Stability Analysis of Host-Parasite System with Functional Response

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Abstract: *In this paper, in a host-parasite system that incorporates functional response and the growth rates would regulate the interaction among the species that might be induce the populations to approach steady states were investigated. To check the biological feasibility of the system, the positivity and boundedness of solutions of the model within deterministic environment are discussed. Moreover, the stability of equilibrium point of deterministic model is investigated. Finally, some numerical simulations to illustrate the analytical results were conducted.*

Keywords*: mathematical model; host-parasite; positivity and boundedness; equilibrium points; stability; Numerical simulation.*

I.   Introduction

It has been the general observation that populations can sometimes undergo drastic changes in their abundance that are not only unexpected but often are equally difficult to explain easily either theoretical or empirical [1]. The rationale behind modeling population growth is to analyze the reason which causes such abrupt and seemingly bizarre changes in the population size.

Recently, the effects of parasites on host population dynamics has became attract the attention of researchers in line with how the parasite induced diminution of host productiveness and survival rates changes the host population dynamics and how such dynamics are applied to predict terrorization to biodiversity in general and endangered species in particular [2–4].

Mathematical model plays an ever more important role in the study of ecology [14], which provides understanding of the underlying mechanisms that species interact and in line with the influence that the parasite changes the fitness of the interaction which can affects directly or indirectly, and, in the process, it suggests to take a measure. It is now widely believed that parasites were responsible for a number of extinctions on large land masses .As a result, ecologists acknowledge the importance of parasites in the dynamics of population.

Mathematical models are increasingly used to guide public health policy decisions and for controlling infectious disease. It is based on the specific property of population growth, the spread rules of infectious diseases, and the related social factors to construct mathematical models reflecting the dynamic properties of infectious diseases, to analyze the dynamical behavior and to do some simulations. Aim of ecological modeling is to understand the prevalence and distribution of a species, together with the factors that determine incidence, spread, and persistence [2].Understanding disease dynamics across hosts is an essential first step in understanding and articulating those conditions under which new diseases can emerge from [3].

General host-parasite models may be judged on several counts, especially on whether the biological assumptions made are valid and whether sufficient assumptions have been made for the outcome to indicate the roles of parasitism in natural interactions [7-10]. It is also important that the model is 'useful' which of course depends on the objectives in mind**.**

Host -parasite interactions have long been the subject of wild interest in the bio mathematical literature. In addition to motivating the models of Anderson and May [1], the analogy has been used to develop theory for how parasites interact with each other within hosts. Parasites can mediate apparent competition between alternative hosts. Additionally, due to tradeoffs between competitive ability and anti-parasite defenses in hosts, parasites can act as keystone species, promoting host species diversity by selectively targeting competitively superior host species [7,8]. The classical model for a prey/Host population of density N and a parasite/predator population of density P be written as;

$$\frac{dN_h}{dt} = r_h N_h - p N_h N_p,$$
$$\frac{dN_p}{dt} = cp N_p N_h - d_p N_p, \tag{1.1}$$

Where r_h-host per capita rate of increase, N_h-number of host, p-predation rate, N_p −number of prey/hosts killed by the predator or parasite, c-host to predator conversion rate.

Now, incorporating this classical model with response we can write as;

$$\frac{dN}{dt} = g(N) - \alpha(N,P)\delta,$$
$$\frac{dP}{dt} = \varepsilon\alpha(N,P)\delta - \sigma P, \qquad (1.2)$$

Where *g(N)* is the growth rate of the host population in the absence of parasite. The functional response $\alpha(N, P)$ represents the instantaneous rate of host reduction per parasite (infection by parasite). The constant ε describes the efficiency of the parasite in infecting host and converting into parasite offspring, while σ denotes the food independent parasite mortality rate.

Since the famous work of Lotka and Volterra [4], there has been extensively investigation on the dynamics of such models. In these works, the functional response, quantifying the amount of host consumed per parasite per unit time, plays an important role in host-parasite dynamics.

