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Prey-Predator model with treatment in Tilapia and Mudfish as predator and disease carrier

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Abstract

The relationship between tilapia and mudfish forms a highly dynamic ecosystem where both predation and disease transmission occur simultaneously. In this system, mudfish not only prey on tilapia but also act as vectors of *streptococcosis* disease, which spreads to tilapia in shared water conditions. This study develops and analyses a coupled predator-prey disease model tailored to the tilapia-mudfish aquatic environment. The model identifies five key equilibrium points: trivial, axial, predator-free, disease-free, and endemic, which explain how the ecological system behaves under various ecological scenarios. The basic reproduction number was computed to characterize conditions that trigger disease persistence or elimination. To improve the health and population of tilapia, optimal control theory was used to introduce treatment as a time-dependent control intervention. By applying Pontryagin's Maximum Principle, the study determined and simulated an optimal treatment strategy, showing how the control effort varies over time, starting with a strong intervention and gradually reducing as the infection level declines. Comparative simulations with and without control further illustrated the effectiveness of treatment. The findings show that treatment can reduce disease prevalence, strengthen the tilapia population, and prevent ecological collapse between the predator and prey. This study focuses on developing a viable treatment framework to enhance the sustainability of aquaculture production, specifically by prioritizing treatment within an integrated aquaculture environment.

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Keywords: Basic reproduction number, optimal control, pontryagin maximum principle, prey-predator, *streptococcosis*

1 Introduction

Predator-prey theory is a branch of ecology that describes the dynamic interaction between predator and prey populations, in which changes in population sizes of one species directly affect the growth and survival of other species. Predator-prey mathematical models allow researchers to investigate

a wide range of cases, including aspects of predation that would be difficult, expensive, or time-consuming to investigate experimentally. When one species acts as a source of food (prey)to another species (predator), the model describing these dynamics is referred to as a predator-prey model. The simplest dynamical interactions between predators and prey were Lotka-Volterra. Further exploration of predator-prey models with the incorporation of disease is an area of great concern; see, for instance, Anderson [3], Abayneh Fentie Bezabih et al [4], Bera [5], Chattopadhyay [6], Hadeler [11], Klempner [**?**], Krishna [14], Sanchayita [18], and Venturino [20]. Several studies have looked at ways of controlling diseases in prey-predator and endemic systems. A study by Diva. A [**?**] showed that harvesting infected prey can help reduce the disease and protect healthy populations. Kebedow et al [12] went further by including predators as potential carriers of the disease, using realistic prey-predator interactions, and showed that controlling infections effectively requires careful planning and considering the costs of interventions. Lashari et al [15] highlighted the importance of treatment limits, demonstrating that when resources are limited, optimal strategies can still reduce infection and minimize costs. While these studies offer valuable insights, they do not fully address real-world aquaculture scenarios where predators can spread disease, and treatment needs to be carefully managed, leaving room for models adapted to systems like tilapia and mudfish.

The dynamical interactions involving tilapia, the prey, and mudfish, the predators, will be examined in this study. The system is influenced by a bacterial infection caused by *Streptococcus iniea*, a well-known pathogen in aquaculture that affects the prey population by introducing complex interactions between disease progression and trophic dynamics. *Streptococcus iniae* is a facultative intracellular bacterial pathogen that is pathogenic to tilapia and causes *streptococcosis* disease. The pathogen is water-borne, especially when the prey are in high density. The mudfish sheds bacteria that infect the tilapia species via the gills. The infected fish shows signs such as gill necrosis, skin haemorrhages, and bulging eyes Amal [1]. The infected tilapia also show behavioural changes, such as sluggish movement, making it easier for predators to capture the infected fish. Tilapia, being omnivores at lower trophic levels, are highly susceptible to the disease. In contrast, mudfish, which occupy higher trophic levels, are more carnivorous. Their scavenging and predatory behaviors, particularly on infected tilapia, can facilitate persistence and spread of the disease within the system. Therefore, controlling these dynamics through treatment is essential for sustainable aquaculture. *Streptococcus iniea* does not naturally infect mudfish Shoemaker [19]. Still, environmental and experimental data suggest that consumption of infected prey can lead to systemic infection, organ failure, or death. Mathematical modelling helps us understand prey, predator, and disease interactions by showing how healthy and infected populations change over time. While used for studying population stability and disease, many models ignore predators or lack realistic transmission and control measures, limiting their relevance to aquaculture.

Dynamical interactions involving prey and predator with control interventions have been studied. For instance Abayneh Fentie Bezabih et al [4] considered a prey-predator model with disease in both species incorporating treatment; the treatment applied had a great impact on the infected species. Optimal levels of treatment were not considered in their study.Optimal levels of treatment were not considered in their study. Prophylactic treatment for a prey-predator system to prevent the propagation of an infectious disease within the population has been studied in a journal article by Cojocaru, M.G. [7].

The spread of an infectious disease was modelled by an SIS model with vital dynamics and infection propagated through contact and predation; the species in this interaction died as a result of the disease. Treatment was applied to the prey and predator, and then to both species. The findings of the study showed that treatment significantly reduced infection, assuming that resources and treatment are provided to both species. Optimal control formulations that minimized both treatment costs and infection levels in both populations were developed. The numerical simulation of the model revealed that treatment reduces infection in both prey and predator. The model developed by Cojocaru, M.G. [7] can be improved by incorporating several control techniques to determine the most cost-effective strategies that minimize the cost of treatment while maximizing the reduction of disease prevalence. Studies by Diva.A [**?**] and Kebedow [12] considered prey-predator models incorporating control strategies. The control strategies were effective in mitigating the spread of infection among the species. Although the previous studies are significant, they do not reflect real-life scenarios where the predators can also transmit disease to the prey. In addition, the models are adapted to real-life aquatic ecosystems involving tilapia and mudfish. This study will consider a prey-predator model adapted to the tilapia(prey) and mudfish(predator) and carrier of *streptocossis* disease. Pontryagin Maximum Principle, a classical tool of control theory, will be utilised in determining the optimal treatment that will minimise infection among infected tilapia. Numerical simulation will be done using Python software to justify theoretical results. The results support informed decision-making and sustainable tilapia aquaculture management.

2 Model Formulation and Analysis

This study describes the variables and parameters as follows; S, I and M represent the susceptible prey, infected prey, and mudfish populations at time t, respectively, $N = S+I$ represents the total prey population; the intrinsic growth rates in the susceptible prey and predator are represented by r_1 and r_2, respectively, while κ_1 and κ_2 denote the predator search rate and predator satiety rate, respectively. The parameter ω represents the conversion efficiency by the predator, while μ represents the natural mortality rate of the species. The constant σ represents the adverse effects on the predator resulting from feeding on infected tilapia, where $0 \leq \sigma \leq 1$, whereas d denotes the death rate of infected prey due to the disease; the force of infection between susceptible and infected prey is represented by β. The rate of treatment among the infected tilapia fish species is represented by $v(t)$. The prey and predator carrying capacities are denoted K_s and K_m, respectively.

2.1 Model Assumption

The model is developed based on the following assumptions;

(i) Infected tilapia harbour a high load of pathogens such that their consumption by mudfish results in systemic infection, organ failure, or death.

