Modelling Dispersal Processes in Impala-Cheetah-Lion Ecosystems with Infection in the Lions

By

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DECLARATION

I, Willard Mbava s210052023, hereby declare that the thesis for PhD Mathematics to be awarded is my own work and that it has not previously been submitted for assessment or completion of any postgraduate qualification to another University or for another qualification.

.....(Signature)

Willard Mbava

Dedication

I dedicate this thesis to my loving wife, Sabina, and our wonderful son, Tinashe Marcel.

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Citation

Teach me your ways, O LORD;

*make them known to m*e.

Teach me to live according to your truth,

for you are my God, who saves me.

I always trust in you.

Psalms 25: 4-5

Abstract

The study involved the predator-prey interaction of three species namely the predator (Cheetah *Acinonyx jubatus*), the super-predator (Lion *Panthera leo*), and their common prey (Impala *Aepyceros melampus*). The study area is the Kruger National Park. The predator being an endangered species, faces a survival problem. It is frequently killed by the super-predator to reduce competition for prey. The super-predator also scares away the predator off its kills. The prey forms the main diet of the predator. The plight of the predator motivated the author to formulate disease and reaction-diffusion models for the species interactions. The purpose of the models were to predict and explain the effect of large competition from the super-predator on the predator population. Important parameters related to additional predator mortality due to presence of super-predator, the disease incidence rate and induced death rate formed the focal points of the analysis.

The dynamics of a predator-prey model with disease in super-predator were investigated. The super-predator species is infected with bovine Tuberculosis. In the study, the disease is considered as biological control to allow the predator population to regain from low numbers. The results highlight that in the absence of additional mortality on the predator by the super-predator, the predator population survives extinction. Furthermore, at current levels of disease incidence, the super-predator population is wiped out by the disease. However, the super-predator population survives extinction if the disease incidence rate is low. Persistence of all populations is possible in the case of low disease incidence rate and no additional mortality imparted on the predator. Furthermore, a two-species subsystem, prey and predator, is considered as a special case to determine the effect of super-predator removal from the system, on the survival of the predator. This is treated as a contrasting case from the smaller parks. The results show that the predator population thrives well in the total absence of its main competitor, with its population rising to at least twice the initial value.

A reaction-diffusion three-species predator-prey model was formulated and analysed. Stability of the temporal and the spatio-temporal systems, existence and non-existence of stationary steady state solutions were studied. Conditions for the emergence of stationary patterns were deduced. The results show that by choosing the diffusion coefficient $d_2 > \bar{D}_2$ sufficiently large, a non-constant positive solution is generated, that is, stationary patterns emerge, depicting dispersal of species. Predators were observed to occupy habitats surrounding prey. However, super-predators were observed to alternate their habitats, from staying away from prey to invading prey habitat.

In the investigation, strategies to determine ways in which the predator species could be saved from extinction and its population improved were devised, and these included isolation of the predator from the super-predator.

Keywords: Predator-prey system, eco-epidemiology, local stability, global stability, infectious disease, reaction-diffusion system, self-diffusion, instability, non-constant positive steady state.

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Chapter 1

Introduction

1.1 Dynamics of predator-prey systems with dispersion

Wildlife conservation is crucial in the fight against extinction for some endangered species. In the case of mammals in which carnivores survive on predation, their conservation becomes essential and paramount to that of other groups of species. An alteration in the levels of competition between carnivores has a direct bearing on the survival of other groups of species [1].

Several large carnivore species populations are facing negative growth [2]. The problems around their conservation can be attributed to direct conflict between humans and predators [3, 4]. The negative growth is indicative of failing efforts in the conservation of carnivores. It appears that perceptions about what is right have super-ceded scientifically proven theories on conservation. Several initial ecological management strategies have imposed a negative effect on species populations [5].

Protected areas are now safe havens for wildlife species. They are reserve areas for wildlife species habitat as they keep away human activities from endangering them.

Apart from these areas, conservation efforts need to be improved where free roaming wildlife are found [6].

To appreciate that predators need to be conserved, there is a need to fully understand their role in wildlife communities. Since predators must kill other animals in order to survive, many myths about them have evolved over the centuries in many cultures.

Extinction of prey species can be attributed to excessive predation or harvesting. Predator diet is usually not limited to one prey species. In general, prey is the common source of food to predators. Extinction of predator species is linked to large scale competition amongst predators and subsequent loss of food. An understanding of the prey and predator interactions is therefore vital in shaping conservation efforts.

Carnivore competition is difficult to analyse. A complementary part to conservation of carnivores is the knowledge of why a species competes for a common resource. Such knowledge assists conservationists to impart an effect on that competition. Carnivore competition is an integral part to species conservation in ecology [1, 7]. It can also take the form of intraguild predation, a common multi-trophic interaction [8, 9, 10]. With intraguild predation, two consumer species in competition for the same resource also involve themselves in another trophic interaction in which one species consumes the other competitor. Such two species can only coexist if there is no dispersal involved, as long as they show a trade-off between competition and predation. Such a trade-off implies that the more powerful consumer makes the less powerful competitor its additional prey [8]. With intra-guild predation, predators tend to expand their prey base. This has a subsequent effect on other trophic levels [1, 11].

Apart from the effects of competition on population densities, the occurrence of infectious diseases in wild animal species has become a serious conservation issue which cannot be ignored. Animal species are not immune to infectious diseases. It is of vital

importance that whilst modelling the species interactions, the consequences of outbreaks of infectious diseases need to be taken into account. The presence of a disease in at least one species alters the way in which that species interacts with others.

When an infectious disease affects the prey species, predation tends to be inclined towards infected rather than healthy prey [12]. The basic assumption is that sick prey are easier to catch than healthy ones. This however, assumes the fact that the disease is not passed on to the predator. In situations where the disease is passed to the predators, the dynamics differ. Infected predators tend to be too weak to hunt successfully and rely on food provided by members of the same group. The presence of disease in one species can be viewed as biological control to alter the population level of another species and avoid extinction [13].

In general, species interaction is not homogeneous in any environment. Predation has an effect on the way in which prey species interact [14, 15, 16] their density, distribution in the environment, and the selection of preferred habitat [17, 18]. The structure of the landscape such as hills, river confluences, and closeness to water, are essential in determining the risk of predation [19]. Predators tend to disperse in the direction of areas where prey density is high. On the other hand, prey tend to disperse to areas in which predator density is low. Predators of a lower guild tend to disperse in areas where the higher guild predators are few. As such, the species interactions tends to be heterogeneous. It is therefore important that spatial considerations of species interactions need to be taken into account in the modeling process. The spatio-temporal dynamics need to be investigated. Spatial distributions can be influenced by topography, resource availability, distribution, and disturbances [20]. The presence of water holes also influences the distribution of wildlife [1, 21].

1.2 The problem in Kruger National Park

The Kruger National Park (KNP) was established in 1898 to protect the wildlife of the South African Lowveld. It is a home to a variety of wildlife species, ranging from trees to mammals, with species numbers estimated as: "336 trees, 49 fish, 34 amphibians, 114 reptiles, 507 birds and 147 mammals" [22]. KNP is South Africa's largest wildlife reserve with an area of approximately 20,000 km^2. The park consists largely of woodland savanna. Recent population estimates (2010-2011) of the numbers of mammals are: lion (*Panthera leo*) 1620-1750; leopard (*Panthera pardus*) 1000; cheetah (*Acinonyx jubatus*) 412 [23]; buffalo(*Synercus caffer*) 37130; impala (*Aepyceros melampus*) 132300-176400 and so forth [24].

The mammals include carnivores and herbivores. The big flesh-eating cats (lion, leopard and cheetah) and dogs (hyaena(*Crocuta crocuta*) and wild dogs (*Lycaon pictus*)) occupy the highest positions on the list of large carnivores. Large and small predators make up a small proportion of the entire animal kingdom in the park. In comparison with the number of herbivores, predators are few. Herbivores can be grouped under either grazers or browsers, although many species can be both. Grazers, like buffalo, have a diet that is made up of grass while browsers, like the giraffe (*Giraffa camelopardalis*), feed on leaves. Some animals, like the elephant (*Loxodonta africana*) and impala, feed on both grass and leaves, depending on the ease of access of the food. In the KNP, the impala provide 29% of lion kills and 44% of cheetah kills [25].

The lion and hyena are the dominant predators of the large predator guild in KNP. The cheetah and wild dog are in the sub-dominant predator level of this guild, and as such, they face immense competition from the lion and wild dog. The effects of this competition can be explained in the way in which the species avoid, interfere and co-exist with each other. The way in which species interfere with each other is closely related to their relative population densities. Species in high abundance prosper at the

expense of those occurring at low densities. This exacerbates the extinction of species with low densities [26]. The low densities of the cheetah and wild dog in the KNP and their failure to regain large numbers can be attributed to this. Furthermore, the low densities can be attributed to loss of food due to large competition, where they lose their kills to the species in the upper guild [1].

In KNP, a predator control programme meant to reduce the number of carnivores was stopped in 1960. The programme was responsible for the death of 269 cheetah. "Early control schemes, and subsequent management policies such as the provision of artificial water holes, tipped the balance in favour of lions at the expense of other predators" [1]. Since then the cheetah population has grown marginally.

Large competition between carnivores has created a major problem for KNP conservation. Lion, an intraguild predator, has a high population in comparison with the cheetah. Due to intra-guild predation by the lions, the cheetah population is low [27]. A reduced lion population could allow the cheetah population to recover. However, an increase in cheetah population could lead to a reduction in prey population levels [1].

1.3 Modelling the dynamics of prey, predator and super predator systems in Kruger National Park

The order of predation of the prey species is such that the top predators usually hunt for the large prey. Buffalo and zebra (*Equus burchelli*) form the main diet for the lion species. However, the lion also hunts for the medium sized prey, which include the impala species. Cheetah usually target medium sized species, such as the impala in particular, occasionally large and small prey. The medium sized prey forms common diet for the lion and cheetah. Impala are an essential source of food for predators [25].

Lion, cheetah and impala occupy different habitats. Lion utilises vast expanses of land depending on prey availability. Cheetah tend to occupy grasslands [28]. Impala is water dependent, and it usually grazes around water holes, as well as open bush land. Prey usually are found near water holes, and predation is concentrated in areas of high prey densities. It has been shown that lions base their territories around water holes [29].

Over the past century, the cheetah has experienced a severe population decline, with global population estimates falling from approximately 100,000 individuals in 1990 to less than 15,000 by 1998, [6, 30]. The excessive decline in cheetah numbers can be attributed to the breakdown of habitat, a shrinking prey base, and conflict with human beings that has lead to huge slaughter [6, 31].

South Africa's cheetah can be divided into either captive or free roaming populations. Cheetah in captive areas include the KNP, Kgalagadi, smaller parks and cheetah relocated to private reserves. There are approximately 357 cheetah in these reserves [32]. The KNP can be considered to fall under the High Prey Density / Competitors Present category, with nice habitat consisting of a combination of woodland and savannah, adequate prey densities, accompanied by a variety of competing large carnivore species, and minimum threat from hunting or poaching. The large carnivore species consist of lions which are considered to be responsible for cheetah cub mortality. It is envisaged that species population growth will be phenomenal as long as there are insignificant stochastic processes or added mortality. However, the presence of competitors is expected to slow down the population growth [33].

The plight of cheetahs symbolises the problems that many predators face throughout the world. Cheetahs are endangered because of loss of habitat and prey to human activities such as farming and development, hunting by farmers as they kill their livestock, and poaching. Even where reserves are able to hold a population of cheetahs,

they fare badly in direct competition with other, more powerful large carnivores such as lions [6, 34]. Cheetahs are less competitive in comparison with their main competitors, hyenas and lions, which are considered to induce their low density [34]. High juvenile, and often adult cheetah mortality within protected areas is attributed to predation by lions [6, 35, 36]. Cheetahs often lose their kills to powerful large carnivores [6, 37]. These large competitors occur in high densities within protected areas, in such a way that cheetah find it difficult to coexist in the same areas [6, 34].

The low density amongst the cheetah species has attracted many conservationists [36] to focus attention on finding the underlying reasons [26]. In the work by Laurenson, it has been established that in the Serengeti National Park, (SNP), Tanzania, juvenile mortality was about 95%, while predation by lions and spotted hyenas contributed 73% to the 95% [36]. This has negatively impacted on the cheetah population, which, has experienced a decline while the lion density has increased [37]. It has been established that predation negatively impacts on the successful reproduction of cheetah [36, 37]. As a result, it is expected that all conservation studies and activities should be targeted toward saving the cheetah especially in regions where the survival of the cheetah is possible [26].

In KNP, the term impala refers to the medium-sized antelope. The KNP is the largest home in South Africa to more than a hundred thousand impala. Impala are generally dominant amongst species in the savannahs. Impala generally inhabit bushland. They are regarded as a water-dependent species, and usually feed in the vicinity of water holes. The impala consume grass, and in the KNP, 90% of their diet consist of grasses. They divert to other food such as flowers, fruits, pods, barks and fallen leaves if necessary. Impala are an essential source of food for predators. In the KNP they provide 29% of lion kills, 28% of leopard kills, 44% of cheetah kills, 75% of wild dog kills and 15% of hyaena kills [25].

Some landscape features enhance the risk of predation. In the case of lions, prey vulnerability is more important to its hunting success than prey availability. The presence of water holes works in favour of lions since water attracts prey species that inhabit dense vegetation, such as areas in the vicinity of rivers [14, 38]. The presence of natural water holes enhances the location of lion kills sites [39]. The spatial distribution of cheetah, in the presence of lion, and in relation to the distribution of prey is of importance.

1.4 Infectious diseases among predator systems

Apart from the ecological aspects there are some important epidemiological aspects affecting animal species within the KNP. There have been persistent outbreaks of animal diseases in the KNP and surrounding areas. Of major importance are the Transboundary Animal Diseases (TADs). An essential TAD affecting species in KNP is Foot-and-Mouth Disease (FMD), "a highly contagious viral disease of cloven-hoofed animals"[40]. Within KNP, wildlife species in which the disease has occurred include buffalo and impala [40, 41, 42, 43].

Amongst other diseases threatening species is Bovine tuberculosis (BTB), caused by *Mycobacterium bovis*. BTB is fast becoming a huge disease problem for wildlife. "The long-term effects of this chronic progressive disease on host populations at sustained high prevalence rates is unknown, but preliminary evidence suggests that it may negatively affect population dynamics or structure in buffalo and lion" [44].

This disease has now spread to and has become endemic in several buffalo populations in South Africa. The lions in the KNP has been diagnosed with BTB. Mycobacterium bovis first appeared in the southern part of the KNP some fifty years ago [45]. It originated from the cattle in the surrounding farming area, and spread to the buffalo. During the same period, the disease spread to the lion population in the KNP [46].

The initial reported outbreak of BTB in lions happened in 1995, and was believed to have originated from feeding on buffalo infected by the same disease [41, 46]. An 80 % disease prevalence in the lion population occupying the southern part of the KNP was reported in the year 2000 [46, 47]. There are two sources of infection within the lion population. Firstly, there is the intrinsic infection resulting from interaction of members within the same pride. Secondly, the extrinsic infection, which results when members from an outside pride infect members from within a pride [48].

Since lions are the apex predators, it has been hypothesized that there may be a change in the lion population, which will ultimately result in changes in the dynamics of other predators, based on their interactions [48]. It is further hypothesized that BTB will impact negatively on the lion population [49].

1.5 Statement of the problem

In this study, the dynamics of a three species population model involving a prey, predator and super-predator corresponding to impala, cheetah and lion respectively, are investigated. The following aspects are taken into consideration:

(a) Cheetah population is under threat of survival, due to large carnivore competition from the lions. Lions scare away cheetah off their kills and even kill them. In addition high cheetah cub mortality rate is due to the lions.

(b) The lion species is affected by BTB.

(c) Impala are the principal prey diet of the cheetah and secondary prey to the lions.

(d) Predation is concentrated in the vicinity of water holes.

The impala population is very large in KNP. Despite the impala providing an abundant source of food for the cheetah, the cheetah population remains very low. The cheetah species is confronted with serious competition from the lions. The effect of BTB disease

in lions on cheetah population requires investigation. As the disease spreads, the effect on additional cheetah mortality needs investigation. It is hypothesized that the presence of disease in lions will lead to an expected recovery of the cheetah population. The problem to be analysed consists of the lions and cheetah feeding on the impala, but the lions inducing an additional mortality on the cheetah to reduce competition. It is assumed that the cheetah is a specialist that feeds only upon the impala, the lion is a generalist intraguild predator that feeds upon the impala, but kills off the cheetah to reduce competition. As such, there is competition for impala between the cheetah and lion. The stability of the continuous time prey-predator-super-predator model needs to be analysed. Furthermore, the spatio-temporal dynamics of this ecosystem needs to be explored, assuming that the population density is not homogeneous in space.

1.6 Objectives

1.6.1 General aim

The aim of this research is to determine the effective management of the impala, cheetah and lion populations, subject to large carnivore competition and presence of an infectious disease in the lion population within the KNP.

1.6.2 Specific objectives

The specific objectives of the study are

(a) To develop a mathematical model to determine the effect of disease infection in the lion species on the dynamics of the system.

(b) To determine the effect of lion removal from the system on the survival of the cheetah species.

(c) To investigate whether the underlying problem for cheetah re-population is the

large lion population which is responsible for additional mortality.

(d) To develop a mathematical model to study the spatio-temporal dynamics and the dispersal processes of the system.

1.7 Justification

The cheetah population varies widely within about 32 countries where cheetahs are still found [30]. "All populations are classified as vulnerable or endangered by the World Conservation Unions (IUCN) Red Data Book and are regulated by the Convention for International Trade in Endangered Species of Wild Fauna and Flora (CITES) as Appendix I" [28, 50]. The appendix ia available on CITES [51]. Such is the case with regards to South Africa, and in particular, the KNP which has a low cheetah population. Possible reasons for the drastic decline of population levels are attributed to the carnivore competition it faces from the lion, high mortality rate of cheetah cubs by the lion, and the dominant diet dependent on impala.

The lion population has been severely affected by the bovine tuberculosis infections. It is of immense importance to determine if the bovine tuberculosis in the lions has an effect on the cheetah populations. Research on the impact of BTB disease in lions on the cheetah population is on-going, but on a low scale. This study will contribute to an understanding of this impact.

1.8 Scope of the study

This study was carried out in South Africa, and data was collected from relevant literature on KNP. The data was used to estimate important parameters for the analysis and simulations of the proposed models. The descriptions of the data and associated parameters are contained in the Table 1.1.

Table 1.1: Parameter descriptions

Species	Parameter description
Impala	intrinsic growth rate, KNP carrying capacity of impala, diffusion coefficient,
Cheetah	natural mortality rate capture rate of the impala handling time of impala, mortality rate by lion, impala biomass conversion rate into cheetah biomass, diffusion coefficient.
Lion	natural mortality rate, capture rate of the impala, handling time of impala, impala biomass conversion rate into lion biomass, BTB disease induced death rate, BTB disease infection rate, diffusion coefficient.

1.9 Methodology

Continuous-time deterministic models were formulated and analysed.

1.9.1 Model 1

Dynamics of impala, cheetah and lion model with disease in lion population.

(a) Model formulation, consisting of a system of coupled non-linear ordinary differential equations.

(b) Analysis of steady states.

(c) Ecological interpretation: effect of super-predator removal and the role of infection.

(d) Parameter estimation using data from literature on KNP as well as studies from other parks with similar environmental conditions.

(e) Numerical analysis using Matlab.

(f) Discussion and conclusion.

1.9.2 Model 2

Dynamics of impala, cheetah and lion model with diffusion.

(a) Model formulation, consisting of a system of non-linear partial differential equations coupled with reaction-diffusion terms.

(b) Analysis of steady states for the corresponding ordinary differential equations (ODE) system.

(c) Analysis of the partial differential equations (PDE) system.

(d) Turing instability.

(e) Parameter estimation using data from literature on KNP as well as studies from other parks with similar environmental conditions.

(f) Numerical analysis using Matlab.

(g) Discussion and conclusion.

Chapter 2

Literature review

2.1 Introduction

All animal species have two limitations to their survival, namely the availability of resources or food, and the presence of natural enemies. In ecological systems where species share common resources or encounter common natural rival species, the species that constantly grows its population when resources are limited or competition from rival species is rife, will make the rest of the other species populations disappear [52].

The interaction of species with each other as well as with the environment determines the population sizes as well as their distribution. The interaction of carnivores with each other can be described in terms of competition, coexistence and avoidance. In this chapter, literature on predation and species survival in KNP is highlighted and essential aspects of conservation dynamics relevant to the survival of these species are identified.

2.2 Competition and predation

Competition between species can be classified in two forms, namely, exploitative competition and interference competition. "Exploitative competition involves indirect negative interactions arising from the use of a common resource" [53]. Every consumer's action is to diminish the resource available to others [54]. "Interference competition involves direct negative interactions arising from territoriality, overgrowth, undercutting, predation or chemical competition" [55]. "Every consumer's action is to reduce access to available resource of other consumers irrespective of the quantity of resource present [54, 56]. Carnivore competition is not uniform but varies with the nature of resource for which the carnivores are competing for [26].

Competition and predation are important factors that shape the structure of terrestrial communities and purpose. They significantly influence the way species interact, their distribution and the form of population dynamics [57, 58, 59]. Competition and predation have become essential elements in understanding species interaction [60]. Of more significance is the interaction between two species that are involved in predator-prey relationship [9, 61, 62]. Holt and Polis [62] defined intraguild predation (IGP) to be the interaction between two species that compete for the same resources but also consume one another [60]. Interference intraguild interactions (IGI) amongst predators often lead to the killing of one of the competitors, but this frequently happens in cases where resources are abundant [26]. Several terrestrial ecosystems consist of two or more higher guild carnivores competing for a number of prey species [63], which may include lower guild carnivores. The lower guild carnivores do not form the main diet of higher guild predators, but may be killed as a result of intensified interference competition [59, 64, 65].

As the population size of lions increase, that of cheetah decreases [66]. In the KNP, the lion population is high whilst the cheetah population is low. Lions act as kleptoparasites

(species that take over and consume food captured by other species) on cheetah. Lions drastically reduce the amount of food consumed by the cheetah. Consequently, cheetah have to devote more energy to hunting [67]. Cheetah may fight to defend their kill or run away but this depends on a variety of factors which include the presence or absence of cubs. Such competition leads to high mortality of cheetah by the lion [1].

2.3 Coexistence

Coexistence between competing species depends on a number of factors. Early studies on interspecific competition suggested that if intra-specific competition is stronger than interspecific competition, coexistence prevails. Besides that, one species often out-competes the other species [34, 68, 69]. "Coexistence requires species to be different in the way they affect, and are affected by resources and natural enemies such that intra-specific competition is stronger than inter-specific competition." [52]. The distinctions between species that can coexist are based on species' niches [70]. Consequently, a basic requirement for coexistence is niche difference or creation of partitions that enhance intra-specific competition other than inter-specific competition [52].

Species that can lead to the creation of niches have ecological distinctions that happen in three separate manners. Firstly, distinct species may concentrate on different resources, (classical resource partitioning [69, 71]) or specialise on density dependent predation [72]. Secondly, distinct species may be restricted by the common resources or natural enemies, but act distinctively in terms of times to consume the resource or react to natural enemies (temporal niche partitioning, [70, 73, 74, 75, 76]). Thirdly, species may be distinct in terms of the place of experiencing and reacting to constraining factors (spatial niche partitioning, [70, 76, 77]). Hence, a species' niche can be defined as consisting of four major axes of the niche space: resources, natural enemies, space and time. The niche can then be described as species reactions to, and their effects on, each point at this niche space [52]. The basic fact that all animals have

an ecological niche and as such coexistence is possible is the important consideration within ecology [1].

Considering the decline in the cheetah population it maybe that this species had its niche restricted to a smaller area of KNP due to human factors which include fencing and park management practises. Cheetah have the ability to stay away from water holes for a prolonged period of time, as they obtain water from consuming prey. On this basis, cheetah can base their territories away from water holes [1].

2.4 Avoidance

The concept of *predator refuges*, where prey search for safe places to avoid predation, is essential for the continued survival of both prey and predator. The concept is applicable to interspecific competition. Furthermore, in a non-homogeneous environment, less powerful species can survive by means of *competitive refuges* where competition is on a low scale [34].

The risk of interference competition or predation has a huge impact on how a species changes its behaviour and spatial distribution [79, 80, 81]. This risk is not uniformly distributed in space and time. It changes with the predators' and competitor's spread, density, and use of habitat [82]. The non-uniformity in risk makes species occupy areas where there is low probability of encountering enemies, or change behaviour in response to varying levels of risk [34, 83]. The response to risk can be described as either reactive or predictive. "A reactive response to risk is based on an animals knowledge of actual, real time risk" [79]. For example, African buffalo are found at waterholes around midday, but not during sunrise nor sunset [84]. "A predictive response, on the other hand, is based on a pre-emptive response to a potential for risk, derived from previous knowledge of the competitors or predators' whereabouts or the habitat types intensively used by them" [79]. Impala are seldom found in areas where there was a

extended chance of predation by lions [18].

The main consequence of competition and intraguild predation is avoidance of the competitors and this is used by cheetah, who will actively seek out areas where lion populations is low [1, 79]. Cheetahs have been observed to regularly avoid areas of high lion population. They are regarded as a 'refugial species'. They use spatial avoidance to minimise encounters with competitively dominant predators [34, 79]. Female cheetahs, however, tend to inhabit areas with high prey densities [85]. In general, all species tend to minimise competition. Since water holes attract high prey densities, competition avoidance by predators is more pronounced there. Availability of water is crucial in conservation efforts of animal species [1]. In the 1960s the KNP installed artificial waterholes. In 1997, with a goal to improve biodiversity, the KNP made an effort to simulate the natural spread of water [86]. Since artificial waterholes were closed, the population of lion has been far higher than that of cheetah. This implied that competition between the carnivores increased due to lack of space, to the point where cheetah population became very low [1].

Competition effects on all animal species can be described in the form of interference, avoidance and co-existence. Population growth is influenced by interference in such a way that large densities grow at the expense of smaller densities, leading to possible extinction. Cheetahs tend to avoid hunting in areas in which prey density is high, as the risk of encountering lions is high [26]. In KNP this may probably provide an explanation as to the failed recovery of cheetah population. Interference may be happen around the captured resource where the lion will aggressively take over cheetah kills.

2.5 Transboundary animal diseases

Transboundary Animal Diseases, TADs, are diseases "that are of significant economic, trade and/or food security importance for a considerable number of countries; which

can easily spread to other countries and reach epidemic proportions; and where control/management, including exclusion, requires co-operation between several countries" [87]. Effective contact between domestic livestock and wildlife is essential for transmission of TADs from surrounding areas to national parks such as the KNP [40]. The diseases affect both the predator and the prey species.

In the KNP, amongst the species being considered in the model, only the lion is affected by BTB [46, 88].

2.6 Predator-Prey interactions with an infectious disease

A number of studies have been done in which an infectious disease runs through the prey species only in a predator-prey model [13, 89, 90, 91, 92]. However, other studies focus on the disease affecting the predator species only [12, 93, 94].

Arino *et al.* [13] proposed an eco-epidemiological model with disease in the prey population. They used ratio-dependent functional responses. It was observed that introduction of diseased prey into the system may save the population from extinction. They concluded that the infected prey population in a classical ratio-dependent predator-prey system can be considered as biological control and save the prey population from extinction. In this study, the proposed model adopts the susceptible-infective (SI) disease dynamics, ratio-dependent predator prey system, and biological control. However, the model considers the introduction of a disease in the super-predator species having a frequency dependent disease incidence. The disease is considered as a biological control in saving the predator from extinction, but not the super-predator itself.

Haque and Venturion [89] studied the role of transmissible diseases in a Holling-Tanner

predator-prey model. They modified the classical Holling-Tanner model (1975), by allowing a disease to spread among the prey species and investigating its effects on the ecosystem. The disease transmission follows mass-action incidence. The prey and predator populations grow logistically with the predator consuming prey according to the Holling type II functional response. Their results suggest that the introduction of a disease in a Holling-Tanner demographic model alters significantly the possible outcomes of the ecosystem. The disease incidence plays an important role for the persistence of the species. They deduced a threshold result to determine when the disease dies out. In this study, the proposed model adopts the same susceptible-infective disease dynamics, but with the disease in super-predator. The disease transmission follows standard incidence. There is no logistic growth in either the predator or super-predator. The super-predator is involved in intra-guild predation with the predator.

Chattopadhyay and Arino [90] studied a three species eco-epidemiological system, with disease in prey. They considered the case in which the predator mainly feeds on the infected prey. Their study was mainly theoretical and did not address any specific situation. They derived conditions for which the populations persist and become extinct. They deduced conditions for which the system enters a Hopf-type bifurcation. Using a Holling-type II functional response for the predator, they deduced that the bifurcated branches were supercritical in a certain parametric region space. In the present model, the SI disease dynamics in predator are followed. Instead of predators targeting infected prey, the infected super-predator's hunting ability is weakened by the disease, as well as its attacking rate on the predator.

Hethcote *et al.* [91] proposed a predator-prey model in which the infected prey were more prone to predation. They investigated the epidemiological and demographic effects of the presence of the disease in prey. The epidemiological model was of susceptible-infective-susceptible (SIS) type, on the assumption that the micro-parasitic infection does not induce immunity. The disease transmission followed the standard

incidence form. It was shown that for some parameter values, the increased predation of the infected prey allowed the disease to die out. Otherwise, without predation of infected prey, the disease would become endemic. In the present study, the model adopts the same standard incidence for disease transmission. The disease dynamics follow, instead, the susceptible-infective form, since lion does not recover from BTB without treatment and become susceptible again. The model has the disease spreading in the super-predator species.

Saenz and Hethcote [92] considered a competing species model with an infectious disease. The model considered was a variant from previous models for a disease in two competing species, since it employed the frequency-dependent incidence and both species were affected by the disease. They considered models with SIS, susceptible-infective-recovered (SIR) and susceptible-infective-recovered-susceptible ($SIRS$) disease formulations. They found that the disease either dies out in both species or remains endemic in both species. They also concluded that the form of the disease incidence strongly affects the asymptotic behavior of a competing species model. In this study, the model considers only the susceptible-infective disease dynamics with disease present in the super-predator species only.

Moreover, other models involve the disease running through the predator species and not the prey species. Venturino [93] proposed and investigated a range of basic models for studying the dynamics of diseases among competing species. In the first case, the disease runs through the prey species and the models were SI and SIS disease formulations in which the disease spread is governed by both mass action incidence and standard incidence independently. In the second case, the disease was allowed to run through the predator species and similar models were formulated. In the model proposed, the susceptible-infective dynamics are adopted for the disease in the super-predator only. The disease does not spread to either the prey or predator.

Haque [12] studied the predator-prey model where the predator is affected by an epidemic. The model incorporates a recovery rate and considers the stability of the positive interior equilibrium point. The predator population is assumed to follow a logistic growth in the presence of alternative food sources. The alternative food sources may enhance the persistence of predator-prey systems. It was shown that the infection in the predator species may save the prey from extinction. The disease reduces the predation rate, and so it acts as a potential method of biological control to ensure persistence of the prey. The model proposed in this study adopts the disease in the super-predator, but a predator free of disease. An investigation is done to determine whether the presence of the disease is considered as biological control in reducing the intra-guild predation and allowing the predator population to grow.

Pal *et al.* [94] studied the dynamics of an eco-epidemiological predator-prey model with disease in predator. SI disease dynamics were used in the model formulation. The mode of disease transmission followed the simple law of mass action. Susceptible and infected predators consume prey according to Holling type I and II functional responses respectively. They carried out the stability and bifurcation analysis. They used normal form theory and center manifold reduction to derive explicit formulae to determine stability and direction of Hopf bifurcation periodic solution. In the model proposed, both the susceptible and infected super-predator consume prey according to Holling type II functional response.

Our proposed model differs from the models discussed above in some respects. Firstly, three species are considered; prey, predator, and super-predator, in which an infectious disease runs through the super-predator. Frequency dependent disease incidence is used for the disease transmission in the super-predator species. Thus, the model extends to include four species. Very little attention has been paid to prey-predator systems involving two competing predators in which intra-guild predation and an infectious disease prevail in one of them.

2.7 Reaction-diffusion modeling and pattern formations

Theoretical models on wildlife that investigate the use of space on several competing predators and mobile prey indicate that higher guild predators are not restricted in their movement and generally match prey distribution. Lower guild predators tend to balance the trade-off between acquiring food and risk of predation [59, 95, 96]. Intraguild competition can change the size of populations and distribution of lower guild predators. This makes the levels of inter- and intra-specific competition to vary across space and time [59, 97]. The relationship between dispersal and species interactions is important in understanding species distribution in spatially structured environments [8]. "When competition for resources is asymmetric, a life-history trade-off between competitive and dispersal abilities can lead to coexistence in a patchy environment" [98].

The temporal modelling of species interactions involving three species have been studied by many authors [99, 100, 101, 102, 103, 104, 105] on the basis of a homogeneous distribution of resources in a given region. The nonlinear system may have a number of steady states. The bahaviour of the system over a long period of time is investigated using the local and global stability of the steady states [106].

In reality, resources are not homogeneously spread through out a given region. The species diffuse from one region of their habitat to another in search of food, and in the process interact with each other and with the environment. This movement has an effect on its interactions with other species [107]. Two types of diffusion which influence the dynamical behaviour of a system of interacting populations are self- and cross-diffusion. Kerner [108] proposed first that there is population pressure experienced by one species as a result of the presence of another species. Self-diffusion defines the movement of species from a region of high concentration to one of low concentra-

tion. Cross-diffusion means the population pressure of one species due to the presence of another species. The cross-diffusion coefficient can assume both positive or negative values. Positive cross-diffusion coefficient denotes movement of one species in the direction of lower concentration of another species, while negative cross-diffusion coefficient represents the population movement of one species towards the region of higher concentration of the other species [109, 110].

To investigate the dynamics of spatial structures on predator-prey distributions, mathematical models on growth and interactions have to incorporate spatial processes including relative motion of species as well as changes in the environment [111]. As species interact, spatial patterns occur naturally. Spatial patterns arise as a result of stochastic processes, environmental disturbances, or deterministic processes. "The deterministic process is intrinsic to the interacting species and results in population-driven and self-organized spatial patterns" [107].

Spatio-temporal dynamics of an ecological system are represented by a system of coupled nonlinear reaction-diffusion equations. The spatio-temporal dynamics of three species with a wide range of functional responses have been studied by several authors [106, 107, 112]. The spatio-temporal system and its corresponding temporal system have the same steady states, but the dynamic behavior may not be the same. The presence of diffusion may cause the stable steady state of the temporal system to lose stability [106]. Turing patterns may form as a result of loss of stability of stable steady state due to diffusion [107]. The existence of non-constant positive steady state and its corresponding stability needs investigation [106, 107, 112]. For a variety of ecological cases, complex stable pattern formations have been established [106, 107].

Gakkhar and Melese [106] investigated the existence and non-existence of non-constant positive steady state for a diffusive three species food web system comprising of two apparently competing prey and a predator. The prey follow logistic growth functions.

The predator adopts modified Leslie-Gower type dynamics and consumes prey species following Holling Type II functional response. The local stability of the constant positive steady state of the temporal system and the spatio-temporal system were discussed. The existence of non-constant positive steady state solution were studied by using the Leray-Schauder degree theory. In this study, the proposed diffusive three species model consists of prey, predator and super-predator, with the super-predator involved in intra-guild predation of the predator. Local and global stability of the steady states of the temporal system are discussed. The local stability of the spatio-temporal system is discussed.

Melese and Gakkhar [107] investigated the formation of patterns in a tri-trophic food chain model with ratio-dependent Michaelis-Menten type functional response and diffusion. Stability and bifurcation analysis were done for the spatially homogeneous steady state. Conditions were derived for Hopf and Turing bifurcation, and for the formation of spatial patterns. The results of numerical simulations revealed the formation of labyrinth patterns and the coexistence of spotted and stripe-like patterns. In the proposed model, a similar stability analysis is performed for the positive steady state of the spatio-temporal system and conditions for the emergence of Turing patterns are derived. However, Holling type II functional responses are employed for the predators, and the model is not a food chain.

Lv *et al.* [109] investigated Turing pattern formation in a three species model involving two prey and generalist predator, under the influence of cross-diffusion. The predator consumes prey according to Holling type I functional response. Prey exert group defense against predators. They showed that in the absence of cross-diffusion, the positive stationary solution was globally asymptotically stable for both the ODE system and reaction-diffusion system. Instability was only possible when cross-diffusion was introduced. Furthermore, the existence and non-existence results concerning non-constant positive steady states of the system were derived. The proposed model differs from Lv

et al model in that the super-predator is involved in intra-guild predation. Predators consume prey according to Holling type II functional response. The system is subject to self-diffusion. There is no prey group defense against predation.

Hei and Yu [112] considered a predator-prey reaction-diffusion system with one resource and two consumers. It was assumed that one consumer species follows Holling type II functional response while the other consumer species follows Beddington-DeAngelis functional response, as they compete for the common resource. They proved that the unique positive constant steady state was stable for the ODE system and the reaction-diffusion system. They derived *a priori* estimates of positive steady state. Conditions for the non-existence of non-constant positive steady state, the existence and bifurcation of non-constant positive steady state were derived. The proposed model differs from Hei and Yu model in that both predators consume prey according to Holling II functional response. There is intraguild predation involving the super-predator.

Guin [113] investigated the spatio-temporal dynamics of reaction-diffusion equations with cross-diffusion for a ratio-dependent predator-prey model. The conditions for diffusion-driven instability were derived. Local and global asymptotic stability results of the unique positive homogeneous steady state without diffusion were established. Numerical results were obtained showing different types of spatial patterns through diffusion-driven instability. In the proposed model, the reaction-diffusion terms for self-diffusion are adopted. However, the functional responses used are prey-dependent Holling type II. A super-predator is included in the predator-prey model. Self-diffusion is considered for the species interactions.

Guin *et al.* [114] discussed the emergence of spatial patterns through diffusion-driven instability in a predator-prey model. They incorporated alternative food source for the predator species in the form of logistic growth, and ratio-dependent Holling type II functional response. They derived the conditions for existence of Turing patterns and

stability properties of the model subject to self and cross-diffusion. Their numerical analysis showed the significant role of self and cross-diffusion. Spatial patterns through Turing instability were obtained. In the proposed model, the predators are specialists. There is no additional food source provided to the predators.

The proposed model differs from the above models in the fact that two competing predators involved. However, the existence and non-existence of non-constant positive steady states of the three species model are investigated. The existence of non-constant positive steady state solution is studied by using the Leray-Schauder degree theory. Conditions for diffusion-driven instability of the steady state are derived.

Chapter 3

Impala, cheetah and lion model with disease in the lions

3.1 Introduction

The sizes of species populations are important in the ecological studies. They are influenced by ecological and epidemiological factors. The ecological factors include species interactions in the form of competition and predation. The epidemiological factors include the spread of infectious diseases [92]. The study of transmissible diseases within an ecological setting is gaining momentum [12]. It is becoming biologically relevant to include the effects of diseases in studies on the behaviour of dynamical ecological systems [115].

The effect of disease in prey on prey-predator systems has been studied by several researchers. For most such models, the paramount assumption is that predation favours infected rather than sound prey [12]. Hethcote *et al.* [91] studied a predator-prey model with SIS disease dynamics in the prey. The infected prey was more vulnerable to predation. They identified thresholds to determine when the predator population survives extinction and when the disease becomes endemic. Mukhopadhyay and Bhattacharyya

[115] studied the dynamics of a delay-diffusion prey-predator model with disease in prey. For their basic model, they established that persistence of the disease depend on the predator death rate and the basic reproduction number. Upon including delay attributed to gestation of the predator, they found out that the predator death rate, basic reproduction number, and equilibrium density of susceptible prey together shape the dynamical behaviour of the system. On the role of diffusion in the delayed model, they deduced that diffusivity coefficients for susceptible and infected prey, together with the perturbation wave number of the general solution, determine the dynamical behaviour of the system. Haque *et al.* [116] investigated an eco-epidemiological predator-prey model with disease in prey. The disease transmission was of standard incidence form from which they derived a threshold property to determine when the disease disappears. They showed that a virulant disease in the prey may allow predators to escape extinction but destabilises a once stable system. Xiao and Chen [117] proposed a predator-prey model with disease in prey. Their model showed that the introduction of a time delay in the coefficient of converting prey into predators has both stabilising and destabilising effects on the positive steady state.

Other researchers consider the situation where the disease spreads among the predator population. The predator-prey interactions are extended to include disease in the predator species. Haque [12] studied the predator-prey model with SIS parasitic infection spreading through the predator species only. Thresholds for the disease progression were identified. The infection in predator was considered as biological control to save prey from extinction. Venturino [93] proposed and investigated several simple models for studying the spread of diseases among competing species. The disease spreads through the prey species only following SI and SIS disease dynamics. The disease spread was governed by both mass action incidence and standard incidence independently. Similar models were formulated to account for the disease spread through the predator species only. Pal *et al.* [94] investigated a predator-prey model with disease present in predator species only. They considered the SI disease dynamics and mass

action transmission form. They used normal form theory and center manifold reduction to derive explicit formulae to determine the stability and direction of Hopf bifurcation periodic solution. Han *et al.* [118] studied four predator prey models in which disease spreads in both the prey and predator. The disease transmission involved both mass action and standard incidence. They identified thresholds for disease persistence and eradication, and established global stability results.

In this chapter, the model proposed is an extension of Haque [12] and Venturino [93] models to include an infectious disease spreading through a super-predator species. The prey, predator and super-predator species are the impala, cheetah and lion respectively. It is considered that the cheetah feeds only upon the impala, the lion feeds upon the impala but kills the cheetah to reduce competition. The impala is the most preferred prey species amongst a host of species consumed by the cheetah [119, 120]. However, it is the third dominant prey species consumed by the lion after zebra and wildebeest in the KNP [121]. Despite the impala occurring in high numbers, the cheetah population remains very low. Moreover, the IUCN (World Conservation Union) Red Data Books listed the cheetah species as vulnerable in South Africa [122].

BTB disease is present in the lion population of KNP [46, 88]. Maas *et al* [46] assessed the impact of feline immunodeficiency virus (FIV) and BTB co-infection in African lions in KNP. They used a multivariable logistic regression model for analysis. They found that BTB does not pose a serious conservation threat to KNP, and that a significant spatio-temporal increase of BTB may impact on lion health.

The aim of this study is to determine whether or not the presence of the disease in the lion population acts as biological control in weakening the lion and indirectly improving the population of the cheetah. With the cheetah confronted with major competition from the lion, as well as extinction, the study also determines the extent to which the presence of the lion affects the cheetah population.

3.2 Model formulation

3.2.1 Assumptions

In this study, the model consists of three populations, the impala, cheetah and lion species whose populations are denoted by $U(t)$, $V(t)$ and $N(t)$, respectively. The following assumptions are considered in formulating the model.

(A1) In the presence of disease, the lion population is divided into two classes, namely susceptible lion and infected lion denoted by $W(t)$ and $B(t)$ respectively. Therefore at time t the total lion population is $N(t) = W(t) + B(t)$.