Functional responses are conventionally modeled as host-dependent, where the host consumption rate by an average parasite is only a function of host density alone. As noted in [4, 22-24], the derived functional response maybe host-dependent under the assumption of spatially homogeneous distributions of both parasite and host. However, when the spatial structure of one or both of the interacting populations are involved, it would be more plausible to take the parasite-dependent functional form, where both parasite and host densities affect the response.

Recently, parasite or dependent responses, a terminology taken from [16-20], have received increasing support from theoretical and empirical study in ecology. Since parasite behavior resembles that of a predator, i.e. the interaction ends with the 'elimination' of the host. Infective offspring are produced shortly after emergence of the vermiform from the host cell. Infection by a single parasite is presumably enough to take life of a host. Based on these features, we chose to explore the system dynamics starting with the modification of the traditional Lotka–Volterra predation model [4] here named model of host–parasite system in which this model is modified in recent paper.

The paper is structured in the following manner. In the next section, basic assumption of the model was considered and the scaled systems which can be used for easier analyze were presented. In section 3 the positivity and boundedness solution of the system and stability analysis of equilibrium points are will be discussed. Finally, numerical examples are carried out and conclusion of the paper with a brief discussion is given in the last section.

II. Basic Assumption And Model Formulation. To

Mathematical model plays an ever more important role in the study of ecology and epidemiology [5, 21], which provides understanding of the underlying mechanisms that influence the spread of disease, and, in the process, it suggests control strategies.

Define $Y(t)$ and $Z(t)$ to be the magnitudes of the host and parasite populations, respectively at time t. In this paper, the continuous population growth, where generations overlap completely was assumed that can be described by differential equations. For instance, protozoan and helminth parasites exhibit continuous time for their growth.

All parasitic species are capable of multiply infecting a proportion of the host population and that the per capita rates of infected hosts are altered by the number of parasites they port would be assumed. The precise functional relationship between the number of parasites harbored and the host's chances of surviving or reproducing varies greatly among different host-parasite associations [20-23]. The rate of parasite induced host mortalities may increase linearly with parasite burden or as a logistic or power law function.

In the majority of host-parasite system it appears to be the death rate rather than the reproductive rate of the host which is influenced by parasitic infection. For instance, many parasitic arthropods also decrease the reproductive power of their hosts, and in certain cases complete parasitic castration occurs [11-13].

Accordingly, the majority of our models assume that the parasite increases the host death rate. Attention is given to the population consequences of parasite induced reduction of host reproductive potential.

The two basic equations, for dy/dt and dz/dt, are constructed from several components, each of which represents specific biological assumptions.

In this paper, the rate of growth of the host population is simply determined by the natural intrinsic rate of increase in the absence of parasitic infection minus the rate of parasite induced host mortalities. Both the host reproductive rates, and the rate of 'natural' mortalities, are represented as constants unaffected by density dependent constraints on population growth. The term 'natural' mortalities to encompass all deaths due to causes other than parasitic infection, e.g. predation and senescence as described in (1.1).

Omission of density dependent constraints on host population growth is deliberate. We recognize that in the real world host population growth would be limited by, among other factors, intra-specific competition for finite resources. Since the aim of this study, however, is to provide qualitative insights into the mechanisms by

which parasites regulate host population growth and such simplification clarifies predictions of biological interest.

Moreover, if the parasite fails to control host population growth, exponential increase of the host population occurs until resource limitation results in the gradual approach to a carrying capacity.

The following Lotka-Volterra system with exponential growth (for host) and decay (for parasite) terms, as well as incorporating with carrying capacity to host species and Holling type I functional response defined as;

$$\frac{dy}{dt} = r_2 y(1 - \frac{y}{k_2}) - \varepsilon yz ;$$
$$\frac{dz}{dt} = -\partial z + \varepsilon kyz \qquad (1.3)$$

This model is structurally unstable but it can be used as a ground work for a more realistic representation.

To model the parasite induced host-parasite system with Holling type II functional response to the system the following assumptions are considered:

(a) In absence of parasite the host population grows logistically with intrinsic growth rate r_2 and environmental carrying capacity k_2.