(ii) In the absence of disease, the susceptible prey and predator populations grow logistically with carrying capacities K_s and K_m, respectively.

(iii) The susceptible prey population becomes infected when it comes into contact with the infected prey, and this contact is assumed to follow standard incidence with β as the rate of contact.

(iv) We assume a Holling type-II functional response to describe predation on both susceptible and infected prey at high prey densities.

(v) There is a constant mortality rate in all interacting species.

(vi) All mudfish are carriers of *streptococcosis* disease.

2.2 Compartmental Diagram

Below is a schematic diagram of the model showing the dynamics of species.

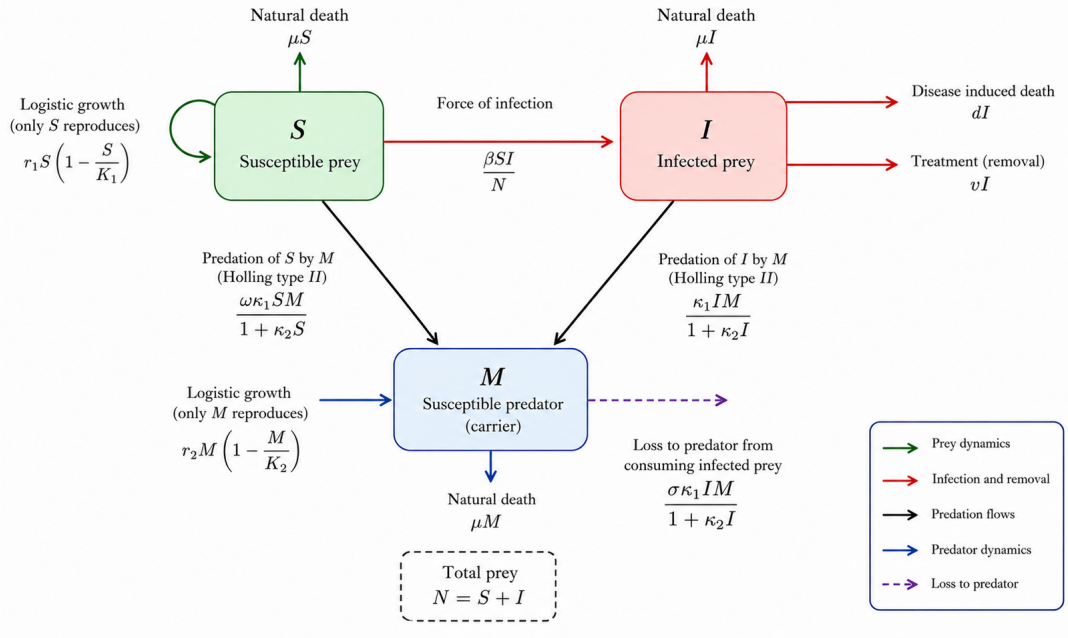


Figure 1: Compartmental diagram with vital dynamics

2.3 Model Equations

The above assumptions were used to formulate the model below;

$$\begin{aligned}\frac{dS}{dt} &= r_1 S(1-\frac{S}{K_s}) - \mu S - \frac{\beta SI}{N} - \frac{\kappa_1 SM}{1+\kappa_2 S} \\ \frac{dI}{dt} &= \frac{\beta SI}{N} - (\mu + d + v)I - \frac{\kappa_1 IM}{1+\kappa_2 I} \\ \frac{dM}{dt} &= r_2 M(1-\frac{M}{K_m}) + \frac{\omega\kappa_1 SM}{1+\kappa_2 S} - \sigma\frac{\kappa_1 IM}{1+\kappa_2 I} - \mu M\end{aligned} \tag{1}$$

2.4 Model Properties

To show that the solutions to model Equation 1 are valid and admissible, boundedness and positivity of solutions will be proved.

2.4.1 Boundedness of Solutions

Theorem 2.1. *Let* $\gamma = (r_1, r_2, \mu, \beta, \kappa_1, v, d, \sigma, K_m, K_s, \Lambda) > 0 \in \mathbb{R}^{+11}$ *be scalar parameters. The Model 1 with initial solution set* $\{S(0) \geq 0, I(0) \geq 0, M(0) \geq 0\} \in \mathbb{R}^{+3}$ *satisfy* $\{S(t) > 0, I(t) > 0, M(t) > 0\} \in \mathbb{R}^{+3}$ *for all* $t > 0$. *The region* $\Omega \subset \mathbb{R}^{+3}$ *is positively invariant with respect to Equation 1.*
$N = S + I + M$ *such that* $\Omega \subset \mathbb{R}^{+3}$ *with* $\Omega^{+3} = \{(S,I,M) \in \mathbb{R}^{+3} : (S + I + M) \leq \frac{\Lambda}{\mu}\}$

Proof. To show boundedness, let the total population of prey-predator be given as,
$N(t) = S(t) + I(t) + M(t)$.
Differentiating $N(t)$ with respect to time t yields,

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dM}{dt} \tag{2}$$

$$\frac{dN}{dt} + \mu N \leq \Lambda \tag{3}$$

where Λ is the total upper bound on the population.
Integrating both sides of Equation 3, we obtain

$$N(t) \leq \frac{\Lambda}{\mu} + C_0 e^{-\mu t} \tag{4}$$

and taking the $\lim_{t\to\infty} N(t)$ of Equation 4, the result is then found to be bounded between;

$$0 \leq N(t) \leq \frac{\Lambda}{\mu} \tag{5}$$

Therefore, the system Equation 1 is well-posed and biologically admissible. □

2.4.2 Positivity of Solutions

Since the model under this study describes the dynamics involving the fish species, the variables S, I, M and all parameters involved must be positive and well defined for all $t > 0$.

Consider

$$\frac{dS}{dt} = r_1 S - \frac{r_1 S^2}{K_s} - \mu S - \frac{\beta SI}{N} - \omega \frac{\kappa_1 SM}{1 + \kappa_2 S} \tag{6}$$

$$\frac{dS}{dt} \geq -(\frac{r_1 S^2}{K_s} + \mu S + \frac{\beta SI}{N} + \frac{\kappa_1 SM}{1 + \kappa_2 S}) \tag{7}$$

$$\begin{aligned} -\frac{dS}{dt} &\leq (\frac{r_1 S^2}{K_s} + \mu S + \frac{\beta SI}{N} + \omega \frac{\kappa_1 SM}{1 + \kappa_2 S}) \\ &\leq r_1 S^2 + \mu S + \beta SI + \omega \kappa_1 SM \\ &\leq S(r_1 S + \mu + \beta I + \omega \kappa_1 M) \end{aligned} \tag{8}$$

Let

$$\mu + \beta I + \omega \kappa_1 M = a \tag{9}$$

by substituting Equation 9 in Equation 8 we obtain,

$$\frac{dS}{dt} \geq -S(r_1 S + a) \tag{10}$$

Separating the variables and solving Equation 10 we obtain;

$$S(t) = \frac{Aa^2 e^{-C_1 t}}{1 - Ae^{-C_1 t} a r_1}$$

where A, a, C_1 are positive constants.