(A2) The disease spreads among the lion population only through in-group and out-group interactions and is not genetically inherited. The infected population do not recover or become immune. The incidence function $\beta(N)$ is assumed to be the nonlinear function $\beta(N) \equiv \frac{1}{N}$, the standard incidence.

(A3) The cheetah and lion consume the impala, although with possibly different search efficiencies, denoted by a and b.

(A4) The cheetah and lion have natural death rates μ and ν respectively. Furthermore, infected lion have disease-induced death rate δ. The susceptible lion kills off the cheetah at a rate p. Infected lions are considered too weak to kill cheetah.

3.2.2 The model

In the lion-cheetah-impala model, the cheetah and lion consume the impala according to the nonlinear Holling type II functional response. The Holling Type II response is the most common type of functional response for predator species [123]. The impala are easier to catch. A solitary lion or cheetah can catch an impala as the impala do not exhibit group defence. On the basis of the assumptions above the following model

is proposed

$$\begin{aligned}
\frac{dU}{dt} &= rU\Big(1-\frac{U}{K}\Big)-\frac{aUV}{1+eU}-\frac{bUN}{1+fU},\\
\frac{dV}{dt} &= \frac{lUV}{1+eU}-pVN-\mu V,\\
\frac{dN}{dt} &= \frac{mUN}{1+fU}-\nu N,
\end{aligned} \tag{3.1}$$

where U, V, and N represent impala, cheetah and lion populations.

The Susceptible-Infective dynamics are incorporated into the model (3.1) for the disease in the lions using the model proposed by Venturino [93]. The model (3.1) becomes

$$\begin{aligned}
\frac{dU}{dt} &= rU\Big(1-\frac{U}{K}\Big)-\frac{aUV}{1+eU}-\frac{bUW}{1+fU}-\frac{cbUB}{1+fU},\\
\frac{dV}{dt} &= \frac{lUV}{1+eU}-pVW-\mu V,\\
\frac{dW}{dt} &= \frac{mUW}{1+fU}-\frac{\beta WB}{N}-\nu W,\\
\frac{dB}{dt} &= \frac{nUB}{1+fU}+\frac{\beta WB}{N}-\nu B-\delta B,\\
\frac{dN}{dt} &= \frac{mUW}{1+fU}+\frac{nUB}{1+fU}-\delta B-\nu N,
\end{aligned} \tag{3.2}$$

where $n = cm$. The parameters used in the model are explained in Table 3.1.

Let S and I denote the fractions of susceptible and infected lions defined by $S = \dfrac{W}{N}$ and $I = \dfrac{B}{N}$ such that $S + I = 1$. Since

$$I' = \Big(\frac{B}{N}\Big)' = \frac{1}{N}\Big[B' - \frac{B}{N}N'\Big], \qquad S' = \Big(\frac{W}{N}\Big)' = \frac{1}{N}\Big[B' - \frac{W}{N}N'\Big]$$

S' and I' can be expressed in terms of W' and B' respectively. The system (3.2) is

Table 3.1: Parameters table for systems (3.1) and (3.2)

Parameter	Description
r	Intrinsic growth rate of the impala
K	Environmental carrying capacity of the impala
a	Capture rate of the impala by the cheetah
b	Capture rate of the impala by the lions;
c	Efficiency of infected lions to capture impala;
e	Handling time of impala by cheetah
f	Handling time of impala by lions
l	Conversion rate of impala biomass into new cheetah
m	Conversion rate of impala biomass into new susceptible lions
n	Conversion rate of impala biomass into new infected lions
p	Mortality rate of cheetah by lions
β	Disease standard incidence
μ	Natural mortality rate of cheetah
ν	Natural mortality rate of lions
δ	Disease-induced mortality rate of infected lions

now rewritten as

$$\begin{aligned}
\frac{dU}{dt} &= rU\Big(1-\frac{U}{K}\Big)-\frac{aUV}{1+eU}-\frac{bUW}{1+fU}-\frac{cbUB}{1+fU},\\
&= r_1U\Big(1-\frac{U}{K}\Big)-\frac{aUV}{1+eU}-\frac{bU[W+cB]}{1+fU},\\
&= r_1U\Big(1-\frac{U}{K}\Big)-\frac{aUV}{1+eU}-\frac{bU[(1-I)N+cIN]}{1+fU},\\
&= U\Big[r_1\Big(1-\frac{U}{K}\Big)-\frac{aV}{1+eU}-\frac{bN[(1-I)+cI]}{1+fU}\Big].
\end{aligned}$$

$$\begin{aligned}
\frac{dV}{dt} &= \frac{lUV}{1+eU}-pVW-\mu V,\\
&= \frac{lUV}{1+eU}-pV(1-I)N-\mu V,\\
&= V\Big[\frac{lU}{1+eU}-p(1-I)N-\mu\Big].
\end{aligned}$$

$$\begin{aligned}
\frac{dN}{dt} &= \frac{mUW}{1+fU} + \frac{nUB}{1+fU} - \delta B - \nu N, \\
&= \frac{mU(1-I)N}{1+fU} + \frac{nUIN}{1+fU} - \delta IN - \nu N, \\
&= N\Big[\frac{[m(1-I)+nI]U}{1+fU} - \delta I - \nu\Big].
\end{aligned}$$

$$\begin{aligned}
\frac{dI}{dt} &= \frac{1}{N}\Big[\frac{nUB}{1+fU} + \frac{\beta WB}{N} - (\nu+\delta)B - \frac{B}{N}N\Big[\frac{[m(1-I)+nI]U}{1+fU} - \delta I - \nu\Big]\Big], \\
&= \frac{1}{N}\Big[\frac{nUIN}{1+fU} + \frac{\beta(1-I)NIN}{N} - (\nu+\delta)IN \\
&\quad -IN\Big[\frac{[m(1-I)+nI]U}{1+fU} - \delta I - \nu\Big]\Big], \\
&= I\Big[\frac{nU}{1+fU} + \beta(1-I) - (\nu+\delta) - \frac{[m(1-I)+nI]U}{1+fU} + \delta I + \nu\Big], \\
&= I\Big[\beta(1-I) + \frac{[n(1-I)-m(1-I)]U}{1+fU} - \delta(1-I)\Big], \\
&= I(1-I)\Big[\beta + \frac{(n-m)U}{1+fU} - \delta\Big].
\end{aligned}$$

Finally, the model can be written as

$$\begin{aligned}
\frac{dU}{dt} &= U\Big[r\Big(1-\frac{U}{K}\Big) - \frac{aV}{1+eU} - \frac{bN[(1-I)+cI]}{1+fU}\Big] = F_1(U,V,N,I), \\
\frac{dV}{dt} &= V\Big[\frac{lU}{1+eU} - p(1-I)N - \mu\Big] = F_2(U,V,N,I), \\
\frac{dN}{dt} &= N\Big[\frac{[m(1-I)+nI]U}{1+fU} - \delta I - \nu\Big] = F_3(U,V,N,I), \\
\frac{dI}{dt} &= I(1-I)\Big[\beta - \frac{(m-n)U}{1+fU} - \delta\Big] = F_4(U,V,N,I),
\end{aligned} \tag{3.3}$$

with the initial conditions $0 \leq U_0 \leq K$, $0 \leq V_0 \leq A$, $0 \leq N_0 \leq N_1$ and $0 \leq I_0 \leq 1$. Thus, the system (3.3) is studied in the region $\Omega = \{(U,V,N,I) \in \mathbb{R}^4_+ : 0 \leq U \leq K, 0 \leq V \leq A, 0 \leq N \leq N_1, 0 \leq I \leq 1\}$.

3.3 Model analysis

3.3.1 Boundedness

All the parameters of system (3.3) are non-negative, and so the corresponding right-hand side of the system is a smooth function of the variables (U, V, N, I) in the region Ω. It follows that local existence and uniqueness properties hold for the solution of the system.

Proposition 3.1 $\mathbb{R}^4_+$ *is an invariant set.*

Proof. The system (3.3) is homogeneous. It follows that the coordinate planes $U = 0$, $V = 0$, $N = 0$ and $I = 0$ are solutions for it. By the existence and uniqueness theorem, any trajectory emanating from $\mathbb{R}^4_+$ stays there and can not cross the coordinates coordinate planes. Thus, $\mathbb{R}^4_+$ is an invariant set. □

Proposition 3.2 $\Omega = \{(U, V, N, I) \in \mathbb{R}^4_+ : 0 \leq U \leq K, 0 \leq V \leq A, 0 \leq N \leq N_1, 0 \leq I \leq 1\}$ *in invariant under the flow (3.3).*

Proof. Consider the system (3.3). The first equation implies that

$$\frac{dU}{dt} \leq Ur\Big(1 - \frac{U}{K}\Big) = U\frac{r}{K}(K - U)$$

with solution $U(t) \leq \frac{KD}{D+e^{-rt}}$ and $\limsup_{t\to\infty} U(t) \leq K$ by the comparison principle for ODEs. From the second equation,

$$\frac{dV}{dt} \leq V\Big(\frac{lU}{1+eU} - \mu\Big) \leq V(l - \mu)$$

if $e > 1$. The corresponding solution is $V(t) \leq Ae^{(l-\mu)t}$ and $\limsup_{t\to\infty} V(t) \leq A$ if $l < \mu$. The third equation implies that

$$\frac{dN}{dt} \leq N\Big[\frac{[m(1-I)+nI]U}{1+fU} - \nu] \leq N(mK + nK - \nu)$$

with solution $N(t) \leq N_1 e^{[K(m+n)-\nu]t}$ and $\limsup_{t\to\infty} N(t) \leq N_1$ when $(m + n)K < \nu$. It follows from the fourth equation that

$$\frac{dI}{dt} \leq \beta I(1 - I)$$

with solution $I(t) \leq \frac{D}{D+e^{-\beta t}}$ and $\limsup_{t\to\infty} I(t) \leq 1$. □

Proposition 3.3 *All solutions of (3.3) starting in $\bar{\Omega}$ are uniformly bounded if $l < a$, $n < m$, $m < b$ and $c < 1$.*

Proof. Define the function

$$\Pi = U + V + N \tag{3.4}$$

Differentiating Π with respect to time along the solutions of (3.3) gives

$$\frac{d\Pi}{dt} = \frac{dU}{dt} + \frac{dV}{dt} + \frac{dN}{dt}.$$

$$\begin{aligned}
\frac{d\Pi}{dt} &= Ur\Big(1-\frac{U}{K}\Big) - \frac{aUV}{1+eU} - \frac{bNU[(1-I)+cI]}{1+fU} + \frac{lUV}{1+eU} - p(1-I)NV - \mu V \\
&\quad + \frac{[m(1-I)+nI]UN}{1+fU} - \delta IN - \nu N], \\
&= Ur\Big(1-\frac{U}{K}\Big) - \frac{(a-l)UV}{1+eU} - \frac{N[(b-m)-[(b-cb)-(m-n)]I]}{1+fU} - p(1-I)NV \\
&\quad -\mu V - \delta IN - \nu N, \\
&\leq Ur\Big(1-\frac{U}{K}\Big) - \mu V - \nu N, \\
&= Ur\Big(1-\frac{U}{K}\Big) + \phi U - \phi U - \mu V + \phi V - \phi V - \nu N + \phi N - \phi N, \\
&= U\Big(r+\phi-\frac{rU}{K}\Big) - (\mu-\phi)V - (\nu-\phi)N - \phi\Pi.
\end{aligned}$$

Thus,

$$\begin{aligned}
\frac{d\Pi}{dt} + \phi\Pi &= -\frac{r}{K}[U^2 - \frac{K(r+\phi)}{r}U] - (\mu-\phi)V - (\nu-\phi)N, \\
&= -\frac{r}{K}\Big(U - \frac{K(r+\phi)}{2r}\Big)^2 + \frac{K(r+\phi)^2}{4r} - (\mu-\phi)V - (\nu-\phi)N, \\
&\leq \frac{K(r+\phi)^2}{4r} - (\mu-\phi)V - (\nu-\phi)N.
\end{aligned}$$

Choosing ϕ to be such that $\phi \leq \min\{\mu, \nu\}$ the right-hand side will be bounded. Let φ be such that

$$\frac{d\Pi}{dt} + \phi\Pi \leq \frac{K(r+\phi)^2}{4r} = \varphi. \tag{3.5}$$

A solution of this is

$$\Pi(t) \leq Ce^{-\phi t} + \frac{\varphi}{\phi},$$

and

$$\Pi(t) \leq \Pi(0)e^{-\phi t} + \frac{\varphi}{\phi}\Big(1 - e^{-\phi t}\Big) \leq \max\Big(\Pi(0), \frac{\varphi}{\phi}\Big).$$

Moreover, $\limsup \Pi(t) \leq \frac{\varphi}{\phi}$ as $t \to \infty$ independent of initial conditions. □

3.3.2 Steady states

Since the focus is on the growth of animal species, there is need for the steady states of the system to satisfy conditions for non-negativity. Furthermore, it is realised that the predators cannot survive in the complete absence of their prey. As such, the steady states $E(0, V, N, I)$, $E(0, 0, N, I)$, $E(0, 0, 0, I)$, $E(0, V, 0, 0)$, $E(0, 0, N, 0)$ and $E(0, 0, 0, I)$ are not feasible. However, the steady state $E(0, 0, 0, 0)$ always exist. The steady states in which impala are present are studied only. The system (3.3) has four biologically feasible nonnegative steady states.

(i) $E_1(U \neq 0, V = 0, N \neq 0, I = 0)$: The first and third equations of (3.3) reduce to

$$\begin{aligned} r\Big(1 - \frac{U}{K}\Big) - \frac{bN}{1 + fU} &= 0, \\ \frac{mU}{1 + fU} - \nu &= 0, \end{aligned}$$

with the result $E_1(U_1, 0, N_1, 0)$ where $U_1 = \frac{\nu}{m - f\nu}$ and $N_1 = \frac{rm(Km - \nu - fK\nu)}{bK(f\nu - m)^2}$. The equilibrium point E_1 exists if

- $m > f\nu$, growth function of lions exceed their death and
- $K(m - f\nu) > \nu$ or $K > \frac{\nu}{m - f\nu} = U_1$, impala population should not exceed the carrying capacity.

(ii) $E_2(U \neq 0, V \neq 0, N \neq 0, I = 0)$: The first, second and third equations of (3.3) reduce to

$$\begin{aligned} r\Big(1 - \frac{U}{K}\Big) - \frac{aV}{1 + eU} - \frac{bN}{1 + fU} &= 0, \\ \frac{lU}{1 + eU} - pN - \mu &= 0, \\ \frac{mU}{1 + fU} - \nu &= 0, \end{aligned} \tag{3.6}$$

with the result $E_2(U_2, V_2, N_2, 0)$ and $U_2 = \frac{\nu}{m-f\nu}$, $N_2 = \frac{l\nu+f\mu\nu-m\mu-e\mu\nu}{p(m+e\nu-f\nu)}$ and $V_2 = \frac{D}{a}$, and

$$D = (1+eU_2)\Big[r\Big(1-\frac{U_2}{K}\Big) - \frac{bN_2}{1+fU_2}\Big].$$

The equilibrium point E_2 exists if

- $m > f\nu$ and $\nu(l-e\mu) > \mu(m-f\nu)$, that is, $\frac{\nu}{m-f\nu} > \frac{\mu}{l-e\mu}$, ratio of lion mortality to impala biomass conversion efficiency exceeds that of the cheetah.
- $r\Big(1-\frac{U_2}{K}\Big) > \frac{bN_2}{1+fU_2}$, the growth function of impala exceeds their predation rate by lions.
- $m + e\nu - f\nu = m - f\nu + e\nu > 0$, that is, $\frac{m}{\nu} > f - e$, the ratio of cheetah's impala biomass conversion efficiency to mortality exceeds the difference in handling times.

(iii) $E_3(U \neq 0, V = 0, N \neq 0, I \neq 0)$: The first, third and fourth equations of (3.3) reduce to

$$\begin{aligned} r\Big(1-\frac{U}{K}\Big) - \frac{bN[(1-I)+cI]}{1+fU} &= 0, \\ \frac{[m(1-I)+nI]U}{1+fU} - \delta I - \nu &= 0, \\ \beta - \frac{(m-n)U}{1+fU} - \delta &= 0, \end{aligned} \tag{3.7}$$

with the result $E_3(U_3, 0, N_3, I_3)$, where $U_3 = \frac{\beta-\delta}{m-n-f\beta+f\delta} = \frac{\beta-\delta}{(m-n)-f(\beta-\delta)}$, $I_3 = \frac{m\beta-m\delta-m\nu+n\nu}{\beta(m-n)} = \frac{m(\beta-\delta)-\nu(m-n)}{\beta(m-n)}$, and $N_3 = \frac{C}{G}$,

$$\begin{aligned} C &= \frac{1}{b}\Big[r\Big(-(m-n)\delta + (m-n)^2\frac{U_3^2}{K} + (m-n)\frac{f\delta U_3^2}{K} - (m-n)^2U_3 \\ &\quad -(m-n)f\delta U_3 + (m-n)\frac{\delta U_3}{K}\Big)\Big], \\ &= \frac{r(m-n)}{b}\Big[(m-n)\frac{U_3^2}{K} + \frac{f\delta U_3^2}{K} - (m-n)U_3 - f\delta U_3 + \frac{\delta U_3 3}{K} - \delta\Big], \\ &= \frac{r(m-n)}{b}\Big[\frac{U_3^2}{K}[(m-n)+f\delta] - U_3[(m-n)+f\delta] + \delta[\frac{U_3}{K}-1]\Big], \\ &= \frac{r(m-n)}{b}\Big[(\frac{U_3}{K}-1)[U_3[(m-n)+f\delta] + \delta[\frac{U_3}{K}-1]\Big], \\ &= \frac{r(m-n)}{b}(\frac{U_3}{K}-1)\Big[U_3[(m-n)+f\delta] + \delta\Big] < 0, \end{aligned}$$

as long as $U_3 < K$; and

$$\begin{aligned} G &= n\beta - m\delta - m\nu + n\nu - (m-n)c\beta I, \\ &= n(\beta + \nu + c\beta I) - m(\delta + \nu + c\beta I), \\ &= (n-m)(\nu + c\beta I) + (n\beta - m\delta) < 0, \end{aligned}$$

if $n\beta - m\delta < 0$ or $\frac{\beta}{\delta} < \frac{m}{n}$, that is, the ratio of disease incidence to disease induced death is less than that of the conversion rate of impala biomass into new susceptible lions to infected ones. Hence, $N_3 > 0$. The equilibrium point E_3 exists if

- $m > n$, conversion rate of impala biomass into new lions for susceptible lions exceeds that of infected ones;
- $\beta > \delta$, disease standard incidence exceeds disease-induced mortality rate of infected lions;
- $(m-n) > f(\beta - \delta)$, $m(\beta - \delta) > \nu(m-n)$, that is, $\frac{m}{\nu}(\beta - \delta) > (m-n) > f(\beta - \delta)$ or $m - f\nu > 0$ (condition for existence of E_3) and $N_3 > 0$.

The mass of the total number of individuals in a species is often referred to as its biomass, here the population times the unit mass.

(iv) $E_4(U \neq 0, V \neq 0, N \neq 0, I \neq 0)$: The system (3.3) becomes

$$\begin{aligned} r\Big(1 - \frac{U}{K}\Big) - \frac{aV}{1+eU} - \frac{bN[(1-I)+cI]}{1+fU} &= 0, \\ \frac{lU}{1+eU} - p(1-I)N - \mu &= 0, \\ \frac{[m(1-I)+nI]U}{1+fU} - \delta I - \nu &= 0, \\ \beta - \frac{(m-n)U}{1+fU} - \delta &= 0, \end{aligned}$$

with $E_4(U_4, V_4, N_4, I_4)$ given by

$$
\begin{aligned}
U_4 &= \frac{\beta-\delta}{m-n-f\beta+f\delta} = \frac{\beta-\delta}{(m-n)-f(\beta-\delta)} > 0,\\
I_4 &= \frac{m\beta-m\delta-m\nu+n\nu}{\beta(m-n)} = \frac{m(\beta-\delta)-\nu(m-n)}{\beta(m-n)} > 0,\\
N_4 &= \frac{\beta(m-n)(\mu-\frac{lU_4}{1+eU_4})}{p(n\beta-m\delta+(n-m)\nu)},\\
V_4 &= \frac{1}{a}\Big(r-\frac{erU_4^2}{K}+erU_4-\frac{rU_4}{K}-\frac{b\beta N_4}{1+fU_4}-\frac{bc\beta I_4N_4}{1+fU_4}-\frac{be\beta U_4N_4}{1+fU_4}\\
&\quad -\frac{bce\beta I_4U_4N_4}{1+fU_4}+\frac{bI_4N_4}{1+fU_4}+be\beta N_4U_4I_4\Big),\\
&= \frac{1}{a}\Big(r(1-\frac{U_4}{K})+erU_4(1-\frac{U_4}{K})+be\beta N_4U_4I_4\\
&\quad +\frac{bN_4}{1+fU_4}(I_4-\beta-c\beta I_4-e\beta U_4-ce\beta I_4U_4)\Big),\\
&= \frac{1}{a}\Big(r(1+eU_4)(1-\frac{U_4}{K})\\
&\quad +be\beta N_4U_4I_4+\frac{bN_4}{1+fU_4}(I_4-\beta(1+cI_4)(1-eU_4))\Big).
\end{aligned}
$$

The equilibrium point E_4 exists if

- $\mu < \frac{lU_4}{1+eU_4}$, cheetah's death rate is less than its growth function, and $(n\beta - m\delta) + (n-m)\nu < 0$. Hence, $N_4 > 0$.
- $U_4 < K$ and $I_4 > \beta(1+cI_4)(1-eU_4)$, in which case $V_4 > 0$.

3.3.3 Analysis of steady states

The Jacobian matrix for the system (3.3) is given by

$$
J = \begin{bmatrix}
j_{11} & \frac{-aU}{1+eU} & -\frac{bU(1+cI-I)}{1+fU} & \frac{bUN}{1+fU}\\
\frac{lV}{(1+eU)^2} & \frac{lU}{1+eU}-Np(1-I)-\mu & -pV(1-I) & pNV\\
\frac{(m-(m-n)I)N}{(1+fU)^2} & 0 & \frac{U(m-mI+nI)}{1+fU}-\delta I-\nu & -\frac{(m-n)UN}{1+fU}-\delta N\\
-\frac{(m-n)I}{(1+fU)^2} & 0 & 0 & \beta-\frac{(m-n)U}{1+fU}-\delta
\end{bmatrix}
$$

where $j_{11} = r - \frac{2rU}{K} - \frac{aV}{(1+eU)^2} - \frac{bN(1+cI-I)}{(1+fU)^2}$.

At any steady state solution, the Jacobian matrix is computed. Let J_k denote the Jacobian evaluated at E_k and $j_{ij}^{[k]}$, $i = 1, 2, 3, 4$, $j = 1, 2, 3, 4$, $k = 1, 2, 3, 4$, the corresponding entries.

Local and global stability of the steady state E_1

For the equilibrium point $E_1(\frac{\nu}{m-f\nu}, 0, \frac{rm(Km-\nu-fK\nu)}{bK(f\nu-m)^2}, 0)$, the Jacobian matrix is given by

$$J_1 = \begin{bmatrix} r - \frac{2rU_1}{K} - \frac{bN_1}{(1+fU_1)^2} & -\frac{aU_1}{1+eU_1} & -\frac{bU_1}{1+fU_1} & \frac{bU_1N_1}{1+fU_1} \\ 0 & -\mu - pN_1 + \frac{lU_1}{1+eU_1} & 0 & 0 \\ -\frac{fmU_1N_1}{1+fU_1} + \frac{mN_1}{1+fU_1} & 0 & \frac{mU_1}{1+fU_1} - \nu & -\delta N_1 - \frac{(m-n)U_1N_1}{1+fU_1} \\ 0 & 0 & 0 & \beta - \delta - \frac{(m-n)U_1}{1+fU_1} \end{bmatrix}$$

The eigenvalues of J_1 are
$\lambda_1^{[1]} = \beta - \delta - \nu + \frac{n\nu}{m}$, $\lambda_2^{[1]} = -\mu - \frac{prm(Km-\nu-fK\nu)}{bK(f\nu-m)^2} + \frac{l\nu}{m+(e-f)\nu}$; $\lambda_3^{[1]} = \frac{-b_1+\sqrt{b_1^2-4b_0b_2}}{2b_0}$ and $\lambda_4^{[1]} = \frac{-b_1-\sqrt{b_1^2-4b_0b_2}}{2b_0}$, for which $\lambda_3^{[1]}$ and $\lambda_4^{[1]}$ are the roots of

$$b_0\lambda^2 + b_1\lambda + b_2 = 0$$

where

$$\begin{aligned} b_0 &= Km(m - f\nu), \\ b_1 &= mr\nu - fKrm\nu + fr\nu^2 + f^2Kr\nu^2, \\ b_2 &= Km^2r\nu - mr\nu^2 + 2fKmr\nu^2 + fr\nu^3 + f^2Kr\nu^3 + 2elrN_1^3 - 2e^2r\mu N_1^3 - aK\mu V_1. \end{aligned}$$

Note that $b_0 > 0$, that is, condition for existence of equilibrium point E_1.

$$\begin{aligned} b_1 &= mr\nu - fKrm\nu + fr\nu^2 + f^2Kr\nu^2, \\ &= r\nu(m + f\nu) - fKr\nu(m - f\nu), \\ &= r\nu[(m + f\nu) - fK(m - f\nu)]. \end{aligned}$$

Thus, $b_1 > 0$ if $\frac{m+f\nu}{m-f\nu} > fK$.

$$
\begin{aligned}
b_2 &= Km^2r\nu - mr\nu^2 + 2fKmr\nu^2 + fr\nu^3 + f^2Kr\nu^3 + 2elrN_1^3 - 2e^2r\mu N_1^3 - aK\mu V_1, \\
&= Kr\nu(m^2 + 2fm\nu + f^2\nu^2) - r\nu^2(m - f\nu) + 2erN_1^3(l - e\mu) - aK\mu V_1, \\
&= Kr\nu[(m + f\nu)^2 + f^2\nu^2) - r\nu^2(m - f\nu) + 2erN_1^3(l - e\mu) - aK\mu V_1, \\
&= r\nu[K(m + f\nu)^2 + f^2\nu^2) - \nu(m - f\nu) + 2erN_1^3(l - e\mu) - aK\mu V_1.
\end{aligned}
$$

Thus, $b_2 > 0$ if $\frac{(m+f\nu)^2}{(m-f\nu)} > \frac{\nu}{K}$ and $2erN_1^3(l - e\mu) > aK\mu V_3$.

Lemma 3.1 *The system (3.3) is locally asymptotically stable about E_1 if*

(a) $\frac{l\nu}{m+(e-f)\nu} < \mu + \frac{prm(Km-\nu-fK\nu)}{bK(f\nu-m)^2}$, *that is, the sum of the cheetah's killing and death rates exceeds their growth rate,*

(b) $\beta + \frac{n\nu}{m} < \delta + \nu$, *that is,* $m\beta + n\nu < m(\delta + \nu)$,

(c) $b_1 > 0$.

Now, the global stability of E_1 is discussed whenever it exists, using the Bendixson-Dulac criterion, Theorem A.1, [124] and the Poincaré-Bendixson Theorem, Theorem A.2, [125].

Proposition 3.4 *E_1 is globally asymptotically stable if $2\nu > mK$ and $m > f\nu$.*

Proof. Consider subsystem (ii) of system (3.3)

$$
\begin{aligned}
\frac{dU}{dt} &= Ur\Big(1 - \frac{U}{K}\Big) - \frac{bN}{1 + fU}, \\
\frac{dN}{dt} &= \frac{mNU}{1 + fU} - \nu N.
\end{aligned} \tag{3.8}
$$

Let $\Phi(U, N) = \frac{1}{UN}$, $h_1(U, N) = Ur\Big(1 - \frac{U}{K}\Big) - \frac{bUN}{1+fU}$, $h_2(U, N) = \frac{mUN}{1+fU} - \mu N$. Now $\Phi(U, N) > 0$ in the interior of the UN plane. It follows that

$$\begin{aligned}
\Delta(U,N) &= \frac{\partial(\Phi h_1)}{\partial U} + \frac{\partial(\Phi h_2)}{\partial N}, \\
&= \frac{\partial}{\partial U}\Big[\frac{r}{N}\Big(1-\frac{U}{K}\Big) - \frac{b}{1+fU}\Big] + \frac{\partial}{\partial N}\Big[\frac{m}{1+fU} - \frac{\nu}{U}\Big], \\
&= -\frac{r}{KN} + \frac{bf}{(1+fU)^2}, \\
&= \frac{bfKN - r(1+fU)^2}{KN(1+fU)^2}.
\end{aligned}$$

Now Δ is not identically zero in the positive quadrant of the UN plane. Thus, $\Delta(U,N) < 0$ and does not change sign if

$$bfKN - r(1+fU)^2 = bfKN - r - 2rfU - rf^2U^2 = -r - (2rU - bKN)f - rf^2U^2 < 0$$

that is, if $2rU - bKN > 0$. This means that at E_1, we have

$$\begin{aligned}
2rU_1 - bKN_1 &= \frac{2r\nu}{m-f\nu} - \frac{rm(Km-\nu-fK\nu)}{(m-f\nu)^2}, \\
&= \frac{2r\nu(m-f\nu) - rm(Km-\nu-fK\nu)}{(m-f\nu)^2}, \\
&= \frac{2r\nu(m-f\nu) - rmK(m-f\nu) + \nu rm}{(m-f\nu)^2}, \\
&= \frac{r(m-f\nu)(2\nu-mK) + \nu rm}{(m-f\nu)^2} > 0,
\end{aligned}$$

if and only if $2\nu > mK$ and $m > f\nu$. $\frac{\nu}{m}$ refers to the ratio of mortality of lions to lion's conversion efficiency of impala biomass to lion biomass. The carrying capacity of impala population must be less than the output/input ratio of lion biomass. Furthermore, the ratio of input/output of lion biomass must outweigh the time the lion spends consuming impala. Thus, subsystem (3.8) does not have a limit cycle in $\bar{\Omega}$. Since E_1 is locally asymptotically stable, the Poincaré-Bendixson Theorem, Theorem A.2, [125] and Proposition 3.3 imply that the equilibrium point E_1 is globally asymptotically stable in the region $\bar{\Omega}$. □

Local and global stability of the steady state E_2

For the equilibrium point $E_2(U_2, V_2, N_2, 0)$ the Jacobian matrix is given by

$$J_2 = \begin{bmatrix} r - \frac{2rU_2}{K} - \frac{bN_2}{(1+fU_2)^2} - \frac{aV_2}{(1+eU_2)^2} & -\frac{aU_2}{1+eU_2} & -\frac{bU_2}{1+fU_2} & \frac{bU_2N_2}{1+fU_2} \\ \frac{lV_2}{(1+eU_2)^2} & \frac{lU_2}{1+eU_2} - \mu - pN_2 & -pV_2 & pN_2V_2 \\ \frac{mN_2}{1+fU_2} & 0 & \frac{mU_2}{1+fU_2} - \nu & -\delta N_2 - \frac{(m-n)U_2N_2}{1+fU_2} \\ 0 & 0 & 0 & \beta - \delta - \frac{(m-n)U_2}{1+fU_2} \end{bmatrix}.$$

The local stability of the disease-free equilibrium at E_2 is deduced from the eigenvalues of the Jacobian matrix J_2. From the fourth row this matrix has the eigenvalue $\beta - \delta - \frac{(m-n)U_2}{1+fU_2}$, and the remaining eigenvalues are derived from the 3×3 Jacobian matrix given by

$$J_2^{'} = \begin{bmatrix} j_{11}^{[2]} & -\frac{aU_2}{1+eU_2} & -\frac{bU_2}{1+fU_2} \\ \frac{lV_2}{(1+eU_2)^2} & \frac{lU_2}{1+eU_2} - \mu - pN_2 & -pV_2 \\ \frac{mN_2}{1+fU_2} & 0 & \frac{mU_2}{1+fU_2} - \nu \end{bmatrix}, \tag{3.9}$$

where $j_{12}^{[2]} < 0$, $j_{13}^{[2]} < 0$, $j_{21}^{[2]} > 0$, $j_{23}^{[2]} < 0$ and $j_{31}^{[2]} > 0$.

The characteristic polynomial of the Jacobian of the system evaluated at this point is given by

$$\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0, \tag{3.10}$$

where

$$\begin{aligned} a_1 &= -(j_{11}^{[2]} + j_{22}^{[2]} + j_{33}^{[2]}) = -\mathrm{Tr}(J_2^{'}), \\ a_2 &= j_{11}^{[2]}j_{22}^{[2]} + j_{11}^{[2]}j_{33}^{[2]} + j_{22}^{[2]}j_{33}^{[2]} - j_{12}^{[2]}j_{21}^{[2]} - j_{13}^{[2]}j_{31}^{[2]}, \\ a_3 &= -j_{12}^{[2]}j_{23}^{[2]}j_{31}^{[2]} + j_{13}^{[2]}j_{22}^{[2]}j_{31}^{[2]} + j_{12}^{[2]}j_{21}^{[2]}j_{33}^{[2]} - j_{11}^{[2]}j_{22}^{[2]}j_{33}^{[2]} = -\mathrm{Det}J_2^{'}. \end{aligned}$$

Now, if $j_{11}^{[2]} < 0$, $j_{22}^{[2]} < 0$, $j_{33}^{[2]} < 0$ then $a_1 > 0$ and $a_2 > 0$. $a_3 > 0$ if $j_{13}^{[2]}j_{22}^{[2]}j_{31}^{[2]} + j_{12}^{[2]}j_{21}^{[2]}j_{33}^{[2]} - j_{11}^{[2]}j_{22}^{[2]}j_{33}^{[2]} > j_{12}^{[2]}j_{23}^{[2]}j_{31}^{[2]}$. If at least one of these conditions is not satisfied, then a_1, a_2 or a_3 is negative and the characteristic polynomial will have at least one positive real root, that is, not all eigenvalues are negative in this case.

Furthermore,

$$\begin{aligned} a_1a_2 - a_3 &= j_{11}^{[2]}j_{12}^{[2]}j_{21}^{[2]} - j_{11}^{2[2]}j_{22}^{[2]} + j_{12}^{[2]}j_{21}^{[2]}j_{22}^{[2]} - j_{11}^{[2]}j_{22}^{2[2]} + j_{11}^{[2]}j_{13}^{[2]}j_{31}^{[2]} + j_{12}^{[2]}j_{23}^{[4]}j_{31}^{[4]} \\ &\quad - j_{11}^{2[2]}j_{33}^{[2]} - 2j_{11}^{[2]}j_{22}^{[2]}j_{33}^{[2]} - j_{22}^{2[2]}j_{33}^{[2]} + j_{13}^{[2]}j_{31}^{[2]}j_{33}^{[2]} - j_{11}^{[2]}j_{33}^{2[2]} - j_{22}^{[4]}j_{33}^{2[4]}, \\ &> 0. \end{aligned}$$

Application of the Routh-Hurwitz criterion [126] leads to the conclusion that the characteristic equation (3.10) has negative real parts. Thus, the following result can be stated.

Lemma 3.2 *The equilibrium point E_2 is locally asymptotically stable if*

(a) $r < \frac{2rU_2}{K} + \frac{bN_2}{(1+fU_2)^2} + \frac{aV_2}{(1+eU_2)^2}$,

(b) $\frac{lU_2}{1+eU_2} < \mu + pN_2$,

(c) $\frac{mU_2}{1+fU_2} - \nu$,

(d) $\beta < \delta + \frac{(m-n)U_2}{1+fU_2}$.

To analyse global stability of the equilibrium point E_2, the last equation in system equation (3.3) is dropped to obtain the system (3.6), since the equilibrium point does not depend on I. The Jacobian matrix of system (3.6) is given by (3.9). The system (3.6) is not competitive and so the geometric approach to global stability problems method is used. The framework for this method is developed in the papers of Smith [127] and Li and Muldowney [128] and was used by Buonomo and Lacitignola [129], Li *et al.* [130] and Tian and Wang [131]. This framework has been used to prove the global stability of the equilibrium point E_2.

The second compound matrix of the system (3.6) is

$$J_2^{'[2]} = \begin{bmatrix} j_{11}^{[2]} + \frac{lU_2}{1+eU_2} - \mu - pN_2 & -pV_2 & \frac{bU_2}{1+fU_2} \\ 0 & j_{11}^{[2]} + \frac{mU_2}{1+fU_2} - \nu & -\frac{aU_2}{1+eU_2} \\ -\frac{mN_2}{1+fU_2} & \frac{lV_2}{(1+eU_2)^2} & \frac{lU_2}{1+eU_2} - \mu - pN_2 + \frac{mU_2}{1+fU_2} - \nu \end{bmatrix}.$$

The matrix function P is set by

$$P(U_2, V_2, N_2) = \mathrm{diag}\Big\{1, \frac{V_2}{N_2}, \frac{V_2}{N_2}\Big\}.$$

Then

$$P_F = \begin{bmatrix} 0 & 0 & 0 \\ 0 & \frac{V_2}{N_2}\Big[\frac{V_2'}{V_2} - \frac{N_2'}{N_2}\Big] & 0 \\ 0 & 0 & \frac{V_2}{N_2}\Big[\frac{V_2'}{V_2} - \frac{N_2'}{N_2}\Big] \end{bmatrix}, \qquad P^{-1} = \begin{bmatrix} 1 & 0 & 0 \\ 0 & \frac{N_2}{V_2} & 0 \\ 0 & 0 & \frac{N_2}{V_2} \end{bmatrix},$$

and

$$P_F P^{-1} = \begin{bmatrix} 0 & 0 & 0 \\ 0 & \frac{V_2'}{V_2} - \frac{N_2'}{N_2} & 0 \\ 0 & 0 & \frac{V_2'}{V_2} - \frac{N_2'}{N_2} \end{bmatrix}.$$

Now

$$P_F J_2^{'[2]} P^{-1} = \begin{bmatrix} j_{11}^{[2]} + \frac{lU_2}{1+eU_2} - \mu - pN_2 & -pN_2 & \frac{bU_2}{1+fU_2}\frac{N_2}{V_2} \\ 0 & j_{11}^{[2]} + \frac{mU_2}{1+fU_2} - \nu & -\frac{aU_2}{1+eU_2} \\ -\frac{mN_2}{(1+fU_2)^2}\frac{V_2}{N_2} & \frac{lV_2}{(1+eU_2)^2} & \frac{lU_2}{1+eU_2} - \mu - pN_2 + \frac{mU_2}{1+fU_2} - \nu \end{bmatrix}.$$

The matrix $Q = P_F P^{-1} + P_F J_2^{'[2]} P^{-1}$ can be written in block form:

$$Q = \begin{bmatrix} Q_{11} & Q_{12} \\ Q_{21} & Q_{22} \end{bmatrix},$$

with $Q_{11} = j_{11}^{[2]} + \dfrac{lU_2}{1+eU_2} - \mu - pN_2$,

$$\begin{aligned} Q_{12} &= \begin{bmatrix} -pN_2 & \frac{bU_2}{1+fU_2}\frac{N_2}{V_2} \end{bmatrix}, \qquad Q_{21} = \begin{bmatrix} 0 \\ -\frac{mN_2}{(1+fU_2)^2}\frac{V_2}{N_2} \end{bmatrix}, \\ Q_{22} &= \begin{bmatrix} j_{11}^{[2]} + \frac{mU_2}{1+fU_2} - \nu + \frac{V_2'}{V_2} - \frac{N_2'}{N_2} & -\frac{aU_2}{1+eU_2} \\ \frac{lV_2}{(1+eU_2)^2} & \frac{lU_2}{1+eU_2} - \mu - pN_2 + \frac{mU_2}{1+fU_2} - \nu + \frac{V_2'}{V_2} - \frac{N_2'}{N_2} \end{bmatrix}. \end{aligned}$$

A norm in $\mathbb{R}^3$ is defined as

$$|u, v, n| = \max\{|u|, |v| + |n|\}$$

for any vector $(u, v, n) \in \mathbb{R}^3$. Let m denote the Lozinskiĭ measure with respect to this norm. Then

$$m(Q) \leq \sup\{g_1, g_2\} \tag{3.11}$$

with

$$\begin{aligned} g_1 &= m_1(Q_{11}) + |Q_{12}|, \\ g_2 &= |Q_{21}| + m_1(Q_{22}), \end{aligned}$$

where $|Q_{12}|$ and $|Q_{21}|$ are matrix norms induced by the L_1 vector norm, and m_1 denotes the Lozinskiĭ measure with respect to the L_1 norm. More specifically,

$m_1(Q_{11}) = j_{11}^{[2]} + \frac{lU_2}{1+eU_2} - \mu - pN_2 = r\Big(1 - \frac{U_2}{K}\Big) - \frac{rU_2}{K} - \frac{(bN_2)^2}{1+fU_2} - \frac{aV_2}{(1+eU_2)^2} + \frac{lU_2}{1+eU_2} - \mu - pN_2,$
$|Q_{12}| = \max\{-pN_2, \frac{bU_2}{1+fU_2}\frac{N_2}{V_2}\} = \overline{a}$, $|Q_{21}| = \frac{mN_2}{(1+fU_2)^2}\frac{V_2}{N_2}$.

To calculate $m_1(Q_{22})$, the absolute value of the off-diagonal elements is added to the diagonal one in each column of Q_{22} and the maximum of the two sums is taken. Thus,

$$\begin{aligned} & m_1(Q_{22}) \\ = \; & \max\Big\{r\Big(1 - \frac{U_2}{K}\Big) - \frac{rU_2}{K} - \frac{(bN_2)^2}{1+fU_2} - \frac{aV_2}{(1+eU_2)^2} + \frac{mU_2}{1+fU_2} - \nu + \frac{V_2'}{V_2} - \frac{N_2'}{N_2} \\ & + \frac{lV_2}{(1+eU_2)^2}; \frac{lU_2}{1+eU_2} - \mu - pN_2 + \frac{mU_2}{1+fU_2} - \nu + \frac{V_2'}{V_2} - \frac{N_2'}{N_2} + \frac{aU_2}{1+eU_2}\Big\}, \\ = \; & \frac{V_2'}{V_2} - \frac{N_2'}{N_2} + \frac{mU_2}{1+fU_2} - \nu + \max\Big\{r\Big(1 - \frac{U_2}{K}\Big) - \frac{rU_2}{K} - \frac{(bN_2)^2}{1+fU_2} \\ & - \frac{aV_2}{(1+eU_2)^2} + \frac{lV_2}{(1+eU_2)^2}; \frac{lU_2}{1+eU_2} - \mu - pN_2 + \frac{aU_2}{1+eU_2}\Big\}, \\ = \; & \frac{V_2'}{V_2} - \frac{N_2'}{N_2} + \frac{mU_2}{1+fU_2} - \nu + \overline{b}, \end{aligned}$$

where $\overline{b} = \max\{r\Big(1 - \frac{U_2}{K}\Big) - \frac{rU_2}{K} - \frac{(bN_2)^2}{1+fU_2} - \frac{aV_2}{(1+eU_2)^2} + \frac{lV_2}{(1+eU_2)^2}; \frac{lU_2}{1+eU_2} - \mu - pN_2 + \frac{aU_2}{1+eU_2}\}$.