(b) Infected host population is not in a state of reproduction and does not compete for the resource.

(c) The interaction between parasite and the host is of Holling type-II form. This combination of functional forms is taken because the capturing of infected host is easier than the susceptible host.

(d) The number of encounter parasite with the host availability is proportional to the density of the host.

(e) The proportionality constant characterizes the ability of the parasite to identify the searching of host.

With the above assumptions, a system (1.3) was modified and formulates the model as:

$$\frac{dy}{dt} = r_2 y \left(1 - \frac{y}{k_2}\right) - \frac{\beta_2 yz}{\alpha_2 + y}$$
$$\frac{dz}{dt} = -\mu z + \frac{\beta_3 zy}{\alpha_3 + y} \qquad (1.4)$$

Where constants $\beta_{i's}$ maximal attacking rate of parasite for susceptible host ,i.e., the parasitic utilization efficiency (part of the functional response), $\alpha_{i's}$ is the half saturation constant, μ constant death rate of the population of parasite, r_2 basal growth rate of the population of host, k_2-size of the carrying capacity of the environment of host populations.

To determine which combinations of parameters control the behavior of the system, dimensionalzing the system (1.4) was carried out, then after manipulation the system (1.4) takes the form;

$$\frac{dy}{dt} = y - \delta y^2 - \frac{\beta yz}{1 + \theta y};$$
$$\frac{dz}{dt} = -kz + \frac{\alpha yz}{1 + \theta y}; \qquad (1.5)$$

III. Dynamical Behaviour Of The System

Since the state variables y and z represent population size, positivity implies that they never become negative. The boundedness may be interpreted as a natural restriction to growth as a consequence of limited resources.

Positivity and boundedness of the solution of the system

In this section, we first discuss some basic dynamical properties of the deterministic model, which is subjected to positive initial conditions; $y(0) \equiv y_0 > 0$, $z(0) \equiv z_0 > 0$. The following lemma was stated and verified as follows.

Lemma 1. Let $(y(t), z(t))$ be the solution of system (1.4) with initial condition. Then (y(t), z(t)) is positive and ultimately bounded for all $t \geqslant 0$.

Proof: First, let us consider $z(t)$ for $t \geqslant 0$. From the equation of system (1.4), such that

$$\frac{dz}{dt} = -\mu z + \frac{\beta_2 yz}{\alpha_3 + y},$$

By separation of variables, this equation becomes;

$$\frac{dz}{z} = \left(-\mu + \frac{\beta_2 y}{\alpha_3 + y}\right) dt$$

Integrating both sides, we can get;

$z(t) \geq z(0)\exp\left\{\int_0^t \left(-(\mu) + \frac{\beta_2 y(s)}{\alpha_3 + y(s)}\right) ds\right\} > 0$

Therefore, with the given positive initial condition we have that $Z(t) > 0$ for all $t \geqslant 0$

For $\frac{dy}{dt} = r_2 y(1 - \frac{y}{k_2}) - \frac{\beta_2 yz}{\alpha_2 + y}$, to show y(t) > 0 for t > 0;

Using theory of differential equation and with similar procedure; the solution of the system (1.4) can be solved analytically and becomes;

$Y(t) = y(0) \exp\int_0^t \left\{r\left(1 - \frac{y(u)}{k_2}\right) - \frac{\beta_2 z(u)}{\alpha_2 + y(u)}\right\} du > 0.$

Hence, it is non-negative.

To show that the systems are ultimately bounded,

The system (1.4) can be written as using system of differential equations Y =F(Y) together with Y(0) =$Y_0 \in R_+^2$, where $Y = (y, z)$, F=$(F_1, F_2)^T$, $F_i(Y) = F_i(y, z), i = 1,2$ with $y_0 \in R_+^2$, say the solution of (1.4), $y(t) = y(t, y_0)$, is such that for all t>0.

