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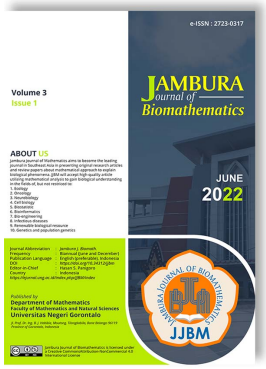

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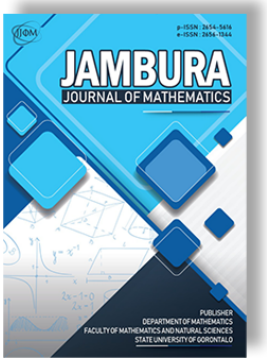

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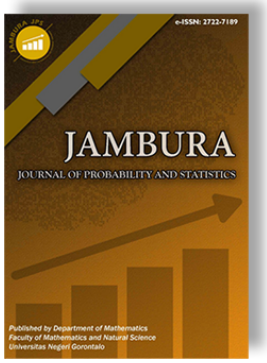

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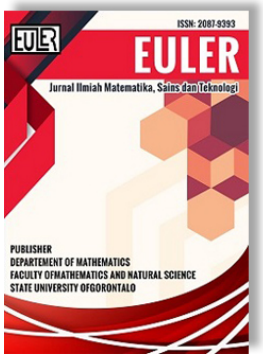

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Research Article

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A Stage-structure Leslie-Gower Model with Linear Harvesting and Disease in Predator

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ABSTRACT. *The growth dynamics of various species are affected by various aspects. Harvesting interventions and the spread of disease in species are two important aspects that affect population dynamics and it can be studied. In this work, we consider a stage-structure Leslie–Gower model with linear harvesting on the both prey and predator. Additionally, we also consider the infection aspect in the predator population. The population is divided into four subpopulations: immature prey, mature prey, susceptible predator, and infected predator. We analyze the existences and stabilities of feasible equilibrium points. Our results shown that the harvesting in prey and the disease in predator impacts the behavioral of system. The situation in the system is more complex due to disease in the predator population. Some numerical simulations are given to confirm our results.*

1. Introduction

In ecological science, the behavioural of species interaction is an essential. One of other factors that impact the reciprocal influence of predator and prey is stage-structure, referring to a size of body species. Regarding to the reason, some researchers have successfully developed and analyzed the stage-structure problem in the species population based on the mathematical model. For examples: Chen and Shi [1], Zhao and Yuan [2], Ma et al. [3] and Liu et al. [4] study dynamics of the stage-structure Lotka-Volterra model. Due to the stage-structure for interaction between prey and predator, Beay and Saija [5], Beay et al. [6], and Moustafa et al. [7] studied the dynamics behavior by using Rosenzweig-MacArthur model. Additionally, the dynamics of the stage-structure Leslie-Gower model studied by Lin et al. [8], Pratiwi et al. [9], Beay and Saija [10], Meng et al. [11], and Salamah et al. [12].

In fact, harvesting intervention could be influence the behaviour of population dynamics of species. By considering stage-structure in species, several works are done. Song and Chen in [13] analyzed optimal harvesting and effect of delay. Next, applying the Monod-Haldane type of functional response and stage-structure for predator species investigated by Pratama et al. [14]. The harvesting on prey species and Allee effect on predator species is introduced and studied by Li et al. [15]. Using the Beddington–DeAngelis functional response and harvesting is worked by Li and Cheng [16]. The combination of the effects of linear harvesting in prey and cannibalism in predator for a Rosenzweig–MacArthur model has studied by Beay and Saija [17], and others problem [18, 19].

On other side, some researchers have investigated the effects of disease on a predator-prey system with stage-structure. Infected population and the death due to disease will be change the number of a species. Mathematically, the disease effect is very interest for studied. In 2019, Majeed studied the impacts of disease on a stage-structured predator-prey model including prey refuge and harvesting [20]. Next, the effects of disease in prey and stage-structure in predator was analyzed by Babakordi and Zangeneh [21]. Kafi and Majeed have studied the dynamical analysis of stage-structure model with disease and refuse in prey [22]. In 2021, Sundari and Valliathal studied the stability of an epidemiological stage-structure system considering Michaelis–Menten funsional response and prey refuge [23]. Due to the application, by using ecoepidemiological SIN model, Kalra and Kaur have studied the stage-structure in predator and control for pest management [24].

Realizing the important role of harvesting and disease that can threaten the existence of species, a study of the interaction behavior of species is carried out mathematically. The model studied takes into account the stage-structure. In addition, the spread of disease among predator species is also a concern in this research. On the other hand, the roles of harvesting parameters on immature prey and susceptible predators that have commercial value are a special subject to be studied. A Leslie-Gower model that integrates the stage-structure in prey, harvesting for immature prey and susceptible predators, as well as the spread of disease in predators is the novelty in our research.

In this paper, we extended the system model (1) in [10]. We considering linear harvesting in the immature prey and susceptible predator. Next, we involving the infected predator in the system. More detail, we organize this paper as follows. In

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Table 1. Description of variable and paremeter

Parameter	Biological interpretation
r	The intrinsic growth rate
c	The growth rate of predator
K	The environmental carrying capacity
v	The maximum value of the per capita reduction rate of predator
n	The environmental protection for immature prey
α	A conversion rate of the immature prey into mature prey
β	The transmission coefficient from susceptible predator to infected predator
δ	The death rate of mature prey
μ	The death rate of predator
k_1	A harvesting rate of immature prey
k_2	A harvesting rate of predator

Section 2, we will show how to construct the a stage-structure Leslie–Gower model considering linear harvesting and infected predator. Furthermore, the fixed point and local stability are investigated in Section 3. In Section 4, we carry out the numerical simulations. Finally, we reach conclusions about our work in Section 5.