$$S(t) > 0, t > \frac{1}{C_1} lnA$$

3 Results and Discussion

3.1 Disease free equilibrium $E_3(S, 0, M)$

In the absence of a disease and at high prey density, the disease-free equilibrium point (DFE) is obtained by setting $I = 0$. By substituting $I = 0$ into Equation 1 we get,

$$r_1 S(1 - \frac{S}{K_s}) - \mu S - \frac{\kappa_1 M}{\kappa_2} = 0 \tag{11}$$

$$r_2 M(1 - \frac{M}{K_m}) - \mu M + \frac{\omega \kappa_1 M}{\kappa_2} = 0 \tag{12}$$

factorizing Equation 12 and solving for M in terms of the basic reproduction number we get,

$$\begin{aligned} M = & \quad (r_2 + \frac{\omega \kappa_1}{\kappa_2} - \mu)\frac{K_m}{r_2} \\ M = & \quad \frac{\beta - R_0(\mu + d + v)}{\kappa_1 R_0} \end{aligned} \tag{13}$$

Using the saturation term $\frac{\kappa_1 M}{\kappa_2}$ and factorizing Equation 11, we get,

$$r_1(1 - \frac{S}{K_s}) - \mu - \frac{\kappa_1 M}{\kappa_2} = 0$$

We solve for S in terms of R_0 and obtain,

$$\begin{aligned} S = & \quad (r_1 - \mu - \frac{\kappa_1 M}{\kappa_2})\frac{K_s}{r_1} \\ S = & \quad K_s(1 - \frac{\mu}{r_1} - \frac{[\beta - R_0(\mu + d + v)]}{r_1 \kappa_2 R_0}) \end{aligned} \tag{14}$$

Therefore, the disease-free equilibrium point is given as,

$$\left(K_s(1 - \frac{\mu}{r_1} - \frac{[\beta - R_0(\mu + d + v)]}{r_1 \kappa_2 R_0}), 0, \frac{\beta - R_0(\mu + d + v)}{\kappa_1 R_0}\right) \tag{15}$$

3.2 Basic Reproduction Number

The basic reproductive number is the number of secondary cases produced when an infected tilapia is introduced into a susceptible population Diekmann [8]. The basic reproduction number R_0 is computed using the next generation method by van den Driessche [**?**] as follows.
The disease compartment is given as

$$\frac{dI}{dt} = \frac{\beta SI}{N} - (\mu + d + v)I - \frac{\kappa_1 IM}{1 + \kappa_2 I} \tag{16}$$

where,

$$F(I) = \frac{\beta SI}{N} \tag{17}$$

$$V(I) = (\mu + d + v)I + \frac{\kappa_1 IM}{1 + \kappa_2 I} \tag{18}$$

are transmission and transition matrices respectively.
Evaluating the Jacobian of Equation 17 and Equation 18 at disease free equilibrium we get,

$$F = \frac{\partial}{\partial I}(\frac{\beta SI}{N})|_{DFE} = \beta \tag{19}$$

$$V = \frac{\partial}{\partial I}[(\mu + d + v)I + \frac{\kappa_1 IM}{1 + \kappa_2 I}]|_{DFE} = (\mu + d + v) + \kappa_1 M \tag{20}$$

Substituting Equation 19 and Equation 20 in $NGM = FV^{-1}$ we get,

$$NGM = \frac{\beta}{(\mu + d + v) + \kappa_1 M} \tag{21}$$

Simplifying further gives the spectral radius of Equation 21 as

$$R_0 = \frac{\beta r_2 \kappa_2}{r_2 \kappa_2 (\mu + d + v) + \omega \kappa_1^2 K_m + \kappa_1 \kappa_2 K_m (r_2 - \mu)} \tag{22}$$

This parameter determines whether *streptococosis*is triggered or eliminated from the population. If $R_0 < 1$, it implies that each infected tilapia infects less than one other fish, and when $R_0 > 1$, it implies that the infection spreads through the tilapia population, with the possibility of reaching an endemic level.

3.3 Local Stability of Predator-Free Equilibrium

Theorem 3.1. *The predator free equilibrium point is unstable provided,* $r_2 + \frac{\omega \kappa_1 N(\mu+d+v)}{\beta + \kappa_2 N(\mu+d+v)} - \frac{\sigma \kappa_2 I_{pf}}{1+\kappa_2 I_{pf}} - \mu > 0, \det B \geq 0$ *and* $trB > 0$*, otherwise its stable.*

Proof. Let the entries of the Jacobian evaluated at the predator-free equilibrium point$E_2 = (S, I, 0)$ be defined as;
$A_{11} = r_1 - \frac{2r_1 N(\mu+d+v)}{\beta K_s} - \mu - \frac{\beta I_{pf}}{N}$
$A_{12} = -(\mu + d + v)$
$A_{13} = -\frac{\omega \kappa_1 N(\mu+d+v)}{\beta + \kappa_2 N(\mu+d+v)}$
$A_{21} = r_1(1 - (\mu + d + v))\frac{N}{K_s \beta}$,
$A_{22} = 0$

$A_{23} = \frac{-\kappa_2 I_{pf}}{1+\kappa_2 I_{pf}}$
$A_{31} = 0$
$A_{32} = 0$
$A_{33} = r_2 + \frac{\omega\kappa_1 N(\mu+d+v)}{\beta+\kappa_2 N(\mu+d+v)} - \frac{\sigma\kappa_2 I_{pf}}{1+\kappa_2 I_{pf}} - \mu$
where I_{pf} denotes infected tilapia population when $M = 0$.
The Jacobian matrix evaluated at predator-free equilibrium point $E_2 = (S, I, 0)$ expressed as block entries is given as;

$$\mathsf{J_{PFE}} = \begin{bmatrix} A_{11} & A_{12} & A_{13} \\ A_{21} & A_{22} & A_{23} \\ 0 & 0 & A_{33} \end{bmatrix} \tag{23}$$

Since $M = 0$, the Jacobian is an upper triangular block which splits to give A_{22} as an eigenvalue of B, which is a $2x2$ matrix whose entries are given as;

$$\mathsf{B} = \begin{bmatrix} A_{11} & A_{12} \\ A_{21} & A_{22} \end{bmatrix} \tag{24}$$

The matrix B is stable if;
$trB = A_{11} + A_{22} < 0$
$\det B = A_{11}A_{22} - A_{12}A_{21} > 0$
The characteristic polynomial for Equation 23 is given as;

$$p(\lambda) = (\lambda - A_{33})(\lambda^2 - trB\lambda + \det B) \tag{25}$$

The polynomial simplifies as:

$$\begin{aligned} p(\lambda) &= \lambda^3 - (A_{11} + A_{22} + A_{33})\lambda^2 + (A_{11}A_{22} - A_{12}A_{21} + A_{33}(A_{11} + A_{22})\lambda \\ &- A_{33}(A_{11}A_{22} - A_{12}A_{21}) \end{aligned} \tag{26}$$

Equation 26 can be written as,

$$p(\lambda) = \lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 \tag{27}$$

where;

$$\begin{aligned} a_1 &= -(A_{11} + A_{22} + A_{33}) = -(trB + A_{33}) \\ a_2 &= \det B + A_{33}trB = (A_{11}A_{22} - A_{12}A_{21}) + A_{33}(A_{11} + A_{22}) \\ a_3 &= -A_{33}\det B = -A_{33}(A_{11}A_{22} - A_{12}A_{21}) \end{aligned}$$

Using the parameters provided, it follows that,

$$A_{11} > 0, A_{12} > 0, A_{21} > 0, A_{22} = 0, A_{33} > 0 \tag{28}$$

From this inequalities we can see that $trB > 0$ and $detB > 0$.