From (1.4), we have $\frac{dy}{dt} \leq r_2 y - \frac{r_2}{k_2} y^2$. By assuming $k_2 = 1$ we can treat this inequality as;

$\frac{dy}{dt} \leq r_2 y - r_2 y^2$ .This become after some algebraic manipulation, $y(t) \leq \frac{1}{1+e^{-(r_2 t + a_1)}}$. Since a_1 is arbitrary constant and $y(t) = y_0$ at $t = t_0$. Hence we can obtain $y(t) \leq \frac{1}{1+\frac{1-y_0}{y_0}e^{-r_2(t+t_0)}}$.

For $t \to \infty$, $y(t) \leq 1$. This indicates that boundedness, because the number 1 is constant.

Moreover, assuming that k= $y + z$ and finding the derivative of k with respect to t along with the solution of the system above,

We get;

$\frac{dk}{dt} = \frac{dy}{dt} + \frac{dz}{dt}$ =$r_2 y \left(1 - \frac{y}{k_2}\right) - \mu z$.

Let multiply k by μ and add with its derivatives, then we can obtain,

$\frac{dk}{dt} + \mu k \leq (r_2 + \mu) y(t) \leq (r_2 + \mu)$.

Now, this leads us to get $k(t) \leq \frac{(r_2+\mu)}{\mu} + ae^{-\mu t}$, where a is arbitrary constant and taking the limit of this system

as $t \to \infty$ we get that $k(t) = y(t) + z(t) \leq \frac{(r_2+\mu)}{\mu}$. Therefore, $z(t) \leq \frac{(r_2+\mu)}{\mu}$. Taking $\frac{(r_2+\mu)}{\mu}$=l, where l is constant. Thus $z(t)$ is ultimately bounded.

3.1. Steady states
The steady states of the system after scaling have the following equilibrium points;

(i) $E_0(0,0)$-washed out of the species.

(ii) $E_1(\frac{1}{\delta}, 0)$-the host species live up to certain time t,

(iii) $E_3\left(\frac{k}{\alpha-\theta k}, \frac{\alpha(\alpha-k(\theta+\delta))}{\beta((\alpha-\theta k)^2)}\right)$ exists when $\alpha > k(\theta + \delta)$,this is the coexistence of the populations.

Community matrix

To analyze the stability of the model we determine the community matrix called Jacobian matrix as follow;

Let $f(y, z) = \frac{dy}{dt}$ and $g(y, z) = \frac{dz}{dt}$, then

$$J=\begin{pmatrix} \frac{\partial f}{\partial y} & \frac{\partial f}{\partial z} \\ \frac{\partial g}{\partial y} & \frac{\partial g}{\partial z} \end{pmatrix}$$ but $f(y, z) = y - \delta y^2 - \frac{\beta yz}{1+\theta y}$ and $g(y, z) = -kz + \frac{\alpha yz}{1+\theta y}$

Thus,

$$J=\begin{pmatrix} \frac{\partial f}{\partial y} & \frac{\partial f}{\partial z} \\ \frac{\partial g}{\partial y} & \frac{\partial g}{\partial z} \end{pmatrix} = \begin{pmatrix} 1 - 2\delta y - \frac{\beta z}{(1+\theta y)^2} & \frac{-\beta y}{1+\theta y} \\ \frac{\alpha z}{(1+\theta y)^2} & -k + \frac{\alpha y}{1+\theta y} \end{pmatrix}$$

3.2. Qualitative analysis of the model.
3.2.1. Local stability of steady state
The system has three non negative steady states. The non –negative equilibriums of the system was listed in section 3.1.above.

The following results can be observed for the system based on the equilibrium points. The vanishing equilibrium point always exists.

Lemma 2.

i.   The trivial equilibrium point is saddle-node (Unstable).

Proof: The characteristic equation at the $E_0(0,0)$ is $(1-\lambda)(-k - \lambda) = 0$.

The eigenvalues are $\lambda = 1, -k$. So, $E_0(0,0)$ is unstable which is saddle in nature . This indicates that there is instability for the host and stability for the parasite, since the eigenvalues are $\lambda_1 > 0$ and $\lambda_2 < 0$.

ii. $E_1(\frac{1}{\delta}, 0)$ is locally asymptotically stable if $\alpha < k(\delta + \theta)$ holds otherwise it is unstable.