2. Model Formulation

In this section, we propose the novelty of our model, i.e. the modification of the stage–structure Leslie-Gower model with linear harvesting and disease in predator. Further we modify the model in [10] as follows:

$$\begin{aligned} \dot{x_1} &= rx_2\left(1-\frac{x_1+x_2}{K}\right)-\alpha x_1-k_1x_1-\frac{vx_1y_1}{n+x_1}, \\ \dot{x_2} &= \alpha x_1-\delta x_2, \\ \dot{y_1} &= cy_1\left(1-\frac{y_1+y_2}{n+x_1},\right)-\beta y_1y_2-k_2y_1, \\ \dot{y_2} &= \beta y_1y_2-\mu y_2, \end{aligned} \tag{1}$$

where: x_1 and x_2 are the populations of immature and mature prey at time t, Next, y_1 and y_2 are the populations of susceptible predator and infected predator at time t. The following assumptions are taken in deriving model (1):

(A1) The growth rate of the immature prey depends entirely on the reproduction by the mature prey, where the growth is assumed to be logistically with constant intrinsic rate r and constant carrying capacity $K > 0$.

(A2) The immature prey is more vulnerable so predators only consume immature prey. The predation mechanism is nonlinear form based on the Holling Type II functional response, where the maximum predation rate is v and the environmental protection for immature prey is denoted by n.

(A3) The immature prey have prospects with the rate of harvesting k_1. Additionally, the immature prey grow up and turn into mature prey with a conversion rate α.

(A4) The growth of mature prey population depends only on the conversion of immature prey into mature prey. The mature prey do not have a risk to be attacked by predator. The death rate of the mature prey population is δ.

(A5) The predator consumes only the immature prey and the growth rate of predator population is assumed to be logistically with constant intrinsic rate c. Next, the susceptible predators have prospects with the rate of harvesting k_2.

(A6) The number of susceptible predators will decrease due to infection with rate β, and the disease causes predatory species to die with a rates μ.

System (1) describes a predator-prey interaction where the susceptible predator only consumes immature prey. One ecological example of such a predator-prey system is the interaction between Moluccan megapode (Eulipoa wallacei) and cats Felis Catus. Felis Catus is reported to prey upon Eulipoa eggs and chicks, and it not easy to attack adult Eulipoa, see [25]. On other side, cats as predator can be infected Feline Leukimia Virus. Continuously, the transmission process serve as sources of infection for other cats. The virus is shed in saliva, nasal secretions, urine, feces, and milk of infected cats. Cat-to-cat transfer of the virus may occur from a bite wound [26]. In addition, cat meat has the prospect of being commercialized. This encourages human hunting for wild cats [27]. For more clarity, the compartment diagram of the model is represented in Figure 1. Next, we explain the all variables and parameters in system (1) by Table 1.

3. Existence of Equilibrium Points and Basic Reproductive Number

3.1. Existence of Equilibrium Points

It is easy to show that system (1) has six non-negative equilibrium points as follows.

- Equilibrium point $P_0 = (0, 0, 0, 0)$, which all species has gone to extinction.
- Equilibrium point

$$P_1 = \left(\frac{\delta K(r\alpha-\delta(\alpha+k_1))}{r\alpha(\alpha+\delta)}, \frac{K(r\alpha-\delta(\alpha+k_1))}{r(\alpha+\delta)}, 0, 0\right),$$

  provided $r\alpha > \delta(\alpha + k_1)$, which there is no predator population in the ecosystem.
- Equilibrium point $P_2 = \left(0, 0, \frac{\mu}{\beta}, \frac{\beta cn-(c\mu+\beta nk_2)}{\beta(\beta n+c)}\right)$, provided $\beta cn > (c\mu + \beta nk_2)$, which there is no prey population in the ecosystem.
- Equilibrium point $P_3 = \left(0, 0, \frac{n(c-k_2)}{c}, 0\right)$, provided $c > k_2$, which there is no prey population in the ecosystem and free disease.

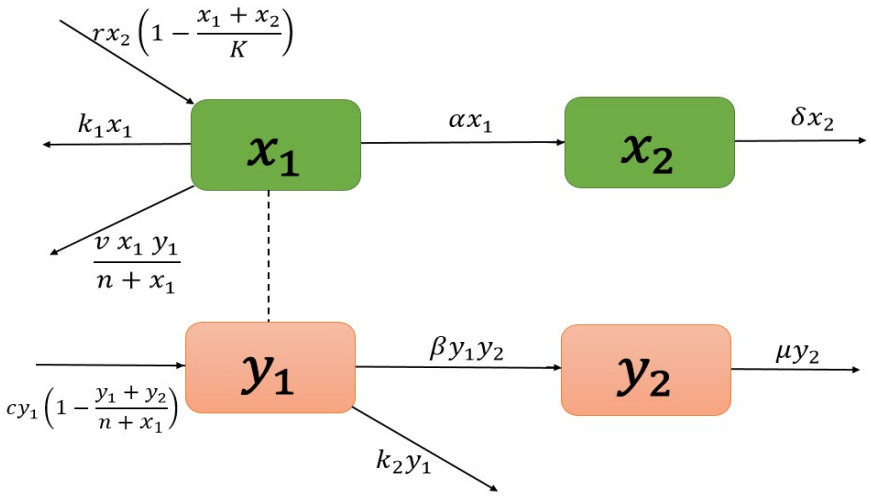


Figure 1. Compartment diagram of the model (1) described the growth of immature prey, mature prey, susceptible predator, and infected predator

