that the exposed population will vanish at some point, it means that the model is LAS when $R_0 < 1$.

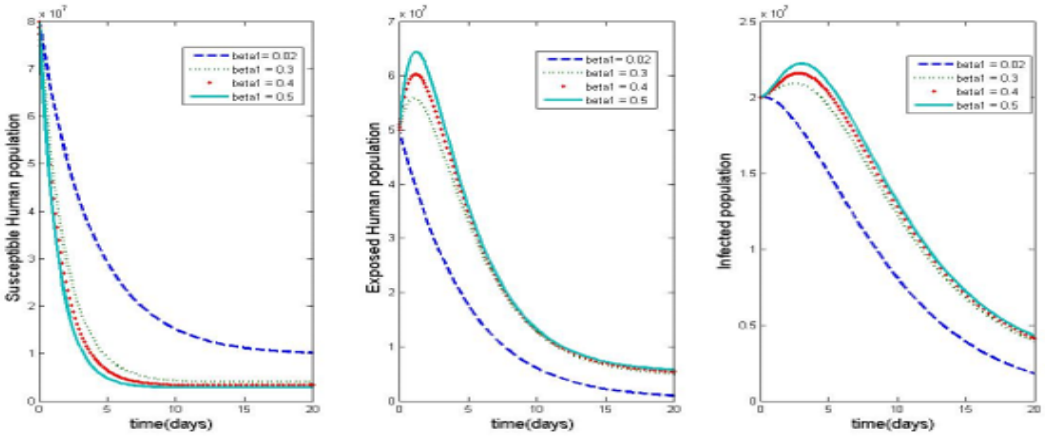


FIGURE 6. Dynamics of Sensitive Parameter On Diseased Population

From the susceptible graph, we observed that increasing the presence of parameter, β in the diseased population leads to decrease in the susceptible class. Then, the population keeps on reducing as the interaction level keep on increasing with time. At the exposed graph, for the fact that the interaction rate has been increased on the susceptible class, then the exposed population will increase gradually with time. At the infected graph, the infectiousness of the disease in the population will increase as well since the exposed class is increased. In general, Figure 6 shows how a variation of the interaction level affects the susceptible, exposed and infected population. From susceptible, it gave a decreasing graph since the interaction increases in the absence of control on the population.

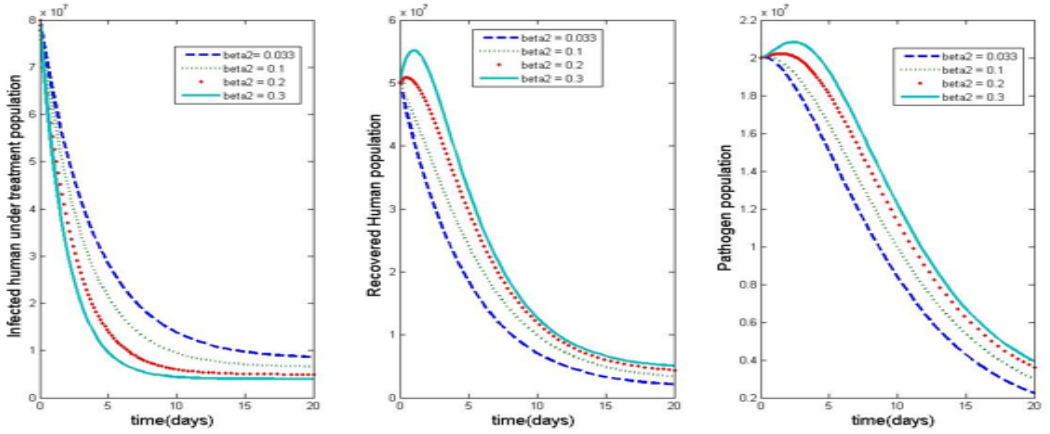


FIGURE 7. Dynamics of Sensitive Parameter On Diseased Population

From the treatment graph, since the infected population here have adopted treatment, then increasing more presence of the parameter, β will reduces the infectiousness of the disease in the population which increase the recovered population. From the pathogens graph, off course increasing more presence of the interaction rate implies increased in the pathogens level.

6. Conclusion

We have presented a fractional order model dynamic of myiasis infection with control measure and the effect of the control measure was felt as demonstrated on the graph. The model captures the causes, the predisposing factors of myiasis disease and a possible way of preventing the interactions. It can be seen in the model that the spread of the disease totally depends on the interaction between the pathogens and the people within the population. It is seen that if the proportion of population that is treated increases, then they will become susceptible again after recovery. The numerical simulations of the fractional order model with different values of α are performed by Caputo's derivative using the predictor corrector method of Adams-Bash forth Moulton type. The dynamics of the compartments have been shown in the graphs obtained. In addition, the results gave an insight that fractional order model is more suitable than its integer-order.

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