We now show that the Routh-Hurwitz criterion holds by showing that the constants a_1, a_2, a_3 in Equation 27 are positive and in addition $a_1 a_2 > a_3$. Since $A_{33} > 0$ and $detB > 0$, then $a_3 < 0$, since $trB > 0$ and $A_{33} > 0$,then $a_1 < 0$ and because $detB > 0$, $A_{33} > 0$ and $trB > 0$,then $a_2 > 0$. It can be seen that $a_1 < 0$ and $a_3 < 0$ fail to satisfy Routh- Hurwitz conditions, implying that the characteristic polynomial has a root which has a positive real part.
Therefore, the predator-free equilibrium point $E_2 = (S, I, 0)$ is unstable. □

3.4 Local stability of Disease-free Equilibrium Point

Theorem 3.2. *Disease free equilibrium point $E_3 = (S, 0, M)$ of Equation 1 is asymptotically stable if $\frac{\beta K_s(r_1-\mu)}{r_1 N}(1 - \frac{\omega\kappa_1\beta}{\kappa_2 R_0(r_1-\mu)}) - (\mu + d + v) - \frac{\kappa_1\beta}{R_0} < 0, trB < 0$ and $\det B > 0$.*

Proof. Let the entries of the Jacobian evaluated at the disease-free equilibrium point $(S, 0, M)$ be defined as;

$A_{11} = -r_1 + \mu - \frac{2\omega(\beta - R_0(\mu+d+v))}{\kappa_2 R_0} - \frac{\omega\kappa_1}{(1+\kappa_2 K_s(1-\frac{\mu}{r_1} - \frac{\omega(\beta - R_0(\mu+d+v))}{r_1\kappa_2 R_0}))^2}$

$A_{12} = -\frac{\beta K_s}{N}(1 - \frac{\mu}{r_1} - \frac{\omega C}{r_1\kappa_2 R_0})$

$A_{13} = -\frac{\omega\kappa_1 K_s(1-\frac{\mu}{r_1} - \frac{\omega C}{r_1\kappa_2 R_0})}{1+\kappa_2 K_s(1-\frac{\mu}{r_1}) - \frac{\omega C}{r_1\kappa_2 R_0}}$

$A_{22} = \frac{\beta K_s}{N}(1 - \frac{\mu}{r_1} - \frac{\omega C}{r_1\kappa_2 R_0}) - (\mu + d + v) - \frac{C}{R_0(1+\kappa_2 K_s(1-\frac{\mu}{r_1}) - \frac{\omega C}{r_1\kappa_2 R_0})^2}$

$A_{31} = \frac{\omega C}{R_0(1+\kappa_2 K_s(1-\frac{\mu}{r_1}) - \frac{\omega C}{r_1\kappa_2 R_0})^2}$

$A_{32} = \frac{\sigma(\beta - R_0(\mu+d+v))}{R_0}$

$A_{33} = r_2(1 - \frac{2C}{\kappa_1 K_m R_0} + \frac{\omega\kappa_1 K_s(1-\frac{\mu}{r_1} - \frac{\omega C}{r_1\kappa_2 R_0})}{1+\kappa_2 K_s})(1 - \frac{\mu}{r_1} - \frac{\omega C}{r_1\kappa_2 R_0}) - \mu$

where $C = \beta - R_0(\mu + d + v) > 0$.
The Jacobian matrix evaluated at the disease -free equilibrium point $E_3 = (S, 0, M)$ expressed as block entries is given as:

$$\mathsf{J_{DFE}} = \begin{bmatrix} A_{11} & A_{12} & A_{13} \\ 0 & A_{22} & 0 \\ A_{31} & A_{32} & A_{33} \end{bmatrix} \tag{29}$$

since Equation 29 has middle row zeros except A_{22}, the characteristic polynomial factors as

$$p(\lambda) = (\lambda - A_{22}) \det(\lambda I - B) \tag{30}$$

Where B is a 2×2 matrix whose entries are given as;

$$\mathsf{B} = \begin{bmatrix} A_{11} & A_{13} \\ A_{31} & A_{33} \end{bmatrix}$$

We simplify the characteristic polynomial $p(\lambda)$ to obtain;

$$p(\lambda) = \lambda^3 - (A_{11} + A_{22} + A_{33})\lambda^2 + (\det B + A_{22}trB)\lambda - A_{22}\det B \tag{31}$$

which can be expressed as;

$$p(\lambda) = \lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 \tag{32}$$

where;
$a_1 = -(trB + A_{22})$
$a_2 = \det B + A_{22}trB$
$a_3 = -A_{22}\det B$
Using the parameters provided, it follows that,

$$A_{11} > 0, A_{13} > 0, A_{31} > 0, A_{22} < 0, A_{33} < 0 \tag{33}$$

we now show that the Routh-Hurwitz criterion for stability $a_1 > 0, a_2 > 0, a_3 > 0$, and $a_1a_2 > a_3$ are satisfied.
Using these conditions, the $trB < 0$, consequently $a_1 > 0$. The determinant of B follows directly from the conditions on A_{11}, A_{31}, A_{33} and A_{31}, $\det B > 0$. This implies that $a_2 = \det B + A_{22}trB > 0$, $a_3 = -A_{22}\det B > 0$ and $a_1a_2 > a_3 > 0$ are satisfied.
Therefore, the DFE point $E_3 = (S, 0, M)$ is asymptotically stable. □

3.5 Local stability of Endemic Equilibrium Point

Theorem 3.3. *The endemic equilibrium point $E_4(S^*, I^*, M^*)$ is locally stable if $A = A_{11}+A_{22}+A_{33} = trJ_{EE} < 0$, $B = A_{11}A_{22} - A_{12}A_{21} + A_{11}A_{33} - A_{13}A_{31} + A_{22}A_{33} - A_{23}A_{32} > 0$,and $C = A_{11}A_{22}A_{33} + A_{12}A_{23}A_{31} + A_{13}A_{21}A_{32} - A_{11} < 0$.*

Proof. Let the entries of the Jacobian evaluated at the Endemic equilibrium point (S^*, I^*, M^*) be defined as;
$A_{11} = r_1(1 - \frac{2S^*}{K_s}) - \mu - \frac{\omega\kappa_1 M^*}{(1+\kappa_2 S^*)^2}$
$A_{12} = -\frac{\beta S^*}{N}$
$A_{13} = -\frac{\omega\kappa_1 S^*}{1+\kappa_2 S^*}$
$A_{21} = \frac{\beta I}{N}$
$A_{22} = \frac{\beta S^*}{N} - (\mu + d + v) - \frac{\kappa_1 M^*}{(1+\kappa_2 I^*)^2}$
$A_{23} = \frac{\kappa_1 I^*}{1+\kappa_2 I^*}$
$A_{31} = \frac{\omega\kappa_1 M^*}{(1+\kappa_2 S^*)^2}$
$A_{32} = \frac{-\sigma\kappa_1 M^*}{(1+\kappa_2 I^*)^2}$
$A_{33} = r_2(1 - \frac{2M^*}{K_m}) + \frac{\omega\kappa_1 S^*}{1+\kappa_2 S^*}) - \frac{\sigma\kappa_1 I^*}{1+\kappa_2 I^*} - \mu$