- Equilibrium point $P_4 = (x_{14}, x_{24}, y_{14}, 0)$ where:

$$\begin{aligned} x_{14} &= \frac{K\delta([k_2-c]\delta v+\alpha cr-c\delta[\alpha+k_1])}{\alpha cr(\alpha+\delta)}, \\ x_{24} &= \frac{K([k_2-c]\delta v+\alpha cr-c\delta[\alpha+k_1])}{cr(\alpha+\delta)}, \\ y_{14} &= \frac{1}{(\alpha+\delta)c^2r\alpha}\left([k_2-c][K\delta^2(v+c(\alpha+k_1))\right. \\ &\quad \left.-(\alpha cr(n(\delta+\alpha)+K\delta))]\right), \end{aligned}$$

provided $k_2 > c$, $\alpha cr > c\delta[\alpha + k_1]$, and $K\delta^2(v + c(\alpha + k_1)) > (\alpha cr(n(\delta + \alpha) + K\delta))$,
which there is no infected predator in the ecosystem.

- The interior equilibrium $P_* = (x_1^*, x_2^*, y_1^*, y_2^*)$, where:

$$\begin{aligned} x_2^* &= \frac{\alpha}{\delta}x_1^*, \\ y_1^* &= \frac{\mu}{\beta}, \\ y_2^* &= \frac{\mu(M^*-m_1)}{\beta(m_2+M^*)}. \end{aligned}$$

where:

$$\begin{aligned} M^* &= x_1^*\alpha cr(\alpha+\delta), \\ m_1 &= Kc\delta^2(\alpha+v+k_1)-K\delta(\alpha cr+\delta vk_2), \\ m_2 &= Kc\delta^2(\alpha+k_1)-K\delta(\alpha cr+\delta v\mu). \end{aligned}$$

Next, x_1^* is the roots from eq. (2):

$$b_1\lambda^2+b_2\lambda+b_3=0, \tag{2}$$

where:

$$\begin{aligned} b_1 &= r\alpha\beta(\alpha+\delta), \\ b_2 &= K\beta\delta(\alpha(\delta-r)+\delta k_1), \\ b_3 &= K\alpha\beta n\delta(\delta-r)+K\delta^2(\beta nk_1+\mu v). \end{aligned}$$

Further, we have the following conditions:

(i) For $b_2^2 - 4b_1b_3 = 0$ and $b_2 < 0$, there is an interior point.
(ii) For $b_2^2 - 4b_1b_3 > 0$ and $b_3 < 0$, there is an interior point.
(iii) For $b_2^2 - 4b_1b_3 > 0$, $b_2 < 0$, and $b_3 > 0$, there are two interior point.

3.2. *Basic Reproductive Number*

A key parameter of the system is the basic reproductive number of the disease in the predator, R_0 [28, 29], defined by:

$$R_0 = \frac{\beta y_{13}}{\mu}, \tag{3}$$

where, βy_{13} is the infection rate of new infectives appearing in a totally susceptible predator population and μ is the death rate of infected predator because of natural death and disease induced mortality.

4. Stability of Equilibrium Points

Let us study the behaviours of local stability of the equilibrium points by the Jacobian matrix:

$$J = \begin{bmatrix} -j_{11} & j_{12} & -j_{13} & 0 \\ \alpha & -\delta & 0 & 0 \\ j_{31} & 0 & j_{33} & -j_{34} \\ 0 & 0 & \beta y_2-\mu & \beta y_1 \end{bmatrix} \tag{4}$$

where:

$$\begin{aligned} j_{11} &= \frac{rx_2}{K}+\alpha+k_1+\frac{vny_1}{(n+x_1)^2}; \\ j_{12} &= r\left(1-\frac{x_1+2x_2}{K}\right); \\ j_{13} &= \frac{vx_1}{n+x_1}; \\ j_{31} &= \frac{cy_1(y_1+y_2)}{x_1^2}; \\ j_{33} &= c-\frac{2y_1+y_2}{x_1}-\beta y_2-k_2; \\ j_{34} &= y_1\left(\beta+\frac{c}{x_1}\right). \end{aligned}$$

Theorem 1. *The extinction point P_0 is unstable.*

Proof. The Jacobian matrix of the system (1) at P_0 has eigenvalues:

$$\begin{aligned}\lambda_{1,2} =& \frac{-l_2 \pm \sqrt{l_2^2 - 4l_1l_3}}{2l_1},\\ \lambda_3 =& \mu,\\ \lambda_4 =& c,\end{aligned}$$

where:

$$l_1 = 1, \quad l_2 = \alpha + \delta, \quad l_3 = \alpha(\delta + k_1).$$

Consequentially, point P_0 is unstable. □

Theorem 2. *P_1 is locally asymptotically stable if:*
(i) $q_j > 0$, *where* $j = 0, 1, ..., 4$
(ii) $q_1q_2 > q_0q_3$
(iii) $q_1q_2q_3 > q_1^2q_4 + q_0q_3^2$.