Therefore

$$\begin{aligned} g_1 &= r\Big(1 - \frac{U_2}{K}\Big) - \frac{rU_2}{K} - \frac{(bN_2)^2}{1+fU_2} - \frac{aV_2}{(1+eU_2)^2} + \frac{lU_2}{1+eU_2} - \mu - pN_2 + \overline{a}, \\ g_2 &= \frac{V_2'}{V_4} - \frac{N_2'}{N_2} + \frac{mU_2}{1+fU_2} - \nu + \overline{b} + \frac{mN_2}{(1+fU_2)^2}\frac{V_2}{N_2}. \end{aligned} \tag{3.12}$$

Rewriting (3.3) with $I = 0$ leads to

$$\frac{V_2'}{V_2} = \frac{lU_2}{1+eU_2} - pN_2 - \mu, \tag{3.13}$$

$$\frac{N_2'}{N_2} = \frac{mU_2}{1+fU_2} - \nu. \tag{3.14}$$

Substituting (3.13) and (3.14) into (3.12) leads to

$$\begin{aligned} g_1 &= \frac{V_2'}{V_2} + r\Big(1-\frac{U_2}{K}\Big) - \frac{rU_2}{K} - \frac{(bN_2)^2}{1+fU_2} - \frac{aV_2}{(1+eU_2)^2} + \overline{a} \le \frac{V_2'}{V_2} - \overline{d}, \\ g_2 &= \frac{V_2'}{V_2} + \overline{b} + \frac{mN_2}{(1+fU_2)^2}\frac{V_2}{N_2} \le \frac{V_2'}{V_2} - \overline{d}. \end{aligned} \tag{3.15}$$

where $\overline{d} = \min\Big\{ -r\Big(1-\frac{U_2}{K}\Big) + \frac{rU_2}{K} + \frac{(bN_2)^2}{1+fU_2} + \frac{aV_2}{(1+eU_2)^2} - \overline{a}, -\frac{mN_2}{(1+fU_2)^2}\frac{V_2}{N_2} - \overline{b}\Big\}$. Therefore

$$m(Q) \le \frac{V_2'}{V_2} - \overline{d},$$

for $t > T$ by (3.11) and (3.15). Along each solution $x(t, x_0)$ to (3.3) such that $x_0 \in K$ and for $t > T$, we thus have

$$\frac{1}{t}\int_0^t m(Q)ds \le \frac{1}{t}\int_0^T m(Q)ds + \frac{1}{t}\log\frac{V(t)}{V(T)} - \overline{d}\ \ \frac{t-T}{t},$$

which implies that $m(Q) \le -\overline{d}/2 < 0$. Hence, the following theorem is established .

Theorem 3.1 *The equilibrium E_2 of the system (3.3) is globally asymptotically stable in Ω if $m > f\nu$, $\frac{\nu}{m-f\nu} > \frac{\mu}{l-e\mu}$, $r\Big(1-\frac{U_2}{K}\Big) > \frac{bN_2}{1+fU_2}$, $m - f\nu + e\nu > 0$, $\beta > \delta + \dfrac{(m-n)U_2}{1+fU_2}$, $d_{11}^{[2]} < 0$, $d_{22}^{[2]} < 0$ and $d_{33}^{[2]} < 0$.*

Local and global stability of the steady state E_3

For the equilibrium point $E_3(U_3, 0, N_3, I_3)$, the Jacobian matrix is given by

$$J_3 = \begin{bmatrix} j_{11}^{[3]} & -\frac{aU_3}{1+eU_3} & -\frac{bU_3(1+(c-1)I_3)}{1+fU_3} & \frac{bU_3N_3}{1+fU_3} \\ 0 & j_{22}^{[3]} & 0 & 0 \\ \frac{[m-(m-n)I_3]N_3}{(1+fU_3)^2} & 0 & j_{33}^{[3]} & -\delta N_3 - \frac{(m-n)U_3N_3}{1+fU_3} \\ -\frac{(m-n)U_3}{(1+fU_3)^2} & 0 & 0 & \beta - \delta - \frac{(m-n)U_3}{1+fU_3} \end{bmatrix},$$

where

$j_{11}^{[3]} = r - \frac{2rU_3}{K} - \frac{bN_3(1+(c-1)I_3)}{(1+fU_3)^2}$, $j_{22}^{[3]} = \dfrac{lU_3}{1+eU_3} - \mu - pN_3(1-I_3)$ and

$j_{33}^{[3]} = \frac{(m-(m-n)I_3)U_3}{1+fU_3} - \delta I_3 - \nu.$

The local stability of the equilibrium at E_3 is established from the eigenvalues of the Jacobian matrix J_3. From the second row this matrix has the eigenvalue $\frac{lU_3}{1+eU_3} - \mu - pN_3(1-I_3)$, and the remaining eigenvalues are derived from the 3×3 Jacobian matrix given by

$$J_3' = \begin{bmatrix} j_{11}^{[3]} & -\frac{bU_3(1+(c-1)I_3)}{1+fU_3} & \frac{bU_3N_3}{1+fU_3} \\ \frac{[m-(m-n)I_3]N_3}{(1+fU_3)^2} & j_{33}^{[3]} & -\delta N_3 - \frac{(m-n)U_3N_3}{1+fU_3} \\ -\frac{(m-n)U_3}{(1+fU_3)^2} & 0 & \beta - \delta - \frac{(m-n)U_3}{1+fU_3} \end{bmatrix}.$$

The characteristic polynomial of the Jacobian of the system evaluated at this point is given by

$$\lambda^3 + h_1\lambda^2 + h_2\lambda + h_3 = 0,$$

$$\begin{aligned} h_1 &= -(j_{11}^{[3]} + j_{33}^{[3]} + j_{44}^{[3]}) = -\mathrm{Tr}(J_3'), \\ h_2 &= j_{11}^{[3]}j_{33}^{[3]} + j_{11}^{[3]}j_{44}^{[3]} + j_{33}^{[3]}j_{44}^{[3]} - j_{13}^{[3]}j_{31}^{[3]} - j_{14}^{[3]}j_{41}^{[3]}, \\ h_3 &= -j_{13}^{[3]}j_{34}^{[3]}j_{41}^{[3]} + j_{14}^{[3]}j_{33}^{[3]}j_{41}^{[3]} + j_{13}^{[3]}j_{31}^{[3]}j_{44}^{[3]} - j_{11}^{[3]}j_{33}^{[3]}j_{44}^{[3]} = -\mathrm{Det}J_3', \end{aligned}$$

Now, if $j_{11}^{[3]} < 0$, $j_{33}^{[3]} < 0$, $j_{44}^{[3]} < 0$, then $h_1 > 0$, $h_2 > 0$ and $h_3 > 0$. If at least one of these conditions is not satisfied, then h_1, h_2 or h_3 is negative and the characteristic polynomial will have at least one positive real root, that is, not all eigenvalues are negative in this case. Furthermore,

$$\begin{aligned} h_1h_2 - h_3 &= j_{11}^{[3]}j_{13}^{[3]}j_{31}^{[3]} - j_{11}^{2[3]}j_{33}^{[3]} + j_{13}^{[3]}j_{31}^{[3]}j_{33}^{[3]} - j_{11}^{[3]}j_{33}^{2[3]} + j_{11}^{[3]}j_{14}^{[3]}j_{41}^{[3]} + j_{13}^{[3]}j_{34}^{[3]}j_{41}^{[3]} \\ &\quad - j_{11}^{2[3]}j_{44}^{[3]} - 2j_{11}^{[3]}j_{33}^{[3]}j_{44}^{[3]} - j_{33}^{2[3]}j_{44}^{[3]} + j_{14}^{[3]}j_{41}^{[3]}j_{44}^{[3]} - j_{11}^{[3]}j_{44}^{2[3]} - j_{33}^{[3]}j_{44}^{2[3]}, \\ &> 0, \end{aligned}$$

if

$j_{11}^{[3]}j_{13}^{[3]}j_{31}^{[3]} - j_{11}^{2[3]}j_{33}^{[3]} + j_{13}^{[3]}j_{31}^{[3]}j_{33}^{[3]} - j_{11}^{[3]}j_{33}^{2[3]} + j_{11}^{[3]}j_{14}^{[3]}j_{41}^{[3]} - j_{11}^{2[3]}j_{44}^{[3]} - 2j_{11}^{[3]}j_{33}^{[3]}j_{44}^{[3]} - j_{33}^{2[3]}j_{44}^{[3]} + j_{14}^{[3]}j_{41}^{[3]}j_{44}^{[3]} - j_{11}^{[3]}j_{44}^{2[3]} - j_{33}^{[3]}j_{44}^{2[3]} > -j_{13}^{[3]}j_{34}^{[3]}j_{41}^{[3]}$.

Thus, the following result can be stated.

Lemma 3.3 *The equilibrium point E_3 is locally asymptotically stable if*

(a) $r < \frac{2rU_3}{K} + \frac{bN_3(1+(c-1)I_3)}{(1+fU_3)^2}$,

(b) $\frac{lU_3}{1+eU_3} < \mu + pN_3(1-I_3)$,

(c) $\frac{(m-(m-n)I_3)U_3}{1+fU_3} < \delta I_3 + \nu$,

(d) $\beta < \delta + \frac{(m-n)U_3}{1+fU_3}$.

The second compound matrix corresponding to $J_3^{'}$ is given by

$$J_3^{'[2]} = \begin{bmatrix} j_{11}^{[3]} + j_{33}^{[3]} & -\delta N_3 - \frac{(m-n)U_3N_3}{1+fU_3} & -\frac{bU_3N_3}{1+fU_3} \\ 0 & j_{11}^{[3]} + \beta - \delta - \frac{(m-n)U_3}{1+fU_3} & -\frac{bU_3(1+(c-1)I_3)}{1+fU_3} \\ \frac{(m-n)I_3}{(1+fU_3)^2} & \frac{[m-(m-n)I_3]N_3}{(1+fU_3)^2} & j_{33}^{[3]} + \beta - \delta - \frac{(m-n)U_3}{1+fU_3} \end{bmatrix}.$$

The matrix function P is set as

$$P(U_3, N_3, I_3) = \text{diag}\Big\{1, \frac{N_3}{I_3}, \frac{N_3}{I_3}\Big\}.$$

Then

$$P_F = \begin{bmatrix} 0 & 0 & 0 \\ 0 & \frac{N_3}{I_3}\left[\frac{N_3'}{N_3} - \frac{I_3'}{I_3}\right] & 0 \\ 0 & 0 & \frac{N_3}{I_3}\left[\frac{N_3'}{N_3} - \frac{I_3'}{I_3}\right] \end{bmatrix}, \qquad P^{-1} = \begin{bmatrix} 1 & 0 & 0 \\ 0 & \frac{I_3}{N_3} & 0 \\ 0 & 0 & \frac{I_3}{N_3} \end{bmatrix},$$

and

$$P_F P^{-1} = \begin{bmatrix} 0 & 0 & 0 \\ 0 & \frac{N_3'}{N_3} - \frac{I_3'}{I_3} & 0 \\ 0 & 0 & \frac{N_3'}{N_3} - \frac{I_3'}{I_3} \end{bmatrix}.$$

Now

$$P_F J_3^{'[2]} P^{-1} = \begin{bmatrix} j_{11}^{[3]} + j_{33}^{[3]} & -\delta I_3 - \frac{(m-n)U_3I_3}{1+fU_3} & -\frac{bU_3I_3}{1+fU_3} \\ 0 & j_{11}^{[3]} + \beta - \delta - \frac{(m-n)U_3}{1+fU_3} & -\frac{bU_3(1+(c-1)I_3)}{1+fU_3} \\ \frac{(m-n)N_3}{(1+fU_3)^2} & \frac{[m-(m-n)I_3]N_3}{(1+fU_3)^2} & j_{33}^{[3]} + \beta - \delta - \frac{(m-n)U_3}{1+fU_3} \end{bmatrix}.$$

The matrix $Q = P_F P^{-1} + P_F J_3^{'[2]} P^{-1}$ can be written in block form:

$$Q = \begin{bmatrix} Q_{11} & Q_{12} \\ Q_{21} & Q_{22} \end{bmatrix},$$

with $Q_{11} = j_{11}^{[3]} + j_{33}^{[3]} = r\left(1 - \frac{U_3}{K}\right) - \frac{rU_3}{K} - \frac{bN_3(1+(c-1)I_3)}{(1+fU_3)^2} + \frac{(m-(m-n)I_3)U_3}{1+fU_3} - \delta I_3 - \nu$,

$$
\begin{aligned}
Q_{12} &= \begin{bmatrix} -\delta I_3 - \frac{(m-n)U_3I_3}{1+fU_3} & -\frac{bU_3I_3}{1+fU_3} \end{bmatrix}, \qquad Q_{21} = \begin{bmatrix} 0 \\ \frac{(m-n)N_3}{(1+fU_3)^2} \end{bmatrix}, \\
Q_{22} &= \begin{bmatrix} j_{11}^{[3]} + \beta - \delta - \frac{(m-n)U_3}{1+fU_3} + \frac{N_3'}{N_3} - \frac{I_3'}{I_3} & -\frac{bU_3(1+(c-1)I_3)}{1+fU_3} \\ \frac{[m-(m-n)I_3]N_3}{(1+fU_3)^2} & j_{33}^{[3]} + \beta - \delta - \frac{(m-n)U_3}{1+fU_3} + \frac{N_3'}{N_3} - \frac{I_3'}{I_3} \end{bmatrix}.
\end{aligned}
$$

A norm in $\mathbb{R}^3$ is defined as

$$|u, n, i| = \max\{|u|, |n| + |i|\}$$

for any vector $(u, n, i) \in \mathbb{R}^3$. Let m denote the Lozinskiĭ measure with respect to this norm. Then

$$m(Q) \leq \sup\{p_1, p_2\}, \tag{3.16}$$

with

$$
\begin{aligned}
p_1 &= m_1(Q_{11}) + |Q_{12}|, \\
p_2 &= |Q_{21}| + m_1(Q_{22}),
\end{aligned}
$$

where $|Q_{12}|$ and $|Q_{21}|$ are matrix norms induced by the L_1 vector norm, and m_1 denotes the Lozinskiĭ measure with respect to the L_1 norm. More specifically,
$m_1(Q_{11}) = r\left(1 - \frac{U_3}{K}\right) - \frac{rU_3}{K} - \frac{bN_3(1+(c-1)I_3)}{(1+fU_3)^2} + \frac{(m-(m-n)I_3)U_3}{1+fU_3} - \delta I_3 - \nu$,
$|Q_{12}| = \max\{-\delta I_3 - \frac{(m-n)U_3I_3}{1+fU_3}, -\frac{bU_3I_3}{1+fU_3}\} = \overline{a}$, $|Q_{21}| = \frac{(m-n)N_3}{(1+fU_3)^2}$. To calculate $m_1(Q_{22})$, the absolute value of the off-diagonal elements is added to the diagonal one in each column of Q_{22} and then take the maximum of the two sums. Thus,

$$
\begin{aligned}
& m_1(Q_{22}) \\
= \; & \max\Big\{r\Big(1-\frac{U_3}{K}\Big)-\frac{rU_3}{K}-\frac{bN_3(1+(c-1)I_3)}{(1+fU_3)^2}+\beta-\delta-\frac{(m-n)U_3}{1+fU_3}+\frac{N_3'}{N_3}-\frac{I_3'}{I_3} \\
& +\frac{[m-(m-n)I_3]N_3}{(1+fU_3)^2}; \frac{(m-(m-n)I_3)U_3}{1+fU_3}-\delta I_3-\nu+\beta-\delta-\frac{(m-n)U_3}{1+fU_3}+\frac{N_3'}{N_3}-\frac{I_3'}{I_3} \\
& +\frac{bU_3(1+(c-1)I_3)}{1+fU_3}\Big\}, \\
= \; & \frac{N_3'}{N_3}-\frac{I_3'}{I_3}+\beta-\delta-\frac{(m-n)U_3}{1+fU_3} \\
& +\max\Big\{r\Big(1-\frac{U_3}{K}\Big)-\frac{rU_3}{K}-\frac{bN_3(1+(c-1)I_3)}{(1+fU_3)^2}+\frac{[m-(m-n)I_3]N_3}{(1+fU_3)^2}; \\
& \frac{(m-(m-n)I_3)U_3}{1+fU_3}-\delta I_3-\nu+\frac{bU_3(1+(c-1)I_3)}{1+fU_3}\Big\}, \\
= \; & \frac{N_3'}{N_3}-\frac{I_3'}{I_3}+\beta-\delta-\frac{(m-n)U_3}{1+fU_3}+\overline{b},
\end{aligned}
$$

where $\overline{b}=\max\Big\{r\Big(1-\frac{U_3}{K}\Big)-\frac{rU_3}{K}-\frac{bN_3(1+(c-1)I_3)}{(1+fU_3)^2}+\frac{[m-(m-n)I_3]N_3}{(1+fU_3)^2}; \frac{(m-(m-n)I_3)U_3}{1+fU_3}-\delta I_3-\nu+\frac{bU_3(1+(c-1)I_3)}{1+fU_3}\Big\}$.

Therefore

$$
\begin{aligned}
p_1 &= r\Big(1-\frac{U_3}{K}\Big)-\frac{rU_3}{K}-\frac{bN_3(1+(c-1)I_3)}{(1+fU_3)^2}+\frac{(m-(m-n)I_3)U_3}{1+fU_3} \\
&\quad -\delta I_3-\nu+\overline{a}, \\
p_2 &= \frac{N_3'}{N_3}-\frac{I_3'}{I_3}+\beta-\delta-\frac{(m-n)U_3}{1+fU_3}+\overline{b}+\frac{(m-n)N_3}{(1+fU_3)^2}.
\end{aligned} \tag{3.17}
$$

Rewriting (3.3) with $V=0$ leads to

$$
\frac{N_3'}{N_3}=\frac{[m(1-I_3)+nI_3]U_3}{1+fU_3}-\delta I_3-\nu, \tag{3.18}
$$

$$
\frac{I_3'}{I_3}=(1-I_3)\Big[\beta+\frac{(n-m)U_3}{1+fU_3}-\delta\Big]. \tag{3.19}
$$

Substituting (3.18) and (3.19) into (3.17) gives

$$
\begin{aligned}
p_1 &= \frac{N_3'}{N_3}+r\Big(1-\frac{U_3}{K}\Big)-\frac{rU_3}{K}-\frac{bN_3(1+(c-1)I_3)}{(1+fU_3)^2}+\overline{a}\le\frac{N_3'}{N_3}-\overline{d}, \\
p_2 &= \frac{N_3'}{N_3}+\frac{I_3'}{1-I_3}+\overline{b}+\frac{(m-n)N_3}{(1+fU_3)^2}\le\frac{N_3'}{N_3}-\overline{d},
\end{aligned} \tag{3.20}
$$

where $\overline{d} = \min\{-r\left(1-\frac{U_3}{K}\right)+\frac{rU_3}{K}+\frac{bN_3(1+(c-1)I_3)}{(1+fU_3)^2})-\overline{a}, -\frac{I_3'}{1-I_3}-\frac{(m-n)N_3}{(1+fU_3)^2}-\overline{b}\}$. Therefore

$$m(Q) \leq \frac{N_3'}{N_3} - \overline{d},$$

for $t > T$ by (3.16) and (3.20). Along each solution $x(t, x_0)$ to (3.3) such that $x_0 \in K$ and for $t > T$, the result is

$$\frac{1}{t}\int_0^t m(Q)ds \leq \frac{1}{t}\int_0^T m(Q)ds + \frac{1}{t}\log\frac{N(t)}{N(T)} - \overline{d}\ \frac{t-T}{t},$$

which implies that $m(Q) \leq -\overline{d}/2 < 0$. Hence, the following theorem is established.

Theorem 3.2 *The equilibrium point E_3 of the system (3.3) is globally asymptotically stable in Ω if $m > n$, $\beta > \delta$, $(m-n) > f(\beta-\delta)$, $m(\beta-\delta) > \nu(m-n)$, $C < 0$ and $G < 0$.*

Local and global stability of the interior steady state E_4

In this section, the stability properties of the interior steady state in the presence of the disease in lions are studied. The local stability of $E_4(U_4, V_4, N_4, I_4)$ is established using the Routh-Hurwitz criterion. The Jacobian matrix of the system (3.3) at E_4 is

$$J_4 = \begin{bmatrix} j_{11}^{[4]} & -\frac{aU_4}{1+eU_4} & -\frac{bU_4(1+(c-1)I_4)}{1+fU_4} & \frac{bU_4N_4}{1+fU_4} \\ \frac{lV_4}{(1+eU_4)^2} & j_{22}^{[4]} & -pV_4(1-I_4) & pN_4V_4 \\ \frac{(m-(m-n)I_4)N_4}{(1+fU_4)^2} & 0 & \frac{(m-(m-n)I_4)U_4}{1+fU_4} - \delta I_4 - \nu & -\delta N_4 - \frac{(m-(m-n)U_4)N_4}{1+fU_4} \\ -\frac{(m-n)U_4}{(1+fU_4)^2} & 0 & 0 & \beta-\delta-\frac{(m-n)U_4}{1+fU_4} \end{bmatrix},$$

where

$j_{11}^{[4]} = r - \frac{2rU_4}{K} - \frac{bN_4(1+(c-1)I_4)}{(1+fU_4)^2} - \frac{aV_4}{(1+eU_4)^2}$ and $j_{22}^{[4]} = \frac{lU_4}{1+eU_4} - \mu - pN_4(1-I_4)$.

Note that $j_{12}^{[4]} < 0$, $j_{13}^{[4]} < 0$, $j_{14}^{[4]} > 0$, $j_{21}^{[4]} > 0$, $j_{23}^{[4]} < 0$, $j_{24}^{[4]} > 0$, $j_{31}^{[4]} > 0$, $j_{34}^{[4]} < 0$ and $j_{41}^{[4]} < 0$. The characteristic polynomial of the linearized system is given by

$$\lambda^4 + m_1\lambda^3 + m_2\lambda^2 + m_3\lambda + m_4 = 0, \tag{3.21}$$

where

$$
\begin{aligned}
m_1 &= -(j_{11}^{[4]} + j_{22}^{[4]} + j_{33}^{[4]} + j_{44}^{[4]}) = -\mathrm{Tr}(J_4), \\
m_2 &= j_{11}^{[4]} j_{22}^{[4]} + j_{11}^{[4]} j_{33}^{[4]} + j_{11}^{[4]} j_{44}^{[4]} + j_{22}^{[4]} j_{33}^{[4]} + j_{22}^{[4]} j_{44}^{[4]} + j_{33}^{[4]} j_{44}^{[4]} - j_{12}^{[4]} j_{21}^{[4]} - j_{13}^{[4]} j_{31}^{[4]} - j_{14}^{[4]} j_{41}^{[4]}, \\
m_3 &= j_{13}^{[4]} j_{22}^{[4]} j_{31}^{[4]} + j_{12}^{[4]} j_{21}^{[4]} j_{33}^{[4]} - j_{11}^{[4]} j_{22}^{[4]} j_{33}^{[4]} - j_{22}^{[4]} j_{33}^{[4]} j_{44}^{[4]} + j_{12}^{[4]} j_{21}^{[4]} j_{44}^{[4]} - j_{12}^{[4]} j_{24}^{[4]} j_{41}^{[4]} - j_{12}^{[4]} j_{23}^{[4]} j_{31}^{[4]} \\
&\quad - j_{11}^{[4]} j_{22}^{[4]} j_{44}^{[4]} - j_{13}^{[4]} j_{34}^{[4]} j_{41}^{[4]} + j_{13}^{[4]} j_{31}^{[4]} j_{44}^{[4]} + j_{14}^{[4]} j_{22}^{[4]} j_{41}^{[4]} + j_{14}^{[4]} j_{33}^{[4]} j_{41}^{[4]} - j_{14}^{[4]} j_{21}^{[4]} j_{42}^{[4]} - j_{11}^{[4]} j_{33}^{[4]} j_{44}^{[4]}, \\
m_4 &= -j_{12}^{[4]} j_{23}^{[4]} j_{34}^{[4]} j_{41}^{[4]} + j_{12}^{[4]} j_{24}^{[4]} j_{33}^{[4]} j_{41}^{[4]} + j_{12}^{[4]} j_{23}^{[4]} j_{31}^{[4]} j_{44}^{[4]} + j_{13}^{[4]} j_{34}^{[4]} j_{41}^{[4]} j_{22}^{[4]} - j_{13}^{[4]} j_{31}^{[4]} j_{22}^{[4]} j_{44}^{[4]} \\
&\quad - j_{14}^{[4]} j_{22}^{[4]} j_{33}^{[4]} j_{41}^{[4]} - j_{12}^{[4]} j_{21}^{[4]} j_{33}^{[4]} j_{44}^{[4]} + j_{11}^{[4]} j_{22}^{[4]} j_{33}^{[4]} j_{44}^{[4]}.
\end{aligned}
$$

When $j_{11}^{[4]} < 0$, $j_{22}^{[4]} < 0$, $j_{33}^{[4]} < 0$ and $j_{44}^{[4]} < 0$, then $m_1 > 0$ and $m_2 > 0$. Furthermore, $m_3 > 0$ if

$$
\begin{aligned}
&j_{13}^{[4]} j_{22}^{[4]} j_{31}^{[4]} + j_{12}^{[4]} j_{21}^{[4]} j_{33}^{[4]} - j_{11}^{[4]} j_{22}^{[4]} j_{33}^{[4]} - j_{22}^{[4]} j_{33}^{[4]} j_{44}^{[4]} + j_{12}^{[4]} j_{21}^{[4]} j_{44}^{[4]} - j_{11}^{[4]} j_{22}^{[4]} j_{44}^{[4]} - j_{13}^{[4]} j_{34}^{[4]} j_{41}^{[4]} \\
&+ j_{13}^{[4]} j_{31}^{[4]} j_{44}^{[4]} + j_{14}^{[4]} j_{22}^{[4]} j_{41}^{[4]} + j_{14}^{[4]} j_{33}^{[4]} j_{41}^{[4]} - j_{11}^{[4]} j_{33}^{[4]} j_{44}^{[4]} > j_{12}^{[4]} j_{24}^{[4]} j_{41}^{[4]} + j_{12}^{[4]} j_{23}^{[4]} j_{31}^{[4]},
\end{aligned}
$$

and $m_4 > 0$ if

$$
\begin{aligned}
&-j_{14}^{[4]} j_{22}^{[4]} j_{33}^{[4]} j_{41}^{[4]} - j_{12}^{[4]} j_{21}^{[4]} j_{33}^{[4]} j_{44}^{[4]} + j_{11}^{[4]} j_{22}^{[4]} j_{33}^{[4]} j_{44}^{[4]} - j_{13}^{[4]} j_{31}^{[4]} j_{22}^{[4]} j_{44}^{[4]} + j_{13}^{[4]} j_{34}^{[4]} j_{41}^{[4]} j_{22}^{[4]} > j_{12}^{[4]} j_{23}^{[4]} j_{34}^{[4]} j_{41}^{[4]} - \\
&j_{12}^{[4]} j_{24}^{[4]} j_{33}^{[4]} j_{41}^{[4]} - j_{12}^{[4]} j_{23}^{[4]} j_{31}^{[4]} j_{44}^{[4]}.
\end{aligned}
$$

Let

$$
\begin{aligned}
q &= (j_{11}^{[4]} + j_{22}^{[4]} + j_{33}^{[4]} + j_{44}^{[4]})(-j_{12}^{[4]} j_{21}^{[4]} - j_{13}^{[4]} j_{31}^{[4]} + j_{22}^{[4]} j_{33}^{[4]} - j_{14}^{[4]} j_{41}^{[4]} + j_{22}^{[4]} j_{44}^{[4]} + j_{33}^{[4]} j_{44}^{[4]} \\
&\quad + j_{11}^{[4]} (j_{22}^{[4]} + j_{33}^{[4]} + j_{44}^{[4]}))(j_{11}^{[4]} j_{22}^{[4]} j_{33}^{[4]} - j_{14}^{[4]} j_{22}^{[4]} j_{41}^{[4]} - j_{14}^{[4]} j_{33}^{[4]} j_{41}^{[4]} + j_{11}^{[4]} j_{22}^{[4]} j_{44}^{[4]} \\
&\quad + j_{11}^{[4]} j_{33}^{[4]} j_{44}^{[4]} + j_{22}^{[4]} j_{33}^{[4]} j_{44}^{[4]} - j_{13}^{[4]} (j_{22}^{[4]} j_{31}^{[4]} - j_{34}^{[4]} j_{41}^{[4]} + j_{31}^{[4]} j_{44}^{[4]}) \\
&\quad + j_{12}^{[4]} (j_{23}^{[4]} j_{31}^{[4]} + j_{24}^{[4]} j_{41}^{[4]} - j_{21}^{[4]} (j_{33}^{[4]} j_{44}^{[4]}))) - (j_{11}^{[4]} j_{22}^{[4]} j_{33}^{[4]} - j_{14}^{[4]} j_{22}^{[4]} j_{41}^{[4]} - j_{14}^{[4]} j_{33}^{[4]} j_{41}^{[4]} \\
&\quad + j_{11}^{[4]} j_{22}^{[4]} j_{44}^{[4]} + j_{11}^{[4]} j_{33}^{[4]} j_{44}^{[4]} + j_{22}^{[4]} j_{33}^{[4]} j_{44}^{[4]} - j_{13}^{[4]} (j_{22}^{[4]} j_{31}^{[4]} - j_{34}^{[4]} j_{41}^{[4]} + j_{31}^{[4]} j_{44}^{[4]}) \\
&\quad - j_{12}^{[4]} (-j_{23}^{[4]} j_{31}^{[4]} - j_{24}^{[4]} j_{41}^{[4]} + j_{21}^{[4]} (j_{33}^{[4]} + j_{44}^{[4]})))^2 - (j_{11}^{[4]} + j_{22}^{[4]} + j_{33}^{[4]} + j_{44}^{[4]})^2 (-j_{14}^{[4]} j_{22}^{[4]} \\
&\quad + j_{33}^{[4]} j_{41}^{[4]} + j_{22}^{[4]} (j_{11}^{[4]} j_{33}^{[4]} j_{44}^{[4]} + j_{13}^{[4]} (j_{34}^{[4]} j_{41}^{[4]} - j_{31}^{[4]} j_{44}^{[4]})) + j_{12}^{[4]} (j_{24}^{[4]} j_{33}^{[4]} j_{41}^{[4]} - j_{21}^{[4]} j_{33}^{[4]} j_{44}^{[4]} \\
&\quad + j_{23}^{[4]} (-j_{34}^{[4]} j_{41}^{[4]} + j_{31}^{[4]} j_{44}^{[4]}))).
\end{aligned}
$$

From the Routh-Hurwitz criterion for a 4×4 Jacobian matrix, all the real parts of roots of (3.21) are negative if and only if the following conditions are satisfied,

$$
m_1 > 0, m_3 > 0, m_4 > 0 \quad \text{and} \quad q = m_1 m_2 m_3 - m_3^2 - m_1^2 m_4 > 0 \tag{3.22}
$$

Now this is true if $j_{11}^{[4]} < 0$, $j_{22}^{[4]} < 0$, $j_{33}^{[4]} < 0$ and $j_{44}^{[4]} < 0$. Thus, the following result can be stated.

Lemma 3.4 *The equilibrium point E_4 is locally asymptotically stable if*

(a) $r < \frac{2rU_4}{K} + \frac{bN_4(1+(c-1)I_4)}{(1+fU_4)^2} + \frac{aV_4}{(1+eU_4)^2}$,

(b) $\frac{lU_4}{1+eU_4} < \mu + pN_4(1-I_4)$,

(c) $\frac{(m-(m-n)I_4)U_4}{1+fU_4} < \delta I_4 + \nu$,

(d) $\beta < \delta + \frac{(m-n)U_4}{1+fU_4}$.

Next, the globally asymptotically stability for the interior equilibrium is discussed.

Theorem 3.3 *The interior equilibrium is globally asymptotically stable if* $M = \frac{aV^*}{(1+eU)(1+eU^*)} + \frac{bN^*[(1-I^*)+cI^*]}{(1+fU)(1+fU^*)} < \frac{r}{K}$.

Proof. A candidate Lyapunov function that has been used by Dubey and Upadhyay [99], Hsu [133], and others is chosen. The following function is considered,

$$\begin{aligned} Z(U,V,N,I) &= \alpha_1\Big(U - U^* - U^*\ln\Big(\frac{U}{U^*}\Big)\Big) + \alpha_2\Big(V - V^* - V^*\ln\Big(\frac{V}{V^*}\Big)\Big) \\ &+ \alpha_3\Big(N - N^* - N^*\ln\Big(\frac{N}{N^*}\Big)\Big) + \alpha_4\Big(I - I^* - I^*\ln\Big(\frac{I}{I^*}\Big)\Big) \end{aligned} \quad (3.23)$$

Define

$$Z_1(U) = U - U^* - U^*\ln\Big(\frac{U}{U^*}\Big), \quad Z_2(V) = V - V^* - V^*\ln\Big(\frac{V}{V^*}\Big),$$

$$Z_3(N) = N - N^* - N^*\ln\Big(\frac{N}{N^*}\Big) \quad Z_4(I) = I - I^* - I^*\ln\Big(\frac{I}{I^*}\Big).$$

Z can be rewritten as

$$Z(U,V,N,I) = \alpha_1 Z_1(U) + \alpha_2 Z_2(V) + \alpha_3 Z_3(N) + \alpha_4 Z_4(I).$$

Differentiating Z with respect to time t along the solutions of model (3.3),

$$\frac{dZ}{dt} = \alpha_1\Big(1 - \frac{U^*}{U}\Big)\frac{dU}{dt} + \alpha_2\Big(1 - \frac{V^*}{V}\Big)\frac{dV}{dt} + \alpha_3\Big(1 - \frac{N^*}{N}\Big)\frac{dN}{dt} + \alpha_4\Big(1 - \frac{I^*}{I}\Big)\frac{dI}{dt} \quad (3.24)$$

The linear approximations $U - U^* \cong 1 + eU \cong 1 + fU$, $V - V^* \cong V$, $N - N^* \cong N$ and $I - I^* \cong I$ are used to compute $\frac{dZ_1(U(t))}{dt}$, $\frac{dZ_2(V(t))}{dt}$, $\frac{dZ_3(N(t))}{dt}$ and $\frac{dZ_4(I(t))}{dt}$ as follows:

$$
\begin{aligned}
\frac{dZ_1}{dt} &= \Big(1 - \frac{U^*}{U}\Big)\Big[r\Big(1 - \frac{U}{K}\Big) - \frac{aV}{1+eU} - \frac{bN[(1-I)+cI]}{1+fU}\Big]U, \\
&= (U - U^*)[r\Big(1 - \frac{U}{K}\Big) - \frac{rU^*}{K} + \frac{rU^*}{K} - \frac{aV}{1+eU} - \frac{aV^*}{1+eU^*} + \frac{aV^*}{1+eU^*} \\
&\quad - \frac{bN[(1-I)+cI]}{1+fU} - \frac{bN^*[(1-I^*)+cI^*]}{1+fU^*} + \frac{bN^*[(1-I^*)+cI^*]}{1+fU^*}\Big], \\
&= (U - U^*)\Big[-\frac{rU}{K} + \frac{rU^*}{K} - \frac{aV}{1+eU} + \frac{aV^*}{1+eU^*} - \frac{bN[(1-I)+cI]}{1+fU} \\
&\quad + \frac{bN^*[(1-I^*)+cI^*]}{1+fU^*}\Big], \\
&= (U - U^*)[-\frac{r}{K}(U - U^*) - \frac{aV(1+eU^*) - aV^*(1+eU)}{(1+eU)(1+eU^*)} \\
&\quad - \frac{bN[(1-I)+cI](1+fU^*) - bN^*[(1-I^*)+cI^*](1+fU)}{(1+fU)(1+fU^*)}\Big], \\
&= (U - U^*)\Big[-\frac{r}{K}(U - U^*) - \frac{a(V-V^*)(1+eU^*) - aV^*(U-U^*)}{(1+eU)(1+eU^*)} \\
&\quad - \frac{b(N-N^*)[(1-I)+cI](1+fU^*) - bN^*[(1-I^*)+cI^*](1+fU)}{(1+fU)(1+fU^*)}\Big], \\
&= -\frac{r}{K}(U - U^*)^2 - \frac{a(U-U^*)(V-V^*)(1+eU^*) - aV^*(U-U^*)^2}{(1+eU)(1+eU^*)} \\
&- \frac{b(N-N^*)[(1-I)+cI](1+fU^*)(U-U^*) - bN^*[(1-I^*)+cI^*](U-U^*)^2}{(1+fU)(1+fU^*)},
\end{aligned}
$$

$$
\begin{aligned}
\frac{dZ_2}{dt} &= \Big(1-\frac{V^*}{V}\Big)\Big[\frac{lU}{1+eU}-p(1-I)N-\mu\Big]V,\\
&= (V-V^*)\Big[\frac{lU}{1+eU}-\frac{lU^*}{1+eU^*}+\frac{lU^*}{1+eU^*}-p(1-I)N-p(1-I^*)N^*\\
&\quad +p(1-I^*)N^*-\mu\Big],\\
&= (V-V^*)\Big[\frac{lU}{1+eU}-\frac{lU^*}{1+eU^*}-p(1-I)N+p(1-I^*)N^*\Big],\\
&= (V-V^*)\Big[\frac{lU(1+eU^*)-lU^*(1+eU)}{(1+eU)(1+eU^*)}-p[(1-I)N-(1-I^*)N^*]\Big],\\
&= (V-V^*)\frac{l(U-U^*)(1+eU^*)-lU^*(U-U^*)}{(1+eU)(1+eU^*)}\\
&\quad -p(V-V^*)[(1-I)N-(1-I^*)N^*],\\
&= -\frac{l[U^*-(1+eU^*)]}{(1+eU)(1+eU^*)}(U-U^*)(V-V^*)-p(V-V^*)[(1-I)N-(1-I^*)N^*,
\end{aligned}
$$

$$
\begin{aligned}
\frac{dZ_3}{dt} &= \Big(1-\frac{N^*}{N}\Big)\Big[\frac{[m(1-I)+nI]U}{1+fU}-\delta I-\nu\Big]N,\\
&= (N-N^*)\Big[\frac{[m(1-I)+nI]U}{1+fU}-\frac{[m(1-I^*)+nI^*]U^*}{1+fU^*}+\frac{[m(1-I^*)+nI^*]U^*}{1+fU^*}\\
&\quad -\delta I-\delta I^*+\delta I^*-\nu\Big],\\
&= (N-N^*)\Big[\frac{[m(1-I)+nI]U}{1+fU}-\frac{[m(1-I^*)+nI^*]U^*}{1+fU^*}-\delta(I-I^*)\Big],\\
&= (N-N^*)\Big[\frac{[m(1-I)+nI](U-U^*)(1+fU^*)-[m(1-I^*)+nI^*]U^*(1+fU)}{(1+fU)(1+fU^*)}\\
&\quad -\delta(I-I^*)\Big],\\
&= (N-N^*)\Big[\frac{[m(1-I)+nI](U-U^*)(1+fU^*)-[m(1-I^*)+nI^*]U^*(U-U^*)}{(1+fU)(1+fU^*)}\\
&\quad -\delta(I-I^*)\Big],\\
&= \frac{[m(1-I)+nI](1+fU^*)-[m(1-I^*)+nI^*]U^*}{(1+fU)(1+fU^*)}(U-U^*)(N-N^*)\\
&\quad -\delta(I-I^*)(N-N^*),\\
&= -\frac{[m(1-I^*)+nI^*]U^*-[m(1-I)+nI](1+fU^*)}{(1+fU)(1+fU^*)}(U-U^*)(N-N^*)\\
&\quad -\delta(I-I^*)(N-N^*),
\end{aligned}
$$

and

$$
\begin{aligned}
\frac{dZ_4}{dt} &= \Big(1-\frac{I^*}{I}\Big)(1-I)\Big[\beta-\frac{(m-n)U}{1+fU}-\delta\Big]I,\\
&= (I-I^*)(1-I)\Big[\beta-\delta-\frac{(m-n)U}{1+fU}+\frac{(m-n)U^*}{1+fU^*}-\frac{(m-n)U^*}{1+fU^*}\Big],\\
&= (I-I^*)(1-I)\Big[-\frac{(m-n)U}{1+fU}+\frac{(m-n)U^*}{1+fU^*}\Big],\\
&= -(I-I^*)(1-I)\Big[\frac{(m-n)U(1+fU^*)-(m-n)U^*(1+fU)}{(1+fU)(1+fU^*)}\Big],\\
&= -\frac{(m-n)(U-U^*)(1+fU^*)(I-I^*)(1-I)}{(1+fU)(1+fU^*)}\\
&\quad +\frac{(m-n)U^*(U-U^*)(I-I^*)(1-I)}{(1+fU)(1+fU^*)},\\
&= -(m-n)\frac{(1+fU^*)-U^*}{(1+fU)(1+fU^*)}(U-U^*)(I-I^*)(1-I).
\end{aligned}
$$

Now,

$$
\begin{aligned}
\frac{dZ}{dt} &= \alpha_1\Big[-\frac{r}{K}(U-U^*)^2-\frac{a(U-U^*)(V-V^*)(1+eU^*)-aV^*(U-U^*)^2}{(1+eU)(1+eU^*)}\\
&\quad -\frac{b(N-N^*)[(1-I)+cI](1+fU^*)(U-U^*)-bN^*[(1-I^*)+cI^*](U-U^*)^2}{(1+fU)(1+fU^*)}\Big]\\
&\quad +\alpha_2\Big[-\frac{l[U^*-(1+eU^*)]}{(1+eU)(1+eU^*)}(U-U^*)(V-V^*)\\
&\quad -p(V-V^*)[(1-I)N-(1-I^*)N^*\Big]\\
&\quad +\alpha_3\Big[-\frac{[m(1-I^*)+nI^*]U^*-[m(1-I)+nI](1+fU^*)}{(1+fU)(1+fU^*)}(U-U^*)(N-N^*)\\
&\quad -\delta(I-I^*)(N-N^*)\Big]\\
&\quad +\alpha_4\Big[-(m-n)\frac{(1+fU^*)-U^*}{(1+fU)(1+fU^*)}(U-U^*)(I-I^*)(1-I)\Big],\\
&= \alpha_1\Big[-\frac{r}{K}+\frac{aV^*}{(1+eU)(1+eU^*)}+\frac{bN^*[(1-I^*)+cI^*]}{(1+fU)(1+fU^*)}\Big](U-U^*)^2\\
&\quad +\Big[-\alpha_1\frac{a(1+eU^*)}{(1+eU)(1+eU^*)}-\alpha_2\frac{l[U^*-(1+eU^*)}{(1+eU)(1+eU^*)}\Big](U-U^*)(V-V^*)\\
&\quad +\Big[-\alpha_1\frac{b[(1-I)+cI](1+fU^*)}{(1+fU)(1+fU^*)}\\
&\quad -\alpha_3\frac{[m(1-I^*)+nI^*]U^*-[m(1-I)+nI](1+fU^*)}{(1+fU)(1+fU^*)}\Big](U-U^*)(N-N^*)\\
&\quad -\alpha_2p(V-V^*)[(1-I)N-(1-I^*)N^*]-\alpha_3\delta(I-I^*)(N-N^*)\\
&\quad -\alpha_4\Big[(m-n)\frac{(1+fU^*)-U^*}{(1+fU)(1+fU^*)}(1-I)\Big](U-U^*)(I-I^*).
\end{aligned}
$$

Let $\alpha_1 = 1$ and $\alpha_4 = 1$, then

$$\alpha_2 \frac{l[(1+eU^*) - U^*]}{(1+eU)(1+eU^*)} = \alpha_1 \frac{a(1+eU^*)}{(1+eU)(1+eU^*)},$$

that is,

$$\alpha_2 = \frac{a(1+eU^*)}{l[(1+eU^*) - U^*]}.$$

Also

$$\alpha_3 \frac{[m(1-I^*) + nI^*]U^* - [m(1-I) + nI](1+fU^*)}{(1+fU)(1+fU^*)} = -\frac{b[(1-I) + cI](1+fU^*)}{(1+fU)(1+fU^*)},$$

that is,

$$\alpha_3 = \frac{b[(1-I) + cI](1+fU^*)}{[m(1-I) + nI](1+fU^*) - [m(1-I^*) + nI^*]U^*}.$$

Hence,

$$\begin{aligned}
\frac{dZ}{dt} &= \Big[-\frac{r}{K} + \frac{aV^*}{(1+eU)(1+eU^*)} + \frac{bN^*[(1-I^*) + cI^*]}{(1+fU)(1+fU^*)} \Big](U-U^*)^2 \\
&\quad - \frac{a(1+eU^*)}{l[(1+eU^*) - U^*} p(V-V^*)[(1-I)N - (1-I^*)N^*] \\
&\quad - \frac{b[(1-I) + cI](1+fU^*)}{[m(1-I) + nI](1+fU^*) - [m(1-I^*) + nI^*]U^*} \delta(I-I^*)(N-N^*) \\
&\quad - \Big[(m-n) \frac{(1+fU^*) - U^*}{(1+fU)(1+fU^*)} (1-I) \Big](U-U^*)(I-I^*).
\end{aligned}$$

The coefficient of $(U-U^*)^2$ is strictly negative if

$$-\frac{r}{K} + \frac{aV^*}{(1+eU)(1+eU^*)} + \frac{bN^*[(1-I^*) + cI^*]}{(1+fU)(1+fU^*)} < 0,$$

that is, if

$$M = \frac{aV^*}{(1+eU)(1+eU^*)} + \frac{bN^*[(1-I^*) + cI^*]}{(1+fU)(1+fU^*)} < \frac{r}{K}.$$

If $\alpha_2 > 0$ and $\alpha_3 > 0$, that is, $e > 1$ and $[m(1-I)+nI](1+fU^*) > [m(1-I^*)+nI^*]U^*$, then the function Z is negative definite. Hence, the interior equilibrium E_4 is globally asymptotically stable. □

3.3.4 Persistence of the system

A system is defined to be uniformly persistence if the minimum of each component $u(t)$ of a positive solution is always greater than some positive constant, i.e. $\liminf_{t\to\infty} u(t) >$

$\epsilon > 0$ [134]. The persistence of a system implies that all the species continue to exist and none of them will become extinct [99]. From Proposition 3.3 the system (3.3) has been proved to be bounded. Now, the system is shown to be persistent. To do so, all the boundary equilibria are shown to be repellers.