Let the Jacobian matrix evaluated at the endemic equilibrium point $E_4 = (S^*, I^*, M^*)$ be defined as,

$$\mathsf{J}_{\mathsf{EE}} = \begin{bmatrix} A_{11} & A_{12} & A_{13} \\ A_{21} & A_{22} & A_{23} \\ A_{31} & A_{32} & A_{33} \end{bmatrix} \tag{34}$$

The characteristic polynomial for 34 is given as;

$$\begin{aligned} P(\lambda) &= \lambda^3 - (A_{11} + A_{22} + A_{33})\lambda^2 \\ &+ (A_{11}A_{22} - A_{12}A_{21} + A_{11}A_{33} - A_{13}A_{31} + A_{22}A_{33} - A_{23}A_{32})\lambda \\ &- (A_{11}A_{22}A_{33} + A_{12}A_{23}A_{31} + A_{13}A_{21}A_{32} - A_{11}A_{23}A_{32} - A_{12}A_{21}A_{33} - A_{13}A_{22}A_{31}) \\ P(\lambda) &= \lambda^3 - A\lambda^2 - B\lambda - C \end{aligned} \tag{35}$$

where;
$A = A_{11} + A_{22} + A_{33} = trJ_{EE}$
$B = A_{11}A_{22} - A_{12}A_{21} + A_{11}A_{33} - A_{13}A_{31} + A_{22}A_{33} - A_{23}A_{32}$
$C = A_{11}A_{22}A_{33} + A_{12}A_{23}A_{31} + A_{13}A_{21}A_{32} - A_{11}A_{23}A_{32} - A_{12}A_{21}A_{33} - A_{13}A_{22}A_{31}$

We now show that the Routh-Hurwitz criterion holds by showing that $A < 0, B > 0, C < 0$ and $AB > C$.
We show that $A < 0$ by defining the variables involved for, instance it is important to note that $A_{22} = \frac{\beta S}{N} - (\mu + d + v) - \frac{\kappa_1 M^*}{(1+\kappa_2 I)^2} < 0$,
$A_{11} < 0$ because of prey density regulation and mortality.

We require that the predator net growth is small to make the overall negative; therefore, we expect $A_{33} < (A_{11} + A_{22})$.
This is a sufficient condition for $A < 0$.
We now show that $B > 0$ by writing the three terms of B as:
$B_1 = A_{11}A_{22} - A_{12}A_{21}$
$B_2 = A_{11}A_{33} - A_{13}A_{31}$
$B_3 = A_{22}A_{33} - A_{23}A_{32}$
We can simplify the sign of B_1 using the sign of $A_{12}A_{21}$:
$B_1 = A_{11}A_{22} + \frac{\beta^2 S^* I^*}{N^2}$.
Since $A_{11}A_{22} > 0$ then $B_1 > 0$. A sufficient condition for $B > 0$ is that the sum of the three B_i terms be positive.

Next we show that the determinant $C < 0$,
$C = A_{11}A_{22}A_{33} + A_{12}A_{23}A_{31} + A_{13}A_{21}A_{32} - A_{11}A_{23}A_{32} - A_{12}A_{21}A_{33} - A_{13}A_{22}A_{31}$
The principal diagonal product $A_{11}A_{22}A_{33} < 0$ since we have shown that $A_{11} < 0, A_{22} < 0, A_{33} < 0$. The other mixed cubic terms are not large compared to the diagonal terms. Thus a sufficient condition for $C < 0$ is:

$A_{11} < 0, A_{33} < 0$ and the magnitude of $A_{11}A_{22}A_{33}$ exceeds the sum of the other mixed terms. Lastly, the last condition $AB < C$ follows directly from the signs of A, B, C shown above. Thus, the endemic equilibrium point $E_4 = (S^*, I^*, M^*)$ is locally asymptotically stable.

The endemic equilibrium point $E_4 = (S^*, I^*, M^*)$ is a positive solution, which shows that the interacting species coexist with each other even if the infection persists.

□

3.6 Optimal Control Analysis

Based on the work done by Samares [2] and Lenhart [16], we formulate an optimal control problem with an appropriate objective functional given by,

$$J(v(t)) = \int_0^T (a_1 S(t) + a_2 I(t) + a_3 M(t) + b_1 v^2(t))dt \tag{36}$$

where a_1, a_2, and a_3 are relative weights representing the importance of susceptible tilapia, infected tilapia, and mudfish population, respectively, whereas b_1 is the weight representing the cost and potential side effects of the treatment strategy for infected tilapia. An assumption that the cost of intervention is non-linear and quadratic is made when formulating the objective functional. The term $b_1 v^2(t)$ represents the cost of treatment of infected tilapia.
We aim to find optimal levels of the control measure needed to minimise disease prevalence as well as the cost of implementing the intervention while maximizing the tilapia population. The integrand in Equation 36 is the Lagrangian for the optimal control problem, which is given by,

$$L(S, I, M, U) = (a_1 S(t) + a_2 I(t) + a_3 M(t) + b_1 v^2(t)) \tag{37}$$

where $v \subset U$
We now define our Hamiltonian function H for the control problem Equation 36 as,

$$\begin{aligned} H(S, I, M, U) &= (a_1 S(t) + a_2 I(t) + a_3 M(t) + b_1 v^2(t)) \\ &+ \lambda_1 (r_1 S(1 - \frac{S}{K_s}) - \mu S - \frac{\beta SI}{N} - \omega \frac{\kappa_1 SM}{1 + \kappa_2 S}) \\ &+ \lambda_2 (\frac{\beta SI}{N} - (\mu + d + v)I - \frac{\kappa_1 IM}{1 + \kappa_2 I}) \\ &+ \lambda_3 (\omega \frac{\kappa_1 SM}{1 + \kappa_2 S} - \sigma \frac{\kappa_1 IM}{1 + \kappa_2 I} - \mu M + r_2 M(1 - \frac{M}{K_m})) \end{aligned} \tag{38}$$

where $\lambda_1.\lambda_2, \lambda_3$ are adjoint variables.
We now need to determine the optimal control function v^* that is in the optimal set $\{U\}$ so that,

$$J(v^*) = minJ(v)|(v) \subset \{U\} \tag{39}$$

subject to the dynamical system in Equation 1 and the set $\{U\}$ is given by
$\{U\} = v(t) : 0 \leq v_i \leq v_i^{max}$. The control set $\{U\}$ is Lebesgue measurable, piecewise continuous on $[0, T]$, and bounded. Using our optimal control problem in Equation 36 and the Hamiltonian in Equation 38, we prove that the optimal control exists and later on characterize the control using the optimality system.