Proof. The Jacobian matrix of the system (1) at P_1 has a characteristics of the polynomial is

$$q_0\lambda^4 + q_1\lambda^3 + q_2\lambda^2 + q_3\lambda = 0,$$

where:

$$\begin{aligned}q_0 &= 1,\\ q_1 &= k_2 + \delta + j_{11_{P1}} - c,\\ q_2 &= j_{11_{P1}}k_2 + \delta(j_{11_{P1}} + k_2) - c(\delta + j_{11_{P1}}) - \alpha j_{12_{P1}},\\ q_3 &= (c - k_2)\left(\alpha j_{12_{P1}} - \delta j_{11_{P1}}\right),\\ q_4 &= 0,\\ j_{11_{P1}} &= \frac{rx_{21}}{K} + \alpha + k_1,\\ j_{12_{P1}} &= r\left(1 - \frac{x_{11} + 2x_{21}}{K}\right).\end{aligned}$$

For $\lambda_i, i = 1, 2, 3, 4$ will be negative whenever $q_j > 0, j = 0, 1, ..., 4$, $q_1q_2 > q_0q_3$, and $q_1q_2q_3 > q_1^2q_4 + q_0q_3^2$. □

Theorem 3. *P_2 is locally asymptotically stable if:*
(i) $g_j > 0$, *where* $j = 0, 1, ..., 4$
(ii) $g_1g_2 > g_0g_3$
(iii) $g_1g_2g_3 > g_1^2g_4 + g_0g_3^2$.

Proof. The Jacobian matrix of the system (1) at P_2 has a characteristics of the polynomial is

$$g_0\lambda^4 + g_1\lambda^3 + g_2\lambda^2 + g_3\lambda + g_4 = 0,$$

where:

$$\begin{aligned}g_0 &= 1,\\ g_1 &= \delta + j_{11_{P2}} - (g^* + \beta y_{12}),\\ g_2 &= \beta y_{12}\left(g^* + \mu - (\delta + j_{11_{P2}} + \beta y_{22})\right) + (\delta - g^*)j_{11_{P2}} - (\alpha r + \delta g^*),\\ g_3 &= \beta j_{11_{P2}} y_{12}(g^* + \mu - \delta) + g^*(\alpha r - \delta j_{11}) + \beta y_{12}(\alpha r + \delta g^* + \delta\mu) - \beta^2 y_{12}y_{22}(\delta + j_{11_{P2}}),\\ g_4 &= \beta\delta j_{11_{P2}} y_{12}((g^* + \mu) - \beta y_{22}) + \alpha\beta y_{12}(\beta r y_{22} - r(g^* + \mu)),\\ g^* &= c - \beta y_{22} - k_2,\\ j_{11_{P2}} &= \alpha + k_1 + \frac{vy_{12}}{n}.\end{aligned}$$

For $\lambda_i, i = 1, 2, 3, 4$ will be negative whenever $g_j > 0, j = 0, 1, ..., 4$, $g_1g_2 > g_0g_3$, and $g_1g_2g_3 > g_1^2g_4 + g_0g_3^2$. □

Theorem 4. *P_3 is locally asymptotically stable if:*
(i) $s_j > 0$, *where* $j = 0, 1, ..., 4$
(ii) $s_1s_2 > s_0s_3$
(iii) $s_1s_2s_3 > s_1^2s_4 + s_0s_3^2$.

Proof. The Jacobian matrix of the system (1) at P_3 has a characteristics of the polynomial is

$$s_0\lambda^4 + s_1\lambda^3 + s_2\lambda^2 + s_3\lambda + s_4 = 0,$$

where:

$$\begin{aligned}s_0 =& 1,\\ s_1 =& n^* + \delta + k_2 - (c + \beta y_{13}),\\ s_2 =& k_2(\delta + n^*) + \delta n^* + \beta_1 y_{13}(c - (\delta + \mu + n^* + k_1 + k_2)) - c(\delta + n^*) - \alpha r,\\ s_3 =& \beta y_{13}(\alpha r + c\delta + cn^* - (\delta\mu + \delta n^* + \delta k_2 + \mu n^* + n^*k_2)) + \alpha cr + \delta n^*k_2 - (\alpha r k_2 + cn^*\delta),\\ s_4 =& (c - (\mu + k_2))(\beta y_{13}(\delta n^* - \alpha r)),\\ n^* =& \alpha + k_1 + vy_{13}.\end{aligned}$$

For $\lambda_i, i = 1, 2, 3, 4$ will be negative whenever $s_j > 0, j = 0, 1, ..., 4$, $s_1s_2 > s_0s_3$, and $s_1s_2s_3 > s_1^2s_4 + s_0s_3^2$. □

Theorem 5. *P_4 is locally asymptotically stable if:*
(i) $u_j > 0$, *where* $j = 0, 1, ..., 4$
(ii) $u_1u_2 > u_0u_3$
(iii) $u_1u_2u_3 > u_1^2u_4 + u_0u_3^2$.