Proof: The characteristic equation at the $E_1(\frac{1}{\delta}, 0)$ is;

(-1-λ) $\left(\frac{\alpha}{\delta+\theta} - k - \lambda\right) = 0$.

The eigenvalues are $\lambda = -1, \frac{\alpha}{\delta+\theta} - k,$

So the equilibrium E_1 is locally asymptotically stable if $\alpha < k(\delta + \theta)$ holds otherwise unstable.

iii. $\quad E_3(y^*, z^*)$, which is the coexistence is stable if $\frac{k[\alpha - k(\theta+\delta)]}{\alpha} > 0$

Proof: The characteristic equation at E_3 is:

$\det(J_3 - \lambda I) = 0$, in this case we have the following;

$\lambda^2 - \lambda v + u = 0$, where $v = -\left[2\delta y^* + \frac{\beta z^*}{(1+\theta y^*)^2} - \frac{\alpha y^*}{1+\theta y^*} + k - 1\right];$

After some algebraic manipulation, we get;

$$v = \frac{-[2\alpha\delta k - k(-k\theta + \alpha)(\theta - \delta)]}{\alpha(\alpha - k\theta)}$$

$$v = \frac{-k[2\alpha\delta - (-k\theta + \alpha)(\theta - \delta)]}{\alpha(\alpha - k\theta)}$$

$$u = \frac{\alpha\beta y^* z^*}{(1 + \theta y^*)(1 + \theta y^*)^2} + \left(\frac{\alpha y^*}{1 + \theta y^*}\right)(1 - 2\delta y^*) - \left(\frac{\alpha y^*}{1 + \theta y^*}\right)\frac{\beta z^*}{(1 + \theta y^*)^2} + 2\delta k y^* + \frac{\beta k z^*}{(1 + \theta y^*)^2} - k$$

$$u = \frac{k[\alpha - k(\theta + \delta)]}{\alpha}$$

Thus,

$\lambda_1 = \frac{v+\sqrt{v^2-4u}}{2}$ and $\lambda_2 = \frac{v-\sqrt{v^2-4u}}{2}$

Using the trace determinant (T-D) criteria, that is $T < 0$ and $T^2 - 4D > 0$, we have two negative eigenvalues if $D > 0$, this holds true if $\alpha > k(\theta + \delta)$.
Hence, under the set criteria the steady state is stable.

3.2.2. Global stability of the steady state
Here, the stability analysis of the model (1.4) is conducted using suitable Lyapunov functions method and the results are presented in the form of theorems followed by their proofs as follows.
Theorem 1: The equilibrium point E_1 is globally asymptotically stable.
Proof: Consider the Lyapunov function derived from the integral form
$$\int_{x^*}^{x} \frac{u - x^*}{u} du .$$
Now, let $v(\text{y}, \text{z}) = \left[y - y^* - y^* ln\left(\frac{y}{y^*}\right)\right].$
On differentiating v with respect to t and after substituting expression for dy/dt gives

$\frac{dv}{dt} = \left(\frac{y-y^*}{y}\right)\left(\frac{dy}{dt}\right)$

$\frac{dv}{dt} = \left(\frac{y-y^*}{y}\right)\left[r_2 y\left(1 - \frac{y}{k_2}\right) - \frac{\beta_2 yz}{\alpha_2+y}\right]$

$\quad = (y - y^*)\left(-\frac{r_2}{k_2}\right)(y - y^*)$

$\quad = -\frac{r_2}{k_2}(y - y^*)^2 < 0;$

Thus, $\frac{dv}{dt} < 0$ i.e., v is positive definite and also $v(y^*, z^*) = 0$. Therefore E_1 is globally asymptotically stable.
Theorem 2: The steady state E_3 is globally asymptotically stable if the condition $s < \beta_2/\beta_3$ is satisfied.
Proof: Consider the Lyapunov functions as $v(y,z) = (y - y^* - y^* ln\frac{y}{y^*}) + s(z - z^* - z^* ln\frac{z}{z^*})$, where s is some positive constant assumed.
Now, the differential of v with respect to t and after some algebraic manipulations reduces to the following form:

$\dot{v} = \left(\frac{y-y^*}{y}\right)\left(\frac{dy}{dt}\right) + s\left(\frac{z-z^*}{z}\right)\left(\frac{dz}{dt}\right)$

$\quad = \left(\frac{y-y^*}{y}\right)\left[r_2 y\left(1 - \frac{y}{k_2}\right) - \frac{\beta_2 yz}{\alpha_2+y}\right] + s\left(\frac{z-z^*}{z}\right)\left[-\mu z + \frac{\beta_3 zy}{\alpha_3+y}\right]$

$\quad = -\left[(y - y^*)^2 \left(\frac{r_2}{k_2} + d\right)\right] + (z - z^*)(y-y^*)\frac{1}{\alpha_3+y}(s\beta_3 - \beta_2), \text{d} = \frac{\beta_2 z^*}{\alpha_2+y}$

Observe that in the expression for $\dot{v}$, the term $-[(y - y^*)^2]$ is negative, where as the expression $(z - z^*)(y-y^*)\frac{1}{\alpha_3+y}$ is positive.

Thus, it is straight forward to conclude that $\frac{dv}{dt} < 0$ if the condition $s\beta_3 - \beta_2 < 0$ holds true.

That is,$s < \beta_2/\beta_3$.

Therefore, the interior equilibrium point is globally asymptotically stable under the set criteria.

IV. Numerical simulations

For substantiation of our earlier discussed analytical results, we here would like to present some numerical replications with the help of some software package.
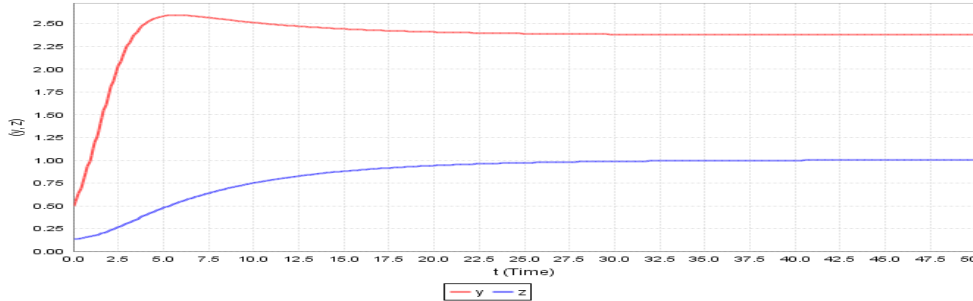

Figure 1: The long dynamics of the two species in which host dominates over the parasite with positive initial conditions.

The value of the parameter is; δ=0.3720, β=0.1720, $\theta = 0.0560$,k=0.0280, $\alpha = 0.0440$.
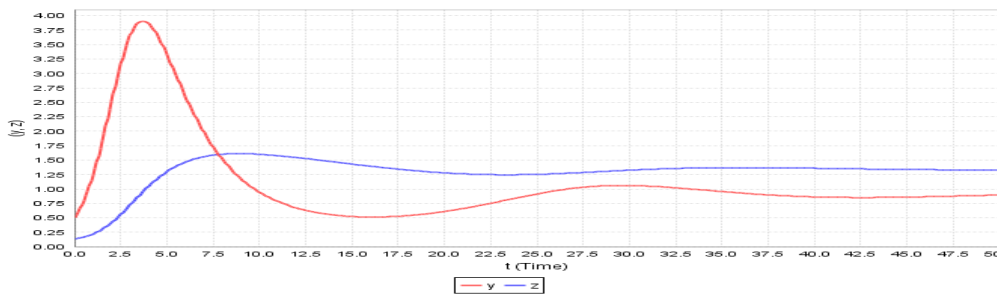
Figure 2: The long dynamics of the population dominated by the dependent species.