3.6.1 Existence of Optimal Control

Theorem 3.4. *The optimal control exists for $v^* \subset U$ such that*
$J(v^*) = min_{v \in U} J(v)$
subject to the control system 1 with its initial conditions.

Proof. Using the results by Fleming [10], the existence of an optimal pair of the system Equation 1 can be determined. In their work, boundedness of the state and control implies compactness, which then implies existence and convexity.

Since we demonstrated that the state variables S, I, M, and control v in Equation 1 are positive, the necessary condition on convexity of the objective functional is satisfied. Further, by definition, the set $\{U\}$ of control v^* is closed and convex. We established earlier that our optimal system is bounded. This boundedness implies compactness of the control problem, which is a requirement for the existence of an optimal control.

We verify that the integrand is convex, The integrand is given as, $L(S, I, M, U) = a_1 S + a_2 I + a_3 M + b_1 v^2$

We must verify that there exist a constant $\omega > 1$ and positive constant η_1 and η_2 such that,

$$\begin{aligned} L(S, I, M, U) &\geq \eta_1 (|v|^2)^{\frac{\omega}{2}} - \eta_2 \\ a_1 S + a_2 I + a_3 M + b_1 v^2 &\geq b_1 v^2 \end{aligned} \tag{40}$$

and

$$b_1 v^2 \geq \eta_1 (|v|^2)^{\frac{\omega}{2}} - \eta_2 \tag{41}$$

This condition is satisfied when $\omega = 2$ and $\eta_2 > 0$. □

3.7 Characterization of Optimal Control

Theorem 3.5. *Given an optimal control* (v^*) *and* S^*, I^*, M^* *the solution of the corresponding system Equation 1 minimizing* $J(v)$ *over* U*. Then there exist costate variables satisfying,*

$$\begin{aligned}
\frac{\partial \lambda_1}{\partial t} &= -\frac{\partial H}{\partial S} = -(\lambda_3 - \lambda_1)\frac{\omega \kappa_1 M}{(1+\kappa_2 S)^2} - (\lambda_2 - \lambda_1)\frac{\beta I}{N} + \mu\lambda_1 + \lambda_1 \frac{2r_1}{K_s} - \lambda_1 r_1 - a_1 \\
\frac{\partial \lambda_2}{\partial t} &= -\frac{\partial H}{\partial I} = (\sigma\lambda_3 - \lambda_2)\frac{\kappa_1 M}{(1+\kappa_2 I)^2} - (\lambda_2 - \lambda_1)\frac{\beta S}{N} + \lambda_2(\mu + d + v) - a_2 \\
\frac{\partial \lambda_3}{\partial t} &= -\frac{\partial H}{\partial M} = -(\lambda_3 - \lambda_1)\frac{\kappa_1 S}{1+\kappa_2 S} - (\sigma\lambda_3 - \lambda_2)\frac{\kappa_1 I}{1+\kappa_2 I} + \lambda_3\mu - \lambda_3 r_2 + 2\lambda_3 \frac{r_2 M}{K_m} - a_3
\end{aligned} \tag{42}$$

with final conditions, $\lambda_1(T) = 0, \lambda_2(T) = 0, \lambda_3(T) = 0$*, for* $0 \le t \le T$*.*
Optimality condition v^* *is given by,*

$$v^* = \begin{cases} \frac{\lambda_2 I^*}{2b_1} & \text{if } 0 \le \frac{\lambda_2 I^*}{2b_1} \le 1. \\ 0 & \text{if } \frac{\lambda_2 I^*}{2b_1} \le 0 \\ 1 & \text{if } \frac{\lambda_2 I^*}{2b_1} \ge 1 \end{cases} \tag{43}$$

Proof. The differential equations governing the costate variables are obtained by differentiating the Hamiltonian function $H(S, I, M, U)$ Equation 38 and evaluating the system at the optimal control. The costate system can be written as

$$\begin{aligned}
\frac{\partial \lambda_1}{\partial t} &= -\frac{\partial H}{\partial S} = -(\lambda_3 - \lambda_1)\frac{\omega \kappa_1 M}{(1+\kappa_2 S)^2} - (\lambda_2 - \lambda_1)\frac{\beta I}{N} + \mu\lambda_1 + \lambda_1 \frac{2r_1}{K_s} - \lambda_1 r_1 - a_1 \\
\frac{\partial \lambda_2}{\partial t} &= -\frac{\partial H}{\partial I} = (\sigma\lambda_3 - \lambda_2)\frac{\kappa_1 M}{(1+\kappa_2 I)^2} - (\lambda_2 - \lambda_1)\frac{\beta S}{N} + \lambda_2(\mu + d + v) - a_2 \\
\frac{\partial \lambda_3}{\partial t} &= -\frac{\partial H}{\partial M} = -(\lambda_3 - \lambda_1)\frac{\kappa_1 S}{1+\kappa_2 S} - (\sigma\lambda_3 - \lambda_2)\frac{\kappa_1 I}{1+\kappa_2 I} + \lambda_3\mu - \lambda_3 r_2 + 2\lambda_3 \frac{r_2 M}{K_m} - a_3
\end{aligned} \tag{44}$$

with transversality conditions $\lambda_1(T) = 0, \lambda_2(T) = 0, \lambda_3(T) = 0$, for $0 \le t \le T$. In the interior of the control set U, where $0 \le v(t) \le v_{max} < \infty$, we have the optimality condition given by

$$\frac{\partial H}{\partial v} = 2b_1 v - \lambda_2 I = 0 \tag{45}$$

solving the optimality condition in Equation 45 $v(t)^*$, we get

$$v(t)^* = \frac{\lambda_2 I^*}{2b_1} \tag{46}$$

Using the bounds of the control $v(t)^*$, its optimal control is given by

$$v^* = \begin{cases} \frac{\lambda_2 I^*}{2b_2} & \text{if } 0 \leq \frac{\lambda_2 I^*}{2b_1} \leq 1. \\ 0 & \text{if} \frac{\lambda_1 I^*}{2b_1} \leq 0 \\ 1 & \text{if } \frac{\lambda_2 I^*}{2b_1} \geq 1 \end{cases} \tag{47}$$

$$v^* = min\left(max\left(\frac{\lambda_2 I^*}{2b_1}, 0\right).v_{max}\right)$$

The Equations 1, 42, together with the optimality conditions, initial conditions, and final time conditions, give the optimality system. □

4 Numerical Simulation

Numerical simulations were carried out to graphically illustrate the effect of treatment on disease transmission and population dynamics in an aquatic environment involving tilapia and mudfish. To ensure that the simulations are realistic, estimated and assumed parameters that accurately represent the ecological interactions between tilapia and mudfish are used. The parameters were used as shown in the table.The parameters were used as shown in the table. The parameter $\beta = 0.2$ was used as a baseline parameter, as β increases the infected tilapia population increases as the susceptible tilapia decrease. The simulations under optimal control were performed using $S_0 = 300, I_0 = 50$ and $M_0 = 30$ as the initial conditions.