Proof. The Jacobian matrix of the system (1) at P_4 has a characteristics of the polynomial is

$$u_0\lambda^4 + u_1\lambda^3 + u_2\lambda^2 + u_3\lambda + u_4 = 0,$$

where:

$$\begin{aligned}u_0 &= 1,\\ u_1 &= \delta + j_{11_{P4}} - (j_{33_{P4}} + \beta y_{14}),\\ u_2 &= j_{13_{P4}}j_{31_{P4}} + \beta j_{33_{P4}}y_{14} + \delta j_{11} - (\delta + j_{11_{P4}})\beta y_{12} - (\alpha j_{12_{P4}} + \delta j_{33_{P4}} + \mu j_{34_{P4}} + j_{11_{P4}}j_{33_{P4}}),\\ u_3 &= \beta y_{14}(\alpha j_{12_{P4}} + \delta j_{33_{P4}} + j_{11_{P4}}j_{33_{P4}} - (j_{13_{P4}}j_{31_{P4}} + \delta j_{11_{P4}})) + \alpha j_{12_{P4}}j_{33_{P4}} + \delta j_{13_{P4}}j_{31_{P4}} - \delta(\mu j_{34_{P4}} + j_{11_{P4}}j_{33_{P4}}) - \mu j_{11_{P4}}j_{34_{P4}},\\ u_4 &= \beta\delta y_{14}(j_{11_{P4}}j_{33_{P4}} - j_{13_{P4}}j_{31_{P4}}) + \alpha j_{12_{P4}}(\mu j_{34_{P4}} - \beta j_{33_{P4}}y_{14}) - \delta\mu j_{11_{P4}}j_{34_{P4}},\\ j_{11_{P4}} &= \frac{rx_{24}}{K} + \alpha + k_1 + \frac{vny_{14}}{(n + x_{14})^2};\end{aligned}$$

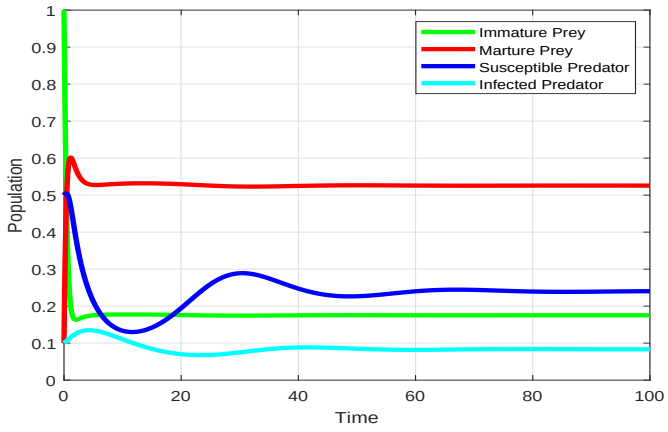


Figure 2. Local asymptotic stability of interior point P_*

$$
\begin{aligned}
j_{12_{P4}} &= r\left(1-\frac{x_{14}+2x_{24}}{K}\right);\\
j_{13_{P4}} &= \frac{vx_{14}}{n+x_{14}};\\
j_{31_{P4}} &= \frac{cy_{14}^2}{x_{14}^2};\\
j_{33_{P4}} &= c-\frac{2y_{14}}{x_{14}}-\beta y_{24}-k_2;\\
j_{34_{P4}} &= y_{14}\left(\beta+\frac{c}{x_{14}}\right).
\end{aligned}
$$

For $\lambda_i, i = 1,2,3,4$ will be negative whenever $u_j > 0, j = 0,1,...,4$, $u_1u_2 > u_0u_3$, and $u_1u_2u_3 > u_1^2u_4 + u_0u_3^2$. □

Theorem 6. *P_* is locally asymptotically stable if:*
(i) $w_j > 0$, *where* $j = 0,1,...,4$
(ii) $w_1w_2 > w_0w_3$
(iii) $w_1w_2w_3 > w_1^2w_4 + w_0w_3^2$.

Proof. The Jacobian matrix of the system (1) at P_* has a characteristics of the polynomial is

$$w_0\lambda^4 + w_1\lambda^3 + w_2\lambda^2 + w_3\lambda + w_4 = 0,$$

where:

$$
\begin{aligned}
w_0 &= 1,\\
w_1 &= \delta + j_{11_{P_*}} - (j_{33_{P_*}} + \beta y_1^*),\\
w_2 &= j_{31_{P_*}}j_{13_{P_*}} + \delta j_{11_{P_*}} + \beta\left(j_{33_{P_*}}y_1^* + j_{34_{P_*}}y_2^*\right)\\
&\quad -\beta y_1^*\left(\delta + j_{11_{P_*}}\right) - \left(\alpha j_{12_{P^*}} + \delta j_{33_{P_*}} + \mu j_{34_{P_*}}\right.\\
&\quad \left.+j_{11_{P_*}}j_{33_{P_*}} + j_{13_{P_*}}j_{31_{P_*}}\right),\\
w_3 &= \beta y_1^*\left(\alpha j_{12_{P_*}} - \delta j_{11_{P_*}}\right)\left(\delta + j_{11_{P_*}}\right)\left(y_1^* j_{33_{P_*}}\right.\\
&\quad \left.+y_2^* j_{34_{P_*}}\right)\beta - j_{13_{P^*}}j_{31_{P^*}}\left(\delta - \beta y_1^*\right)\\
&\quad +\alpha j_{12_{P_*}}j_{33_{P_*}} - \mu j_{34_{P_*}}\left(\delta + j_{11_{P_*}}\right) - \delta j_{11_{P_*}}j_{33_{P_*}},\\
w_4 &= \left(y_1^* j_{33_{P_*}} + y_2^* j_{34_{P_*}}\right)\left(\delta j_{11_{P_*}} - \alpha j_{12_{P_*}}\right)\beta\\
&\quad +\alpha\mu j_{12_{P_*}}j_{34_{P_*}} - \left(\mu j_{11_{P_*}}j_{34_{P_*}} + \beta y_1^* j_{13_{P_*}}j_{31_{P_*}}\right),\\
j_{11_{P_*}} &= \frac{rx_2^*}{K} + \alpha + k_1 + \frac{vny_1^*}{(n+x_1^*)^2};
\end{aligned}
$$

$$
\begin{aligned}
j_{12_{P_*}} &= r\left(1-\frac{x_1^*+2x_2^*}{K}\right);\\
j_{13_{P_*}} &= \frac{vx_1^*}{n+x_1^*};\\
j_{31_{P_*}} &= \frac{cy_1^*(y_1^*+y_2^*)}{x_1^{*2}};\\
j_{33_{P_*}} &= c-\frac{2y_1^*+y_2^*}{x_1^*}-\beta y_2^*-k_2;\\
j_{34_{P_*}} &= y_1^*\left(\beta+\frac{c}{x_1^*}\right).
\end{aligned}
$$