Theorem 3.4 *If the following conditions hold*

(i) $\beta > \delta$,

(ii) $\frac{lU_3}{1+eU_3} > \mu + pN_3(1 - I_3)$,

then the system is persistent.

Proof:

From the Jacobian matrix associated with $E_1(U_1, 0, N_1, 0)$ the following eigenvalue $\lambda_1^{[1]} = \beta - \delta - \nu + \frac{n\nu}{m} = \beta - \delta + \frac{\nu}{m}(n - m)$ was found. But equilibrium point E_3 exists if $m > n$ and $\beta > \delta$, making $\lambda_1^{[1]} > 0$. Hence, existence of E_3 implies that E_1 is unstable. For the equilibrium point $E_2(U_2, V_2, N_2, 0)$, from the Jacobian matrix J_2, there is an eigenvalue $\beta - \delta - \frac{(m-n)U_2}{1+fU_2} = \beta - \delta - (m - n)\frac{\nu}{m}$. Using the same argument as above, E_2 is unstable if E_3 exists.

For the equilibrium point $E_3(U_3, 0, N_3, I_3)$, from the Jacobian matrix J_3, an eigenvalue $j_{22}^{[3]} = \frac{lU_3}{1+eU_3} - \mu - pN_3(1 - I_3)$ exists. If $\frac{lU_3}{1+eU_3} > \mu + pN_3(1 - I_3)$ then the eigenvalue is positive, and E_3 is unstable.

Thus, all the boundary equilibria of system (3.3) are repellers if the conditions stated in the theorem hold. □

3.4 Dynamics of subsystems of system (3.2)

3.4.1 The role of infection on the lion, cheetah, impala system

To observe the influence of infection in the lion-cheetah-impala system, a subsystem with no infection is considered. Setting $I = 0$ in system (3.3) results in an extended

Lotka-Volterra type super-predator, predator-prey system.

$$\begin{aligned}\frac{dU}{dt} &= U\Big[r(1-\frac{U}{K})-\frac{aV}{1+eU}-\frac{bW}{1+gU}\Big],\\ \frac{dV}{dt} &= V\Big[\frac{lU}{1+eU}-pW-\mu\Big],\\ \frac{dW}{dt} &= W\Big[\frac{mU}{1+gU}-\nu\Big].\end{aligned} \tag{3.25}$$

This reduced system has five nonnegative equilibria namely: $\hat{E}_0(0,0,0)$, $\hat{E}_2(U_2,0,0)$, $\hat{E}_3(U_3,V_3,0)$, $\hat{E}_4(U_4,0,W_4)$ and $\hat{E}_5(U_5,V_5,W_5)$. However, equilibrium points $\hat{E}_4(U_4,0,W_4)$ and $\hat{E}_5(U_5,V_5,W_5)$ are equivalent to E_3 and E_4 discussed in the previous section. The equilibrium points $\hat{E}_0(0,0,0)$, $\hat{E}_2(U_2,0,0)$, $\hat{E}_3(U_3,V_3,0)$ are discussed in the next section for a system in which the lions are absent.

3.4.2 The effect of lion removal on the eco-epidemiological system

In the absence of the lions, the system (3.2) reduces to a impala-cheetah sub-system given by

$$\begin{aligned}\frac{dU}{dt} &= U\Big[r(1-\frac{U}{K})-\frac{aV}{1+eU}\Big],\\ \frac{dV}{dt} &= V\Big[\frac{lU}{1+eU}-\mu\Big].\end{aligned} \tag{3.26}$$

This subsystem has three biologically feasible equilibria namely: (i) $\bar{E}_0(0,0)$, (ii) $\bar{E}_1(K,0)$ and (iii) $\bar{E}_2(U_2,V_2)$ where $U_2=\frac{\mu}{l-e\mu}$ and $V_2=\frac{rl(Kl-\mu-eK\mu)}{aK(e\mu-l)^2}$). The first two equilibria always exist, and $\bar{E}_2$ exists if $l>e\mu$ and $l-e\mu>\frac{\mu}{K}$ or $U_2<K$. The Jacobian of the system (3.26) is given by

$$J(U,V)=\begin{bmatrix} r(1-\frac{2U}{K})-\frac{aV}{(1+eU)^2} & -\frac{aU}{1+eU}\\ \frac{lV}{(1+eU)^2} & \frac{lU}{1+eU}-\mu\end{bmatrix}. \tag{3.27}$$

(a) $\bar{E}_0$ is always unstable with eigenvalues r and $-\mu$. It is a saddle point whose stable manifold is the V-axis.

(b) $\bar{E}_1$ has eigenvalues $-r$ and $\frac{Kl}{1+eK} - \mu$. Thus, $\bar{E}_1$ is unstable in the V-axis if $\frac{Kl}{1+eK} > \mu$, that is, $l - e\mu > \frac{\mu}{K}$ and $\bar{E}_2$ exists. The growth function of cheetah when impala population is at carrying capacity, exceeds death rate. $\bar{E}_1$ is locally asymptotically stable if $l - e\mu < \frac{\mu}{K}$ and $\bar{E}_2$ does not exist.

Next, the global stability of $\bar{E}_1$ is established. In order to do so, Theorem A.3 and Lemma A.1 that are essential in finding a candidate Lyapunov function and proving the global stability are used. Define $L : \{(U, V) \in \Omega : U > 0\} \to \mathbb{R}$ by

$$L(U, V) = \frac{1}{2}V^2.$$

The time derivative of L computed along solutions of (3.26) is

$$\begin{aligned} L'(U,V) &= V \cdot V' = V^2\Big[\frac{lU}{1+eU} - \mu\Big], \\ &= V^2\Big[\frac{lU - \mu(1+eU)}{1+eU}\Big], \\ &= V^2\Big[\frac{(l - e\mu) - \frac{\mu}{U}}{U(1+eU)}\Big]. \end{aligned}$$

It follows that $L'(U, V) \leq 0$ if $U = K$, that is, $l - e\mu - \frac{\mu}{K} < 0$. Hence, L is a Lyapunov function on Ω. Substituting $V = 0$ in the first equation of (3.26) leads to

$$\frac{dU}{dt} = Ur\Big(1 - \frac{U}{K}\Big),$$

which shows that $U \to K$ as $t \to \infty$. Therefore, it follows from the LaSalle's Invariance Principle, that every solution of the equations in the model (3.26), with initial conditions in Ω, approaches $\bar{E}_1$ as $t \to \infty$. Hence, it follows that the point $\bar{E}_1$ is globally asymptotically stable in Ω if $l - e\mu - \frac{\mu}{K} < 0$, i.e, if $\frac{\mu}{l-e\mu} > K$, impala population exceeds carrying capacity.

(c) $\bar{E}_2$ has eigenvalues $\lambda_1 = \frac{-a_1 + \sqrt{a_1^2 - 4a_0a_2}}{2a_0}$ and $\lambda_2 = \frac{-a_1 - \sqrt{a_1^2 - 4a_0a_2}}{2a_0}$, the roots of the quadratic equation

$$a_0\lambda^2 + a_1\lambda + a_2 = 0,$$

where $a_0 = Kl(l - e\mu)$, $a_1 = lr\mu - eKlr\mu + er\mu^2 + e^2Kr\mu^2$ and
$a_2 = Kl^2r\mu - lr\mu^2 - 2eKlr\mu^2 + er\mu^3 + e^2Kr\mu^3$.

Now, $a_0 > 0$, since it is derived from a condition for existence of $\bar{E}_2$.

$$\begin{aligned} a_1 &= lr\mu - eKlr\mu + er\mu^2 + e^2Kr\mu^2, \\ &= r\mu(l + e\mu) - eKr\mu(l - e\mu), \\ &= r\mu[(l + e\mu) - eK(l - e\mu)]. \end{aligned}$$

$a_1 > 0$ if $\frac{l+e\mu}{l-e\mu} > eK$.

$$\begin{aligned} a_2 &= Kl^2r\mu - lr\mu^2 - 2eKlr\mu^2 + er\mu^3 + e^2Kr\mu^3, \\ &= Kr\mu(l^2 - 2el\mu + e^2\mu^2) - r\mu^2(l - e\mu), \\ &= Kr\mu(l - e\mu)^2 - r\mu^2(l - e\mu), \\ &= (l - e\mu)r\mu[K(l - e\mu) - \mu], \\ &= (l - e\mu)r\frac{\mu}{K}[(l - e\mu) - \frac{\mu}{K}]. \end{aligned}$$

Thus, $a_2 > 0$ if $l - e\mu > \frac{\mu}{K}$, that is, $\frac{\mu}{l-e\mu} < K$, impala population is less than their carrying capacity. The following result can be stated.

Lemma 3.5 *$\bar{E}_2$ is locally asymptotically stable when it exists if $a_1 > 0, a_2 > 0$, that is, $\frac{l+e\mu}{l-e\mu} > eK$ and $l - e\mu > \frac{\mu}{K}$.*

Furthermore,

$$\begin{aligned} \mathrm{Tr}(J(U_2, V_2)) &= -\mu - \frac{r\mu}{K(l - e\mu)} + \frac{elr\mu(Kl - \mu - eK\mu)}{K(l - e\mu)^3(1 + \frac{e\mu}{l-e\mu})^2} + \frac{l\mu}{(l - e\mu)(1 + \frac{e\mu}{l-e\mu})} \\ &\quad - \frac{lr(Kl - \mu - eK\mu)}{K(l - e\mu)^2(1 + \frac{e\mu}{l-e\mu})} + r(1 + \frac{\mu}{K(l - e\mu)}), \end{aligned}$$

$$\begin{aligned} \mathrm{Det}(J(U_2, V_2)) &= -r\mu - \frac{2r\mu^2}{K(l - e\mu)} - \frac{el^2r\mu^2(l - e\mu - \frac{\mu}{K})}{(l - e\mu)^3(1 + \frac{e\mu}{l-e\mu})^2} \\ &\quad + \frac{lr\mu(l - e\mu - \frac{3\mu}{K})}{(l - e\mu)^2(1 + \frac{e\mu}{l-e\mu})} + \frac{lr\mu}{(l - e\mu)(1 + \frac{e\mu}{l-e\mu})}. \end{aligned}$$

The type of interior equilibrium point depends on the sign of

$$\begin{aligned} \Delta(\bar{U}_2, \bar{V}_2) &= \mathrm{Tr}^2(J(U_2, V_2)) - 4\mathrm{Det}(J(U_2, V_2)), \\ &= \frac{1}{K^2l^2(l - e\mu)^2}\Big[r\mu(r\mu(l + e\mu)^2 - K^2(l - e\mu)^2(4l^2 - 4el\mu - e^2r\mu) \\ &\quad + 2K\mu(l - e\mu)[2l^2 - e^2r\mu - el(r + 2\mu)])\Big]. \end{aligned}$$

If $\Delta(\bar{U}_2, \bar{V}_2) \geq 0$ then the interior equilibrium is a stable node, otherwise it is a spiral point. The existence condition of $\bar{E}_2$ renders $l > e\mu$.
Let $\delta(x) = d_2x^2 + d_1x + d_0$ where $d_2 = -K^2(4l^2 - 4el\mu - e^2r\mu)$, $d_1 = 2K\mu(2l^2 - e^2r\mu - el(r + 2\mu))$ and $d_0 = r\mu(l + e\mu)^2$. $d_0 > 0$. The discriminant

$$d_1^2 - 4d_0d_2 = 4K^2r\mu(l+e\mu)^2(4l^2-4el\mu-e^2r\mu)+4K^2\mu^2(2l^2-e^2r\mu-el(r+2\mu))^2 > 0$$

if $4l(l - e\mu) > e^2r\mu$.

Now, the global stability of $\bar{E}_2$ is discussed whenever it exists, using the Bendixson-Dulac criterion, Theorem A.1, [124] and the Poincaré-Bendixson Theorem, Theorem A.2, [125].

Proposition 3.5 *$\bar{E}_2$ is globally asymptotically stable if $2\mu > lK$.*

Proof. Consider the system (3.26). Let $\Phi(U, V) = \frac{1}{UV}$,
$h_1(U, V) = Ur\Big(1 - \frac{U}{K}\Big) - \frac{aUV}{1+eU}$, $h_2(U, V) = \frac{lUV}{1+eU} - \mu V$. Now $\Phi(U, V) > 0$ in the interior of the $U - V$ plane. Then

$$\begin{aligned}
\Delta(U, V) &= \frac{\partial(\Phi h_1)}{\partial U} + \frac{\partial(\Phi h_2)}{\partial V}, \\
&= \frac{\partial}{\partial U}\Big[\frac{r}{V}\Big(1 - \frac{U}{K}\Big) - \frac{a}{1 + eU}\Big] + \frac{\partial}{\partial V}\Big[\frac{l}{1 + eU} - \frac{\mu}{U}\Big], \\
&= -\frac{r}{KV} + \frac{ae}{(1 + eU)^2}, \\
&= \frac{aeKV - r(1 + eU)^2}{KV(1 + eU)^2}.
\end{aligned}$$

Now Δ is not identically zero in the positive quadrant of the $U - V$ plane. $\Delta(U, V) < 0$ and does not change sign if

$$aeKV - r(1+eU)^2 = aeKV - r - 2reU - e^2U^2 = -r - (2rU - aKV)e - re^2U^2 < 0$$

that is, if $2rU - aKV > 0$. This means that at E_2, we have

$$\begin{aligned} 2rU_2 - aKV_2 &= \frac{2r\mu}{l - e\mu} - \frac{rl(Kl - \mu - eK\mu)}{(l - e\mu)^2}, \\ &= \frac{2r\mu(l - e\mu) - rl(Kl - \mu - eK\mu)}{(l - e\mu)^2}, \\ &= \frac{2r\mu(l - e\mu) - rlK(l - e\mu) + \mu rl}{(l - e\mu)^2}, \\ &= \frac{r(l - e\mu)(2\mu - lK) + \mu rl}{(l - e\mu)^2} > 0, \end{aligned}$$

if and only if $2\mu > lK$ and $l > e\mu$. $\frac{\mu}{l}$ refers to the ratio of mortality of cheetah to cheetah conversion efficiency of impala biomass to cheetah biomass. The carrying capacity of impala population must be less than the output/input ratio of cheetah biomass. Furthermore, the ratio of input/output of cheetah biomass must outweigh the time the cheetah spends consuming impala. Thus, subsystem (3.26) does not have a limit cycle in $\bar{\Omega}$. Since $\bar{E}_2$ is locally asymptotically stable, the Poincaré-Bendixson Theorem and Proposition 3.3 imply that the equilibrium point $\bar{E}_2$ is globally asymptotically stable in the region $\bar{\Omega}$. □

3.5 Numerical analysis

It was estimated that 1684 lions lived in the KNP in 2005 and 2006 [24]. The lion population in KNP appears to be stable at around 1700 since 2005. The KNP has a current lion population of approximately 1700. Keet et al. [135] estimated that about 500 of the 1700 lions reside in areas with buffalo whose BTB prevalence is high [88]. The initial population densities for each species are chosen to be positive at $U(0) = 133,000$ [24], $V(0) = 412$ [23], $N(0) = 1700$ [24], $W(0) = 1200$ and $B(0) = 500$. Some parameter values which were not readily available were computed on the basis of the steady states and stability conditions, and these include impala conversion rates into cheetah and lion biomass.

3.5.1 Parameter estimates

The literature on the dynamics of the three species in KNP as well as studies from other parks with similar environmental conditions (e.g. the SNP) provide the following information:

(i) Carrying capacity for impala

The impala population was estimated to lie between 132, 300 and 176, 400 between the years 2010 and 2011, [24]. In this study,the carrying capacity was estimated to be 200, 000.

(ii) In a study on impala in KNP, Fairall [136] deduced that fecundity lies around 95% in mature females and is drastically low in two-year old females. A sample of 100 females studied produced 244 lambs during their lifetime. A female was observed to produce offspring for a period of 10 years up to the age of 12 years. At an early reproducing age of two years, approximately 45% of the mortality had taken place. In this study, the growth rate of the impala was estimated to be 0.01342.

(iii) Mortality rates

The life-expectancy of cheetah in protected areas is estimated at 18 years [137]. In this study the mortality rate of cheetah was estimated as the reciprocal of the life-expectancy. Hence, $\mu = \frac{1}{18} = 0.0556$. Once the juvenile cheetah emerge from the lair, mortality rate due to predation by lions is estimated at 50% [33]. In this study the cheetah mortality rate by the lions was estimated as $p = 0.5$. The lifespan for lions in KNP is about 15 years [48]. The mortality rate of lions was estimated as the reciprocal of the life-expectancy. Hence, $\nu = \frac{1}{15} = 0.0667$.

(iv) Predation rates

In a study on feeding ecology of cheetah in south eastern KNP, Mills et.al [119] deduced that impala were the dominant prey killed out of nine prey species se-

lected. Seven adult cheetahs were monitored in the same region between 1987 and 1990 [138]. Kill rates were observed for different coalitions. It was deduced that on average a cheetah consumes 1.4 kg of meat per day [138]. Out of the total weight of an adult impala, only 60% was estimated to be consumable [119]. The body masses of adult male and female impala were estimated as 54.4kg and 40.9kg [120, 139], giving an average weight of 47.65kg. Thus, an average cheetah eats 17.87 impala per year. The predation rate is considered as the number of prey killed/consumed per predator per year. In this study, the cheetah predation rate of the impala was considered as 0.0001787 after re-scaling.

The impala is the third dominant prey species consumed by the lion after zebra and wildebeest in the KNP [121]. Between 1986 and 1995, the following impala kill rates per lion per year were observed in the KNP: Pride females 13.0 (1986-1989), pride females 8.7 (1992-1995), territorial males 1.3 and non-territorial males 15.1 [140]. In this study it was estimated that the impala kill rate per lion per year to be the average 9.525. In this study, the lion predation rate of the impala was considered as 0.00009525 after re-scaling.

(v) Kill retention times

Kill retention times are influenced by how large the group and prey are, densities of predators, and awareness of the presence of competing predators [119]. The cheetah kill retention time was 165 min [119]. In this study, we estimate the cheetah handling time as kill retention time per day, $e = \frac{165}{1440} = 0.1146$ per day, or $e = \frac{165}{1440\times365} = 0.0003$ per year. The lion is expected to have a longer retention time as it has no competitors. In this study we estimate the kill retention time to be 220 min, giving $f = 0.1528$ per day or $f = 0.0004$ per year.

(vi) Conversion rates

The cheetah and lion biomass conversion rates l and m are estimated in relation with the bounds for existence of equilibrium points E_2 and E_3. For E_2, we have

$l > e\mu = 1.668 \times 10^{-5}$. For E_3, we have $m > f\nu = 2.668 \times 10^{-5}$. In this study, it was estimated that $l = 1.705 \times 10^{-5}$ and $m = 2.7 \times 10^{-5}$.

(vii) Disease standard incidence and disease-induced mortality rate

"The initial frequency of diseased animals is defined as the proportion of animals in the population that contract the disease (with or without clinical symptoms) and are infected with the pathogen", [48]. In a study by Keet (unpublished data 1999-2004), 16 animals tested positive and were observed for a period of five years. Of the 16,7 out of 10 changed stage from infected to diseased, whilst one lioness succumbed to injuries though still infectious. Thus, 80% of infected lions became diseased within five years [47], leading to a deduction that 8 % of lions became diseased within a period of six months. The initial frequency of diseased animals was estimated to be 0.16 per year.

The mortality from disease at the end of infectious period was 100% . 14 diseased lions were monitored to check the possibility of recovery. However, all succumbed to the disease in a period of six months [48]. In this study, it was estimated that the disease-induced mortality rate to be $\delta = 0.0767$.

The parameters values used in the numerical analysis of model (3.2) and subsystem (3.26) are given in Table 3.2.

Table 3.2: Parameter Description and Values

Symbol	Description	Value	Units	Reference
r	Intrinsic growth rate of the impala	0.01342	yr^{-2}	[136]
K	Environmental carrying capacity of the impala	200 000	yr^{-1}	Estimate
a	Capture rate of the impala by the cheetah	0.0001787	yr^{-1}	[119]
b	Capture rate of the impala by the lion	0.00009525	yr^{-1}	[140]
c	Efficiency of infected lion to capture impala	0.5		Estimate
e	Handling time of impala by cheetah	0.0003	yr	[119]
f	Handling time of impala by lion	0.0004	yr	Estimate
l	Impala biomass conversion efficiency into new cheetah	0.00001705		Computed
m	Impala biomass conversion efficiency into new susceptible lion	0.000027		Computed
n	Impala biomass conversion efficiency into new infected lion	0.0000135		Computed
p	Mortality rate of cheetah by lion	0.5	yr^{-1}	[33]
β	Disease standard incidence	0.16	yr^{-1}	[48]
ν	Natural mortality rate of lion	0.0667	yr^{-1}	[48]
μ	Natural mortality rate of cheetah	0.0556	yr^{-1}	[137]
δ	Disease-induced mortality rate of infected lion	0.0767	yr^{-1}	[48]

Keeping the rest of the parameters at their fixed levels, some predation and epidemiological parameters are varied, namely p, β, δ, a and b to try to understand their influence on the dynamics of system (3.2). The re-scaled species population in relation to its initial value, $U(t)/U(0)$ for instance, was plotted as a function of time.

3.5.2 The effect of lion removal on the system dynamics

In the absence of the lion, impala and cheetah populations and phase diagrams are shown in Figure 3.1. It can be observed that the positive equilibrium $\bar{E}_2$ is reached as t tends to infinity. The impala population rises rapidly to reach a plateau below the carrying capacity, before it falls down and settle to equilibrium value above the initial level. The cheetah population rises to more than double the initial value. The trajectories indicate the global stability of the equilibrium point $\bar{E}_2$.

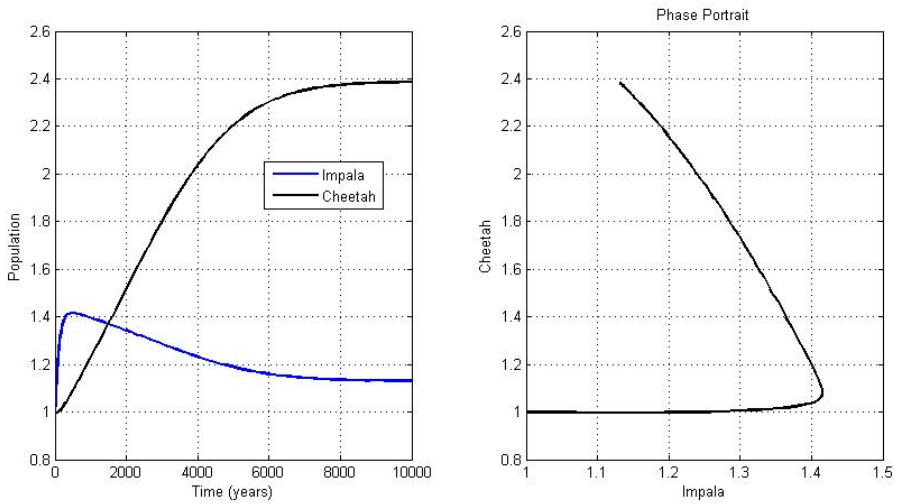

Figure 3.1: *Global stability of the system (3.26) around the equilibrium point $\bar{E}_2$, (a) scaled population, (b) trajectory tending to $\bar{E}_2$.*

3.5.3 The effect of cheetah mortality by lion on cheetah population density

In this section the effect on the system of changes in the cheetah mortality by lion, p, is described. As the mortality rate increases in the wide range $0 \leq p \leq 0.5$, the cheetah population becomes depressed and eventually goes extinct. For instance for $p = 0$, that is, in the absence of lion kills, the cheetah population is stable. The population

of the impala grows to below carrying capacity. However, the lion population becomes extinct in 50 years. Increasing p from 0 to 0.00001, the cheetah population takes a slight setback and eventually stabilises at about 350. As p takes the value 0.0001, the cheetah population drops significantly. This shows the effect the cheetah mortality by lion has on the cheetah population. Thus, the added cheetah mortality by lions has the potential of eliminating the cheetah species from the system. There is only a quantitative change, qualitatively, the system's behaviour remains the same. This behaviour is shown in Figures 3.2, 3.3 and 3.4. In all the three cases both lion populations decline and tend to zero. This occurs as a result of high disease incidence rate.

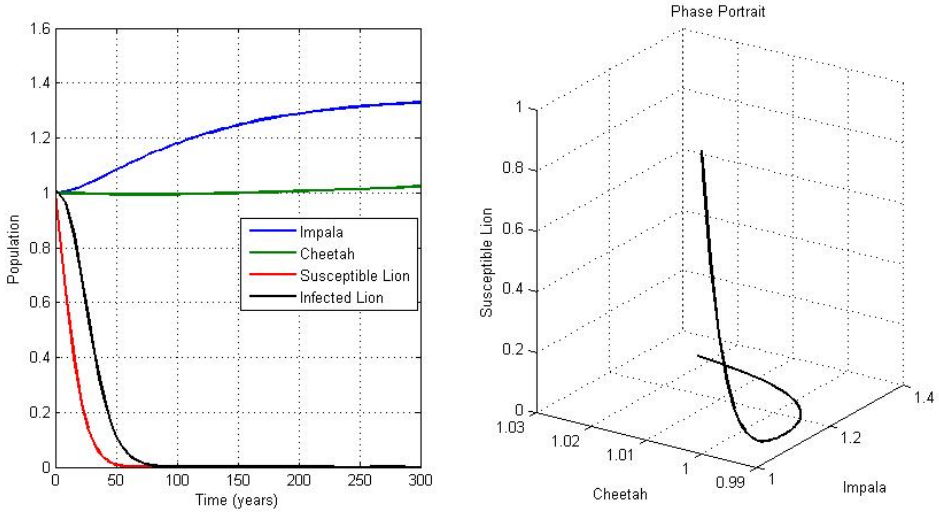

Figure 3.2: *(a) Variation of scaled population, (b) Phase space trajectory, when cheetah mortality by lion,* $p = 0$ *and all other parameter values remain unchanged*

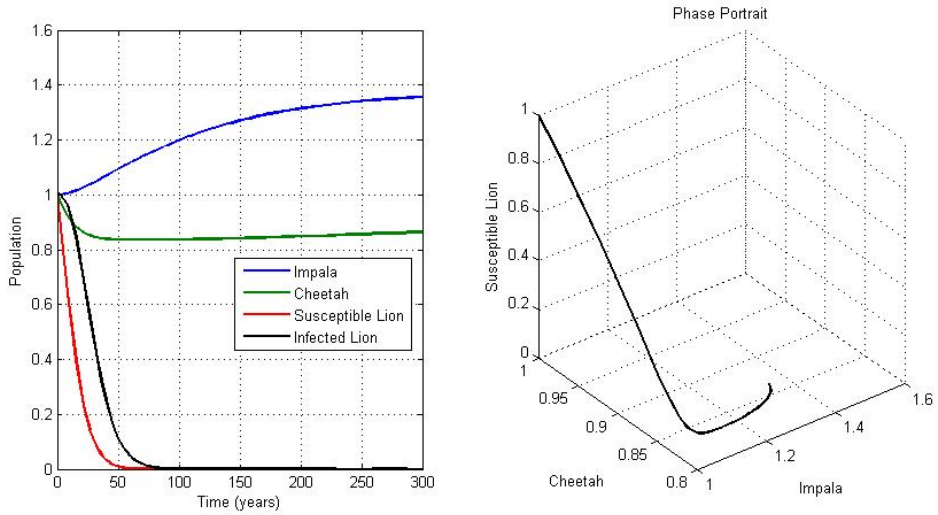

Figure 3.3: *(a) Variation of scaled population, (b) Phase space trajectory, when cheetah mortality by lion,* $p = 0.00001$ *and all other parameter values remain unchanged*

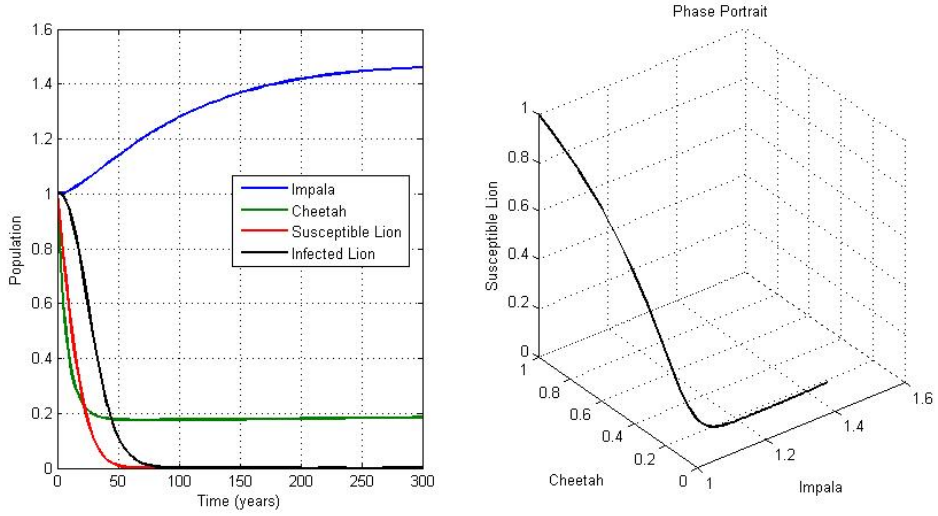

Figure 3.4: *(a) Variation of scaled population, (b) Phase space trajectory, when cheetah mortality by lion,* $p = 0.0001$ *and all other parameter values remain unchanged*

3.5.4 Effect of epidemiological parameters on the system dynamics

In this section the effects on the system of changes in the disease-related parameters, β and δ, are described. The system changes are considered in the absence of cheetah kills by lions, $p = 0$, as the cheetah population stabilises and does not face extinction.

Disease standard incidence in lions, β

The disease standard incidence, β, is 0.16. At this value, the total lion population does not survive long enough, but declines continuously and becomes extinct in 50 years. When the magnitude of β is decreased from 0.16 to 0.08 in the absence of cheetah kills by lions, $p = 0$ and all other parameter values remaining constant, the healthy lions survive but the infected lions becomes extinct in 150 years, twice the initial period. Thus, the disease is eliminated. The cheetah population survives. Fewer lions are becoming infected and as such the healthy lions survive. This behaviour is shown in Figures 3.5 and 3.6. As such, a decrease in the disease incidence rate has a positive effect on the lion population, negative effect on the impala population, and has insignificant effect on the cheetah population. The effects remain the same when β is reduced further to $\beta = 0.02$.

Disease-induced death rate, δ

The disease-induced mortality rate, δ, is 0.0767. When the value of δ is decreased from 0.0767 to 0.00767 while keeping $p = 0$ and all other parameters remaining constant, the susceptible lion population are still eliminated from the system. The diseased lions survive longer. Since the rate of infection has not changed, more susceptible lions become diseased and are eliminated from the system. A further decrease in δ to 0.000767 is accompanied by only a marginal increase in the diseased lion population. However, the entire lion population still becomes extinct after 100 years. This behaviour is shown in Figures 3.7 and 3.8.

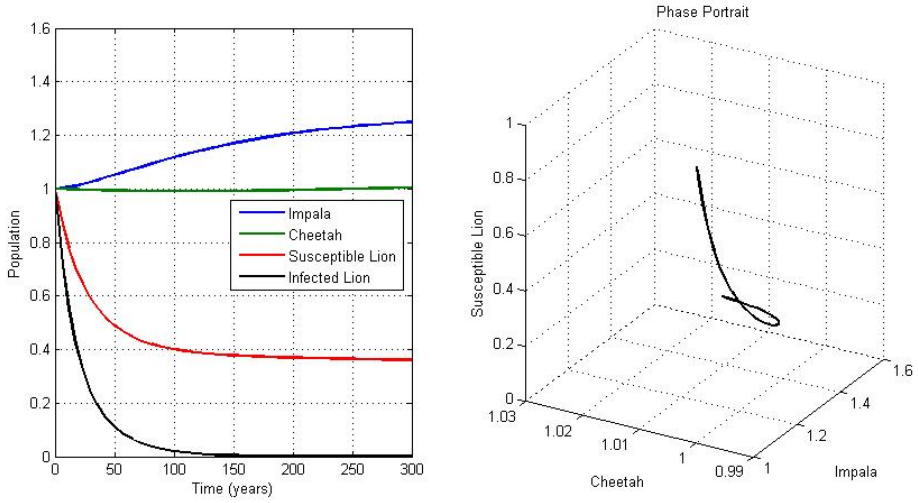

Figure 3.5: *Effect of disease incidence on (a) variation of scaled population, (b) phase space trajectory, when* $\beta = 0.08$ *and* $p = 0$ *and all other parameter values remain unchanged*

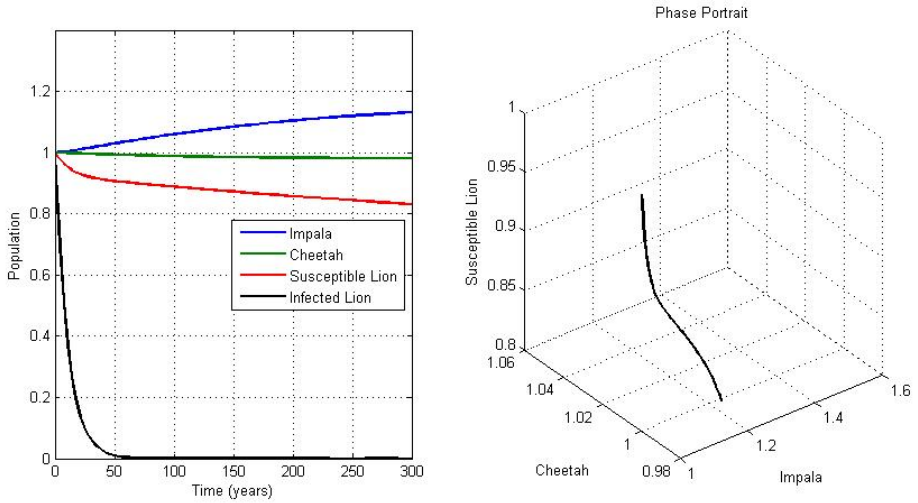

Figure 3.6: *Effect of disease incidence on (a) variation of scaled population, (b) phase space trajectory, when* $\beta = 0.02$ *and* $p = 0$ *and all other parameter values remain unchanged*

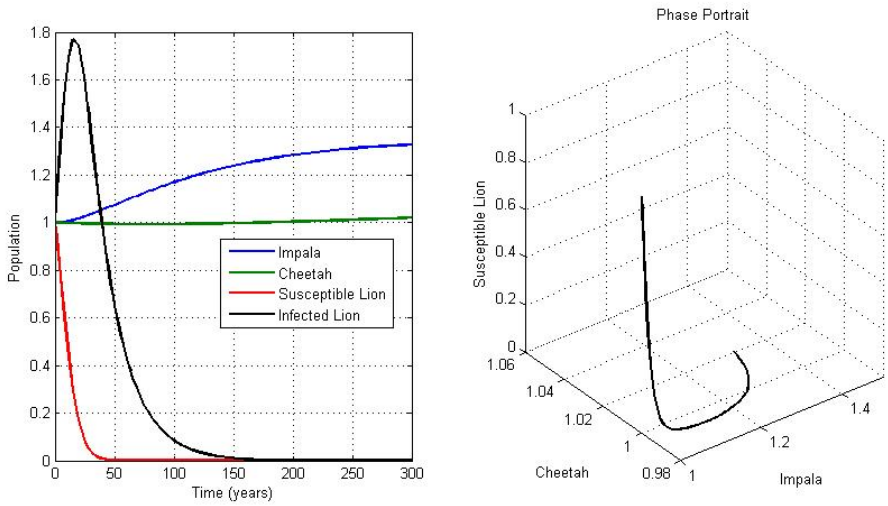

Figure 3.7: *Effect of disease induced mortality rate on (a) scaled population, (b) phase space trajectory, when* $\delta = 0.00767$ *and* $p = 0$ *and all other parameter values remain unchanged*

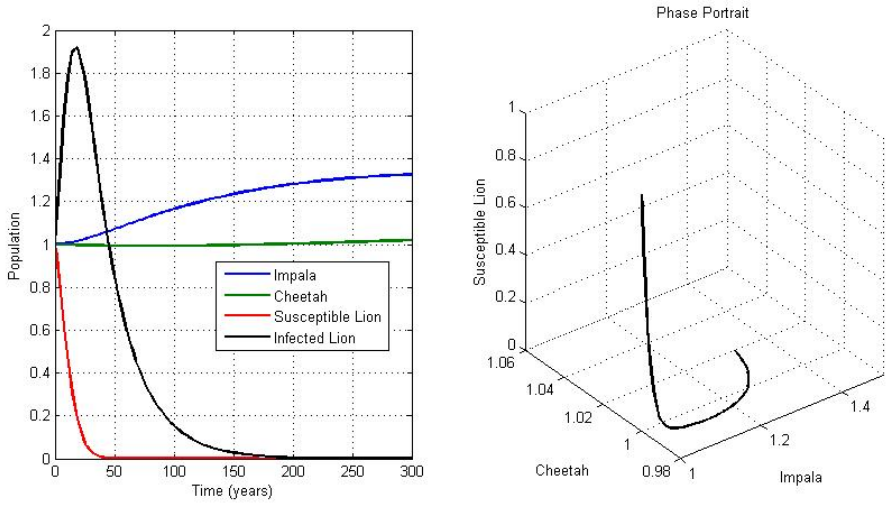

Figure 3.8: *Effect of disease induced mortality rate on (a) scaled population, (b) phase space trajectory, when* $\delta = 0.000767$ *and* $p = 0$ *and all other parameter values remain unchanged*

3.5.5 The effect of predation on the system dynamics

In this section the effects on the system of changes in the predation rate parameters, a and b, are described.

Predation of impala by cheetah, a

The predation rate of the impala by the cheetah, a, is 0.0001767. When a is increased by a factor of 10, the impala species becomes extinct in about 75 years. This may be attributed to over predation. The cheetah population is also eliminated due to unavailability of food. An increase in the cheetah predation rate is accompanied by a decrease in impala population which eventually becomes extinct. The extinction in impala is largely due to increased predation. However, the cheetah population becomes extinct in 150 years. When a is further increased to $a = 0.01767$, the impala species becomes extinct in a much shorter time due to over predation. This behaviour is shown in Figures 3.9 and 3.10.

Predation of impala by susceptible lion, b

The predation rate of the impala by the susceptible lion, b, is 0.00009525. An increase of b by a factor of 10 is accompanied by reduced population levels of both the impala and the cheetah. The impala population survives at below carrying capacity, and this may be attributed to over-predation. The reduction in cheetah population may be as a result of increased competition. When b is further increased to $b = 0.009525$, both the impala and cheetah population eventually become extinct. As such, heavier lion predation has a negative effect on the population levels of both the impala and the cheetah. The lion populations also rapidly becomes extinct. This behaviour is shown in Figures 3.11 and 3.12.