Table 5.1
Model parameters

Parameters	Description	Value	Justification
r_1	Intrinsic growth rate of susceptible prey	$0.8year^{-1}$	Assumed.
r_2	Intrinsic growth rate of predator	$0.4year^{-1}$	Assumed.
K_s	Prey carrying capacity	1000 tilapia	Estimate.
K_m	Predator carrying capacity	300 mudfish	Estimate.
μ	Natural mortality rate of species	0.01 $year^{-1}$	Assumed.
d	Disease induced death rate of infected prey	$0.05year^{-1}$	Assumed.
β	Transmission rate of infection	0.2 contacts $year^{-1}$	Assumed.
ω	Conversion efficiency	0.1 predator $prey^{-1}$	Assumed.
υ	Treatment effectiveness	0.2 prey $year^{-1}$	Assumed.
κ_1	Predator search rate	0.005prey $predator^{-1}year^{-1}$	Assumed.
κ_2	Predator satiety rate	0.5 prey $predator^{-1}year^{-1}$	Assumed.
σ	Side effects of feeding on infected prey	0.05 predators $prey^{-1}year^{-1}$	Assumed.

4.1 Simulation of Equilibrium points

From Figure 2, the disease remains endemic in the absence of predators. This state is driven by the consumption of infected prey, which becomes toxic to the predators. This equilibrium point represents a scenario of predator extinction, which occurs as a result of the toxicity of infected prey.

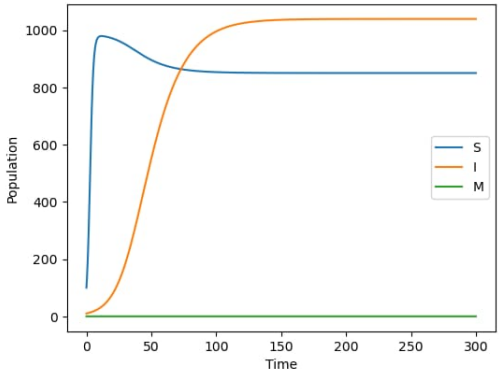


Figure 2: Predator-Free Equilibrium

Figure 3 indicates that as the disease dies out, the number of susceptible prey in the population increases over time. The disease-free equilibrium point represents a healthy ecosystem where tilapia survive and reproduce normally as the predator population adjusts depending on the prey availability.

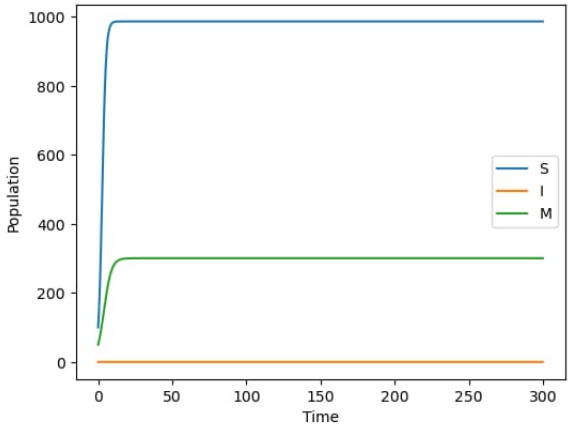


Figure 3: Disease Free Equilibrium

Figure 4 shows that even with the presence of the disease, the interacting species approaches a particular stable point away from the origin, indicating that the population remains positive with time. This is a coexistence equilibrium point, depicting that the disease cannot be completely eradicated, but the populations adjust and coexist with each other.

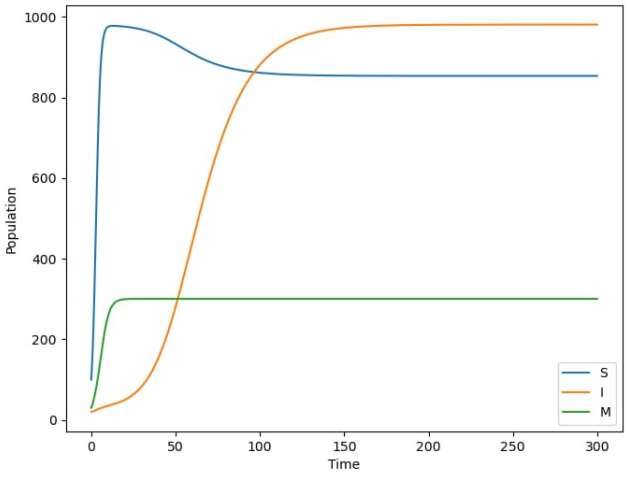
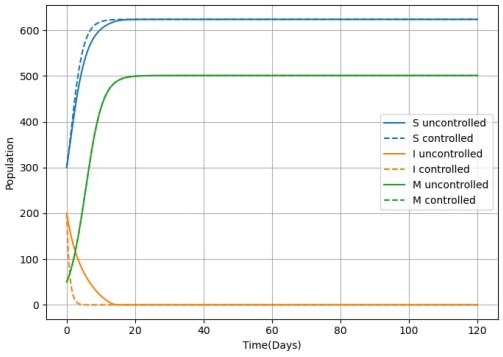


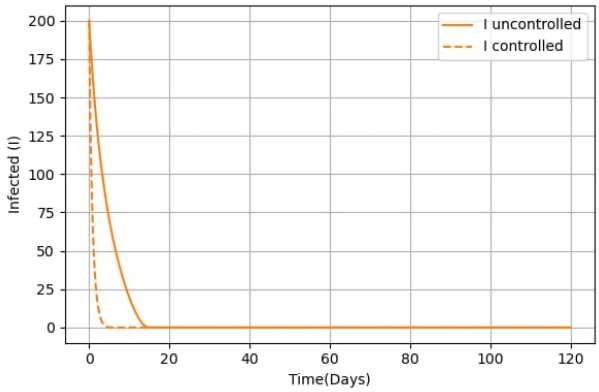
Figure 4: Endemic Equilibrium

4.2 Simulation of Model 1 with and without control parameters

From Figure 5a and Figure 5b, we see that the susceptible tilapia rise under control but decline without control; the infected tilapia rise sharply without control but remain low when the control is used; and many mudfish rise under control and fluctuate without control. These figures imply that without treatment, infection reduces the biomass of the prey, destabilising the predator-prey interactions. Treatment, on the other hand, results in healthy prey, leading to a more stable predator. In the uncontrolled scenario, infection rises and peaks; treatment suppresses it. The parameter $\beta = 0.2$ was an assumed parameter for numerical simulation



(a) Graphical representation of the effect of control on the populations $\beta = 0.2$



(b) Effect of control on Infected populations $\beta = 0.2$

Figure 5: Effect of Control

4.3 Simulation of optimal control profile $v^*(t)$ over time

In this section, we perform a simulation of an optimal control profile over time to provide insight into how control efforts should be administered to achieve the desired outcome. The time-dependent control obtained illustrates when and how strongly treatment should be applied in the simulation, informing us when the control is most effective.