For $\lambda_i, i = 1,2,3,4$ will be negative whenever $w_j > 0, j = 0,1,...,4$, $w_1w_2 > w_0w_3$, and $w_1w_2w_3 > w_1^2w_4 + w_0w_3^2$. □

4.1. Disease Control

Lemma 1. *If $\frac{\beta y_{13}}{\mu} < 1$, then the disease will be eradicated from the ecosystem.*

Proof. Using the fourth equation of system (1), we get

$$\dot{y_2} = y_2\left(\beta y_1 - \mu\right).$$

Thus, if $\frac{\beta y_{13}}{\mu} < 1$, then $\frac{dy_2}{dt} < 0$ and $I \to 0$ as $t \to \infty$. Hence ultimately there will be no infection and disease will disappear from the ecosystem. To explain it conveniently we recall the basic reproductive number R_0 defined by

$$R_0 = \frac{\beta y_{13}}{\mu} \tag{5}$$

Thus $R_0 < 1$ implies that infected predator will disappear from the ecosystem and disease will be eradicated from the predator population. □

5. Example and Numerical Simulation

For examples to confirm the previous analytical results, several numerical simulations have been carried out. The model (1) is solved using the initial conditions and some hypothetical values of the parameters. We first consider the following parameter values:

$$r = 1.9;\ c = 0.5;\ K = 1;\ v = 0.25;\ n = 0.4;\ \alpha = 1.5;$$
$$\beta = 0.5;\ \delta = 0.5;\ \mu = 0.12;\ k_1 = 0.1;\ k_2 = 0.1.$$