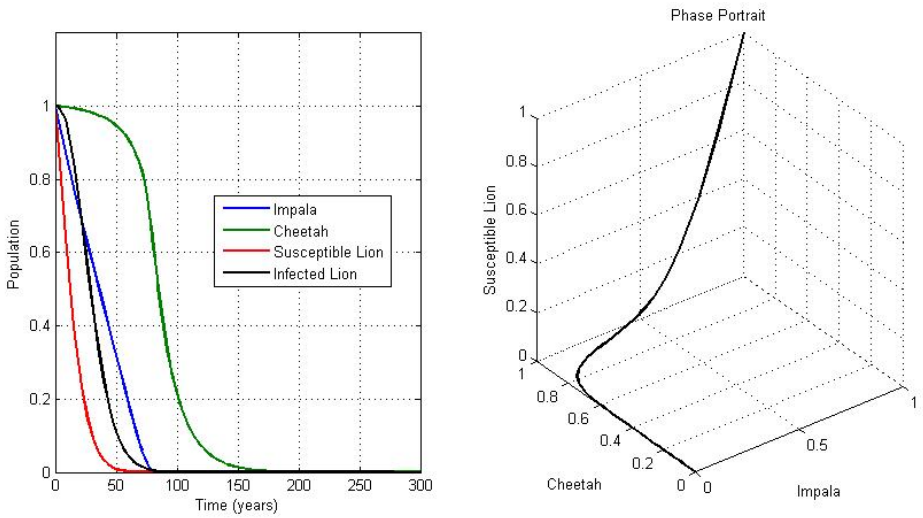

Figure 3.9: *Effect of cheetah predation rate on (a) scaled population, (b) phase space trajectory, when* $a = 0.001787$ *and* $p = 0$ *and all other parameter values remain unchanged*

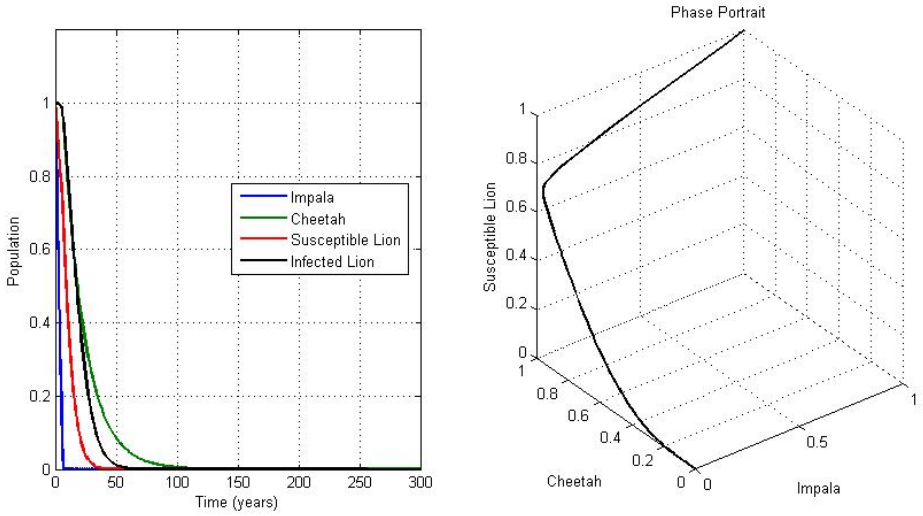

Figure 3.10: *Effect of cheetah predation rate on (a) scaled population, (b) phase space trajectory, when* $a = 0.01787$ *and* $p = 0$ *and all other parameter values remain unchanged*

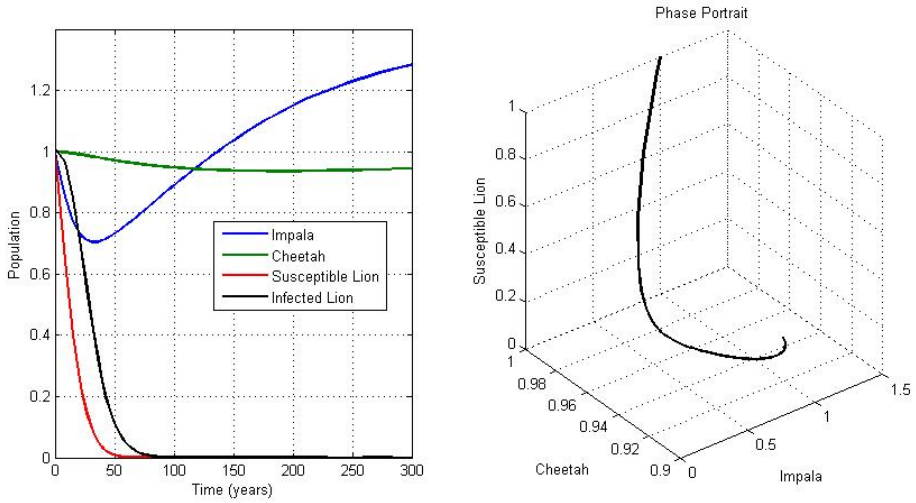

Figure 3.11: *Effect of lion predation rate on (a) scaled population, (b) phase space trajectory, when* $b = 0.0009525$ *and* $p = 0$ *and all other parameter values remain unchanged*

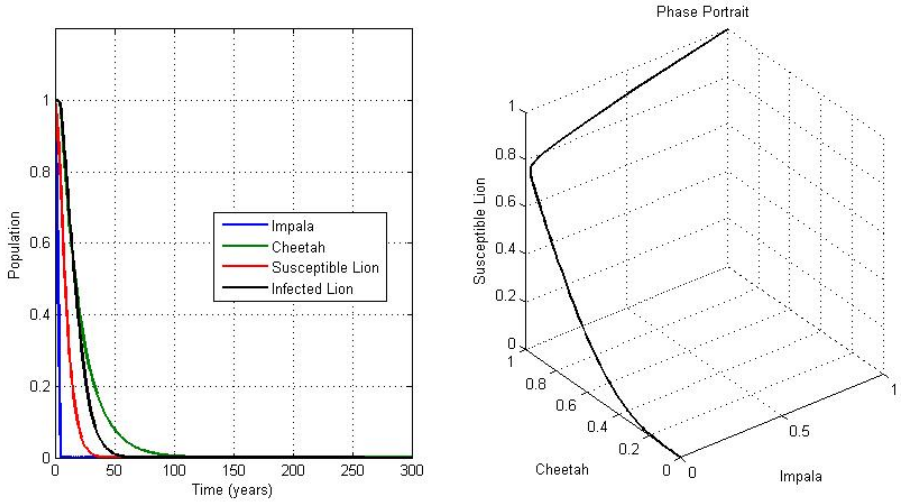

Figure 3.12: *Effect of lion predation rate on (a) scaled population, (b) phase space trajectory, when* $b = 0.009525$ *and* $p = 0$ *and all other parameter values remain unchanged*

3.6 Discussion and conclusion

In relation to cheetah mortality by the lions, p, the cheetah in KNP can only survive in the absence of lion killings, $p = 0$, or killings are kept at minimum levels $p = 0.0001$. It is recommended that the two species be separated to ensure that the cheetah species does not become extinct. It appears that the current population level of the cheetah is too low to ensure growth beyond its initial value even with a situation in which there is no competition by lions.

The disease standard incidence, β, value is high resulting in that the susceptible lions become extinct in about 25 years. At the same time the entire lion population becomes diseased. The prediction is that the entire lion population disappear in 50 years. When $\beta = 0.08$, the lion population persists but is not able to grow beyond its initial value. Efforts must be targeted on vaccination of all healthy lions to prevent infection, and treatment of sick lions to reduce further infection. The reduced infection rate is accompanied by a decline in impala population growth, as more healthy lions hunt.

The disease-induced mortality rate, δ, for lions is high. All infected lions are dying early. At $\delta = 0.00767$ the diseased lions stay longer but are infectious, and further spreading the disease to the healthy lions. The reduced mortality rate leads to an increase in diseased lion before they die.

Most notable is the conclusion that changes to the epidemiological parameter values have an insignificant effect on the cheetah population. The increased cheetah predation rate, a leads to the extinction of the impala population. However, with reduced impala, the cheetah population becomes constant in the presence of impala, but disappears as impala become extinct.

A gradual increase in the lion predation rate, b results in reduced growth of impala and

cheetah populations. A further increase leads to extinction of all species. Increased predation in the presence of disease does not assist increasing the lion population. As long as the disease is present and the disease incidence is high, the lion population remains suppressed and eventually becomes extinct.

Chapter 4

Impala, cheetah and lion model with diffusion

4.1 Introduction

In natural systems the distribution of resources is seldom uniform. In reality, natural systems show some spatial variation even though it may not be apparent [141]. There is no definite pattern in the distribution of resources in an ecosystem. The densities of resources tend to be randomly distributed in a habitat [106]. Spatial variation is vital as it drives the movement, distribution and continued survival of species. It influences the interaction between interdependent species. Unlike species interact in a way that destroy spatial homogeneity and lead to heterogeneous distributions [141]. It is vital to consider population and ecological models when species move from one region to another. The main examples involve ecological invasions, where one species takes over control of another species' habitat (as with grey and red squirrels in the UK [142]), or modelling the outbreak and transmission of infectious diseases [143].

Modelling spatio-temporal pattern formation in natural systems has gained immense popularity amongst researchers from biology, ecology and mathematical biology. Tem-

poral mathematical models on prey-predator systems focus on dynamics of growth and interactions. In order to understand dynamics in spatial structure, the temporal models are extended to include spatial processes. The main spatial processes include the movement of species as well as variations in their habitats [20]. As animal species interact in the form of competition or predation, they disperse through the environment. The dispersion leads to variations in population densities in their habitats. However, the rate of dispersion varies with species, and spatial patterns of their movements emerge. Mathematical ecological modelling of such spatio-temporal dynamics makes use of a system of coupled nonlinear reaction-diffusion equations [106].

Several models on spatio-temporal dynamics of species interactions have been proposed [59, 79, 144, 145]. Vanak *et al.* [59] built multivariate models to examine movement decisions of four large mammalian carnivore species in Karongwe Game Reserve, (KGR). The carnivores were lion, leopard, cheetah and wild-dog. They found that the strength of intraguild interactions did not influence species distribution, as spatial movements and survival techniques of lower guild predators varied with risk of encounter of higher guild predators, and availability of prey. Lions were found to enjoy free movement and positioned themselves in areas of high density of their principal prey and also of lower guild predators. Cheetah were found to overlap with areas occupied by lions but reduced their risk of encounter by using avoidance techniques and limiting hunting. They also showed the spatial overlap of regions occupied by each predator and prey using their respective relative probability of occurrence. Movement decisions were also shown to be functions of choice of prey. In the proposed deterministic partial differential equation model, conditions for the emergence of spatial distributions of species are deduced. The effects of the presence of lions on such distribution is also investigated.

Broekhuis *et al.* [79] studied the risk of predation or interference competition as main drivers of the shape of spatial distributions. They classified an animal's response to risk as either reactive or predictive. They examined whether a cheetah's response to

the presence of lions was reactive or predictive. They built a Generalised Linear Mixed Model, and used simultaneous Global Positioning System (GPS) data to test the hypothesis that cheetah avoided areas occupied by lions; cheetahs avoided interactions with lions; cheetahs' choice of habitats relied on lion habitat; and that cheetahs' response to risk was a function of habitat type. Their results indicated that cheetahs' response to risk was a function of nature of predator, type of habitat and urgency of the risk. Cheetahs' response was found to be reactive rather than predictive.

Liu and Lin [144] studied a predator-prey model with Holling type III response function under the influence of cross-diffusion and subject to the homogeneous Neumann boundary conditions. *A priori* estimates of positive upper and lower bounds of positive steady states were given. The non-existence and existence results of non-constant positive steady states were established as the cross-diffusion coefficient was varied. This implied that cross-diffusion was responsible for the emergence of stationary patterns. Cross-diffusion was introduced to describe the mutual interferences between individuals. The proposed model is different from the Liu and Lin model in that no cross diffusion is considered. However, the same stability analysis is performed.

Guin [145] investigated the emergence of spatial patterns through Turing instability in a reaction-diffusion predator-prey model. The predator-prey interactions were coupled with intra-specific competition among predators as well as self and cross-diffusion. The intra-specific competition was found to have a significant effect on the emergence of spatial patterns around the unique positive equilibrium.

The major objective of this work is to investigate the existence and non-existence of non-constant positive steady state of the three species prey, predator and super-predator model. Conditions for diffusion-driven instability of the steady state are derived. The prey, predator and super-predator species are the impala, cheetah and lion respectively.

4.2 The model

Consider a impala-cheetah-lion system in which the cheetah and lion consume impala according to the Holling type II functional response, that allows movement of different species across the niche as they search for food. Let $U(X,Y,T)$, $V(X,Y,T)$ and $W(X,Y,T)$ denote the densities of the impala, cheetah and lions respectively at time T and position (X,Y) in the habitat. To take into account the inhomogeneous distribution of the predators and the impala in different spatial locations within a fixed bounded domain Ω in $\mathbb{R}^2$, with smooth boundary $\partial\Omega$ at any given time, and the natural tendency of each species to diffuse to a smaller population concentration, the following associated reaction-diffusion (PDE) system is considered:

$$\begin{aligned}
\frac{\partial U}{\partial T} - D_1\big(\frac{\partial^2 U}{\partial X^2} + \frac{\partial^2 U}{\partial Y^2}\big) &= rU\Big(1 - \frac{U}{K}\Big) - \frac{a_1 UV}{1+b_1U} - \frac{a_2 UW}{1+b_2U}, \\
\frac{\partial V}{\partial T} - D_2\big(\frac{\partial^2 V}{\partial X^2} + \frac{\partial^2 V}{\partial Y^2}\big) &= \frac{e_1 UV}{1+b_1U} - pVW - m_1 V, \\
\frac{\partial W}{\partial T} - D_3\big(\frac{\partial^2 W}{\partial X^2} + \frac{\partial^2 W}{\partial Y^2}\big) &= \frac{e_2 UW}{1+b_2U} - m_2 W,
\end{aligned} \tag{4.1}$$

with the following boundary conditions

$$\begin{aligned}
\frac{\partial U}{\partial \mathbf{n}}|_{\partial\Omega} &= \frac{\partial V}{\partial \mathbf{n}}|_{\partial\Omega} = \frac{\partial W}{\partial \mathbf{n}}|_{\partial\Omega} = 0, T > 0, \\
U(X,Y,0) &= U_0(X,Y) \geq 0, V(X,Y,0) = V_0(X,Y) \geq 0, \\
W(X,Y,0) &= W_0(X,Y) \geq 0, (X,Y) \in \Omega,
\end{aligned}$$

where the positive parameters $r, K, a_1, a_2, b_1, b_2, e_1, e_2, m_1, m_2, p, D_1, D_2$ and D_3 are interpreted in Table 4.1. $\frac{\partial}{\partial \mathbf{n}}|_{\partial\Omega}$ is the outward normal of $\partial\Omega$.

To reduce the number of parameters, the system (4.1) is non-dimensionalised using the transformations $u = \frac{U}{K}$, $t = rT$, $v = \frac{a_1 V}{r}$, $w = \frac{a_2 W}{r}$, $x = X\sqrt{\frac{r}{D_1}}$, $y = Y\sqrt{\frac{r}{D_1}}$, to obtain the following transformed system

Table 4.1: Parameters for system (4.1)

Parameter	Description
r	Intrinsic growth rate of the impala
K	Environmental carrying capacity of the impala
a_1	Capture rate of the impala by the cheetah
a_2	Capture rate of the impala by the lion;
b_1	Handling time of impala by cheetah
b_2	Handling time of impala by lion
e_1	Conversion rate of impala biomass into new cheetah
e_2	Conversion rate of impala biomass into new lion
p	Mortality rate of cheetah by lion
m_1	Natural mortality rate of cheetahs
m_2	Natural mortality rate of lion
D_1	Diffusion coefficient of impala
D_2	Diffusion coefficient of cheetah
D_3	Diffusion coefficient of lion

$$\begin{aligned}
\frac{\partial u}{\partial t} - \frac{D_1}{r}\left(\frac{\partial^2 u}{\partial X^2} + \frac{\partial^2 u}{\partial Y^2}\right) &= u(1-u) - \frac{a_1 uV}{r(1+b_1 Ku)} - \frac{a_2 uW}{r(1+b_2 Ku)}, \\
\frac{\partial V}{\partial t} - \frac{D_2}{r}\left(\frac{\partial^2 V}{\partial X^2} + \frac{\partial^2 V}{\partial Y^2}\right) &= \frac{e_1 K}{r}\frac{uV}{(1+b_1 Ku)} - \frac{p}{r}VW - \frac{m_1}{r}V, \\
\frac{\partial W}{\partial t} - \frac{D_3}{r}\left(\frac{\partial^2 W}{\partial X^2} + \frac{\partial^2 W}{\partial Y^2}\right) &= \frac{e_2 K}{r}\frac{uW}{(1+b_2 Ku)} - \frac{m_2}{r}W.
\end{aligned}$$

The above system becomes

$$\begin{aligned}
\frac{\partial u}{\partial t} - \left(\frac{\partial^2 u}{\partial x^2} + \frac{\partial^2 u}{\partial y^2}\right) &= u(1-u) - \frac{uv}{(1+\beta_1 u)} - \frac{uw}{1+\beta_2 u}, \\
\frac{\partial v}{\partial t} - \frac{D_2}{D_1}\left(\frac{\partial^2 v}{\partial x^2} + \frac{\partial^2 v}{\partial y^2}\right) &= \frac{e_1 K}{r}\frac{uv}{(1+\beta_1 u)} - \frac{p}{a_2}vw - \frac{m_1}{r}v, \\
\frac{\partial w}{\partial t} - \frac{D_3}{D_1}\left(\frac{\partial^2 w}{\partial x^2} + \frac{\partial^2 w}{\partial y^2}\right) &= \frac{e_2 K}{r}\frac{uw}{(1+\beta_2 u)} - \frac{m_2}{r}w.
\end{aligned}$$

Finally, the system can be written as

$$\begin{aligned}
\frac{\partial u}{\partial t} - \big(\frac{\partial^2 u}{\partial x^2} + \frac{\partial^2 u}{\partial y^2}\big) &= u(1-u) - \frac{uv}{(1+\beta_1 u)} - \frac{uw}{1+\beta_2 u}, \\
\frac{\partial v}{\partial t} - d_2\big(\frac{\partial^2 v}{\partial x^2} + \frac{\partial^2 v}{\partial y^2}\big) &= \frac{\mu_1 uv}{1+\beta_1 u} - \nu vw - \delta_1 v, \\
\frac{\partial w}{\partial t} - d_3\big(\frac{\partial^2 w}{\partial x^2} + \frac{\partial^2 w}{\partial y^2}\big) &= \frac{\mu_2 uw}{1+\beta_2 u} - \delta_2 w,
\end{aligned} \tag{4.2}$$

with boundary conditions

$$\begin{aligned}
\frac{\partial u}{\partial \mathbf{n}}|_{\partial\Omega} &= \frac{\partial v}{\partial \mathbf{n}}|_{\partial\Omega} = \frac{\partial w}{\partial \mathbf{n}}|_{\partial\Omega} = 0, t > 0, \\
u(x,y,0) &= u_0(x,y) \geq 0, v(x,y,0) = v_0(x,y) \geq 0, \\
w(x,y,0) &= w_0(x,y) \geq 0, (x,y) \in \Omega,
\end{aligned}$$

where $\beta_1 = Kb_1$, $\beta_2 = Kb_2$, $\delta_1 = \frac{m_1}{r}$, $\delta_2 = \frac{m_2}{r}$, $\nu = \frac{p}{a_2}$, $\mu_1 = \frac{e_1 K}{r}$, $\mu_2 = \frac{e_2 K}{r}$, $d_2 = \frac{D_2}{D_1}$ and $d_3 = \frac{D_3}{D_1}$. The homogeneous Neumann boundary condition means that system (4.2) is isolated and no population can move across the boundary of Ω.

It is necessary to investigate the temporal dynamics of the system before studying the spatio-temporal system (4.2). In the absence of population gradient the spatio-temporal system (4.2) is reduced to a system which is written as follows

$$\begin{aligned}
\frac{du}{dt} &= u(1-u) - \frac{uv}{(1+\beta_1 u)} - \frac{uw}{1+\beta_2 u} = G_1(u,v,w), \\
\frac{dv}{dt} &= \frac{\mu_1 uv}{1+\beta_1 u} - \nu vw - \delta_1 v = G_2(u,v,w), \\
\frac{dw}{dt} &= \frac{\mu_2 uw}{1+\beta_2 u} - \delta_2 w = G_3(u,v,w), \\
u(0) &= u_0 \geq 0, v(0) = v_0 \geq 0, w(0) = w_0 \geq 0.
\end{aligned} \tag{4.3}$$

Denoting $\mathbf{u} = (u,v,w)^T$ and $\mathbf{G}(\mathbf{u}) = (G_1(\mathbf{u}), G_2(\mathbf{u}), G_3(\mathbf{u}))$, the system (4.3) can be written as:

$$\begin{cases} \dfrac{d\mathbf{u}}{dt} = \mathbf{G}(\mathbf{u}), \\ \mathbf{u}(0) = \mathbf{u}_0 \geq 0. \end{cases} \tag{4.4}$$

4.2.1 Boundedness

Since all the parameters of system (4.3) are non-negative, the right-hand side is a smooth function of the variables (u, v, w) in the positive octant $\bar{\Gamma} = \{(u, v, w) : u > 0, v > 0, w > 0\}$. Thus local existence and uniqueness properties hold for its solution. The following claim is made.

Proposition 4.1 *$\bar{\Gamma}$ is an invariant set.*

Proof. The system (4.3) is homogeneous, so that the coordinate planes $u = 0$, $v = 0$, $w = 0$ all solve it. By the existence and uniqueness theorem, any trajectory starting in the first octant remains there and will not cross the coordinate planes. Thus, $\bar{\Gamma}$ is an invariant set. □

Proposition 4.2 *All solutions of (4.3) starting in $\bar{\Gamma}$ are uniformly bounded if $\mu_1 < 1$ and $\mu_2 < 1$.*

Proof. Define the function

$$\psi = u + v + w. \tag{4.5}$$

Differentiating ψ with respect to time along the solutions of (4.3) gives

$$\frac{d\psi}{dt} = \frac{du}{dt} + \frac{dv}{dt} + \frac{dw}{dt}$$

or

$$\begin{aligned}
\frac{d\psi}{dt} &= u(1-u) - \frac{uv}{(1+\beta_1 u)} - \frac{uw}{1+\beta_2 u} + \frac{\mu_1 uv}{1+\beta_1 u} - \nu vw - \delta_1 v + \frac{\mu_2 uw}{1+\beta_2 u} - \delta_2 w, \\
&= u(1-u) - \frac{(1-\mu_1)uv}{(1+\beta_1 u)} - \frac{(1-\mu_2)uw}{1+\beta_2 u} - \nu vw - \delta_1 v - \delta_2 w, \\
&\leq u(1-u) - \delta_1 v - \delta_2 w, \\
&= u(1-u) + \phi u - \phi u - \delta_1 v + v\phi - v\phi - \delta_2 w + w\phi - w\phi, \\
&= u[(1+\phi) - u] - (\delta_1 - \phi)v - (\delta_2 - \phi)w - \phi\psi.
\end{aligned}$$

Thus,

$$\begin{aligned}\frac{d\psi}{dt}+\phi\psi &\leq -[u^2-u(1+\phi)]-(\delta_1-\phi)v-(\delta_2-\phi)w,\\ &= -\big(u-\frac{(1+\phi)}{2}\big)^2+\frac{(1+\phi)^2}{4}-(\delta_1-\phi)v-(\delta_2-\phi)w,\\ &\leq \frac{(1+\phi)^2}{4}-(\delta_1-\phi)v-(\delta_2-\phi)w.\end{aligned}$$

Choosing ϕ to be such that $\phi \leq \min\{\delta_1, \delta_2\}$ the right-hand side will be bounded. Let φ be such that

$$\frac{d\psi}{dt}+\phi\psi \leq \frac{(1+\phi)^2}{4}=\varphi. \tag{4.6}$$

Solving this we obtain

$$\psi(t) \leq Ce^{-\phi t}+\frac{\varphi}{\phi}$$

and

$$\psi(t) \leq \psi(0)e^{-\phi t}+\frac{\varphi}{\phi}(1-e^{-\phi t}) \leq \max(\psi(0), \frac{\psi}{\phi}).$$

Moreover, $\limsup \psi(t) \leq \frac{\varphi}{\phi}$ as $t \to \infty$ independent of initial conditions. □

4.2.2 Steady states

The system (4.3) has five nonnegative equilibria

(i) $E_0(u=0, v=0, w=0)$ (trivial equilibrium): $E_0(u_0, v_0, w_0) = E_0(0,0,0)$

(ii) $E_1(u \neq 0, v=0, w=0)$ (axial equilibrium): From (4.3) the system reduces to

$$1-u=0$$

with the result $E_1(u_1, v_1, w_1) = E_1(1,0,0)$.

(iii) $E_2(u \neq 0, v \neq 0, w=0)$ (boundary equilibrium): From (4.3) the system reduces to

$$\begin{aligned}1-u-\frac{v}{(1+\beta_1 u)}&=0\\ \frac{\mu_1 u}{1+\beta_1 u}-\delta_1&=0\end{aligned}$$

with the result $E_2(u_2, v_2, w_2) = E_2\Big(\frac{\delta_1}{\mu_1-\beta_1\delta_1}, \frac{\mu_1(\mu_1-\beta_1\delta_1-\delta_1)}{(\mu_1-\beta_1\delta_1)^2}, 0\Big)$. The boundary equilibrium E_2 exists if $\mu_1 > \delta_1(\beta_1 + 1)$.

(iv) $E_3(u \neq 0, v = 0, w \neq 0)$ (boundary equilibrium): From (4.3)

$$1 - u - \frac{w}{1 + \beta_2 u} = 0$$
$$\frac{\mu_2 u}{1 + \beta_2 u} - \delta_2 = 0$$

with the result $E_3(u_3, v_3, w_3) = E_3\Big(\frac{\delta_2}{\mu_2-\beta_2\delta_2}, 0, \frac{\mu_2(\mu_2-\beta_2\delta_2-\delta_2)}{(\mu_2-\beta_2\delta_2)^2}\Big)$. The boundary equilibrium E_3 exists if $\mu_2 > \delta_2(\beta_2 + 1)$.

(v) The positive (interior) equilibrium point $E_4(u \neq 0, v \neq 0, w \neq 0)$ or constant positive solution exists if there is a positive solution to the following set of non-linear equations from the system (4.3):

$$1 - u - \frac{v}{(1 + \beta_1 u)} - \frac{w}{1 + \beta_2 u} = 0 \tag{4.7a}$$
$$\frac{\mu_1 u}{1 + \beta_1 u} - \nu w - \delta_1 = 0 \tag{4.7b}$$
$$\frac{\mu_2 u}{1 + \beta_2 u} - \delta_2 = 0 \tag{4.7c}$$

whose solution yields the point $E_4(u_4, v_4, w_4)$ where

$$\begin{cases} u_4 = \dfrac{\delta_2}{\mu_2 - \beta_2\delta_2}, \\ v_4 = 1 - \beta_1 u_4^2 + u_4 + \beta_1 u_4 - \dfrac{w_4}{1 + u_4} - \dfrac{u_4 w_4}{1 + u_4}, \\ w_4 = \dfrac{-\beta_1\delta_1\delta_2 + \beta_2\delta_1\delta_2 + \delta_2\mu_1 - \delta_1\mu_2}{\nu(\beta_1\delta_2 - \beta_2\delta_2 + \mu_2)} = \dfrac{u_4(\mu_1 - \beta_1\delta_1) - \delta_1}{\nu(\beta_1 u_4 + 1)}, \end{cases} \tag{4.8}$$

The constant positive solution E_4 exists if and only if

$$\begin{cases} \mu_2 > \beta_2\delta_2, \\ \mu_2 + \beta_1\delta_2 > \beta_2\delta_2, \\ \delta_2\mu_1 > \delta_1(\mu_2 + \beta_1\delta_2 - \beta_2\delta_2), \\ 1 > \beta_1 u_4^2 + \dfrac{w_4}{1 + u_4} + \dfrac{u_4 w_4}{1 + u_4}, \end{cases} \tag{4.9}$$

that is,

$$\begin{cases} \frac{\mu_2}{\delta_2} > \beta_2, \\ \frac{\mu_2}{\delta_2} > \beta_2 - \beta_1, \\ \frac{\mu_1}{\delta_1} - \beta_1 > \frac{\mu_2}{\delta_2} - \beta_2, \\ 1 > \beta_1 u_4^2 + \dfrac{w_4}{1+u_4} + \dfrac{u_4 w_4}{1+u_4}. \end{cases} \tag{4.10}$$

Firstly, the ratio of input/output of lion biomass must outweigh the time the lion spends consuming impala. Secondly, the ratio of input/output of cheetah biomass must outweigh the difference in time the cheetah and lion spend consuming impala. Thirdly, the ratio of input/output of cheetah biomass less the time the cheetah spends consuming impala exceeds that of the lion.

Furthermore, the existence of the positive solution was established using the approach of Dubey and Upadhyay [99]. Two functions $f(u,v)$ and $g(u,v)$ which intersect at the equilibrium point $E_4(u_4, v_4, w_4)$ are deduced. Equations (4.7a) and (4.7b) can be rewritten as

$$w = \frac{(1-u-v+u\beta_1+u^2\beta_1)(1+u\beta_2)}{1+u\beta_1}, \tag{4.11}$$

$$w = \frac{u\mu_1 - u\beta_1\delta_1 - \delta_1}{\nu(1+u\beta_1)}. \tag{4.12}$$

From (4.7a) and (4.7c),

$$w = \frac{u(1-u-v+u\beta_1+u^2\beta_1)\mu_2}{(1+u\beta_1)\delta_2}. \tag{4.13}$$

From (4.11) and (4.12),

$$f(u,v) = \frac{(1-u-v+u\beta_1+u^2\beta_1)(1+u\beta_2)}{1+u\beta_1} - \frac{u\mu_1 - u\beta_1\delta_1 - \delta_1}{\nu(1+u\beta_1)} = 0. \tag{4.14}$$

From (4.11) and (4.13) define

$$g(u,v) = \frac{(1-u-v+u\beta_1+u^2\beta_1)(1+u\beta_2)}{1+u\beta_1} - \frac{u(1-u-v+u\beta_1+u^2\beta_1)\mu_2}{(1+u\beta_1)\delta_2} = 0. \tag{4.15}$$

Two functions of u and v have been obtained as a result of reducing the system of equations. The existence of the equilibrium point is based on the existence of

an intersection point (u_4, v_4) of the functions $f(u, v)$ and $g(u, v)$ in the positive quadrant. The value of z_4 can be deduced once the existence of (u_4, v_4) has been established. From (4.14), as $u \to 0$, $v \to v_f$ given by

$$v_f = \frac{\nu + \delta_1}{\nu}. \tag{4.16}$$

Note that v_f is positive and $v_f > 1$. In a similar way, from (4.15) as $u \to 0$, $v \to v_g$ given by

$$v_g = 1 \tag{4.17}$$

The functions v_f and v_g are the points where the functions $f(u, v)$ and $g(u, v)$ cross the v-axis of the uv-plane respectively. From equation (4.14),

$$\frac{dv}{du} = -\frac{\partial f}{\partial u} \Big/ \frac{\partial f}{\partial v}$$

where

$$\begin{aligned}
\frac{\partial f}{\partial u} &= \frac{(1 - u - v + u\beta_1 + u^2\beta_1)\beta_2}{1 + u\beta_1} - \frac{(1 - \beta_1 + 2u\beta_1)(1 + u\beta_2)}{1 + u\beta_1} \\
&\quad - \frac{\beta_1(1 - u - v + u\beta_1 + u^2\beta_1)(1 + u\beta_2)}{(1 + u\beta_1)^2} - \frac{\mu_1 - \beta_1\delta_1}{\nu(1 + u\beta_1)} + \frac{\beta_1(u\mu_1 - u\beta_1\delta_1 - \delta_1)}{\nu(1 + u\beta_1)^2}, \\
&= \frac{[(1 + u\beta_1)(u - 1) + v](1 + u\beta_2)[\beta_1 - (1 + u\beta_1)(1 + \beta_2 + \beta_1(2u - 1))]\nu - \mu_1}{\nu(1 + u\beta_1)^2},
\end{aligned}$$

and

$$\frac{\partial f}{\partial v} = -\frac{1 + \beta_2 u}{1 + \beta_1 u} < 0.$$

Now, $\frac{dv}{du} < 0$ if $\frac{\partial f}{\partial u} < 0$ which are obtained by setting $2u > 1$ and $\beta_1 < (1 + u\beta_1)(1 + \beta_2 + \beta_1(2u - 1))$. From equation (4.15), we can write

$$\frac{dv}{du} = -\frac{\partial g}{\partial u} \Big/ \frac{\partial g}{\partial v}$$

where

$$\begin{aligned}\frac{\partial g}{\partial u} &= \frac{(1-u-v+u\beta_1+u^2\beta_1)\beta_2}{1+u\beta_1} - \frac{(1-\beta_1+2u\beta_1)(1+u\beta_2)}{1+u\beta_1} \\ &+\frac{\beta_1(1-u-v+u\beta_1+u^2\beta_1)(1+u\beta_2)}{(1+u\beta_1)^2} + \frac{u(1-\beta_1+2u\beta_1)\mu_2}{(1+u\beta_1)\delta_2} \\ &-\frac{u\beta_1(1-u-v+u\beta_1+u^2\beta_1)\mu_2}{(1+u\beta_1)^2\delta_2} - \frac{(1-u-v+u\beta_1+u^2\beta_1)\mu_2}{(1+u\beta_1)\delta_2}, \\ &= \frac{[(1+u\beta_1)(u-1)+v][(\beta_1-\beta_2)\delta_2+\mu_2]}{(1+u\beta_1)^2\delta_2} \\ &+\frac{(1+u\beta_1)(1+\beta_1(2u-1))[u\mu_2-\delta_2(1+u\beta_2)]}{(1+u\beta_1)^2\delta_2}\end{aligned}$$

and

$$\frac{\partial g}{\partial v} = \frac{\mu_2 u - \delta_2(1+\beta_2 u)}{\delta_2(1+\beta_2 u)}.$$

Now, $\frac{dv}{du} < 0$ if (a) $\frac{\partial g}{\partial u} > 0$ and $\frac{\partial g}{\partial v} < 0$ which are obtained by setting $u > 1$, $\beta_1 > \beta_2$ and $u\mu_2 > \delta_2(1+u\beta_2)$; (b) $\frac{\partial g}{\partial u} < 0$ and $\frac{\partial g}{\partial v} > 0$ which are obtained by setting $u < 1$ and $u\mu_2 < \delta_2(1+u\beta_2)$. If follows that $f(u,v)$ and $g(u,v)$ intersect at a positive point (u,v) since for $f(u,v)$, $\frac{dv}{du} < 0$, $v_f > v_g$ and for $g(u,v)$, $\frac{dv}{du} > 0$.

4.2.3 Analysis of steady states

The dynamical behaviour of the system can be studied by computing the Jacobian corresponding to each equilibrium point. The Jacobian is given by

$$J = \begin{bmatrix} j_{11} & -\frac{u}{1+u\beta_1} & -\frac{u}{1+u\beta_2} \\ \frac{v\mu_1}{1+u\beta_1} - \frac{uv\mu_1\beta_1}{(1+u\beta_1)^2} & \frac{\mu_1 u}{1+u\beta_1} - \nu w - \delta_1 & -\nu v \\ \frac{w\mu_2}{1+u\beta_2} - \frac{uw\beta_2\mu_2}{(1+u\beta_2)^2} & 0 & \frac{\mu_2 u}{1+u\beta_2} - \delta_2 \end{bmatrix},$$

where $j_{11} = 1 - 2u + \frac{uw\beta_2}{(1+u)^2} - \frac{w}{1+u\beta_2} + \frac{uv\beta_1}{(1+u\beta_1)^2} - \frac{v}{1+u\beta_1}$.

Denote by $J_k = J$ the Jacobian evaluated at E_k and similarly for $j_{ij}^{[k]} = j_{ij}$, $i = 1,2,3$, $j = 1,2,3$, $k = 1,2,3,4$. The Jacobian matrix is evaluated at each equilibrium point and then used to determine the stability of that point.

Local stability of E_1

For the axial equilibrium point $E_1(1,0,0)$, the Jacobian is given by

$$J_1 = \begin{bmatrix} -1 & -\frac{1}{1+\beta_1} & -\frac{1}{1+\beta_2} \\ 0 & \frac{\mu_1}{1+\beta_1} - \delta_1 & 0 \\ 0 & 0 & \frac{\mu_2}{1+\beta_2} - \delta_2 \end{bmatrix}.$$

The corresponding eigenvalues are -1, $-\delta_1 + \frac{\mu_1}{1+\beta_1}$ and $-\delta_2 + \frac{\mu_2}{1+\beta_2}$. Thus, the axial equilibrium point is unstable, a saddle point, since $-\delta_1 + \frac{\mu_1}{1+\beta_1} > 0$ (or $\mu_1 - \delta_1 - \delta_1\beta_1 > 0$, condition for existence of E_2) and $-\delta_2 + \frac{\mu_2}{1+\beta_2} > 0$ (or $\mu_2 - \delta_2 - \delta_2\beta_2 > 0$, condition for existence of E_3). Thus, whenever E_1 is asymptotically stable, the equilibrium points E_2 and E_3 do not exist.

Local and global asymptotic stability of E_2

For the boundary equilibrium $E_2(u_2, v_2, 0)$ the Jacobian is given by

$$J_2 = \begin{bmatrix} 1 - 2u_2 - \frac{v_2}{(1+u_2\beta_1)^2} & -\frac{u_2}{1+u_2\beta_1} & -\frac{u_2}{1+u_2\beta_2} \\ \frac{v_2\mu_1}{(1+u_2\beta_1)^2} & \frac{\mu_1 u_2}{1+u_2\beta_1} 0 - \delta_1 & -\nu v_2 \\ 0 & 0 & \frac{\mu_2 u_2}{1+u_2\beta_2} - \delta_2 \end{bmatrix},$$

where $u_2 = \frac{\delta_1}{\mu_1 - \beta_1\delta_1}$ and $v_2 = \frac{\mu_1(\mu_1 - \beta_1\delta_1 - \delta_1)}{(\beta_1\delta_1 - \mu_1)^2}$. The corresponding eigenvalues are $\lambda_1 = -\delta_2 + \frac{\mu_2 u_2}{1+u_2} = -\delta_2 - \frac{\mu_2\delta_1}{(\beta_1\delta_1 - \mu_1)(1 - \frac{\beta_2\delta_1}{\beta_1\delta_1 - \mu_1})}$, and the roots of

$$a_0\lambda^2 + a_1\lambda + a_2 = 0$$

where $a_0 = \beta_1\delta_1\mu_1 - \mu_1^2$, $a_1 = -\beta_1\delta_1^2 - \beta_1^2\delta_1^2 - \delta_1\mu_1 + \beta_1\delta_1\mu_1$ and $a_2 = -\beta_1\delta_1^3 - \beta_1^2\delta_1^3 + \delta_1^2\mu_1 + 2\beta_1\delta_1^2\mu_1 - \delta_1\mu_1^2$. Thus $\lambda_2 = \frac{-a_1 - \sqrt{a_1^2 - 4a_0a_2}}{2a_0}$ and $\lambda_3 = \frac{-a_1 + \sqrt{a_1^2 - 4a_0a_2}}{2a_0}$. The conditions so that the system has a stable equilibrium point E_2 are deduced. Since $a_0 < 0$, stability is ensured by requiring that $a_1 < 0$, and $\delta_2 > \frac{\mu_2\delta_1}{(\beta_1\delta_1 - \mu_1)(1 - \frac{\beta_2\delta_1}{\beta_1\delta_1 - \mu_1})}$. Furthermore, the equilibrium point E_2 is a stable node if $a_1^2 - 4a_0a_2 > 0$ and a stable spiral $a_1^2 - 4a_0a_2 < 0$.

Now the global stability of E_2 is established using the Bendixson-Dulac criterion, Theorem A.1, [124] and the Poincaré-Bendixson Theorem, Theorem A.2, [125].

Proposition 4.3 *E_2 is globally asymptotically stable if $2\delta_1 > \mu_1$.*

Proof. Consider subsystem (i) of system (4.3)

$$\begin{aligned} \frac{du}{dt} &= u(1-u) - \frac{uv}{(1+\beta_1 u)}, \\ \frac{dv}{dt} &= \frac{\mu_1 uv}{1+\beta_1 u} - \delta_1 v. \end{aligned} \tag{4.18}$$

Let $\Phi(u,v) = \frac{1}{uv}$, $h_1(u,v) = u(1-u) - \frac{uv}{1+\beta_1 u}$, $h_2(u,v) = \frac{\mu_1 uv}{1+\beta_1 u} - \delta_1 v$. Now $\Phi(u,v) > 0$ in the interior of the $u-v$ plane. Then

$$\begin{aligned} \Delta(u,v) &= \frac{\partial(\Phi h_1)}{\partial u} + \frac{\partial(\Phi h_2)}{\partial v}, \\ &= \frac{\partial}{\partial u}\Big(\frac{1-u}{v} - \frac{1}{1+\beta_1 u}\Big) + \frac{\partial}{\partial v}\Big(\frac{1}{1+\beta_1 u} - \frac{\delta_1}{u}\Big), \\ &= -\frac{1}{v} + \frac{\beta_1}{(1+\beta_1 u)^2}, \\ &= \frac{\beta_1 v - (1+\beta_1 u)^2}{v(1+\beta_1 u)^2}. \end{aligned}$$

Now Δ is not identically zero in the positive quadrant of the $u-v$ plane. A claim is made that $\Delta(u,v) < 0$ and does not change sign if

$$\beta_1 v - (1+\beta_1 u)^2 = \beta_1 v - 1 - 2\beta_1 u - u^2 = -1 - (2u - v)\beta_1 - u^2 < 0$$

that is, if $2u - v > 0$. This means

$$\begin{aligned} 2u - v &= \frac{2\delta_1}{\mu_1 - \beta_1\delta_1} - \frac{\mu_1(\mu_1 - \beta_1\delta_1 - \delta_1)}{(\mu_1 - \beta_1\delta_1)^2}, \\ &= \frac{2\delta_1(\mu_1 - \beta_1\delta_1) - \mu_1(\mu_1 - \beta_1\delta_1) + \mu_1\delta_1}{(\mu_1 - \beta_1\delta_1)^2}, \\ &= \frac{(\mu_1 - \beta_1\delta_1)(2\delta_1 - \mu_1) + \mu_1\delta_1}{(\mu_1 - \beta_1\delta_1)^2} \\ &> 0, \end{aligned}$$

if and only if $2\delta_1 > \mu_1$. Thus, subsystem (4.18) does not have a limit cycle in $\bar{\Gamma}$. Since E_2 is locally asymptotically stable, the Poincaré-Bendixson Theorem, Theorem A.2, [125] and Proposition 4.2 imply that the equilibrium point E_2 is globally asymptotically stable in the region $\bar{\Gamma}$. □

Local and global asymptotic stability of E_3

For the boundary equilibrium $E_3(u_3, 0, w_3)$ the Jacobian is given by

$$J_3 = \begin{bmatrix} 1 - 2u_3 - \frac{w_3}{(1+u_3\beta_2)^2} & -\frac{u_3}{1+u_3\beta_1} & -\frac{u_3}{1+u_3\beta_2} \\ 0 & \frac{\mu_1 u_3}{1+u_3\beta_1} - \nu w_3 - \delta_1 & 0 \\ \frac{w_3\mu_2}{(1+u_3\beta_2)^2} & 0 & \frac{\mu_2 u_3}{1+u_3\beta_2} - \delta_2 \end{bmatrix},$$

where $u_3 = \frac{\delta_2}{\mu_2 - \beta_2\delta_2}$ and $w_3 = \frac{\mu_2(\mu_2-\beta_2\delta_2-\delta_2)}{(\beta_2\delta_2-\mu_2)^2}$. The corresponding eigenvalues are $\lambda_1 = -\delta_1 + \frac{\mu_1 u_3}{1+u_3} - \nu w_3 = -\delta_1 - \frac{\mu_1\delta_2}{(\beta_2\delta_2-\mu_2)(1-\frac{\beta_2\delta_2}{\beta_2\delta_2-\mu_2})} - \frac{\nu\mu_2(\mu_2-\beta_2\delta_2-\delta_2)}{(\beta_2\delta_2-\mu_2)^2}$, and the roots of

$$b_0\lambda^2 + b_1\lambda + b_2 = 0,$$

where $b_0 = \beta_2\delta_2\mu_2 - \mu_2^2$, $b_1 = -\beta_2\delta_2^2 - \beta_2^2\delta_2^2 - \delta_2\mu_2 + \beta_2\delta_2\mu_2$ and $b_2 = -\beta_2\delta_2^3 - \beta_2^2\delta_2^3 + \delta_2^2\mu_2 + 2\beta_2\delta_2^2\mu_2 - \delta_2\mu_2^2$. Thus $\lambda_2 = \frac{-b_1-\sqrt{b_1^2-4b_0b_2}}{2b_0}$ and $\lambda_3 = \frac{-b_1+\sqrt{b_1^2-4b_0b_2}}{2b_0}$. Conditions so that the system has a stable equilibrium point E_3 are deduced. Since $b_0 < 0$, stability is ensured by requiring that $b_1 > 0$ and $\delta_1 + \nu w_3 > \frac{\mu_1 u_3}{1+u_3}$. Further, the equilibrium point E_3 is a stable node if $b_1^2 - 4b_0b_2 > 0$ and a stable spiral $b_1^2 - 4b_0b_2 < 0$.