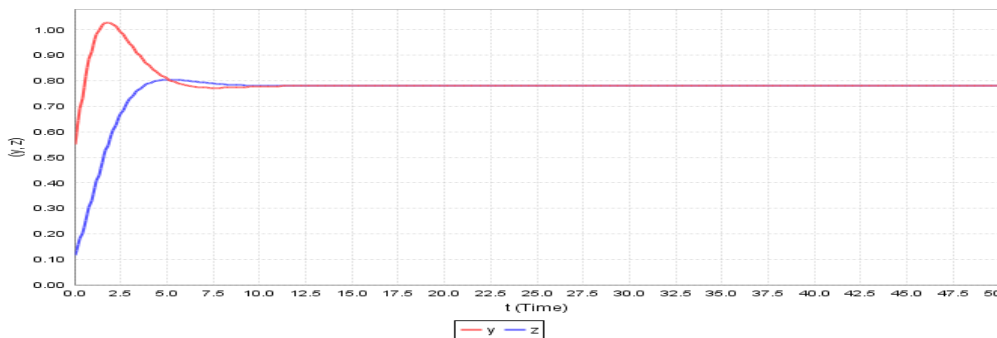The value of the parameter is; δ=0.1000, β=0.7720, $\theta = 0.1250$,k=0.0560, $\alpha = 0.0310$

Figure 3: the two populations exist together for a long dynamics.

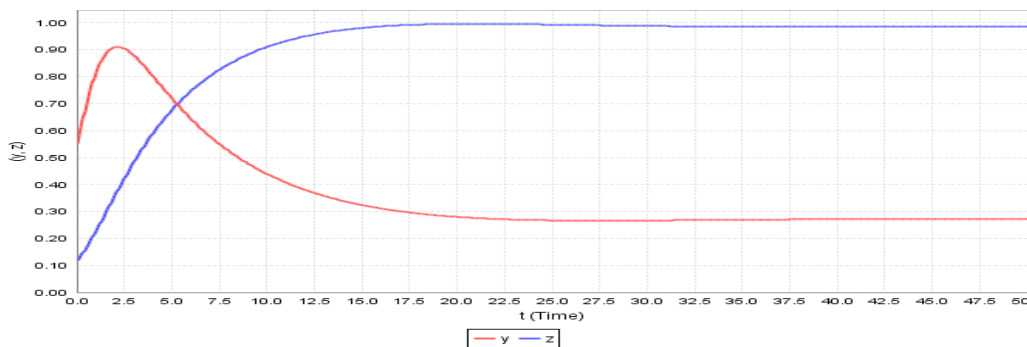The value of the parameter is; δ=0.9030, β=0.9470, $\theta = 0.4440$,k=0.4280, $\alpha = 0.0190$

Figure 4: The parasite dominates over the host population

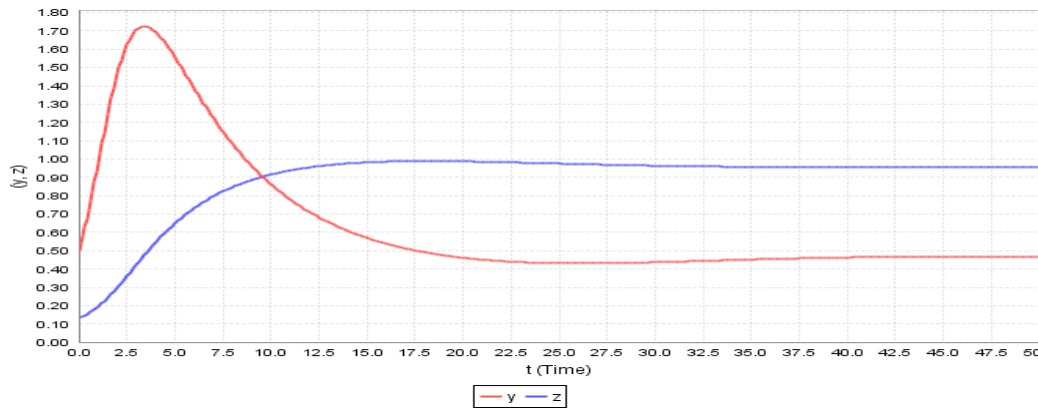The value of the parameter is; δ=0.9030, β=0.9470, $\theta = 0.1780$,k=0.0340, $\alpha = 0.0560$