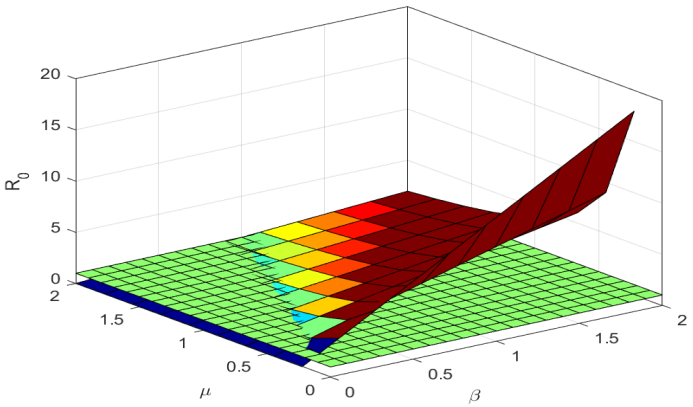


Figure 3. The relationship among β, μ, and R_0

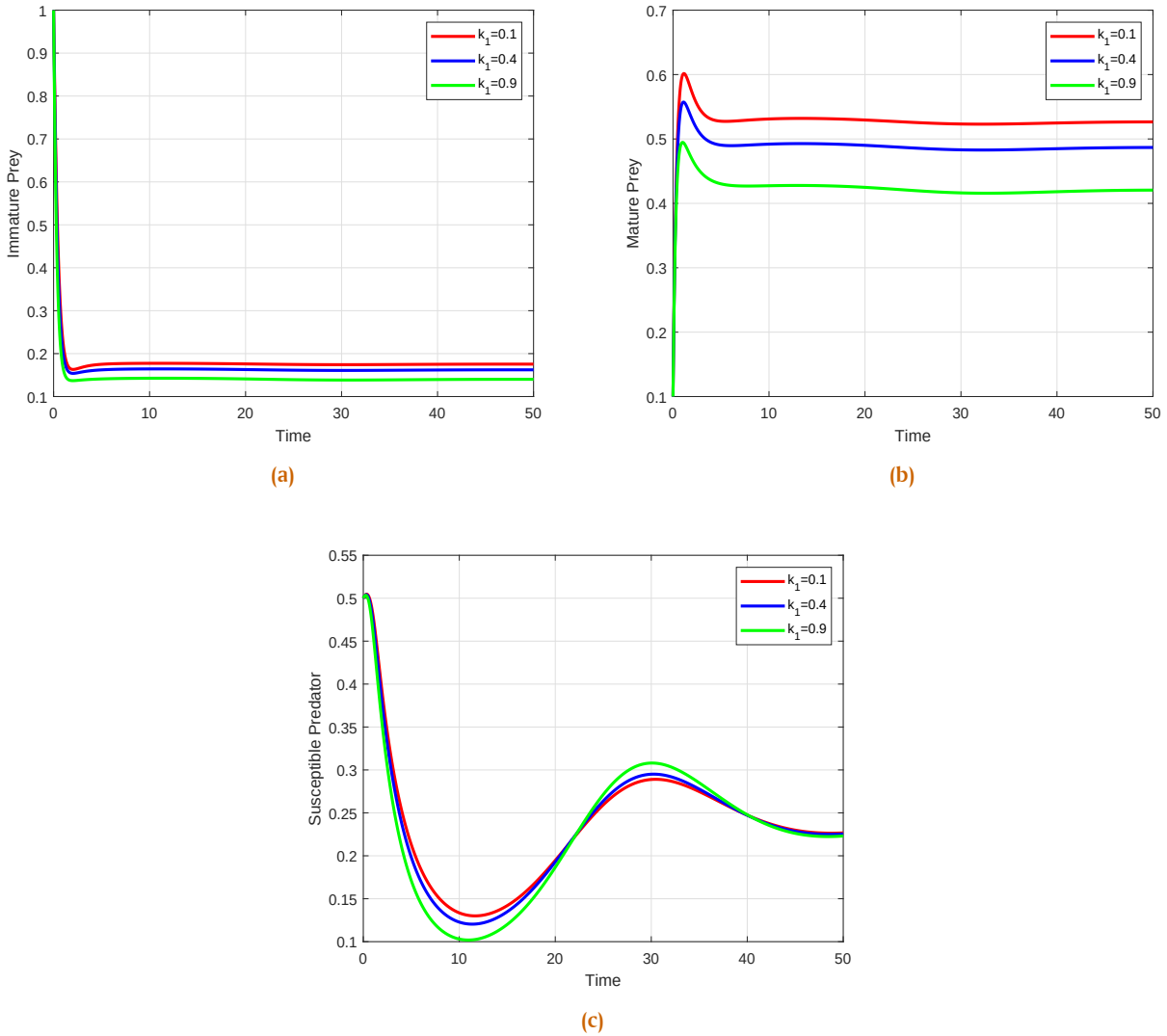
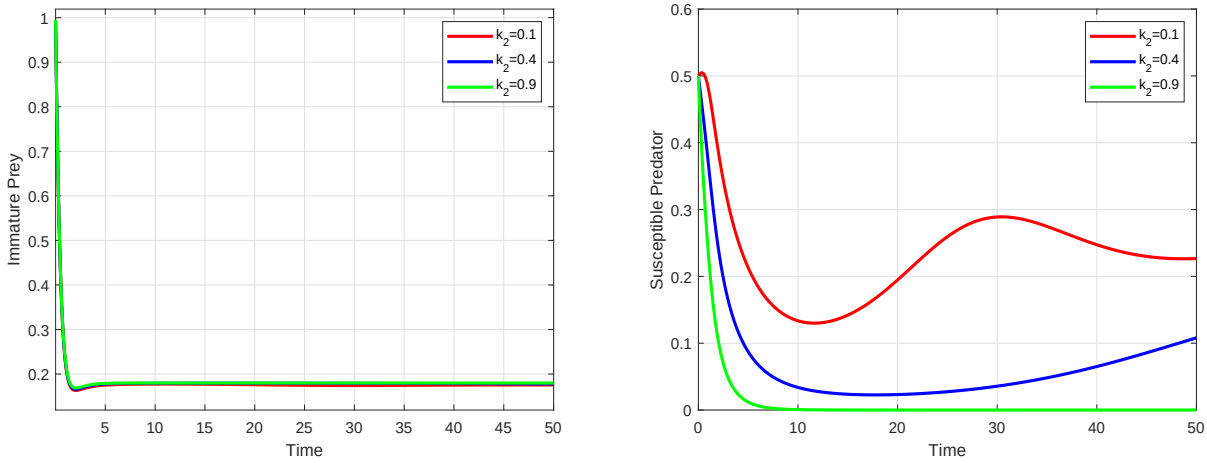
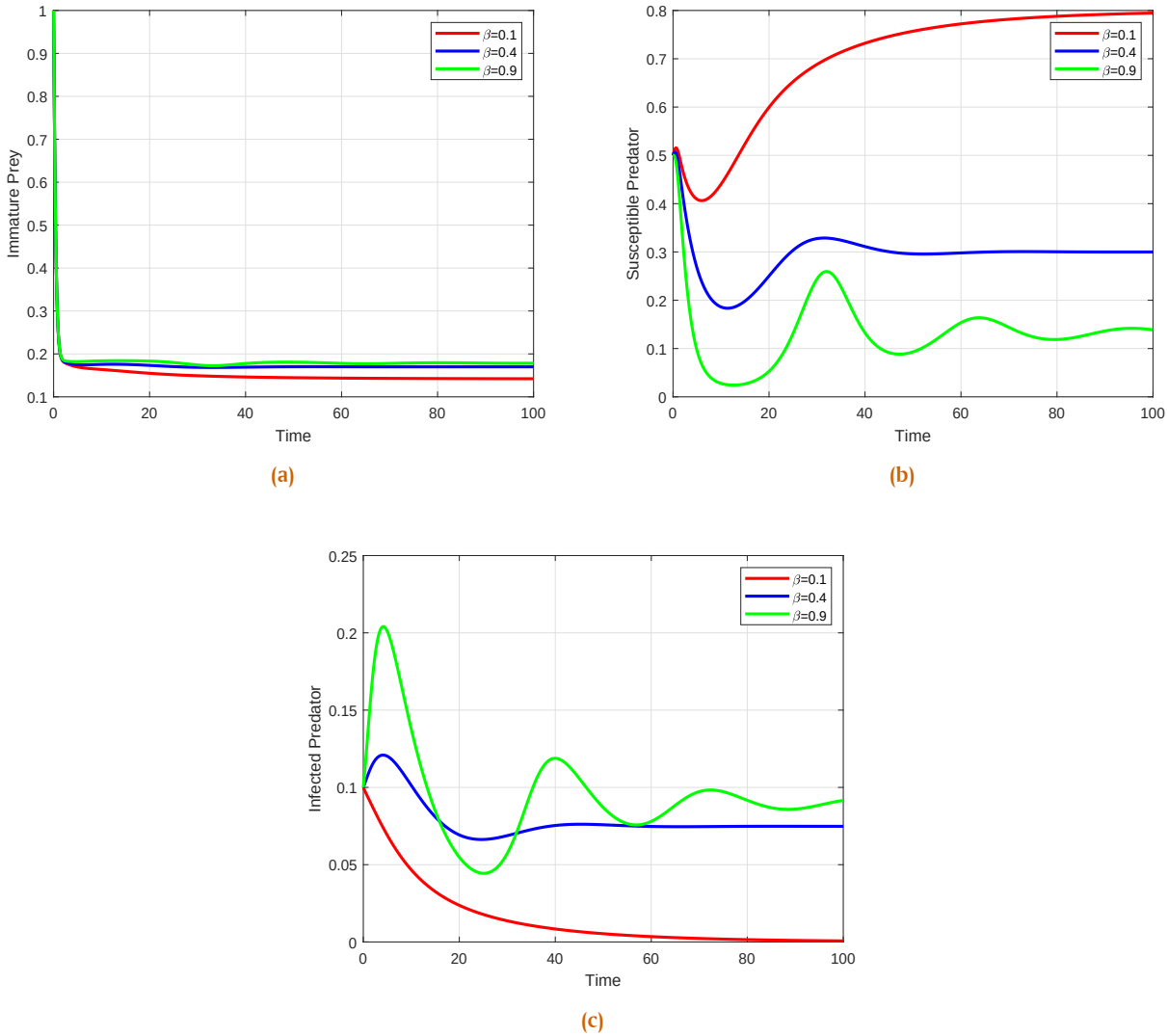