As with E_2 the global stability of E_3 is established using the Bendixson-Dulac criterion, Theorem A.1, [124] and the Poincaré-Bendixson Theorem, Theorem A.2, [125].

Proposition 4.4 *E_3 is globally asymptotically stable if $2\delta_2 > \mu_2$.*

Proof. Consider subsystem (ii) of system (4.3)

$$\begin{aligned} \frac{du}{dt} &= u(1-u) - \frac{uw}{(1+\beta_2 u)}, \\ \frac{dw}{dt} &= \frac{\mu_2 uw}{1+\beta_2 u} - \delta_2 w. \end{aligned} \tag{4.19}$$

Let $\Psi(u, w) = \frac{1}{vw}$, $g_1(u, w) = u(1-u) - \frac{uw}{(1+\beta_2 u)}$, $g_2(u, w) = \frac{\mu_2 uw}{1+\beta_2 u} - \delta_2 w$. Now $\Psi(u, w) > 0$ in the interior of the $u - w$ plane. Then

$$\begin{aligned}\Delta(u,w) &= \frac{\partial(\Psi g_1)}{\partial v}+\frac{\partial(\Psi g_2)}{\partial w},\\ &= \frac{\partial}{\partial u}\Big(\frac{1-u}{w}-\frac{1}{1+\beta_2 u}\Big)+\frac{\partial}{\partial w}\Big(\frac{1}{1+\beta_2 u}-\frac{\delta_2}{u}\Big),\\ &= -\frac{1}{w}+\frac{\beta_2}{(1+\beta_2 u)^2},\\ &= \frac{\beta_2 w-(1+\beta_2 u)^2}{w(1+\beta_2 u)^2}.\end{aligned}$$

Now Δ is not identically zero in the positive quadrant of the $u-w$ plane. A claim is made that $\Delta(u,w)<0$ and does not change sign if

$$\beta_2 w-(1+\beta_2 u)^2=\beta_2 w-1-2\beta_2 u-u^2=-1-(2u-w)\beta_2-u^2<0,$$

that is, if $2u-w>0$. This means

$$\begin{aligned}2u-w &= \frac{2\delta_2}{\mu_2-\beta_2\delta_2}-\frac{\mu_2(\mu_2-\beta_2\delta_2-\delta_2)}{(\mu_2-\beta_2\delta_2)^2},\\ &= \frac{2\delta_2(\mu_2-\beta_2\delta_2)-\mu_2(\mu_2-\beta_2\delta_2)+\mu_2\delta_2}{(\mu_2-\beta_2\delta_2)^2},\\ &= \frac{(\mu_2-\beta_2\delta_2)(2\delta_2-\mu_2)+\mu_2\delta_2}{(\mu_2-\beta_2\delta_2)^2},\\ &> 0,\end{aligned}$$

if and only if $2\delta_2>\mu_2$. Thus, subsystem (4.19) does not have a limit cycle in $\bar{\Gamma}$. Since E_3 is locally asymptotically stable, the Poincaré-Bendixson Theorem, Theorem A.2, [125] and Proposition 4.2 imply that the equilibrium point E_3 is globally asymptotically stable in the region $\bar{\Gamma}$. □

Local and global asymptotic stability of interior equilibrium E_4

The eigenvalues for the positive equilibrium $E_4(u_4,v_4,w_4)$ are not available in explicit form. The Jacobian matrix is given by

$$J_4=\begin{bmatrix} 1-2u_4-\frac{v_4}{(1+u_4\beta_1)^2}-\frac{w_4}{(1+\beta_2 u_4)^2} & -\frac{u_4}{1+u_4\beta_1} & -\frac{u_4}{1+u_4\beta_2}\\ \frac{\mu_1 v_4}{(1+\beta_1 u_4)^2} & \frac{\mu_1 u_4}{1+\beta_1 u_4}-\nu w_4-\delta_1 & -\nu v_4\\ \frac{w_4\mu_2}{(1+u_4\beta_2)^2} & 0 & \frac{\mu_2 u_4}{1+u_4\beta_2}-\delta_2\end{bmatrix}, \qquad (4.20)$$

where
$j_{11}^{[4]} = 1 - 2u_4 + \frac{u_4 v_4 \beta_1}{(1+u_4\beta_1)^2} - \frac{v_4}{1+u_4\beta_1} + \frac{u_4 w_4 \beta_2}{(1+\beta_2 u_4)^2} - \frac{w_4}{1+u_4\beta_2}$, $j_{12}^{[4]} = -\frac{u_4}{1+u_4\beta_1}$,
$j_{13}^{[4]} = -\frac{u_4}{1+u_4\beta_2}$, $j_{21}^{[4]} = \frac{v_4\mu_1}{1+\beta_1 u_4} - \frac{u_4\mu_1\beta_1 v_4}{(1+\beta_1 u_4)^2}$, $j_{22}^{[4]} = \frac{\mu_1 u_4}{1+\beta_1 u_4} - \nu w_4 - \delta_1$, $j_{23}^{[4]} = -\nu v_4$,
$j_{31}^{[4]} = \frac{w_4\mu_2}{1+\beta_2 u_4} - \frac{u_4\beta_2 w_4\mu_2}{(1+u_4\beta_2)^2}$ and $j_{33}^{[4]} = \frac{\mu_2 u_4}{1+u_4\beta_2} - \delta_2$.
We note that $j_{12}^{[4]} < 0$, $j_{13}^{[4]} < 0$, $j_{21}^{[4]} > 0$, $j_{23}^{[4]} < 0$, $j_{31}^{[4]} > 0$.

The characteristic equation of the Jacobian matrix about E_4 is given by

$$\lambda^3 + A_1\lambda^2 + A_2\lambda + A_3 = 0,$$

where

$$\begin{cases} A_1 = -(j_{11}^{[4]} + j_{22}^{[4]} + j_{33}^{[4]}) = -\mathrm{Tr}(J_4), \\ A_2 = j_{11}^{[4]}j_{22}^{[4]} + j_{11}^{[4]}j_{33}^{[4]} + j_{22}^{[4]}j_{33}^{[4]} - j_{12}^{[4]}j_{21}^{[4]} - j_{13}^{[4]}j_{31}^{[4]}, \\ A_3 = -j_{12}^{[4]}j_{23}^{[4]}j_{31}^{[4]} + j_{13}^{[4]}j_{22}^{[4]}j_{31}^{[4]} + j_{12}^{[4]}j_{21}^{[4]}j_{33}^{[4]} - j_{11}^{[4]}j_{22}^{[4]}j_{33}^{[4]} = -\det J_4. \end{cases} \tag{4.21}$$

According to Routh-Hurwitz criterion, the necessary and sufficient conditions for local stability of equilibrium point E_4 are

$$A_1 > 0, A_2 > 0, A_3 > 0 \quad \text{and} \quad A_1A_2 - A_3 > 0. \tag{4.22}$$

Now, $j_{12}^{[4]} < 0$, $j_{13}^{[4]} < 0$, $j_{21}^{[4]} > 0$, $j_{23}^{[4]} < 0$ and $j_{31}^{[4]} > 0$. Thus, $A_1 > 0$ and $A_2 > 0$ if $j_{11}^{[4]} < 0$, $j_{22}^{[4]} < 0$ and $j_{33}^{[4]} < 0$. $A_3 > 0$ if $j_{13}^{[4]}j_{22}^{[4]}j_{31}^{[4]} + j_{12}^{[4]}j_{21}^{[4]}j_{33}^{[4]} - j_{11}^{[4]}j_{22}^{[4]}j_{33}^{[4]} > j_{12}^{[4]}j_{23}^{[4]}j_{31}^{[4]}$.
The last condition of (4.22) will be satisfied provided that

$$\begin{aligned} &j_{11}j_{12}j_{21} - j_{11}^2j_{22} + j_{12}j_{21}j_{22} - j_{11}j_{22}^2 + j_{11}j_{13}j_{31} - j_{11}^2j_{33} - 4j_{11}j_{22}j_{33} - j_{22}^2j_{33} + j_{13}j_{31}j_{33} \\ &- j_{11}j_{33}^2 - j_{22}j_{33}^2 \\ &> 2j_{13}j_{22}j_{31} - j_{21}j_{23}j_{31} + 2j_{12}j_{21}j_{33}. \end{aligned}$$

Thus, the feasible equilibrium point E_4 is locally asymptotically stable if the conditions (4.22) are satisfied.

To show the local asymptotic stability of the equilibrium point $E_4(u_4, v_4, w_4)$, the method of first approximation [89] is used.

Proposition 4.5 *The system (4.3) around the interior equilibrium E_4 is asymptotically stable if*

(a) $1+\frac{u_4v_4\beta_1}{(1+u_4\beta_1)^2}-\frac{v_4}{1+u_4\beta_1}+\frac{u_4w_4\beta_2}{(1+u_4\beta_2)^2}+\frac{\mu_1u_4}{1+\beta_1u_4}<2u_4+2w_4\nu+\delta_1,$

(b) $1+\frac{u_4v_4\beta_1}{(1+u_4\beta_1)^2}+\frac{u_4w_4\beta_2}{(1+u_4\beta_2)^2}+\frac{\mu_2u_4}{1+u_4\beta_2}<2u_4+\frac{2v_4}{1+u_4\beta_1}+\frac{w_4}{1+u_4\beta_2}+\delta_2,$

(c) $\frac{u_4\mu_1}{1+u_4\beta_1}+\frac{u_4}{v_4}(-\frac{u_4v_4\beta_1\mu_1}{(1+u_4\beta_1)^2}+\frac{v_4\mu_1}{1+u_4\beta_1})+\frac{u_4\mu_2}{1+u_4\beta_2}+\frac{u_4}{w_4}(\frac{u_4w_4\beta_2\mu_2}{(1+u_4\beta_2)^2}+\frac{w_4\mu_2}{1+u_4\beta_2})$
$<w_4\phi+\delta_1+\delta_2,$

(d) $j_{11}^{[4]}<0$, $j_{22}^{[4]}<0$ and $j_{33}^{[4]}<0$.

Proof. The Method of First Approximation [89] is used. In this method it is shown that (a) the second compound matrix $J^{[2]}(E_4)$ of J_4 is stable and (b) $\det J_4<0$.
The second compound matrix $J^{[2]}(E_4)=(c_{ij})_{3\times3}$ of J_4 is given by

$$J^{[2]}(E_4)=\begin{bmatrix} c_{11} & c_{12} & c_{13}\\ c_{21} & c_{22} & c_{23}\\ c_{31} & c_{32} & c_{33}\end{bmatrix}=\begin{bmatrix} j_{11}^{[4]}+j_{22}^{[4]} & j_{23}^{[4]} & -j_{13}^{[4]}\\ j_{32}^{[4]} & j_{11}^{[4]}+j_{33}^{[4]} & j_{12}^{[4]}\\ -j_{31}^{[4]} & j_{21}^{[4]} & j_{22}^{[4]}+j_{33}^{[4]}\end{bmatrix}.$$

Now for $E_4(u_4,v_4,w_4)$ and the diagonal matrix $D=\mathrm{diag}(u_4,v_4,w_4)$, the matrix $J^{[2]}(E_4)$ is similar to $DJ^{[2]}(E_4)D^{-1}=(a_{ij})_{3\times3}$, where $a_{11}=c_{11}$, $a_{12}=c_{12}\frac{w_4}{v_4}$, $a_{13}=c_{13}\frac{w_4}{u_4}$, $a_{21}=c_{21}\frac{v_4}{w_4}$, $a_{22}=c_{22}$, $a_{23}=c_{23}\frac{v_4}{u_4}$, $a_{31}=c_{31}\frac{u_4}{w_4}$, $a_{32}=c_{32}\frac{u_4}{v_4}$, $a_{33}=c_{33}$. The matrix $J^{[2]}(E_4)$ is stable if and only if $DJ^{[2]}(E_4)D^{-1}$ is stable. Since the diagonal elements of the matrix $DJ^{[2]}(E_4)D^{-1}$ are negative, the matrix will be stable if it is diagonally dominant in rows. Set $\mu^*=\max\{g_1,g_2,g_3\}$, where

$$\begin{aligned}
g_1 &= a_{11}+a_{12}+a_{13}=1-2u_4-2w_4\nu+\frac{u_4v_4\beta_1}{(1+u_4\beta_1)^2}-\frac{v_4}{1+u_4\beta_1}+\frac{u_4w_4\beta_2}{(1+u_4\beta_2)^2}\\
&\quad+\frac{\mu_1u_4}{1+\beta_1u_4}-\delta_1,\\
g_2 &= a_{21}+a_{22}+a_{23}=1-2u_4+\frac{u_4v_4\beta_1}{(1+u_4\beta_1)^2}-\frac{2v_4}{1+u_4\beta_1}+\frac{u_4w_4\beta_2}{(1+u_4\beta_2)^2}-\frac{w_4}{1+u_4\beta_2}\\
&\quad+\frac{\mu_2u_4}{1+u_4\beta_2}-\delta_2,\\
g_3 &= a_{31}+a_{32}+a_{33}=-w_4\phi-\delta_1-\delta_2+\frac{u_4\mu_1}{1+u_4\beta_1}+\frac{u_4}{v_4}(-\frac{u_4v_4\beta_1\mu_1}{(1+u_4\beta_1)^2}+\frac{v_4\mu_1}{1+u_4\beta_1})\\
&\quad+\frac{u_4\mu_2}{1+u_4\beta_2}+\frac{u_4}{w_4}(\frac{u_4w_4\beta_2\mu_2}{(1+u_4\beta_2)^2}+\frac{w_4\mu_2}{1+u_4\beta_2}).
\end{aligned}$$

Now when the above conditions hold true then $\mu^* < 0$, which implies the diagonal dominance and thus verifies the first condition. Again, since $j_{12}^{[4]} < 0$, $j_{13}^{[4]} < 0$, $j_{21}^{[4]} > 0$, $j_{23}^{[4]} < 0$, $j_{31}^{[4]} > 0$, the determinant of J_4 is given by

$$\begin{aligned}
|J_4| &= j_{11}^{[4]} j_{22}^{[4]} j_{33}^{[4]} + j_{12}^{[4]} j_{23}^{[4]} j_{31}^{[4]} - j_{12}^{[4]} j_{21}^{[4]} j_{33}^{[4]} - j_{13}^{[4]} j_{22}^{[4]} j_{31}^{[4]} \\
&= \big(- \frac{u_4 w_4 \mu_2 \beta_2}{(1+u_4\beta_2)^2} + \frac{w_4\mu_2}{1+u_4\beta_2}\big)\big(\frac{u_4 v_4 \nu}{1+u_4\beta_1} - \frac{u_4 w_4 \nu}{1+u_4\beta_2} + \frac{u_4^2\mu_1}{(1+u_4\beta_1)(1+u_4\beta_2)} \\
&\quad - \frac{u_4\delta_1}{1+u_4\beta_2}\big) + (\frac{\mu_2 u_4\beta_2}{1+u_4} - \delta_2)\Big(\big(1 - 2u_4 + \frac{u_4 v_4 \beta_1}{(1+u_4\beta_1)^2} - \frac{v_4}{1+u_4\beta_1} \\
&\quad + \frac{u_4 w_4 \beta_2}{(1+u_4\beta_2)^2} - \frac{w_4}{1+u_4\beta_2}\big)\big(\frac{\mu_1 u_4}{1+\beta_1 u_4} - \nu w_4 - \delta_1\big) \\
&\quad + \frac{u_4\big(-\frac{u_4 v_4 \beta_1 \mu_1}{(1+u_4\beta_1)^2} + \frac{v_4\mu_1}{1+u_4\beta_1}\big)}{1+u_4\beta_1}\Big) < 0,
\end{aligned}$$

if $j_{11}^{[4]} < 0$, $j_{22}^{[4]} < 0$ and $j_{33}^{[4]} < 0$. □

Next, the global stability of E_4 basing on Theorem A.3 and Lemma A.1 that are essential in finding a candidate Lyapunov function.

Theorem 4.1 *The interior equilibrium is globally asymptotically stable if* $\beta_1 > 1$ *and* $M = \frac{v^*}{(1+\beta_1 u)(1+\beta_1 u^*)} + \frac{w^*}{(1+\beta_2 u)(1+\beta_2 u^*)} < 1$.

Proof. A candidate Lyapunov function that has been used by Dubey and Upadhyay [99], Hsu [133], and others is chosen. The following function is considered,

$$\begin{aligned}
V(u,v,w) &= \alpha_1(u - u^* - u^*\ln(\frac{u}{u^*})) + \alpha_2(v - v^* - v^*\ln(\frac{v}{v^*})) \\
&\quad + \alpha_3(w - w^* - w^*\ln(\frac{w}{w^*})).
\end{aligned} \tag{4.23}$$

Defining

$$V_1(u) = u - u^* - u^*\ln(\frac{u}{u^*}), \quad V_2(v) = v - v^* - v^*\ln(\frac{v}{v^*}) \quad \text{and} \quad V_3(z) = w - w^* - w^*\ln(\frac{w}{w^*}),$$

then V can be rewritten as

$$V(u,v,w) = \alpha_1 V_1(u) + \alpha_2 V_2(v) + \alpha_3 V_3(w).$$

Differentiating V with respect to time t along the solutions of model (4.3),

$$\frac{dV}{dt} = \alpha_1\Big(1 - \frac{u^*}{u}\Big)\frac{du}{dt} + \alpha_2\Big(1 - \frac{v^*}{v}\Big)\frac{dv}{dt} + \alpha_3\Big(1 - \frac{w^*}{w}\Big)\frac{dw}{dt}. \tag{4.24}$$

The linear approximations $u - u^* \cong 1 + \beta_1 u \cong 1 + \beta_2 u$, $v - v^* \cong v$ and $w - w^* \cong w$ are used to compute $\frac{dV_1(u(t))}{dt}$, $\frac{dV_2(v(t))}{dt}$ and $\frac{dV_3(w(t))}{dt}$ as follows:

$$\begin{aligned}
\frac{dV_1}{dt} &= \Big(1 - \frac{u^*}{u}\Big)\Big[1 - u - \frac{v}{1+\beta_1 u} - \frac{w}{1+\beta_2 u}\Big]u, \\
&= (u-u^*)[1 - u + u^* - u^* - \frac{v}{1+\beta_1 u} + \frac{v^*}{1+\beta_1 u^*} - \frac{v^*}{1+\beta_1 u^*} \\
&\quad - \frac{w}{1+\beta_2 u} + \frac{w^*}{1+\beta_2 u^*} - \frac{w^*}{1+\beta_2 u^*}\Big], \\
&= (u-u^*)[-u + u^* - \frac{v}{1+\beta_1 u} + \frac{v^*}{1+\beta_1 u^*} - \frac{w}{1+\beta_2 u} + \frac{w^*}{1+\beta_2 u^*}\Big], \\
&= (u-u^*)[-(u-u^*) - \frac{v(1+\beta_1 u^*) - v^*(1+\beta_1 u)}{(1+\beta_1 u)(1+\beta_1 u^*)} \\
&\quad - \frac{w(1+\beta_2 u^*) - w^*(1+\beta_2 u)}{(1+\beta_2 u)(1+\beta_2 u^*)}\Big], \\
&= (u-u^*)[-(u-u^*) - \frac{(v-v^*)(1+\beta_1 u^*) - v^*(u-u^*)}{(1+\beta_1 u)(1+\beta_1 u^*)} \\
&\quad - \frac{(w-w^*)(1+\beta_2 u^*) - w^*(u-u^*)}{(1+\beta_2 u)(1+\beta_2 u^*)}\Big], \\
&= -(u-u^*)^2 - \frac{(v-v^*)(u-u^*)(1+\beta_1 u^*) - v^*(u-u^*)^2}{(1+\beta_1 u)(1+\beta_1 u^*)} \\
&\quad - \frac{(w-w^*)(u-u^*)(1+\beta_2 u^*) - w^*(u-u^*)^2}{(1+\beta_2 u)(1+\beta_2 u^*)},
\end{aligned}$$

$$\begin{aligned}
\frac{dV_2}{dt} &= \Big(1 - \frac{v^*}{v}\Big)\Big[\frac{\mu_1 u}{1+\beta_1 u} - \nu w - \delta_1\Big]v, \\
&= (v-v^*)[\frac{\mu_1 u}{1+\beta_1 u} - \frac{\mu_1 u^*}{1+\beta_1 u^*} + \frac{\mu_1 u^*}{1+\beta_1 u^*} - \nu w + \nu w^* - \nu w^* - \delta_1\Big], \\
&= (v-v^*)[\frac{\mu_1 u}{1+\beta_1 u} - \frac{\mu_1 u^*}{1+\beta_1 u^*} - \nu w + \nu w^*\Big], \\
&= (v-v^*)[\frac{\mu_1 u(1+\beta_1 u^*) - \mu_1 u^*(1+\beta_1 u)}{(1+\beta_1 u)(1+\beta_1 u^*)} - \nu(w-w^*)\Big], \\
&= (v-v^*)[\frac{\mu_1(u-u^*)(1+\beta_1 u^*) - \mu_1 u^*(u-u^*)}{(1+\beta_1 u)(1+\beta_1 u^*)}\Big] - \nu(v-v^*)(w-w^*), \\
&= \frac{\mu_1[(1+\beta_1 u^*) - u^*](u-u^*)(v-v^*)}{(1+\beta_1 u)(1+\beta_1 u^*)} - \nu(v-v^*)(w-w^*), \\
&= -\frac{\mu_1[u^* - (1+\beta_1 u^*)](u-u^*)(v-v^*)}{(1+\beta_1 u)(1+\beta_1 u^*)} - \nu(v-v^*)(w-w^*),
\end{aligned}$$

and

$$\begin{aligned}
\frac{dV_3}{dt} &= \Big(1-\frac{w^*}{w}\Big)\Big[\frac{\mu_2 u}{1+\beta_2 u}-\delta_2\Big]w,\\
&= (w-w^*)[\frac{\mu_2 u}{1+\beta_2 u}-\frac{\mu_2 u^*}{1+\beta_2 u^*}+\frac{\mu_2 u^*}{1+\beta_2 u^*}-\delta_2\Big],\\
&= (w-w^*)[\frac{\mu_2 u}{1+\beta_2 u}-\frac{\mu_2 u^*}{1+\beta_2 u^*}\Big],\\
&= (w-w^*)[\frac{\mu_2 u(1+\beta_2 u^*)-\mu_2 u^*(1+\beta_2 u)}{(1+\beta_2 u)(1+\beta_2 u^*)}\Big],\\
&= (w-w^*)[\frac{\mu_2 (u-u^*)(1+\beta_2 u^*)-\mu_2 u^*(u-u^*)}{(1+\beta_2 u)(1+\beta_2 u^*)}\Big],\\
&= -\frac{\mu_2[u^*-(1+\beta_2 u^*)](u-u^*)(w-w^*)}{(1+\beta_1 u)(1+\beta_1 u^*)}.
\end{aligned}$$

Now,

$$\begin{aligned}
\frac{dV}{dt} &= \alpha_1\Big[-(u-u^*)^2-\frac{(v-v^*)(u-u^*)(1+\beta_1 u^*)-v^*(u-u^*)^2}{(1+\beta_1 u)(1+\beta_1 u^*)}\\
&\quad -\frac{(w-w^*)(u-u^*)(1+\beta_2 u^*)-w^*(u-u^*)^2}{(1+\beta_2 u)(1+\beta_2 u^*)}\Big]\\
&\quad +\alpha_2\Big[-\frac{\mu_1[u^*-(1+\beta_1 u^*)](u-u^*)(v-v^*)}{(1+\beta_1 u)(1+\beta_1 u^*)}-\nu(v-v^*)(w-w^*)\Big]\\
&\quad +\alpha_3\Big[-\frac{\mu_2[u^*-(1+\beta_2 u^*)](u-u^*)(w-w^*)}{(1+\beta_1 u)(1+\beta_1 u^*)}\Big],
\end{aligned}$$

$$\begin{aligned}
&= \alpha_1\Big[-1+\frac{v^*}{(1+\beta_1 u)(1+\beta_1 u^*)}+\frac{w^*}{(1+\beta_2 u)(1+\beta_2 u^*)}\Big](u-u^*)^2\\
&\quad +\Big[-\alpha_1\frac{(1+\beta_1 u^*)}{(1+\beta_1 u)(1+\beta_1 u^*)}-\alpha_2\mu_1\frac{[u^*-(1+\beta_1 u^*)]}{(1+\beta_1 u)(1+\beta_1 u^*)}\Big](u-u^*)(v-v^*)\\
&\quad +\Big[-\alpha_1\frac{(1+\beta_2 u^*)}{(1+\beta_2 u)(1+\beta_2 u^*)}-\alpha_3\mu_2\frac{[u^*-(1+\beta_2 u^*)]}{(1+\beta_2 u)(1+\beta_2 u^*)}\Big](u-u^*)(w-w^*)\\
&\quad -\alpha_2\nu(v-v^*)(w-w^*).
\end{aligned}$$

Let $\alpha_1 = 1$, then

$$-\alpha_2\mu_1\frac{[u^*-(1+\beta_1 u^*)]}{(1+\beta_1 u)(1+\beta_1 u^*)}=\frac{\alpha_1}{1+\beta_1 u},$$

that is,

$$\alpha_2=\frac{1+\beta_1 u^*}{\mu_1[(1+\beta_1 u^*)-u^*]}.$$

Also

$$\alpha_3\mu_2\frac{[(1+\beta_2 u^*)]-u^*}{(1+\beta_2 u)(1+\beta_2 u^*)}=\frac{\alpha_1}{1+\beta_2 u},$$

that is,

$$\alpha_3 = \frac{1+\beta_2 u^*}{\mu_2[(1+\beta_2 u^*) - u^*]}.$$

Hence,

$$\begin{aligned}\frac{dV}{dt} &= \Big[-1 + \frac{v^*}{(1+\beta_1 u)(1+\beta_1 u^*)} + \frac{w^*}{(1+\beta_2 u)(1+\beta_2 u^*)}\Big](u-u^*)^2 \\ &\quad - \frac{1+\beta_1 u^*}{\mu_1[1+u^*(\beta_1-1)]}\nu(v-v^*)(w-w^*).\end{aligned}$$

The coefficient of $(v-v^*)(w-w^*)$ is strictly negative if $\beta_1 > 1$ and the coefficient of $(u-u^*)^2$ is strictly negative if

$$-1 + \frac{v^*}{(1+\beta_1 u)(1+\beta_1 u^*)} + \frac{w^*}{(1+\beta_2 u)(1+\beta_2 u^*)} \leq -1 + M < 0,$$

where

$$M = \frac{v^*}{(1+\beta_1 u)(1+\beta_1 u^*)} + \frac{w^*}{(1+\beta_2 u)(1+\beta_2 u^*)} < 1.$$

If $\alpha_2 > 0$ and $\alpha_2 > 0$, that is, $\beta_1 > 1$ and $\beta_2 > 1$, then the function V is negative definite. Hence, the interior equilibrium is globally asymptotically stable. □

4.3 Stability of the constant positive steady state for the reaction-diffusion system

In this section, the local stability of the constant positive steady state $\mathbf{u}$ of the spatio-temporal system (4.2) is investigated. Let $0 = \mu_0 < \mu_1 < \mu_2 < \mu_3 < \dots$ be the eigenvalues of the operator $-\Delta$ on Ω with the homogeneous Neumann boundary condition, and $\dim E(\mu_i)$ be the eigenspace corresponding to μ_i in $C^1(\overline{\Omega})$. Let $\mathbf{X} = \{\mathbf{u} \in [C^1(\overline{\Omega})]^3 | \frac{\partial \mathbf{u}}{\partial \mathbf{n}} = 0 \text{ on } \partial\Omega\}$, $\{\phi_{i,j}; j = 1, \dots, \dim E(\mu_i)\}$ an orthonormal basis of $E(\mu_i)$, and $\mathbf{X}_{i,j} = \{c\phi_{i,j} | c \in \mathbb{R}^3$. Then,

$$\mathbf{X} = \bigoplus_{i=1}^{\infty} \mathbf{X}_i \quad \text{and} \quad \mathbf{X}_i = \bigoplus_{j=1}^{\dim E(\mu_i)} \mathbf{X}_{ij} \tag{4.25}$$

Theorem 4.2 *Assume that the parameters in (4.3) satisfy (4.8) and (4.22). Then the constant positive steady state* $\mathbf{u}^*$ *of the spatio-temporal system (4.2) is uniformly asymptotically stable.*

Proof. Let $\mathbf{D} = \text{diag}(1, d_2, d_3)$, $\mathbf{L} = \mathbf{D}\Delta + \mathbf{G_u}(\mathbf{u}^*)$. The linearisation of the system (4.2) about the constant positive steady state $\mathbf{u}^*$ is given by

$$\mathbf{u}_t = \mathbf{L}\mathbf{u}.$$

The eigenspace $\mathbf{X}_i, i > 0$, is invariant under the operator $\mathbf{L}$. λ_i is an eigenvalue of $\mathbf{L}$ on $\mathbf{X}_i$ if and only if it is an eigenvalue of the matrix $-\mu_i\mathbf{D} + \mathbf{G_u}(\mathbf{u}^*)$. The characteristic polynomial of $-\mu_i\mathbf{D} + \mathbf{G_u}(\mathbf{u}^*)$ is

$$\psi_i(\lambda) = \lambda^3 + A_{1i}\lambda^2 + A_{2i}\lambda + A_{3i},$$

with

$$\begin{aligned}
A_{1i} &= \mu_i(1 + d_2 + d_3) + A_1, \\
A_{2i} &= \mu_i^2(d_2 + d_3 + d_2d_3) - \mu_i((j_{22}^{[4]} + j_{33}^{[4]}) + d_2(j_{11}^{[4]} + j_{33}^{[4]}) + d_3(j_{11}^{[4]} + j_{22}^{[4]})) + A_2, \\
A_{3i} &= \mu_i^3(d_2d_3) + \mu_i^2(d_2j_{33}^{[4]} - d_3j_{22}^{[4]} - d_2d_3j_{11}^{[4]}) + \mu_i((j_{22}^{[4]}j_{33}^{[4]}) \\
&\quad + d_2(j_{11}^{[4]}j_{33}^{[4]} - j_{13}^{[4]}j_{31}^{[4]}) + d_3(j_{11}^{[4]}j_{22}^{[4]} - j_{12}^{[4]}j_{21}^{[4]})) + A_3,
\end{aligned}$$

where the $j_{ij}^{[4]}, A_1$ are as given in (4.20) and (4.21). From the condition (4.22), it follows that

$$A_{1i} > 0, A_{2i} > 0, A_{3i} > 0.$$

An algebraic calculation of $E_i = A_{1i}A_{2i} - A_{3i} > 0$ yields

$$E_i = B_1\mu_i^3 + B_2\mu_i^2 + B_3\mu + A_1A_2 - A_3,$$

where

$$\begin{aligned}
B_1 &= d_2 + d_2^2 + d_3 + 2d_2d_3 + d_2^2d_3 + d_3^2 + d_2d_3^2 > 0, \\
B_2 &= -(j_{22}^{[4]} + j_{33}^{[4]}) - (j_{11}^{[4]} + j_{33}^{[4]})d_2^2 + (d_2 + d_3 + d_2d_3)A_1 - (j_{11}^{[4]} + j_{22}^{[4]})d_3^2 \\
&\quad + (d_2 + d_3 + d_2d_3)A_2 > 0, \\
B_3 &= -j_{22}^{[4]}j_{33}^{[4]} + (j_{13}^{[4]}j_{31}^{[4]} - j_{11}^{[4]}j_{33}^{[4]})d_2 + (j_{12}^{[4]}j_{21}^{[4]} - j_{11}^{[4]}j_{22}^{[4]})d_3 + (1 + d_2 + d_3)A_1 \\
&\quad - ((j_{22}^{[4]} + j_{33}^{[4]}) + (j_{11}^{[4]} + j_{33}^{[4]})d_2 + (j_{11}^{[4]} + j_{22}^{[4]})d_3)A_2 > 0
\end{aligned}$$

Note that $E_i > 0$ as $A_1A_2 - A_3 > 0$ under the given conditions. From the Routh-Hurwitz criterion it follows that, for each $i \geq 0$, all the three roots $\lambda_{i,1}, \lambda_{i,2}, \lambda_{i,3}$ of $\psi_i(\lambda) = 0$ have negative real parts. There exists a positive constant σ such that

$$\mathrm{Re}\{\lambda_{i,1}\}, \mathrm{Re}\{\lambda_{i,2}\}, \mathrm{Re}\{\lambda_{i,3}\} \leq -\sigma, \ \ \forall i \geq 1. \tag{4.26}$$

Thus, the spectrum of $\mathbf{L}$, which consists of eigenvalues, lies in $\{\mathrm{Re}\lambda \leq -\sigma\}$. The local stability of $\mathbf{u}^*$ is concluded by applying Theorem 5.1.1 of Henry [146].

The proof of (4.26) is given below. Let $\lambda = \mu_i \xi$, then

$$\psi_i(\lambda) = \mu_i^3 \xi^3 + A_{1i}\mu_i^2 \xi^2 + A_{2i}\mu_i \xi + A_{3i} = \tilde{\psi}_1(\xi),$$

Since $\mu_i \to \infty$ as $i \to \infty$, it follows that

$$\frac{\tilde{\psi}_1(\xi)}{\mu_i^3} = \xi^3 + \frac{A_{1i}}{\mu_i}\xi^2 + \frac{A_{2i}}{\mu_i^2}\xi + \frac{A_{3i}}{\mu_i^3},$$

and

$$\lim_{i\to\infty}\{\frac{\tilde{\psi}_1(\xi)}{\mu_i^3}\} = \xi^3 + (1 + d_2 + d_3)\xi^2 + (d_1d_2 + d_1d_3 + d_2d_3)\xi + d_1d_2d_3 = \bar{\psi}_1(\xi).$$

Upon applying the Routh-Hurwitz criterion, it follows that the three roots ξ_1, ξ_2, ξ_3 of $\bar{\psi}_1(\xi) = 0$ all have negative real parts ($S_1 = 1 + d_2 + d_3 > 0$, $S_2 = d_2 + d_3 + d_2d_3 > 0$, $S_3 = d_2d_3 > 0$) and

$$\begin{aligned} S_1S_2 - S_3 &= (1 + d_2 + d_3)(d_2 + d_3 + d_2d_3) - d_2d_3 \\ &= 2d_2d_3 + (d_2 + d_3) + d_2^2(1 + d_3) + d_3^2(1 + d_2) > 0 \end{aligned}$$

Thus, there exists a positive constant $\bar{\sigma}$ such that $\mathrm{Re}\{\xi_1\}, \mathrm{Re}\{\xi_2\}, \mathrm{Re}\{\xi_3\} \leq -\bar{\sigma}$. Continuity implies that there exists i_0 such that the three roots $\xi_{i,1}, \xi_{i,2}, \xi_{i,3}$ of $\bar{\psi}_1(\xi) = 0$ satisfy

$$\mathrm{Re}\{\xi_{i,1}\}, \mathrm{Re}\{\xi_{i,2}\}, \mathrm{Re}\{\xi_{i,3}\} \leq -\bar{\sigma}, \ \ \forall i \geq i_0.$$

In turn, $\mathrm{Re}\{\lambda_{i,1}\}, \mathrm{Re}\{\lambda_{i,2}\}, \mathrm{Re}\{\lambda_{i,3}\} \leq -\mu_i\bar{\sigma} = -\kappa_i$ for all $i \geq i_0$.

The inequality (4.26) holds for

$$\sigma = \min\{\kappa_i\}.$$

This completes the proof. □

As a consequence of Theorem 4.2, problem (4.2) has no non-constant positive steady state in some neighbourhood of $\mathbf{u}^*$ if (4.8) and (4.22) hold.

4.4 *A priori* estimates of positive steady state

The corresponding steady-state problem of (4.2) is

$$\begin{cases} -\Delta u = u(1-u) - \dfrac{uv}{(1+\beta_1 u)} - \dfrac{uw}{1+\beta_2 u} = G_1(\mathbf{u}), \\ -d_2\Delta v = \dfrac{\mu_1 uv}{1+\beta_1 u} - \nu vw - \delta_1 v = G_2(\mathbf{u}), \\ -d_3\Delta w = \dfrac{\mu_2 uw}{1+\beta_2 u} - \delta_2 w = G_3(\mathbf{u}), \\ \dfrac{\partial u}{\partial \mathbf{n}}|_{\partial\Omega} = \dfrac{\partial v}{\partial \mathbf{n}}|_{\partial\Omega} = \dfrac{\partial w}{\partial \mathbf{n}}|_{\partial\Omega} = 0, t > 0, \\ u(x,y,0) = u_0(x,y), v(x,y,0) = v_0(x,y), w(x,y,0) = w_0(x,y), (x,y) \in \Omega. \end{cases} \tag{4.27}$$

For convenience, let Λ represent the collective constants $(\beta_1, \beta_2, \mu_1, \mu_2, \delta_2, \delta_2, \nu)$.
In this section *a priori* positive upper and lower bounds for the positive solutions of (4.27) are deduced on the basis of two important results, Proposition A.1 (Maximum Principle [147]), and Proposition A.2 (Harnack Inequality [148]). The result for upper bounds is contained in the following theorem.

Theorem 4.3 *(Upper bounds) For any positive solution (u, v, w) of (4.27),*

$$\max_{\overline{\Omega}} u \le 1, \qquad \max_{\overline{\Omega}} v \le \frac{\mu_1(4d_1(\nu C_1 + \delta_1) + d_2)}{4(\nu C_1 + \delta_1)}, \qquad \max_{\overline{\Omega}} w \le \frac{\mu_1 - \beta_1\delta_1}{\nu(\beta_1 + 1)}. \tag{4.28}$$

Proof. Since

$$u(1-u) - \frac{uv}{(1+\beta_1 u)} - \frac{uw}{1+\beta_2 u} \le u(1-u),$$

application of the Maximum Principle Proposition A.1(i) gives $\max_{\overline{\Omega}} u \le 1$. From (4.8)

$$w = \frac{u(\mu_1 - \beta_1\delta_1) - \delta_1}{\nu(\beta_1 u + 1)} \le \frac{u(\mu_1 - \beta_1\delta_1)}{\nu(\beta_1 u + 1)} \le \frac{u(\mu_1 - \beta_1\delta_1)}{\nu(\beta_1 u + u)} = \frac{\mu_1 - \beta_1\delta_1}{\nu(\beta_1 + 1)}.$$

Hence, $\max_{\overline{\Omega}} w \leq \dfrac{\mu_1 - \beta_1\delta_1}{\nu(\beta_1+1)}$. Let $\phi = \mu_1 u + d_2 v$, then

$$\begin{aligned} -\Delta\phi &= \mu_1\Delta u + d_2\Delta v = \mu_1 u(1-u) - \frac{\mu_1 uw}{1+\beta_2 u} - \nu vw - \delta_1 v, \qquad x \in \Omega \\ \partial_v \phi &= 0, \qquad x \in \Omega. \end{aligned}$$

Let $\phi(u_0) = \max_{\overline{\Omega}} \phi(u)$, then an application of the Maximum Principle Proposition A.1 (i) yields

$$(\nu w + \delta_1)v(u_0) \leq \mu_1 u(1-u) - \frac{\mu_1 uw}{1+\beta_2 u} \leq \mu_1 u(1-u) \leq \frac{\mu_1}{4},$$

that is,

$$v(u_0) \leq \frac{\mu_1}{4(\nu w + \delta_1)} \leq \frac{\mu_1}{4(\nu \underline{C} + \delta_1)},$$

where $\underline{C} < \min w$. It follows that

$$d_2 \max_{\overline{\Omega}} v(u) \leq \max_{\overline{\Omega}} \phi(u) = \phi(u_0) = \mu_1 u(u_0) + d_2 v(u_0) \leq \mu_1 + \frac{d_2\mu_1}{4(\nu\underline{C} + \delta_1)}.$$

Hence,

$$\max_{\overline{\Omega}} v(u) \leq \frac{\mu_1(4(\nu\underline{C} + \delta_1) + d_2)}{4(\nu\underline{C} + \delta_1)}.$$

□

For the lower bounds, the following theorem is considered.