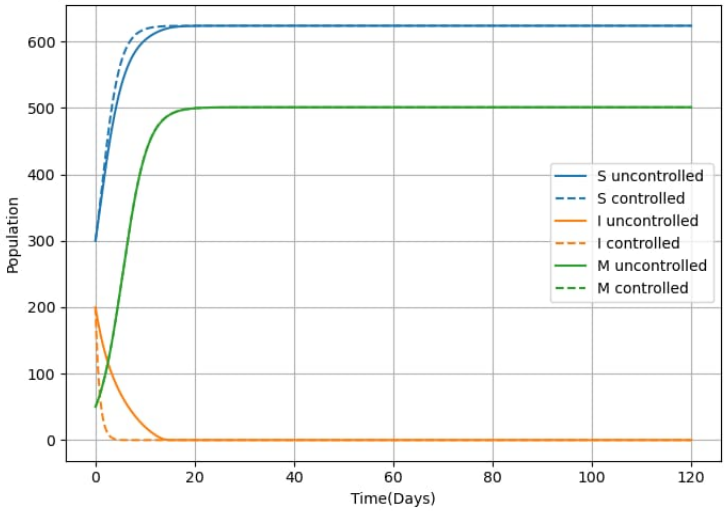


Figure 6: Graphical representation of the optimal control profile

From Figure 6, we can see that treatment effort $v(t)$ rises at initial states of the outbreak, and the control effort $v(t)$ decreases towards zero as the infection decreases. This profile has ecological meaning; for instance, better treatment at the onset of streptococcus disease is cost-effective and prevents the growth of the disease to endemic levels.

5 Conclusion and Recommendations

5.1 Conclusion

This study evaluated a well-posed tilapia–mudfish predator–prey model with disease dynamics. We derived R_0 using the next-generation matrix, proving stability for axial, disease-free, and endemic equilibria, while trivial and predator-free states remained unstable. Application of Pontryagin's Maximum Principle demonstrated that optimal treatment elevates susceptible prey populations and suppresses infection. Rather than attempting complete pathogen or predator eradication, sustainable aquaculture management must maintain $R_0 < 1$ and foster controlled coexistence to balance ecosystem integrity with economic productivity.

5.2 Recommendations

Our research explored how prey growth, predator-prey relationships, and disease transmission interact within tilapia and mudfish aquaculture. Building on this work, future studies could incorporate critical environmental drivers like water quality, pH, and oxygen levels to better mirror actual farming conditions. Expanding the framework to feature multiple predator species and novel transmission networks would reveal richer ecological dynamics. Additionally, accounting for stochastic processes would better reflect random shifts in fish stocks and disease spillovers. Finally, testing strategies like

vaccination or selective breeding—and modeling multi-cage patch networks with moving populations—would significantly broaden the practical value of this approach.

Declaration of Interest

The authors declare that they do not have any known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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References

[1] Amal, M. N. A., & Zamri-Saad, M. (2011). Streptococcosis in tilapia (*Oreochromis niloticus*): A review. *Pertanika Journal of Tropical Agricultural Science, 34*(2), 195–206.

[2] Anal, C. S., & P. (2023). A predator-prey model for the optimal control of fish harvesting through imposition of a tax. *An International Journal of Optimization and Control: Theories and Applications, 13*(1), 68–80. https://doi.org/10.11121/ijocta.2023.1218

[3] May, R. M., & Anderson, R. M. (1978). Regulation and stability of host-parasite population interactions. II. Destabilizing processes. *Journal of Animal Ecology, 47*(1), 249–267. https://doi.org/10.2307/3934

[4] Bezabih, A. F., Edessa, G. K., & Koya, P. R. (2021). Ecoepidemiological model and analysis of prey-predator system. *Journal of Applied Mathematics, 2021*, Article 6679686, 1–17. https://doi.org/10.1155/2021/6679686

[5] Bera, S. P., Maiti, A., & Samanta, G. P. (2015). A prey-predator model with infection in both prey and predator. *Filomat, 29*(8), 1753–1767. https://doi.org/10.2298/FIL1508753B

[6] Chattopadhyay, J., & Arino, O. (1999). A predator-prey model with disease in the prey. *Nonlinear Analysis: Theory, Methods & Applications, 36*(6), 747–766. https://doi.org/10.1016/S0362-546X(98)00126-6

[7] Cojocaru, M.-G., Migot, T., & Jaber, A. (2020). Controlling infection in predator-prey systems with transmission dynamics. *Infectious Disease Modelling, 5*, 1–11. https://doi.org/10.1016/j.idm.2019.12.002

[8] Diekmann, O., Heesterbeek, J. A. P., & Metz, J. A. J. (1990). On the definition and computation of the basic reproduction ratio R_0 in models for infectious diseases in heterogeneous populations. *Journal of Mathematical Biology, 28*(4), 365–382.

[9] Amalia, R. U. D., Fatmawati, Windarto, & Arif, D. K. (2018). Optimal control of predator-prey mathematical model with infection and harvesting on prey. *Journal of Physics: Conference Series, 974*(1), Article 012050. https://doi.org/10.1088/1742-6596/974/1/012050

[10] Fleming, W. H., & Rishel, R. W. (1975). Stochastic differential equations and Markov diffusion processes. In *Deterministic and stochastic optimal control* (pp. 106–150). Springer.

[11] Hadeler, K. P., & Freedman, H. I. (1989). Predator-prey populations with parasitic infection. *Journal of Mathematical Biology, 27*(6), 609–631. https://doi.org/10.1007/BF00276947

[12] Kebedow, K. G., Cheru, S. L., & Ega, T. T. (2025). Optimal control strategies in a diseased prey-predator model with Holling type-II dynamics. *Journal of Applied Mathematics, 2025*, Article 5535536. https://doi.org/10.1155/jama/5535536

[13] Klempner, M. S., & Shapiro, D. S. (2004). Crossing the species barrier—one small step to man, one giant leap to mankind. *New England Journal of Medicine, 350*, 1171–1172.

[14] Das, K. P. (2011). A mathematical study of a predator-prey dynamics with disease in predator. *ISRN Applied Mathematics, 2011*, Article 807486, 1–16. https://doi.org/10.5402/2011/807486

[15] Lashari, A. A. (2016). Optimal control of an SIR epidemic model with a saturated treatment. *Applied Mathematics & Information Sciences, 10*(1), 185–191. https://doi.org/10.18576/amis/100117

[16] Lenhart, S., & Workman, J. T. (2007). *Optimal control applied to biological models*. Chapman & Hall/CRC.

[17] Mukhopadhyay, B., & Bhattacharyya, R. (2010). On an eco-epidemiological model with prey harvesting and predator switching: Local and global perspectives. *Nonlinear Analysis: Real World Applications, 11*(5), 3824–3833.

[18] Pramanick, S., Bhattacharyya, J., & Pal, S. (2020). A prey-predator model with pathogen infection on predator population. In R. P. Mondaini (Ed.), *Trends in biomathematics: Modeling cells, flows, epidemics, and the environment* (pp. 275–297). Springer. https://doi.org/10.1007/978-3-030-46306-9_18

[19] Shoemaker, C. A., Klesius, P. H., & Evans, J. J. (2001). Prevalence of *Streptococcus iniae* in tilapia, hybrid striped bass, and channel catfish on commercial fish farms in the United States. *American Journal of Veterinary Research, 62*(2), 174–177. https://doi.org/10.2460/ajvr.2001.62.174

[20] Venturino, E. (2001). Effect of disease on competing species. *Mathematical Biosciences, 174*(1), 111–131.