Figure 4. Effects of parameter harvesting k_1 for: (a) Immature prey; (b) Mature prey; (c) Susceptible predator



(a) (b)

Figure 5. Effects of parameter harvesting k_2 for: (a) Immature prey; (b) Susceptible predator



(a) (b)

(c)

Figure 6. Effects of parameter infection rate β for: (a) Immature prey; (b) Susceptible predator; (c) Infected predator

Based on the simulation results for above parameter values, we get the conditions of Theorem 6 are satisfied and consequently $P_* = (0.1752; 0.5257; 0.2403; 0.0834)$ is locally asymptotically stable (see Figure 2), where:

$$\begin{aligned} w_0 &= 1, \\ w_1 &= 3.380056109, \\ w_2 &= 2.676998194, \\ w_3 &= 0.491531614, \\ w_4 &= 0.045520489, \\ w_1 w_2 &= 9.048404099, \\ w_0 w_3 &= 0.491531614, \\ w_1 w_2 w_3 &= 4.447576674, \\ w_1^2 w_4 + w_0 w_3^2 &= 0.761664874. \end{aligned}$$

For this case, we obtained that $R_0 = 1.333333334$. It is indication that the existence of disease transmission in the population. We can see in the Figure 3 that parameter β (infection rate of predator) and parameter μ (the death of predator due to disease) are two parameters has important role in the spread of disease. The higher the value of infection parameter β, the greater the number of infected predators. This is parallel to the value of R_0 which continues to increase. Changes in parameter μ also cause changes in population dynamics in predators related to death due to disease.

The increasing parameter k_1 (harvesting for immature prey) causes the prey population to decrease. Increased harvesting of immature prey has a direct impact so that the population size is increasingly smaller. At the same time, an indirect impact occurs on mature preys which continues to shrink due to the number of immature prey into adults which are significantly reduced due to harvesting. Moreover, the predator population is also decreasing due to the decreasing number of prey as a food source. This case is illustrated in the Figure 4.

Next, the increasing parameter k_2 (harvesting for susceptible predator) causes the predator population to decrease. Nevertheless, this provides benefits to the ever-increasing prey population (see Figure 5). The number of immature prey populations as a food source for predators will grow larger because the value of parameter k_2 continues to increase. In other words, harvesting for susceptible predators has a positive impact on prey population growth, generally. Furthermore, the presence of disease in the predator population also provides an advantage to the prey which increases in number. As the value of parameter β increases, the immature prey population size will decrease. Conversely, the population density of susceptible predators is getting smaller. Thus, disease transmission in predator populations has a significant impact on the growth of prey population. This case illustrated in Figure 6.

6. Conclusion

The dynamical behaviour of a stage–structure Leslie Gower model with linear harvesting and disease in predator has investigated. The model has six equilibrium which are conditionally locally asymptotically stable, i.e. the non-predator point (P_1); the non-prey point (P_2); the existence of susceptible predator point (P_3); the disease-free point (P_4); and the interior point (P_*), except the extinction of all species point (P_0) is always unstable. The parameters k_1, k_2, and β has been shown the influence for dynamics of population's size. Increasing the value of parameter k_1 will reduce the population densities of immature prey and susceptible predator. Furthermore, increasing the values of parameters k_2 and β will reduce the predator population, but the prey population will be increases. In addition, if the value of parameter β increases then the value of R_0 will follow suit. The value of R_0 is threshold for disease spread in predator populations.

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