Theorem 4.4 *(Lower bounds) Let Λ and d be fixed positive constants such that $d \leq \min\{1, \underline{d}_2, \underline{d}_3\}$. Then there exist positive constants $C_i^*, i = 1, 2, 3$, which are dependent on Λ and d, such that every positive solution (u, v, w) of (4.27) satisfies*

$$\min_{\overline{\Omega}} u(x) \geq C_1^*, \qquad \min_{\overline{\Omega}} v(x) \geq C_2^*, \qquad \min_{\overline{\Omega}} w(x) \geq C_3^*. \tag{4.29}$$

Proof. Let $u(x_0) = \min_{\overline{\Omega}} u(x)$, $v(y_0) = \min_{\overline{\Omega}} v(x)$, $w(z_0) = \min_{\overline{\Omega}} w(x)$ and $w(y_1) = \max_{\overline{\Omega}} w(x)$, then applying the maximum principle yields

$$\begin{cases} (1-u(x_0)) - \dfrac{v(x_0)}{(1+\beta_1 u(x_0))} - \dfrac{w(x_0)}{1+\beta_2 u(x_0)} \leq 0, \\ \dfrac{\mu_1 u(y_0)}{1+\beta_1 u(y_0)} - \nu w(y_0) - \delta_1 \leq 0, \\ \dfrac{\mu_2 u(z_0)}{1+\beta_2 u(z_0)} - \delta_2 \leq 0, \\ \dfrac{\mu_1 u(y_1)}{1+\beta_1 u(y_1)} - \nu w(y_1) - \delta_1 \geq 0. \end{cases} \tag{4.30}$$

The second inequality gives

$$w(y_0) \geq \frac{1}{\nu}\left(\frac{\mu_1 u(y_0)}{1+\beta_1 u(y_0)} - \delta_1\right). \tag{4.31}$$

The fourth inequality in (4.30) gives

$$w(y_1) \leq \frac{1}{\nu}\left(\frac{\mu_1 u(y_1)}{1+\beta_1 u(y_1)} - \delta_1\right) \leq \frac{1}{\nu}(\mu_1 u(y_1) - \delta_1)) \leq \frac{1}{\nu}(\mu_1 \max_{\overline{\Omega}} u - \delta_1) \leq \frac{1}{\nu}(\mu_1 - \delta_1). \tag{4.32}$$

The first inequality in (4.30) imply

$$1 - u(x_0) \leq \frac{v(x_0)}{1+\beta_1 u(x_0)} + \frac{w(x_0)}{1+\beta_2 u(x_0)},$$

that is,

$$1 - u(x_0) \leq v(x_0) + w(x_0). \tag{4.33}$$

Define,

$$\begin{aligned} c_1(x) &= \Big(1 - u - \frac{v}{(1+\beta_1 u)} - \frac{w}{1+\beta_2 u}\Big), \\ c_2(x) &= d_2^{-1}\Big(\frac{\mu_1 u}{1+\beta_1 u} - \nu w - \delta_1\Big), \\ \text{and} \quad c_3(x) &= d_3^{-1}\Big(\frac{\mu_2 u}{1+u} - \delta_2\Big). \end{aligned}$$

Now, (4.28) implies that there exists a positive constant $\overline{C}(d,\Lambda)$ such that $\|c_1\|_\infty, \|c_2\|_\infty, \|c_3\|_\infty \leq \overline{C}$, if $1, d_2, d_3 \geq d$. Thus, as u, v, w satisfy

$$\begin{aligned} \Delta u + c_1(x)u &= 0, \\ \Delta v + c_2(x)v &= 0, \\ \Delta w + c_3(x)w &= 0, \quad u \in \partial\Omega; \\ \frac{\partial u}{\partial \mathbf{n}} &= 0, \quad \frac{\partial v}{\partial \mathbf{n}} = 0, \quad \frac{\partial w}{\partial \mathbf{n}} = 0, \quad u \in \partial\Omega. \end{aligned}$$

The Harnack Inequality in Proposition A.2 shows that there exists a positive constant $C_* = C_*(\Lambda, d)$ such that

$$\max_{\overline{\Omega}} u(x) \leq C_* \min_{\overline{\Omega}} u(x), \ \max_{\overline{\Omega}} v(x) \leq C_* \min_{\overline{\Omega}} v(x), \ \max_{\overline{\Omega}} w(x) \leq C_* \min_{\overline{\Omega}} w(x). \tag{4.34}$$

From (4.33), it follows that

$$\begin{aligned}
1 - u(x_0) &\leq v(x_0) + w(x_0), \\
1 - u(x_0) &\leq v(y_0) + w(y_1), \\
1 - u(x_0) &\leq v(y_0) + \frac{\mu_1}{\nu} \max_{\overline{\Omega}} u(x) - \frac{\delta_1}{\nu}, \\
1 + \frac{\delta_1}{\nu} - \frac{\mu_1}{\nu} \max_{\overline{\Omega}} u(x) - u(x_0) &\leq v(y_0), \\
1 + \frac{\delta_1}{\nu} - \frac{\mu_1}{\nu} \max_{\overline{\Omega}} u(x) - \frac{1}{C_*} \max_{\overline{\Omega}} u(x) &\leq v(y_0).
\end{aligned}$$

From (4.28) $\max_{\overline{\Omega}} u \leq 1$. Hence, it follows that

$$C_2^* = \big(1 + \frac{\delta_1}{\nu} - \frac{\mu_1}{\nu} - \frac{1}{C_*}\big) \leq \min_{\overline{\Omega}} v(x).$$

Furthermore, from (4.33), it follows that

$$\begin{aligned}
1 - u(x_0) &\leq v(x_0) + w(y_1), \\
1 - u(x_0) &\leq v(x_0) + \frac{\mu_1}{\nu} \max_{\overline{\Omega}} u(x) - \frac{\delta_1}{\nu}, \\
1 + \frac{\delta_1}{\nu} - v(x_0) &\leq u(x_0) + \frac{\mu_1}{\nu} \max_{\overline{\Omega}} u(x), \\
1 + \frac{\delta_1}{\nu} - v(x_0) &\leq \min_{\overline{\Omega}} u(x) + \frac{\mu_1}{\nu} C_* \min_{\overline{\Omega}} u(x), \\
1 + \frac{\delta_1}{\nu} - C_2^* &\leq (1 + \frac{\mu_1}{\nu} C_*) \min_{\overline{\Omega}} u(x).
\end{aligned}$$

Hence, it follows that

$$C_1^* = \frac{1 + \frac{\delta_1}{\nu} - C_2^*}{1 + \frac{\mu_1}{\nu} C_*} \leq \min_{\overline{\Omega}} u(x).$$

From (4.31),it follows that

$$\min_{\overline{\Omega}} w(x) = w(z_0) \geq w(y_0) \geq \frac{1}{\nu}(\frac{\mu_1 u(y_0)}{1 + \beta_1 u(y_0)} - \delta_1) \geq \frac{1}{\nu}(\frac{\mu_1 C_1^*}{1 + \beta_1 C_1^*} - \delta_1) = C_3^*.$$

□

4.5 Non-existence of non-constant positive solution

In this section the energy method is used to deduce the results of non-existence of nonconstant positive solution of (4.27). Let $0 = \mu_0 < \mu_1 < \mu_2 < \cdots$ be the eigenvalues of the operator $-\Delta$ with the homogeneous Neumann condition. The following result is used to prove the subsequent theorem. For any $\varphi \in L^1(\Omega)$, let

$$\bar{\varphi} = \frac{1}{|\Omega|}\int_\Omega \varphi dx. \tag{4.35}$$

Theorem 4.5 *Let d_2^* and d_3^* be positive arbitrary constants that satisfy $\alpha_1 d_2^* \geq \dfrac{\mu_1}{1+\beta_1}$, $\alpha_1 d_3^* \geq \dfrac{\mu_2}{1+\beta_2}$. Then there exists a positive constant $D_1 = D_1(\Lambda, d_2^*, d_3^*)$ such that when $1 > D_1$, $d_2 \geq d_2^*$ and $d_3 \geq d_3^*$ problem (4.27) has no non-constant positive solution.*

Proof. Let (u, v, w) be a positive solution of (4.27) and $(\bar{u}, \bar{v}, \bar{w})$ be the average of (u, v, w) over Ω. Multiplying the equations of (4.27) by $(u - \bar{u})$, $(v - \bar{v})$ and $(w - \bar{w})$ respectively, and integrating over Ω gives

$$\begin{aligned}
&\int_\Omega \{|\nabla u|^2 + d_2|\nabla v|^2 + d_3|\nabla w|^2\}dx \\
= &\int_\Omega \{(G_1(u,v,w) - G_1(\bar{u},\bar{v},\bar{w}))(u-\bar{u})\}dx \\
&+ \int_\Omega \{(G_2(u,v,w) - G_2(\bar{u},\bar{v},\bar{w}))(v-\bar{v})\}dx \\
&+ \int_\Omega \{(G_3(u,v,w) - G_3(\bar{u},\bar{v},\bar{w}))(w-\bar{w})\}dx, \\
= &\int_\Omega \{[u(1-u) - \frac{uv}{(1+\beta_1 u)} - \frac{uw}{1+\beta_2 u}] \\
&-[\bar{u}(1-\bar{u}) - \frac{\bar{u}\bar{v}}{(1+\beta_1\bar{u})} - \frac{\bar{u}\bar{w}}{1+\beta_2\bar{u}}]\}(u-\bar{u})dx \\
&+ \int_\Omega \{[\frac{\mu_1 uv}{1+\beta_1 u} - \nu vw - \delta_1 v] - [\frac{\mu_1\bar{u}\bar{v}}{1+\beta_1\bar{u}} - \nu\bar{v}\bar{w} - \delta_1\bar{v}](v-\bar{v})\}dx \\
&+ \int_\Omega \{[\frac{\mu_2 uw}{1+u} - \delta_2 w] - [\frac{\mu_2\bar{u}\bar{w}}{1+\bar{u}} - \delta_2\bar{w}](w-\bar{w})\}dx,
\end{aligned}$$

$$
\begin{aligned}
= & \int_\Omega \{[1-(u+\bar{u})](u-\bar{u})^2 - \frac{v(u-\bar{u})^2 + (\bar{u}+\beta_1 u\bar{u})(v-\bar{v})(u-\bar{u})}{(1+\beta_1 u)(1+\beta_1\bar{u})} \\
& - \frac{w(u-\bar{u})^2 + (\bar{u}+\beta_2 u\bar{u})(w-\bar{w})(u-\bar{u})}{(1+\beta_2 u)(1+\beta_2\bar{u})}\}dx \\
& + \int_\Omega \{\frac{\mu_1 v(u-\bar{u})(v-\bar{v}) + \mu_1(\bar{u}+\beta_1 u\bar{u})(v-\bar{v})^2}{(1+\beta_1 u)(1+\beta_1\bar{u})} \\
& - \nu w(v-\bar{v})^2 - \nu\bar{v}(v-\bar{v})(w-\bar{w}) - \delta_1(v-\bar{v})^2\}dx \\
& + \int_\Omega \{\frac{\mu_2 w(u-\bar{u})(w-\bar{w}) + \mu_2(\bar{u}+\beta_2 u\bar{u})(w-\bar{w})^2}{(1+\beta_2 u)(1+\beta_2\bar{u})} - \delta_2(w-\bar{w})^2\}dx.
\end{aligned}
$$

To proceed with the proof the ϵ-Young Inequality, Proposition A.3, is used. Then using Proposition (4.3) and the ϵ-Young Inequality

$$
\begin{aligned}
& \int_\Omega \{|\nabla u|^2 + d_2|\nabla v|^2 + d_3|\nabla w|^2\}dx \\
\leq & \int_\Omega \{(1+C)(u-\bar{u})^2 + (\frac{\mu_1(\bar{u}+\beta_1 u\bar{u})}{(1+\beta_1 u)(1+\beta_1\bar{u})} + \varepsilon_1)(v-\bar{v})^2 \\
& + (\frac{\mu_2(\bar{u}+\beta_2 u\bar{u})}{(1+\beta_2 u)(1+\beta_2\bar{u})} + \varepsilon_2)(w-\bar{w})^2\}dx \\
\leq & \int_\Omega \{(1+C)(u-\bar{u})^2 + (\frac{\mu_1}{1+\beta_1} + \varepsilon_1)(v-\bar{v})^2 \\
& + (\frac{\mu_2}{1+\beta_2} + \varepsilon_2)(w-\bar{w})^2\}dx \qquad (4.36)
\end{aligned}
$$

for some positive constant $C = C(\Lambda, d_2^*, d_3^*, \varepsilon_1, \varepsilon_2)$ where $\varepsilon_1, \varepsilon_2$ are the arbitrary small positive constants arising from Young's inequality. Using the Poincare Inequality, Proposition A.4 [144], where $\bar{g}$ is similar to $\bar{\varphi}$ in (4.35), it follows from (4.36) that

$$
\begin{aligned}
& \alpha_1 \int_\Omega \{(u-\bar{u})^2 + d_2(v-\bar{v})^2 + d_3(w-\bar{w})^2\}dx, \\
\leq & \int_\Omega \{(1+C)(u-\bar{u})^2 + (\frac{\mu_1}{1+\beta_1} + \varepsilon_1)(v-\bar{v})^2 \\
& + (\frac{\mu_2}{1+\beta_2} + \varepsilon_2)(w-\bar{w})^2\}dx. \qquad (4.37)
\end{aligned}
$$

If $\varepsilon_1, \varepsilon_2 > 0$ are chosen very small such that

$$
\alpha_1 d_2^* \geq \frac{\mu_1}{1+\beta_1} + \varepsilon_1, \;\; \alpha_1 d_3^* \geq \frac{\mu_2}{1+\beta_2} + \varepsilon_2,
$$

then (4.37) implies that $v = \bar{v} =$ constant, $w = \bar{w} =$ constant, and $u = \bar{u} =$ constant if $1 > D_1 \triangleq \alpha^{-1}(1+C)$. □

4.6 Existence of non-constant positive solution

In this section the main aim is to discuss the existence of non-constant positive solutions to the system (4.27) by using Leray-Schauder degree theory [149]. In the last section, Theorem 4.5 implies that when the assumptions of the theorem hold then (4.27) will not have non-constant positive solution. In addition to this, as a consequence of Theorem 4.2 the system (4.2) will not have a non-constant positive steady state in some neighborhood of $\mathbf{u}^*$ if the existence condition (4.9) holds. However, by properly choosing the parameters and contradicting the condition (4.22), it is possible to obtain a non-constant positive solution for (4.27) and hence a non-constant positive steady state of (4.2). The existence of non-constant positive classical solutions to the system (4.27) is discussed when the diffusion coefficient d_2 vary while the parameters Λ, d_3 are kept fixed. In view of this reason, this discussion is restricted to the case where Λ satisfy (4.9) and $j_{11}^{[4]} > 0, j_{22}^{[4]} > 0, j_{33}^{[4]} > 0$. This ensures that stationary patterns can arise as a result of diffusion.

The linearization of (4.27) is studied at $\mathbf{u}^*$. Let $\mathbf{X}$ be as given in Section 4.3, and define

$$\begin{aligned} \mathbf{X}^+ &= \{\mathbf{u} \in \mathbf{X} | u, v, w > 0 \text{ on } \bar{\Omega}\}, \\ \mathcal{B}(C) &= \{\mathbf{u} \in \mathbf{X} | c^{-1} < u, v, w < c \text{ on } \bar{\Omega}\}, c > 0, \end{aligned}$$

where c is a positive constant that is guaranteed to exist by Theorems 4.3 and 4.4. Then (4.27) can be written as

$$\begin{cases} -\mathbf{D}\Delta\mathbf{u} = \mathbf{G}(\mathbf{u}), & x \in \Omega \ , \\ \partial_{\mathbf{n}}\mathbf{u} = 0, & x \in \partial\Omega \ . \end{cases} \tag{4.38}$$

Applying the fixed point index method [106, 112], it is seen that finding positive solutions of (4.27) is equivalent to finding positive solutions of the equation

$$\mathbf{F}(\mathbf{u}) \triangleq \mathbf{u} - (\mathbf{I} - \Delta)^{-1}\{\mathbf{D}^{-1}\mathbf{G}(\mathbf{u}) + \mathbf{u}\} = 0 \text{ in } \mathbf{X}^+,$$

where $\mathbf{I}$ is the identity map from $C^1(\Omega)$ to itself and $(\mathbf{I} - \Delta)^{-1}$ is the inverse of $\mathbf{I} - \Delta$ in $\mathbf{X}$ subject to Neumann boundary condition. As $\mathbf{F}(\cdot)$ is a compact perturbation of

the identity operator, for any $\mathcal{B} = \mathcal{B}(C)$, the Leray-Schauder degree $\deg(\mathbf{F}(\cdot), 0, B)$ is well-defined if $\mathbf{F}(\mathbf{u}) \neq 0$ on ∂B.

Moreover,

$$\mathbf{D_u F}(\tilde{\mathbf{u}}) = \mathbf{I} - (\mathbf{I} - \Delta)^{-1}\{\mathbf{D}^{-1}\mathbf{G}(\tilde{\mathbf{u}}) + \mathbf{u}\}.$$

Suppose that $\mathbf{D_u F}(\tilde{\mathbf{u}})$ is invertible, then the index of $\mathbf{F}$ at $\tilde{\mathbf{u}}$ is defined as $\text{index}(\mathbf{F}(\cdot), \tilde{\mathbf{u}}) = (-1)^{\gamma}$, where γ is the total number of eigenvalues with negative real parts (counting multiplicities) of $\mathbf{D_u F}(\tilde{\mathbf{u}})$ [150].

The decomposition of (4.25) is referred to in the ensuing discussion of the eigenvalues of $\mathbf{D_u F}(\tilde{\mathbf{u}})$. Firstly, note that, for each integer $i \geq 1$ and each integer $1 \leq j \leq \dim E(\mu_i)$, $\mathbf{X}_{ij}$ is invariant under $\mathbf{D_u F}(\tilde{\mathbf{u}})$, and λ is an eigenvalue $\mathbf{D_u F}(\tilde{\mathbf{u}})$ on $\mathbf{X}_{ij}$ if and only if it is an eigenvalue of the matrix

$$\mathbf{I} - \frac{1}{1+\mu_i}[\mathbf{D}^{-1}\mathbf{G}_u(\mathbf{u}) + \mathbf{I}] = \frac{1}{1+\mu_i}[\mu_i\mathbf{I} - \mathbf{D}^{-1}\mathbf{G}_u(\tilde{\mathbf{u}})].$$

Thus, $\mathbf{D_u F}(\tilde{\mathbf{u}})$ is invertible if and only if, for all $i \geq 1$, the matrix $\mathbf{I} - \frac{1}{1+\mu_i}[\mathbf{D}^{-1}\mathbf{G}_u(\mathbf{u}) + \mathbf{I}]$ is non-singular. Writing

$$H(\mu) = H(\tilde{\mathbf{u}}; \mu) \triangleq \det\{\mu_i\mathbf{I} - \mathbf{D}^{-1}\mathbf{G}_u(\tilde{\mathbf{u}})\} = \frac{1}{d_2 d_3}\det\{\mu\mathbf{D} - \mathbf{G}_u(\tilde{\mathbf{u}})\}. \tag{4.39}$$

Note, furthermore, that if $H(\mu_i) \neq 0$, then for each $1 \leq j \leq \dim E(\mu_i)$, the number of negative eigenvalues of $\mathbf{D_u F}(\tilde{\mathbf{u}})$ on $\mathbf{X}_{ij}$ is odd if and only if $H(\mu_i) < 0$. In conclusion, the following result is stated which can also be found in [151].

Proposition 4.6 *Suppose that, $i \geq 1$, the matrix $\mu_i\mathbf{I} - \mathbf{D}^{-1}\mathbf{G}_u(\tilde{\mathbf{u}})$ is non-singular. Then*

$$index\mathbf{F}((\cdot), \tilde{\mathbf{u}}) = (-1)^{\gamma}, \ \ where\ \gamma = \sum_{i \geq 1, H(\mu_i) < 0} \dim E(\mu_i).$$

To facilitate the computation of $\text{index}(\mathbf{F}(\cdot), \tilde{\mathbf{u}})$, consider carefully the sign $H(\mu_i)$. The direct calculation gives

$$\det\{\mu\mathbf{D} - \mathbf{G}_u(\tilde{\mathbf{u}})\} = A_3(d_2)\mu^3 + A_2(d_2)\mu^2 + A_1(d_2)\mu - \det\{\mathbf{G}_u(\tilde{\mathbf{u}})\} \triangleq \mathcal{A}(d_2; \mu) \tag{4.40}$$

with

$$\begin{cases} A_3(d_2) = d_2 d_3, \\ A_2(d_2) = -(j_{33}^{[4]} d_2 + j_{22}^{[4]} d_3 + j_{11}^{[4]} d_2 d_3), \\ A_1(d_2) = j_{22}^{[4]} j_{33}^{[4]} - j_{13}^{[4]} j_{31}^{[4]} d_2 + j_{11}^{[4]} j_{33}^{[4]} d_2 - j_{12}^{[4]} j_{21}^{[4]} d_3 + j_{11}^{[4]} j_{22}^{[4]} d_3, \end{cases}$$

where $j_{ij}^{[4]}$ are as given in (4.20).

Consider the dependence of $\mathcal{A}$ on d_2. Let $\tilde{\mu}_1(d_2)$, $\tilde{\mu}_2(d_2)$ and $\tilde{\mu}_3(d_2)$ be the three roots of $\mathcal{A}(d_2;\mu) = 0$ with $\mathrm{Re}\{\tilde{\mu}_1(d_2)\} \le \mathrm{Re}\{\tilde{\mu}_2(d_2)\} \le \mathrm{Re}\{\tilde{\mu}_3(d_2)\}$. Then

$$\tilde{\mu}_1(d_2)\tilde{\mu}_2(d_2)\tilde{\mu}_3(d_2) = \frac{\det\{\mathbf{G}_u(\tilde{\mathbf{u}})\}}{A_3(d_2)}.$$

Since $\det\{\mathbf{G}_u(\tilde{\mathbf{u}})\} < 0$ from (4.21) and (4.22), and $A_3(d_2) > 0$, one of $\tilde{\mu}_1(d_2)$, $\tilde{\mu}_2(d_2)$, $\tilde{\mu}_3(d_2)$ is real and negative, and the product of the other two is positive. For a sufficiently large d_2, i.e $d_2 \to \infty$, consider the following limits:

$$\lim_{d_2\to\infty} \frac{A_3(d_2)}{d_2} = d_3, \qquad \lim_{d_2\to\infty} \frac{A_2(d_2)}{d_2} = -j_{33}^{[4]} - j_{11}^{[4]} d_3, \qquad \lim_{d_2\to\infty} \frac{A_1(d_2)}{d_2} = j_{11}^{[4]} j_{33}^{[4]} - j_{13}^{[4]} j_{31}^{[4]},$$

$$\begin{aligned} \lim_{d_2\to\infty} \frac{\mathcal{A}_1(d_2)}{d_2} &= d_3\mu^3 - (j_{33}^{[4]} + j_{11}^{[4]} d_3)\mu^2 + (j_{11}^{[4]} j_{33}^{[4]} - j_{13}^{[4]} j_{31}^{[4]})\mu \\ &= \mu[d_3\mu^2 - (j_{33}^{[4]} + j_{11}^{[4]} d_3)\mu + j_{11}^{[4]} j_{33}^{[4]} - j_{13}^{[4]} j_{31}^{[4]}]. \end{aligned}$$

Note that $j_{11}^{[4]} j_{33}^{[4]} - j_{13}^{[4]} j_{31}^{[4]} > 0$. If the parameters Λ, d_2, d_3 satisfy $j_{33}^{[4]} + j_{11}^{[4]} d_3 > 0$, the following proposition can be established.

Proposition 4.7 *Assume that (4.9) holds, and $j_{11}^{[4]} > 0, j_{22}^{[4]} > 0, j_{33}^{[4]} > 0$. Then there exists a positive constant $\bar{D}_2$ such that when $d_2 \ge \bar{D}_2$, the three roots $\tilde{\mu}_1(d_2)$, $\tilde{\mu}_2(d_2)$, $\tilde{\mu}_3(d_2)$ of $\mathcal{A}(d_2;\mu) = 0$ are all real and satisfy*

$$\begin{cases} \lim_{d_2\to\infty} \tilde{\mu}_1(d_2) = \dfrac{j_{33}^{[4]} + j_{11}^{[4]} d_3 - \sqrt{(j_{33}^{[4]} + j_{11}^{[4]} d_3)^2 - 4d_3(j_{11}^{[4]} j_{33}^{[4]} - j_{13}^{[4]} j_{31}^{[4]})}}{2d_3} \triangleq \hat{\mu}, \\ \lim_{d_2\to\infty} \tilde{\mu}_2(d_2) = 0, \\ \lim_{d_2\to\infty} \tilde{\mu}_3(d_2) = \dfrac{j_{33}^{[4]} + j_{11}^{[4]} d_3 + \sqrt{(j_{33}^{[4]} + j_{11}^{[4]} d_3)^2 - 4d_3(j_{11}^{[4]} j_{33}^{[4]} - j_{13}^{[4]} j_{31}^{[4]})}}{2d_3} \triangleq \check{\mu}. \end{cases} \tag{4.41}$$

Moreover, if $j_{11}^{[4]}j_{33}^{[4]} - j_{13}^{[4]}j_{31}^{[4]} < 0$, *then*

$$\begin{cases} -\infty < \tilde{\mu}_1(d_2) < 0 < \tilde{\mu}_2(d_2) < \tilde{\mu}_3(d_2), \\ \mathcal{A}(d_2;\mu) < 0, \ \ \textit{when } \mu \in (-\infty, \tilde{\mu}_1(d_2)) \cup (\tilde{\mu}_2(d_2), \tilde{\mu}_3(d_2)), \\ \mathcal{A}(d_2;\mu) > 0, \ \ \textit{when } \mu \in (\tilde{\mu}_1(d_2), \tilde{\mu}_2(d_2)) \cup (\tilde{\mu}_3(d_2), +\infty,). \end{cases} \tag{4.42}$$

The following theorem proves the existence of non-constant positive solutions of (4.27) for some fixed positive constants Λ, d_3, when d_2 is sufficiently large.

Theorem 4.6 *Assume that the parameters* Λ, d_3 *are fixed,* $j_{11}^{[4]} > 0, j_{22}^{[4]} > 0, j_{33}^{[4]} > 0$, *(4.9), (4.29) hold, and* $j_{11}^{[4]}j_{33}^{[4]} - j_{13}^{[4]}j_{31}^{[4]} < 0$, $\breve{\mu} \in (\mu_n, \mu_{n+1})$ *for some* $n \geq 1$, *and the sum* $\gamma_n = \sum_{i=1}^{n} \dim E(\mu_i)$ *is odd. Then there exists a positive constant* $\bar{D}_2$ *such that, if* $d_2 \geq \bar{D}_2$, *the system (4.27) has at least one non-constant positive solution.*

Proof. If $j_{11}^{[4]}j_{33}^{[4]} - j_{13}^{[4]}j_{31}^{[4]} > 0$, from Proposition 4.7, it follows that there exists a positive constant $\bar{D}_2$, such that when $d_2 \geq \bar{D}_2$, (4.42) holds and

$$0 = \mu_0 < \tilde{\mu}_1(d_2) < \mu_2, \ \ \tilde{\mu}_3(d_2) \in (\mu_n, \mu_{n+1}) \tag{4.43}$$

Now it is established that for any $d_2 \geq \bar{D}_2$, (4.27) admits at least one non-constant positive solution. To develop the proof, the homotopy invariance of the topological degree is invoked to generate a contradiction. Suppose the assertion is not true for some $d_2 = \tilde{d}_2 \geq \bar{D}_2$. Fix $d_2 = \bar{d}_2$. By Theorem 4.5, a positive constant $D_1 = D_1(\Lambda, d_2^*, d_3^*)$ is obtained such that

$$d_2^* \geq \frac{\mu_1}{\alpha_1(1+\beta_1)}, \ \ d_3^* \geq \frac{\mu_2}{\alpha_1(1+\beta_2)}.$$

Fix $\hat{d}_2 \geq d_2^*$, $\hat{d}_3 \geq \max\{d_3^*, d_3\}$, $1 > D_1$, For $t \in [0,1]$, define
$\mathbf{D}(t) = \text{diag}(1, d_2(t), d_3(t))$ with $d_i(t) = td_i(t) + (1-t)\hat{d}_i(t), i = 2, 3$ and consider the problem

$$\begin{cases} -\mathbf{D}(t)\Delta\mathbf{u} = \mathbf{G}(\mathbf{u}), & x \in \Omega, \\ \partial_{\mathbf{n}}\mathbf{u} = 0, & x \in \partial\Omega. \end{cases} \tag{4.44}$$

Thus, $\mathbf{u}^*$ is a non-constant positive solution of (4.27) if and only if it is a positive solution of (4.44) for $t = 1$. Hence, $\tilde{\mathbf{u}}^*$ is the unique constant positive solution of (4.44)

for any $0 \leq t \leq 1$. For any $0 \leq t \leq 1$, $\mathbf{u}^*$ is a positive solution of (4.27) if and only if it is a solution of the following problem

$$\mathbf{F}(t;\mathbf{u}) \triangleq \mathbf{u} - (\mathbf{I}-\Delta)^{-1}\{\mathbf{D}^{-1}(t)\mathbf{G}(\mathbf{u}) + \mathbf{u}\} = 0 \text{ in } \mathbf{X}^+.$$

Note that $\mathbf{F}(1;\mathbf{u}) = \mathbf{F}(\mathbf{u})$. But from Theorem 4.5 it is deduced that $\mathbf{F}(0;\mathbf{u}) = 0$ has only the positive solution $\tilde{\mathbf{u}}^*$ in $\mathbf{X}^+$. It follows that

$$D_{\mathbf{u}}\mathbf{F}(t;\mathbf{u}) \triangleq \mathbf{I} - (\mathbf{I}-\Delta)^{-1}\{\mathbf{D}^{-1}(t)\mathbf{G}_{\mathbf{u}}(\mathbf{u}) + \mathbf{I}\}.$$

In particular,

$$\begin{aligned} D_{\mathbf{u}}\mathbf{F}(0;\tilde{\mathbf{u}}) &= \mathbf{I} - (\mathbf{I}-\Delta)^{-1}\{\hat{\mathbf{D}}^{-1}\mathbf{G}_{\mathbf{u}}(\tilde{\mathbf{u}}) + \mathbf{I}\}, \\ D_{\mathbf{u}}\mathbf{F}(1;\tilde{\mathbf{u}}) &= \mathbf{I} - (\mathbf{I}-\Delta)^{-1}\{\mathbf{D}^{-1}\mathbf{G}_{\mathbf{u}}(\tilde{\mathbf{u}}) + \mathbf{I}\} = D_{\mathbf{u}}\mathbf{F}(\tilde{\mathbf{u}}), \end{aligned}$$

where $\hat{\mathbf{D}}$ =diag$(1, \hat{d}_2, \hat{d}_3)$. From (4.39) and (4.40)

$$H(\mu) = \frac{1}{d_2 d_3}\mathcal{A}(d_1;\mu). \tag{4.45}$$

For $t = 1$, it follows from (4.42) and (4.43) that

$$\begin{cases} H(\mu_0) = H(0) > 0, \\ H(\mu_i) < 0, 1 \leq i \leq n \quad , \\ H(\mu_i) > 0, i \geq n+1. \end{cases}$$

Hence, 0 is not an eigenvalue of the matrix $\mu_i\mathbf{I} - \mathbf{D}^{-1}\mathbf{G}_{\mathbf{u}}(\tilde{\mathbf{u}})$ for all $i \geq 0$, and

$$\sum_{i\geq 1, H(\mu_i)<0} \dim E(\mu_i) = \sum_{i=1}^{n} \dim E(\mu_i) = \gamma_n,$$

which is odd. By Proposition 4.6

$$\text{index}(\mathbf{F}(1;\cdot), \tilde{\mathbf{u}}) = (-1)^{\gamma} = (-1)^{\gamma_n} = -1. \tag{4.46}$$

There is need to prove that

$$\text{index}(\mathbf{F}(0;\cdot), \tilde{\mathbf{u}}) = (-1)^0 = 1. \tag{4.47}$$

From (4.9), we fix $0 < \beta_0 < \beta_2$ such that

$$\mu_2 > \beta_2\delta_2 > \beta_0\delta_2. \tag{4.48}$$

Define $\beta_2(s) = s\beta_2 + (1-s)\beta_0$ for $s \in [0,1]$, and consider problem (4.27) where $(1, d_2, d_3)$ and β_2 are replaced by $(1, \hat{d}_2, \hat{d}_3)$ and $\beta_2(s)$, respectively. This problem is relabeled as (4.27s), and denoting the corresponding non-linear term $\mathbf{G}(\mathbf{u})$ by $\mathbf{G}(s;\mathbf{u})$. As $\beta_2(s) \le \beta_2$ for all $s \in [0,1]$, Theorem 4.3 holds for the problem (4.27s). Similar to the proof of Theorem 4.5, $\tilde{\mathbf{u}}$ is the only positive solution of (4.27s) for all $s \in [0,1]$. In the same way as done above, define

$$\widehat{\mathbf{F}}(s;\mathbf{u}) \triangleq \mathbf{u} - (\mathbf{I}-\Delta)^{-1}\{\widehat{\mathbf{D}}^{-1}(t)\mathbf{G}(s;\mathbf{u}) + \mathbf{u}\} = 0 \text{ in } \mathbf{X}^+.$$

Then $\widehat{\mathbf{F}}(1;\cdot) = \mathbf{F}(0;\cdot)$, and $\tilde{\mathbf{u}}$ is the only positive solution of $\widehat{\mathbf{F}}(s;\mathbf{u}) = 0$ for all $s \in [0,1]$. The homotopy invariance of the topological degree asserts that

$$\text{index}(\widehat{\mathbf{F}}(1;\cdot), \tilde{\mathbf{u}}) = \text{index}(\mathbf{F}(0;\cdot), \tilde{\mathbf{u}}). \tag{4.49}$$

Since $\beta_2(0) = \beta_0$ and β_0 satisfies (4.48), $j_{11}^{[4]}(\beta_0) < 0$, $j_{33}^{[4]}(\beta_0) < 0$, then $\det(\mu_i\widehat{\mathbf{D}} - \mathbf{G}_{\mathbf{u}}(0;\tilde{\mathbf{u}})) > 0$, for all $i \ge 1$. Consequently, by Proposition 4.6, $\text{index}(\mathbf{F}(0;\cdot) = (-1)^0 = 1$ because, in this case, the corresponding $\gamma = 0$. Applying $\widehat{\mathbf{F}}(1;\cdot) = \mathbf{F}(0;\cdot)$ and (4.49) we see that (4.47) holds.

Since the positive solution is bounded, Theorems 4.3 and 4.4, there exists a positive constant c such that, for all $0 \le t \le 1$, the positive solutions of (4.43) satisfy $1/c < u, v, w < c$. Therefore, $\mathbf{F}(t;\mathbf{u}) \ne 0$ on $\partial\mathcal{B}(C)$ for all $0 \le t \le 1$. By the homotopy invariance of the topological degree,

$$\deg(\mathbf{F}(1;\cdot), 0, \mathcal{B}(C)) = \deg(\mathbf{F}(0;\cdot), 0, \mathcal{B}(C)). \tag{4.50}$$

Besides, on the basis of our assumption, the two equations $\mathbf{F}(1;\mathbf{u}) = 0$ and $\mathbf{F}(0;\mathbf{u}) = 0$ are satisfied by one positive solution $\tilde{\mathbf{u}}$ in $\mathcal{B}(C)$, and hence, by (4.46) and (4.47),

$$\begin{aligned} \deg(\mathbf{F}(0;\cdot), 0, \mathcal{B}(C)) &= \text{index}(\mathbf{F}(0;\cdot), \tilde{\mathbf{u}}) = 1, \\ \deg(\mathbf{F}(1;\cdot), 0, \mathcal{B}(C)) &= \text{index}(\mathbf{F}(1;\cdot), \tilde{\mathbf{u}}) = -1. \end{aligned}$$

This yields a contradiction to (4.50) and the system has one non-constant positive solution. □

4.7 Turing instability

A reaction-diffusion system exhibits diffusion-driven instability, generally known as Turing instability, when the homogeneous steady state remains stable under small perturbations in the absence of diffusion but becomes unstable under small spatial perturbations in the presence of diffusion [153]. In this section the necessary and sufficient conditions for diffusion-driven instability of the steady state and the development of spatial pattern for the general system (4.2) are derived. Consider the two-dimensional domain defined by $0 < x < p$, $0 < y < q$ whose rectangular boundary is denoted by $\partial\Omega$. Linearising the spatio-temporal system (4.2) at the spatially homogeneous steady state E_4 for small space and time-dependent fluctuations leads to

$$\begin{aligned} u(x,y,t) &= u_4 + \overline{u}(x,y,t);\ |\overline{u}(x,y,t)| \ll u_4, \\ v(x,y,t) &= v_4 + \overline{v}(x,y,t);\ |\overline{v}(x,y,t)| \ll v_4, \\ w(x,y,t) &= w_4 + \overline{w}(x,y,t);\ |\overline{w}(x,y,t)| \ll w_4. \end{aligned}$$

Let $\overline{\mathbf{u}} = (\overline{u}, \overline{v}, \overline{w})$, then the linearised system becomes

$$\overline{\mathbf{u}}_t = A\overline{\mathbf{u}} + \mathbf{D}\nabla^2\overline{\mathbf{u}}. \tag{4.51}$$

To solve this system of equations subject to the boundary conditions (4.2), firstly $\overline{\mathbf{U}}(x,y)$ is defined to be the time-independent solution of the spatial eigenvalue problem defined by

$$\nabla^2\overline{\mathbf{U}} + k^2\overline{\mathbf{U}} = 0, \quad (\mathbf{n}\cdot\nabla)\overline{\mathbf{U}} = 0 \quad \text{for} \quad (x,y) \quad \text{on} \quad \partial\Omega, \tag{4.52}$$

where k is an eigenvalue. The corresponding eigenfunctions are

$$\overline{\mathbf{U}}_k(x,y) = \alpha_i \cos(k_x x)\cos(k_y y).$$

Assume solutions of the form

$$\overline{\mathbf{u}} = \sum_i e^{\lambda(k^2)t}\overline{\mathbf{U}}_k(x,y) = \sum_i \alpha_i e^{\lambda(k^2)t}\cos(k_x x)\cos(k_y y), \tag{4.53}$$

i.e.

$$\begin{aligned}\overline{u}(x,y,t) &= \alpha_0 e^{\lambda t}\cos(k_x x)\cos(k_y y)\\ \overline{v}(x,y,t) &= \alpha_1 e^{\lambda t}\cos(k_x x)\cos(k_y y)\\ \overline{w}(x,y,t) &= \alpha_2 e^{\lambda t}\cos(k_x x)\cos(k_y y)\end{aligned}$$

where λ is the growth rate of perturbation in time t, $\alpha_i (i = 0, 1, 2)$ represent the amplitudes, $k_x = \dfrac{n\pi}{p}$ and $k_y = \dfrac{m\pi}{q}$ are the wave numbers of the solutions, and $k^2 = k_x^2 + k_y^2$. This form of solution satisfies zero flux conditions at $x = 0$, $x = p$, $y = 0$ and $y = q$. Substituting this form (4.53) into (4.51) and cancelling $e^{\lambda t}$ yields

$$\begin{aligned}\lambda\overline{\mathbf{U}} &= J\overline{\mathbf{U}} + D\nabla^2\overline{\mathbf{U}}\\ &= J\overline{\mathbf{U}} - Dk^2\overline{\mathbf{U}}\end{aligned}$$

The corresponding linearised system has the characteristic equation

$$|J - k^2 D - \lambda I| = 0 \tag{4.54}$$

that is, with $J = J_4$,

$$\begin{vmatrix} j_{11}^{[4]} - k^2 - \lambda & j_{12}^{[4]} & j_{13}^{[4]} \\ j_{21}^{[4]} & j_{22}^{[4]} - k^2 d_2 - \lambda & j_{23}^{[4]} \\ j_{31}^{[4]} & 0 & j_{33}^{[4]} - k^2 d_3 - \lambda \end{vmatrix} = 0.$$

or

$$(j_{11}^{[4]}-k^2-\lambda)(j_{22}^{[4]}-k^2-\lambda)(j_{33}^{[4]}-k^2-\lambda)+j_{12}^{[4]}j_{23}^{[4]}j_{31}^{[4]}-(j_{33}^{[4]}-k^2-\lambda)j_{12}^{[4]}j_{21}^{[4]}-(j_{22}^{[4]}-k^2-\lambda)j_{13}^{[4]}j_{31}^{[4]} = 0$$

The characteristic equation corresponding to E_4 is

$$\lambda^3 + h_2(k^2)\lambda^2 + h_1(k^2)\lambda + h_0(k^2) = 0, \tag{4.55}$$

with

$$h_2(k^2) = k^2(1 + d_2 + d_3) - (j_{11}^{[4]} + j_{22}^{[4]} + j_{33}^{[4]}),$$

$$\begin{aligned}h_1(k^2) &= j_{11}^{[4]}j_{22}^{[4]} + j_{11}^{[4]}j_{33}^{[4]} + j_{22}^{[4]}j_{33}^{[4]} - j_{12}^{[4]}j_{21}^{[4]} - j_{13}^{[4]}j_{31}^{[4]} - j_{23}^{[4]}j_{32}^{[4]}\\ &\quad -k^2(j_{22}^{[4]}(1 + d_3) + j_{33}^{[4]}(1 + d_2) + j_{11}^{[4]}(d_2 + d_3)) + k^4(d_2 + d_3 + d_2 d_3),\end{aligned}$$

$$
\begin{aligned}
h_0(k^2) &= -j_{11}^{[4]}j_{22}^{[4]}j_{33}^{[4]} - j_{12}^{[4]}j_{23}^{[4]}j_{31}^{[4]} - j_{13}^{[4]}j_{21}^{[4]}j_{32}^{[4]} + j_{11}^{[4]}j_{23}^{[4]}j_{32}^{[4]} + j_{12}^{[4]}j_{21}^{[4]}j_{33}^{[4]} + j_{13}^{[4]}j_{22}^{[4]}j_{31}^{[4]} \\
&\quad -k^2((j_{23}^{[4]}j_{32}^{[4]} - j_{22}^{[4]}j_{33}^{[4]})d_1 + (j_{13}^{[4]}j_{31}^{[4]} - j_{11}^{[4]}j_{33}^{[4]})d_2 + (j_{12}^{[4]}j_{21}^{[4]} - j_{11}^{[4]}j_{22}^{[4]})d_3) \\
&\quad -k^4(j_{33}^{[4]}d_2 + j_{22}^{[4]}d_3 + j_{11}^{[4]}d_2d_3) + k^6d_2d_3.
\end{aligned}
$$

Reaction-diffusion systems are associated with instabilities or bifurcations. The Hopf and Turing bifurcations are two types of symmetry-breaking bifurcations that lead to the formation of patterns. Hopf bifurcation is space-independent and collapses the temporal symmetry of a system leading to oscillations that are uniform in space and periodic in time. Turing bifurcation collapses spatial-symmetry giving rise to the emergence of patterns that are stationary in time and oscillatory in space [107, 154, 155]. Equation (4.55) is the dispersion relation. The conditions under which the real parts of λ are negative are investigated. When the conditions are satisfied for all k, the homogeneous steady state (4.3) is stable to small perturbations.

According to the Routh-Hurwitz criteria $\mathrm{Re}(\lambda(k)) < 0$ if and only if

$$
h_0(k^2) > 0, h_2(k^2) > 0, h_1(k^2)h_2(k^2) - h_0(k^2) > 0. \tag{4.56}
$$

If at least one of the conditions above is violated, then the implication is that there exists an eigenvalue with positive real part, which is instability. Turing instability happens if the homogeneous steady state is stable when there is no diffusion ($k^2 = 0$) but becomes unstable in the presence of diffusion ($k^2 > 0$). Therefore, conditions which support a Turing instability are deduced. These are such that the spatially uniform steady state remains stable when subjected to small perturbations, that is, all $\lambda(k^2)$ in equation (4.55) have $\mathrm{Re}(\lambda(k^2 = 0)) < 0$, and that, only patterns of a particular spatial extent, that is, patterns within a definite range of wave length k, can begin to form, with $\mathrm{Re}(\lambda(k^2 \neq 0)) > 0$ [107].

The homogeneous steady state E_4 is locally asymptotically stable if and only if $h_0(0) > 0$, $h_2(0) > 0$ and $h_1(0)h_2(0) - h_0(0) > 0$. However, it becomes unstable due to diffusion if at least any one of the conditions in equation (4.56) is violated. Note that instability

due to diffusion cannot happen by contradicting $h_2(k^2) > 0$ since the trace of the matrix J_4 is negative for stability. Conditions which can reverse the sign of the other two conditions in equation (4.56) are sought. Each expression for $h_0(k^2)$ and $h_1(k^2)h_2(k^2) - h_0(k^2)$ is a cubic function of k^2 that takes the form [107]:

$$P(k^2) = P_3k^6 + P_2k^4 + P_1k^2 + P_0, \quad P_3 > 0, P_0 > 0. \tag{4.57}$$

The coefficients $P_i(i = 0, 1, 2, 3)$ are given in Tables 4.2 and 4.3.