Figure 5: The dependent species dominates the host population.
The value of the parameter is; δ=0.3720, β=0.9660, θ = 0.1000,k=0.0280, α = 0.0440.
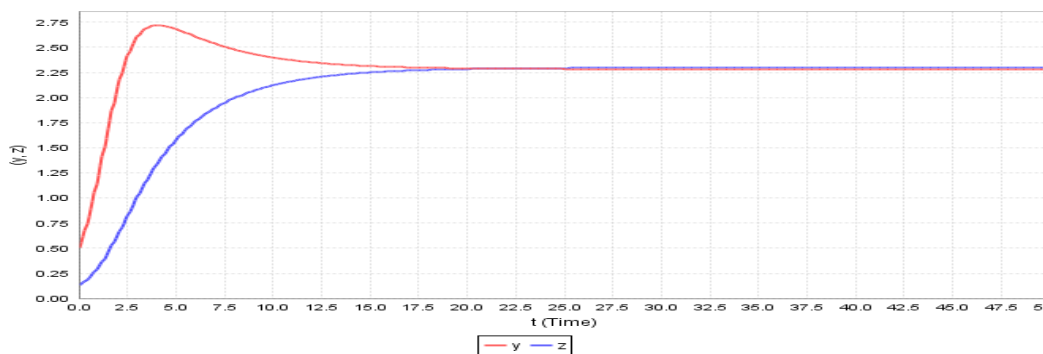

Figure 6: The coexistence of the two populations for long dynamics with different initial conditions.
The value of the parameter is; δ=0.3720, β=0.1720, θ = 0.2440,k=0.0280, α = 0.0940.

V.   Result and Conclusions

In this paper, a two dimensional host-parasite system consider for investigation. Based on the assumptions mathematical model was formulated. The validity of the model was conducted. To have biological relevance, we have should ensured that for any finite time, a unique solution to the model exists and that state variables should always be non-negative and ultimately bounded. The steady states are evaluated for the purpose of stability analysis. The stability analyses of the steady state were investigated. Simulation study is conducted to support the analytical results. Moreover, the following conclusions were drawn.

Figure 2, 4, 5 shows that when the value of is θ lies between 0.1 and 0.2 and β is relative large, the parasite dominates the host population. That is, when the half saturation rate or handling rate of time per unite time of the host lies between 0.1 and 0.2 and the parasitic utilization efficiency (part of the functional response) is somehow large as shown in the figure.

Moreover, when the parametric value of is less than 0.1 the host population dominates over the dependent species which is shown as in figure 1.

But when this parameter is greater than 0.2 the populations exist together for a long time as shown in figure 3 and 6

From the figures one can deduce that, with the given parameters chosen when the number of parasite population exceeds the number of its host fitness there will be a decline of host species as observed. Moreover, the species behave asymptotical behavior which was indicating in the figure as time become large. Beside this, the models contain the central assumption that the parasite increases the rate of host mortalities. The parasite induced changes in this rate are formulated as functions of the parasite numbers per host. The parameters influencing the ability of the parasite to regulate the growth of its host's population were not completely mentioned here, this is our further investigation.

The population models of host-parasite interactions are all characterized by the central assumption that parasites cause host mortalities. In particular, we have assumed that the net rate of such mortalities is related to the average parasite burden of the members of a host population. Species which exhibit such characteristics may in certain circumstances play an important role in regulating or controlling the growth of their host population.

In this paper, in conclusion, the researcher has demonstrated that the population processes are of particular significance in stabilizing the dynamical behavior of a host parasite interaction and enhancing the regulatory influence of the parasite.

This study can be extended by considering other functional response curves. That is limit cycle that leads to periodic solution and bifurcations condition will be our next investigation.

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