Table 4.2: Values of $P_i(i = 0, 1, 2, 3)$ for h_0

	h_0
P_3	d_2d_3
P_2	$-j_{11}^{[4]}d_2d_3 - j_{22}^{[4]}d_3 - j_{33}^{[4]}d_2$
P_1	$(j_{22}^{[4]}j_{33}^{[4]} - j_{23}^{[4]}j_{32}^{[4]}) + d_2(j_{11}^{[4]}j_{33}^{[4]} - j_{13}^{[4]}j_{31}^{[4]}) + d_3(j_{11}^{[4]}j_{22}^{[4]} - j_{12}^{[4]}j_{21}^{[4]})$
P_0	$h_0(0)$

Table 4.3: Values of $P_i(i = 0, 1, 2, 3)$ for $h_1h_2 - h_0$

	$h_1h_2 - h_0$
P_3	$(1 + d_3)(d_2 + d_3)(1 + d_2)$
P_2	$-j_{11}^{[4]}(2d_2 + d_2^2 + 2d_3 + 2d_2d_3 + d_3^2) - j_{22}^{[4]}(2d_2 + 2d_3 + 2d_2d_3 + 1 + d_3^2)$ $-j_{33}^{[4]}(1 + 2d_2 + d_2^2 + 2d_3 + 2d_2d_3)$
P_1	$j_{11}^{[4]^2}(d_2 + d_3) + j_{22}^{[4]^2}(1 + d_3) + j_{33}^{[4]^2}(1 + d_2) - j_{13}^{[4]}j_{31}^{[4]}(1 + d_3) - j_{12}^{[4]}j_{21}^{[4]}(1 + d_2)$ $-j_{23}^{[4]}j_{32}^{[4]}(d_2 + d_3) + 2(1 + d_2 + d_3)(j_{11}^{[4]}j_{22}^{[4]} + j_{11}^{[4]}j_{33}^{[4]} + j_{22}^{[4]}j_{33}^{[4]})$
P_0	$h_1(0)h_2(0) - h_0(0)$

Theorem 4.7 *The spatio-temporal system (4.2) will experience Turing instability at the homogeneous steady state E_4 if the following two conditions are met:*

1. $P_1 < 0$ *or* $(P_2 < 0$ *and* $P_2^2 > 3P_1P_3)$

2. $2P_2^3 - 9P_1P_2P_3 + 27P_3^2P_0 - 2(P_2^2 - 3P_1P_3)^{3/2} < 0$

Proof. If for some strictly positive real number k^2, $P(k^2)$ is negative, then the associated minimum must be negative. The minimum is located at a positive zero of $P'(k^2) = 0$, i.e.

$$3P_3(k^2)^2 + 2P_2(k^2) + P_1 = 0,$$

given by

$$k^2 = k_c^2 = \frac{-P_2 + (P_2^2 - 3P_1P_3)^{\frac{1}{2}}}{3P_3}. \tag{4.58}$$

Now k_c^2 is real and positive if

$$P_1 < 0 \text{ or } (P_2 < 0 \text{ and } P_2^2 > 3P_1P_3). \tag{4.59}$$

It follows that

$$k^4 = (k_c^2)^2 = \frac{2P_2^2 - 2P_2(P_2^2 - 3P_1P_3)^{\frac{1}{2}} - 3P_1P_3}{9P_3^2},$$

and

$$k^6 = (k_c^2)^3 = \frac{-4P_2^3 + (4P_2^2 - 3P_1P_3)(P_2^2 - 3P_1P_3)^{\frac{1}{2}} + 9P_1P_2P_3}{27P_3^3}.$$

Thus,

$$\begin{aligned} P(k_c^2) &= \frac{-4P_2^3 + (4P_2^2 - 3P_1P_3)(P_2^2 - 3P_1P_3)^{\frac{1}{2}} + 9P_1P_2P_3}{27P_3^2} \\ &\quad + \frac{2P_2^3 - 2P_2^2(P_2^2 - 3P_1P_3)^{\frac{1}{2}} - 3P_1P_2P_3}{9P_3^2} \\ &\quad + \frac{-P_1P_2 + P_1(P_2^2 - 3P_1P_3)^{\frac{1}{2}}}{3P_3} + P_0 \\ &= \frac{-4P_2^3 + (4P_2^2 - 3P_1P_3)(P_2^2 - 3P_1P_3)^{\frac{1}{2}} + 9P_1P_2P_3}{27P_3^2} \\ &\quad + \frac{6P_2^3 - 6P_2^2(P_2^2 - 3P_1P_3)^{\frac{1}{2}} - 9P_1P_2P_3}{27P_3^2} \\ &\quad + \frac{-9P_1P_2P_3 + 9P_1P_3(P_2^2 - 3P_1P_3)^{\frac{1}{2}}}{27P_3^2} + \frac{27P_3^2P_0}{27P_3^2}, \end{aligned}$$

which reduces to

$$P_{\min} = P(k_c^2) = \frac{2P_2^3 - 9P_1P_2P_3 + 27P_3^2P_0 - 2(P_2^2 - 3P_1P_3)^{3/2}}{27P_3^2}. \tag{4.60}$$

Hence, $P(k_c^2) < 0$ if

$$2P_2^3 - 9P_1P_2P_3 + 27P_3^2P_0 - 2(P_2^2 - 3P_1P_3)^{3/2} < 0. \tag{4.61}$$

The condition for bifurcation occurs is $P_{\min} = 0$. The inequality (4.61) reduces to an equation given by

$$2P_2^3 - 9P_1P_2P_3 + 27P_3^2P_0 - 2(P_2^2 - 3P_1P_3)^{3/2} = 0. \tag{4.62}$$

□

4.8 Numerical analysis

It was estimated that 1684 (95 % CI:1617 - 1751) lions lived in the Kruger National Park (KNP) in 2005 and 2006 [48]. The initial population densities for each species are chosen to be positive at $U(0) = 133,000$ [24], $V(0) = 412$ [23], $W(0) = 1700$ [48]. The parameters values used in the numerical analysis of model (4.1) are given in Table 4.4. Some parameter values which were not readily available were computed on the basis of the steady states and stability conditions, and these include prey conversion rates into predator biomass.

4.8.1 Parameter estimates

Conversion rates

The cheetah and lion biomass conversion rates μ_1 and μ_2 are estimated in relation with the bounds for existence of equilibrium points E_2 and E_3. For E_2, we have $\mu_1 > \beta_1\delta_1 = 298.2$. For E_3, we have $\mu_2 > \beta_2\delta_2 = 331.44$. In this study, it was estimated that $\mu_1 = 320$ and $\mu_2 = 340$.

Table 4.4: Parameter values

Symbol	Description	Value	Reference
D_1	Diffusion coefficient of impala	0.5	estimate
D_2	Diffusion coefficient of cheetah	1.0	estimate
D_3	Diffusion coefficient of lion	0.8	estimate

Based on the parameter values given in Tables 3.2 and 4.4, the non-dimensional parameter values used in the simulations were calculated and are given in Table 4.5 .

Table 4.5: Parameters for system (4.2)

Parameter	Description	Values
d_2	Diffusion coefficient of impala	2.0
d_3	Diffusion coefficient of cheetah	1.6
β_1	Handling time of impala by cheetah	60
β_2	Handling time of impala by lion	80
μ_1	Conversion rate of impala biomass into new cheetahs	320
μ_2	Conversion rate of impala biomass into new lions	340
δ_1	Natural mortality of cheetah	4.97
δ_2	Natural mortality of lion	4.143
ν	Mortality rate of cheetah by lion	0.525

4.8.2 Population variations

The population distributions for impala, cheetah and lion in the ODE system corresponding to system (4.1) are plotted. The initial values used are $U_0 = 133,000$, $V_0 = 412$, and $W_0 = 1700$. In the absence of lion kills, $p = 0$, all the species populations persist as shown in Figure 4.1. However, when the lion kills are present, $p = 0.0005$, the cheetah will be extinct in about forty years as shown in Figure 4.2. The point in time of extinction is a function of rate of cheetah kills. The more cheetah are killed in a given time, the quicker will they become extinct.

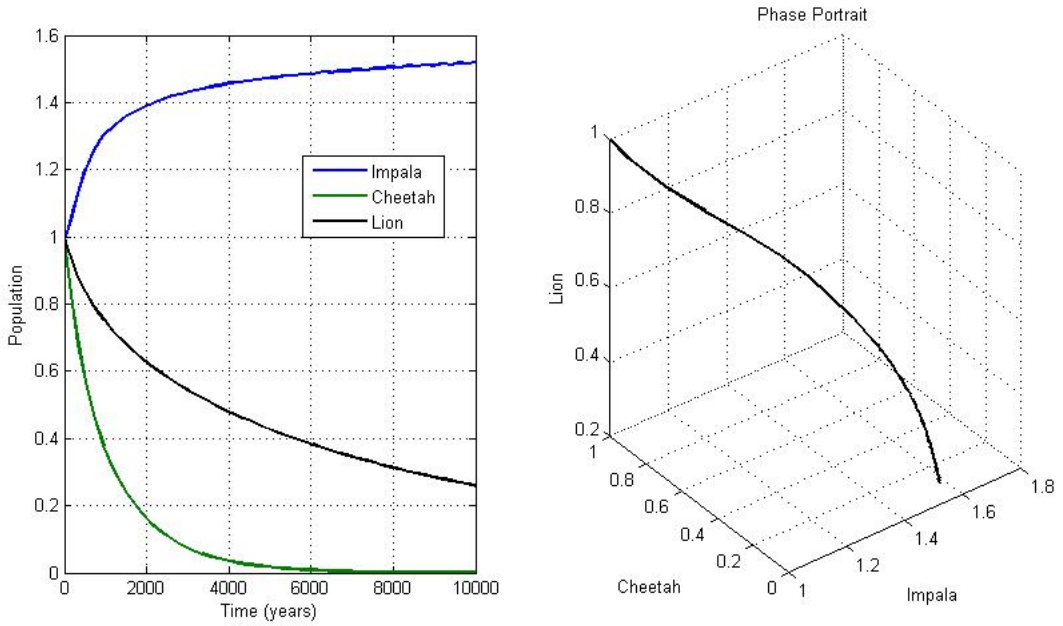

Figure 4.1: *(a) Variation of scaled population, (b) Phase space trajectory, when* $p = 0$

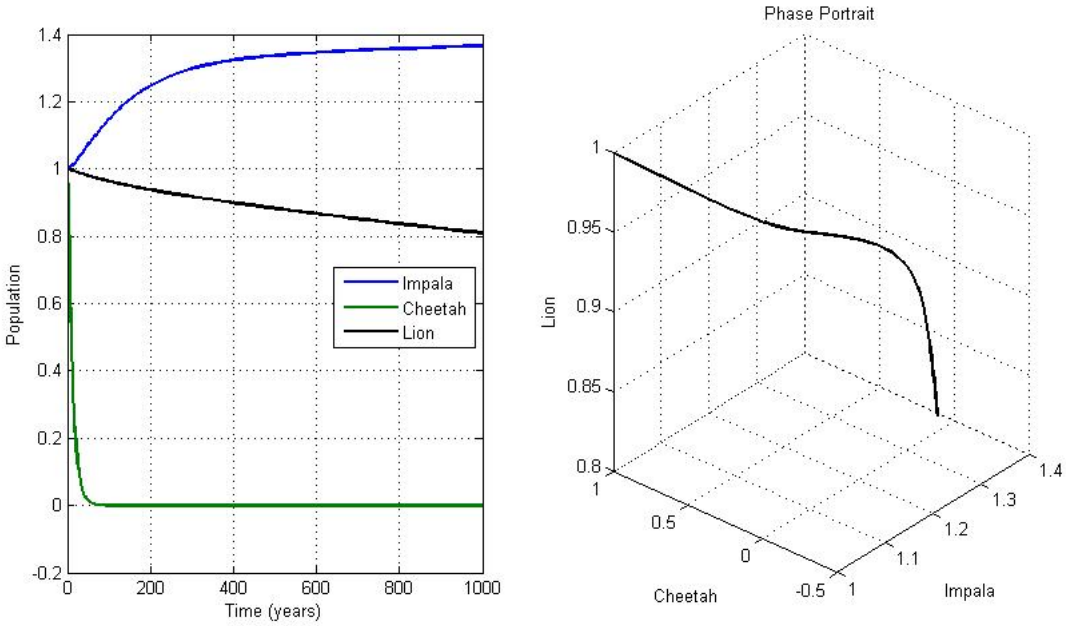

Figure 4.2: *(a) Variation of scaled population, (b) Phase space trajectory, when* $p =$ 0.00005

4.8.3 Spatio-Temporal Pattern Formation

For the numerical simulations, a MATLAB code adapted from Schneider [156] was used on a two dimensional square grid. Two sub-systems corresponding to the spatio-temporal system (4.2) were separately solved numerically in two-dimensional space using a second-order finite difference approximation for the spatial derivatives and a Forward Euler method for the time integration. A 100×100 grid points and grid size of 4×4 were used. Four tests were run at 31250 time steps per run where $\Delta t = 0.08$. The initial conditions were generated randomly as $u(x, y, 0) = 0.5+$ rand$[0, 2]$, $v(x, y, 0) = 0.5+$ rand$[0, 2]$ and $w(x, y, 0) = 0.5+$ rand$[0, 2]$.

Consider the impala-cheetah subsystem

$$\begin{aligned} \frac{\partial u}{\partial t} - \big(\frac{\partial^2 u}{\partial x^2} + \frac{\partial^2 u}{\partial y^2}\big) &= u(1-u) - \frac{uv}{(1+\beta_1 u)}, \\ \frac{\partial v}{\partial t} - d_2\big(\frac{\partial^2 v}{\partial x^2} + \frac{\partial^2 v}{\partial y^2}\big) &= \frac{\mu_1 uv}{1+\beta_1 u} - \delta_1 v, \end{aligned} \tag{4.63}$$

with boundary conditions

$$\begin{aligned} \frac{\partial u}{\partial \mathbf{n}}|_{\partial\Omega} &= \frac{\partial v}{\partial \mathbf{n}}|_{\partial\Omega} = 0, t > 0, \\ u(x, y, 0) &= u_0(x, y) \geq 0, v(x, y, 0) = v_0(x, y) \geq 0, \\ &\quad (x, y) \in \Omega. \end{aligned}$$

The patterns are shown in Figures 4.3 to 4.6. It is observed that cheetah tend to be located around areas occupied by impala. Where impala are densely populated, cheetah are also densely populated but surrounding impala. Where impala are sparsely populated, cheetah are not found there.

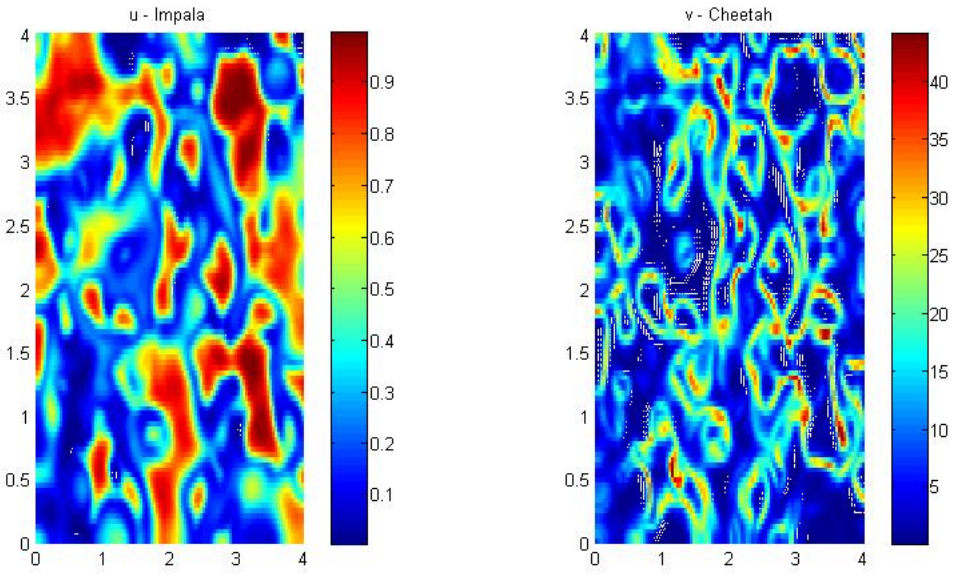

Figure 4.3: *Spatial distribution of impala and cheetah at* $T = 2500$.

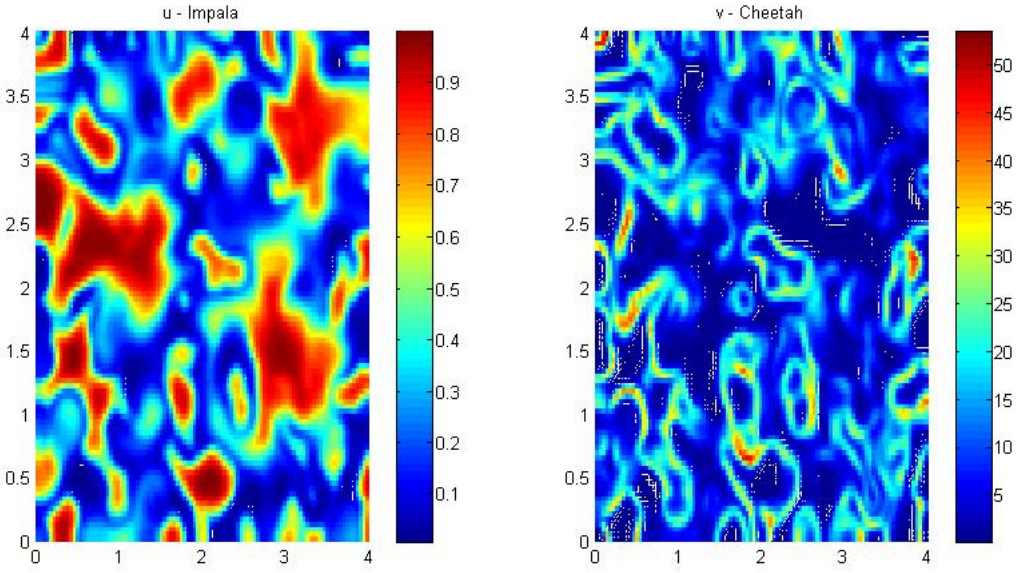

Figure 4.4: *Spatial distribution of impala and cheetah at* $T = 5000$.

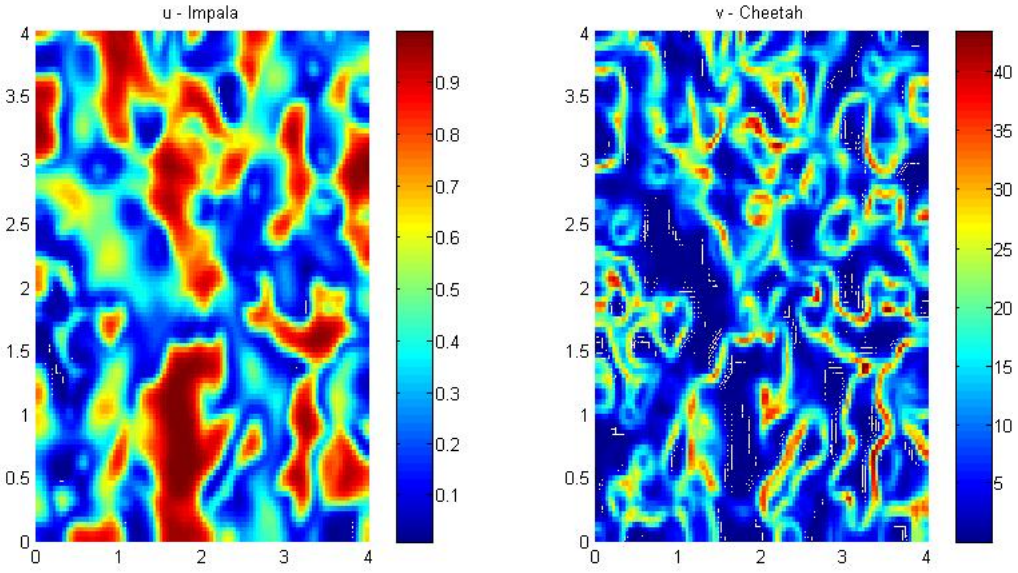

Figure 4.5: *Spatial distribution of impala and cheetah at* $T = 7500$.

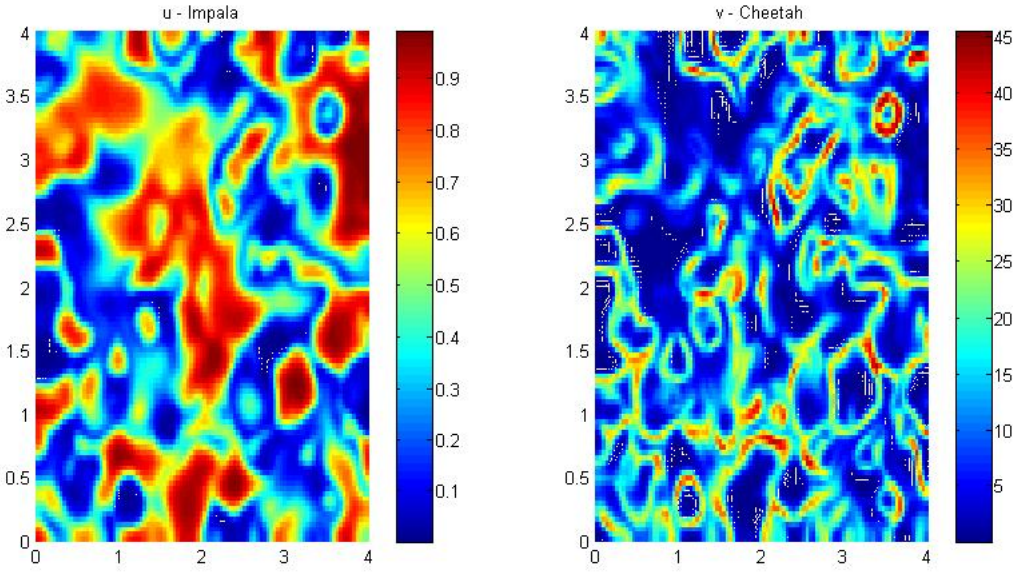

Figure 4.6: *Spatial distribution of impala and cheetah at* $T = 10000$.

For the impala-lion subsystem

$$\begin{aligned} \frac{\partial u}{\partial t} - (\frac{\partial^2 u}{\partial x^2} + \frac{\partial^2 u}{\partial y^2}) &= u(1-u) - \frac{uw}{1+\beta_2 u}, \\ \frac{\partial w}{\partial t} - d_3(\frac{\partial^2 w}{\partial x^2} + \frac{\partial^2 w}{\partial y^2}) &= \frac{\mu_2 uw}{1+\beta_2 u} - \delta_2 w, \end{aligned} \tag{4.64}$$

with boundary conditions

$$\begin{aligned} \frac{\partial u}{\partial \mathbf{n}}|_{\partial\Omega} &= \frac{\partial w}{\partial \mathbf{n}}|_{\partial\Omega} = 0, t > 0, \\ u(x,y,0) &= u_0(x,y) \geq 0, \\ w(x,y,0) &= w_0(x,y) \geq 0, (x,y) \in \Omega. \end{aligned}$$

The patterns are shown in Figures 4.7 to 4.10.

It can be observed that lions tend to change location with time. Initially, they are found in areas with low impala density. Later, they occupy areas with high impala density. Thus, lions' habitat selection alternates between high impala density and low impala density areas.

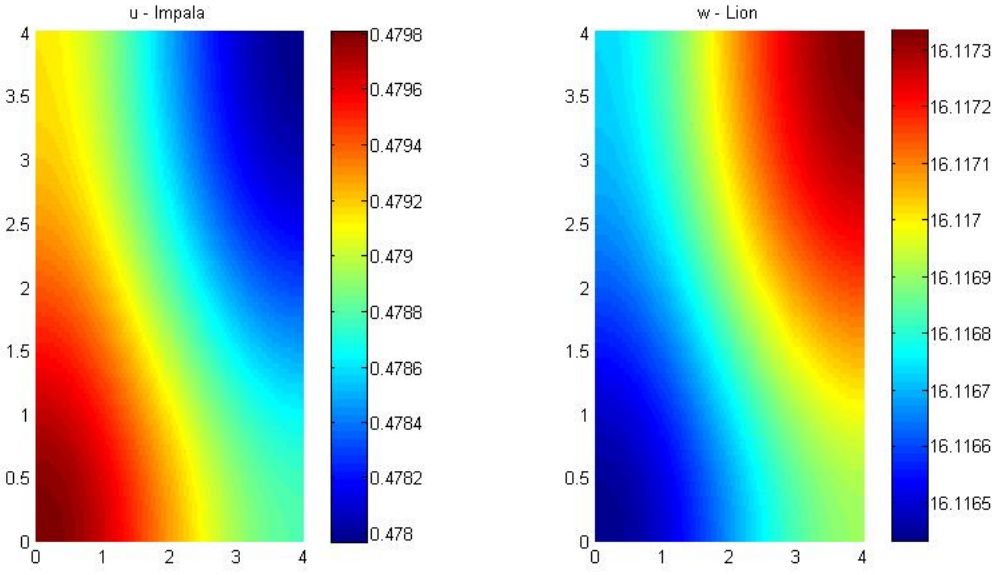

Figure 4.7: *Spatial distribution of impala and lion at* $T = 2500$.

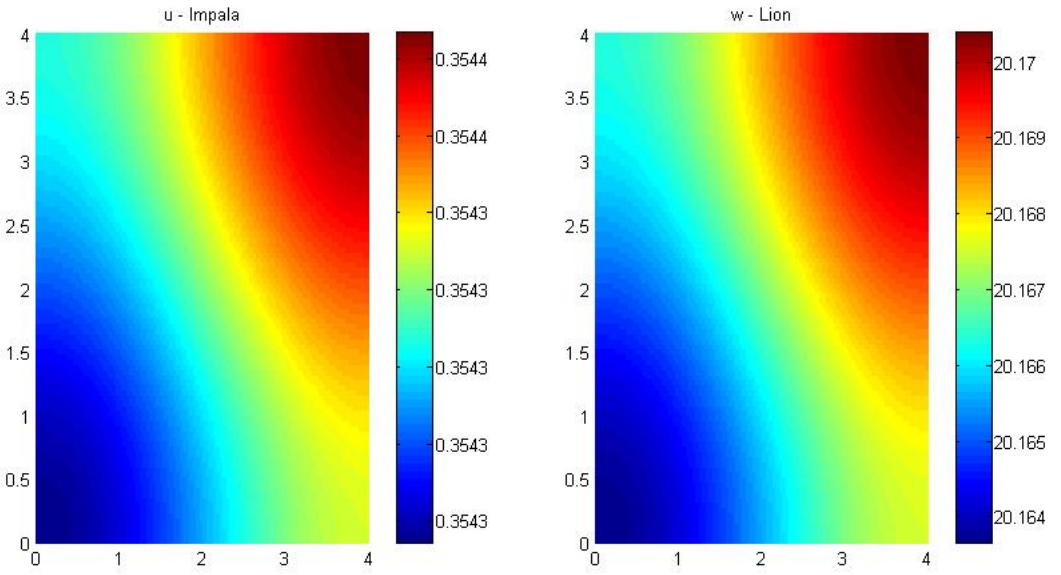

Figure 4.8: *Spatial distribution of impala and lion at* $T = 5000$.

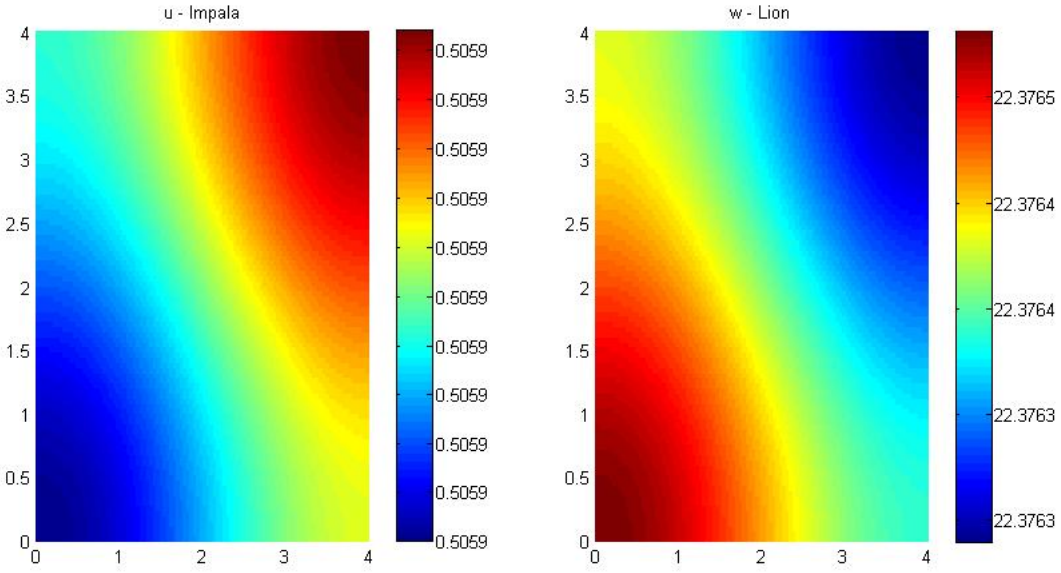

Figure 4.9: *Spatial distribution of impala and lion at* $T = 7500$.

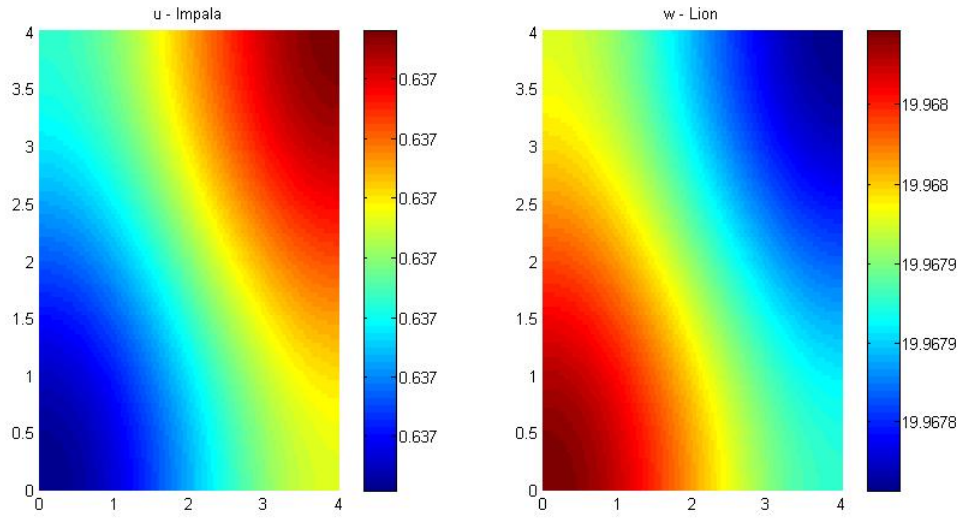

Figure 4.10: *Spatial distribution of impala and lion at* $T = 10000$.

4.9 Discussion and conclusion

The temporal system (4.3) corresponding to the reaction diffusion system (4.2) was analysed. Equilibrium points were identified and their corresponding global stability results derived. In particular, the positive steady state was globally asymptotically stable for the ODE system. The time series plots of the temporal system indicate that cheetah become extinct with time.

However, the temporal system became unstable when diffusion was taken into account. The implication is that self-diffusion can lead to the disappearance of stability and the emergence of patterns. The reaction-diffusion system (4.2) was analysed to establish conditions for existence and non-existence of non-constant stationary points. Furthermore, Turing instability conditions were derived for the positive steady state having been perturbed. The patterns indicate that both lions and cheetah follow impala but somehow in different ways. Cheetah tend to surround impala always. Lions spend time away from impala and also within areas occupied by impala. Lions and cheetah do not necessarily occupy the same territories.

Chapter 5

Discussion, conclusions and recommendations

5.1 Methods employed

The deterministic mathematical modelling techniques employed in this study provide a framework for describing the interactions between the three species involved. However, this is neither a unique way of representing such interactions, nor the best way. The representation of interactions has largely been based on theoretical explanations from ecology and epidemiology. Mathematical models have been formulated to explore effects of presence of disease in one species on the other species, as well as spatial considerations to account for species distributions.

Mathematical analysis of the models to establish stability conditions relied on a number of theorems and basic concepts of species interactions. These included linearisation, Lyapunov, Lasalle Invariance Principle, Routh-Hurwitz stability criterion, Bendixson-Dulac, Poincare-Bendixson, Geometric approach to global stability problems, Maximum Principle, Harnack Inequality, Energy method, Young Inequality, Poincare Inequality, Leray-Schauder degree theory and Turing Instability.

The numerical analysis was done in MATLAB. Some in-built MATLAB functions were employed to solve the systems for time-series and phase plots.

5.2 Results of the study

In Chapter 3 the interactions between impala, cheetah and lion were modelled in the presence of a disease in the lion population. The key aspects of the study were to examine the effects of the presence of disease in lions and added cheetah mortality by lions on cheetah population. Possible biologically feasible steady states for the main disease model (3.3), and for the subsystem (3.26) involving absence of lions were deduced. The respective global stability results were derived. Numerical simulations were performed for both systems.

In Chapter 4, spatio-temporal dynamics between impala, cheetah and lions were discussed. The study involved establishing stability conditions of the steady states of the temporal system, as well as positive steady state of the spatio-temporal system. Analysis of the spatio-temporal system involved establishing *a priori* estimates, the existence and non-existence of the stationary steady state solutions, and the conditions for Turing instability to occur.

5.2.1 On the role of additional mortality on cheetah by the lion

In the presence of the lion species in the system, it is worthwhile noting that the added cheetah mortality due to the lions ensured that cheetah become extinct in a very short period. When added mortality can be reduced to very low or is absent, cheetah survive and avoid extinction. This is shown in Figures 3.2 and 3.3. Cheetah and lion can coexist as long as added mortality is very low, though the cheetah population declines

slowly over time.

5.2.2 On the role of disease in the lion

The disease infection rate ensured that when it is low, extinction of lions is not possible. Cheetah and lion co-exist as long as disease infection rate assumes very low values. Cheetah population persists, but grows marginally with time. Furthermore, the presence of disease in lions reduces the pressure lions exert on cheetah, resulting in cheetah able to survive longer, but only as long as added mortality is kept very low. This is shown in Figures 3.2, 3.5 and 3.6. The equilibrium point E_4 for the system (3.3) was found to be locally asymptotically stable, using Routh Hurwitz criterion. Furthermore, the global asymptotic stability was established using the Lyapunov method. Conditions were established when the Lyapunov function would be negative definite.

The model without disease in lion, temporal system, studied in chapter 4 can be compared with the model with disease in lion covered in chapter 3. As in Proposition 4.5, the interior equilibrium E_4 for the system (4.3) is locally asymptotically stable if the Routh Hurwitz conditions are satisfied. Moreover, as stated in Theorem 4.1, the interior equilibrium is globally asymptotically stable on the basis of a Lyapunov function. From Figure 4.1, it was shown that with no added cheetah mortality due to the lions, the cheetah population declines, though slowly over a long time. It does not recover, until it becomes extinct.

Comparing Figures 3.2 and 4.1, it can be observed that the presence of the disease in lion keeps the population of the cheetah steady, though the lion population is driven to extinction. However, the impala population reaches a reduced level of about 1.4 times the initial level. In the absence of disease, the cheetah population slowly declines over a long time.

As such, the presence of disease in lions, when infection rate is high, drives the lion

population to extinction. The cheetah population grows. Unlike Maas *et al.* [46] who in their paper found out that disease in lion does not pose a serious conservation issue, in this study, the disease cannot be ignored as the lion is driven to extinction at the current levels of infection.

5.2.3 On the effect of lion removal from the system

In the absence of lion in the system (3.3), the cheetah population in subsystem (3.26) recovered from low value. Whenever the equilibrium point $\bar{E}_1$ was found to be stable, $\bar{E}_2$ did not exist. $\bar{E}_1$ was locally and globally stable when $l - e\mu - \frac{\mu}{K} < 0$. The impala population grew to carrying capacity. However, as in Lemma 3.5, equilibrium point $\bar{E}_2$ is locally asymptotically stable if $\frac{l+e\mu}{l-e\mu} > eK$ and $l - e\mu > \frac{\mu}{K}$, that is, $\frac{\mu}{l-e\mu} < K$, and $\bar{E}_1$ does not exist. Stability is assured if impala population is less than carrying capacity. Furthermore, as in Proposition 3.5, the global asymptotic stability of $\bar{E}_2$ is assured if $2\mu > lK$.

Thus, the absence of lion in the system allowed the cheetah population to rise significantly to more than double the initial value. The equilibrium point $\bar{E}_2$ is reached as shown in Figure 3.1. The results indicate that cheetah thrives very well in the absence of large competition from the lion. These findings are in agreement with the studies by Linnell and Strand [26]. Removal of large predators from an ecosystem may allow small to medium predator density to improve. However, accompanied by this is the enhanced predation of prey populations [26, 157, 158]. The impala population rises to about 1.18 times the initial value, lower than the 1.4 times the initial value in the presence of the lion. This can be seen upon comparing Figures 3.1, 3.2 and 3.3.

5.2.4 The spatio-temporal system

Analysis of the temporal system was based on establishing stability conditions of the steady states. The steady state E_2 was globally asymptotically stable if $2\delta_1 > \mu_1$ as

stated in Proposition 4.3. Furthermore, the steady state E_3 was globally asymptotically stable if $2\delta_2 > \mu_2$ as stated in Proposition 4.4. For the interior equilibrium, global stability was established using a Lyapunov function and Lasalle Invariance Principle.

Analysis of the reaction-diffusion system was based on the positive steady state. As stated in Theorem 4.2, the constant positive steady state was uniformly asymptotically stable. The stability was based on linearisation of the system and use of Routh Hurwitz criterion. *A priori* estimates of the positive steady state were deduced, that is, upper and lower bounds for the species populations. The non-existence of non-constant positive solution was established in Theorem 4.5 using the Poincare Inequality. The existence of non-constant positive solution was established in Theorem 4.6 using the Leray-Schauder degree theory. Keeping all other parameters fixed and allowing d_2 to vary, together with some conditions satisfied, there exists a positive constant $\bar{D}_2$ such that, if $d_2 \geq \bar{D}_2$, the system (4.27) has at least one non-constant positive solution as stated in Theorem 4.6.

Patterns showing species distributions were plotted on a square lattice. These were done for sub-systems involving impala and cheetah, and impala and lion respectively. For the impala cheetah subsystem, it was deduced that cheetah always track impala habitats but always surrounding them. Cheetah would not be found within impala territories. However, for the impala-lion subsystem, lions behaved in two different ways. Firstly, lions infiltrated impala habitats. Secondly, they showed a tendency to stay away form impala habitats. As such, a conclusion that cheetah and lions did not occupy the same territory was reached. This supports the notion that cheetah avoid lion territories at all costs.

5.3 Recommendations of the study

It has been found that cheetah thrive well in the absence of the lion. It is recommended that the cheetah be separated from the lion to ensure their continued survival and persistence. In the presence of the lion, cheetah will become extinct, though the population decline is dependent on the intensity of the added cheetah mortality due to lions. Predation by lions is a major contributor to the decline of the cheetah population.

The presence of disease in lions has insignificant effect on the cheetah population. However, the disease affects the lion species. At high levels of disease incidence, lion population becomes extinct quickly. The continued survival of the lions depend on the levels of the disease incidence and the disease-induced mortality rates. Without human intervention, the study showed the lions becoming extinct. Urgent treatment of disease is essential to save the lion from possible extinction.

5.4 Extensions of the study

The study offers insights into the survival of cheetah in the presence of their main competitor, the lion. However, there are a number of improvements to the proposed models to bring them closer to reality.

Logistic growth can be factored in for the lion species since impala is not their primary prey. Model (3.3) can be extended to include spatial considerations. This enables a clearer understanding of the effects of disease in lion on spatial distribution of the cheetah. There is need to incorporate water holes in the model. These are predation prone areas for impala. Simulating distribution of species would possibly show the location of cheetah relative to lion. Expectations are that cheetah occupy different areas from lion's territory, with some overlapping regions.

There is need to consider provision of additional food to cheetah, to compensate for loss of kills to the lions. The food can be in the form of slaughtered prey made available at some selected feeding places within their habitat. This can be incorporated into the model and an optimal control problem formulated. The problem to be solved involve seeking optimal strategy to increase the population of cheetah to desired level using quantity and quality of additional food as control variables.

It appears to be economically unviable to create patches within KNP to separate the cheetah from the lion. The results for the subsystem (3.26) which involve removal of lion suggest that cheetah survive well in the absence of their main competitor. The model can be extended to become a patchy model where lions are separated from cheetah.

There is urgent need to provide treatment to sick lions to reduce transmission rate and possibly eradicate the disease. The results show that even the cheetah survive extinction if the rate of disease infection in lion is low as supported by the Figures 3.5 and 3.6. If the infection rate remains as it is, the results suggest that in the long term, the infection has the potential to drive the lion population to extinction. As such, treatment of sick lions can be factored in the model.

The disease model can be extended to include susceptible-exposed-infective (SEI) dynamics. This enables the analysis to identify the class between the exposed and infective that can be used as biological control to improve the cheetah population.

Appendix A

A.1 Auxiliary and standard results of the theory of Ordinary Differential Equations

Theorem A.1 *(Bendixson-Dulac criterion)*
Let $h_1(u,v), h_2(u,v)$ and $\Phi(u,v)$ be C^1 functions in a simply connected domain $D \subset \mathbb{R}^2$ such that

$$\frac{\partial(\Phi h_1)}{\partial u} + \frac{\partial(\Phi h_2)}{\partial v}$$

does not change sign in D and vanishes at most on set of measure zero. Then the system

$$\begin{aligned} \frac{du}{dt} &= h_1(u,v), \\ \frac{dv}{dt} &= h_2(u,v), \end{aligned}$$

has no periodic orbits in D.

Theorem A.2 *(Poincaré-Bendixson)*
Let the functions h_1 and h_2 have continuous first partial derivatives in a domain D of the uv-plane. Let D_1 be a bounded sub-domain in D, and let R be the region that consists of D_1 plus its boundary (all points of R are in D). Suppose that R contains

no critical point of the system

$$\begin{aligned} \frac{du}{dt} &= h_1(u,v), \\ \frac{dv}{dt} &= h_2(u,v). \end{aligned}$$

If there exists a constant t_0 such that $x = \phi(t)$, $y = \psi(t)$ is a solution of the system that exists and stays in R for all $t \geq t_0$, then either $x = \phi(t)$, $y = \psi(t)$ is a periodic solution (closed trajectory), or $x = \phi(t)$, $y = \psi(t)$ spirals toward a closed trajectory as $t \to \infty$. In either case, the system has a periodic solution in R.

Theorem A.3 *[132] Let E be an open subset of $\mathbb{R}^n$ containing $\mathbf{x}_0$. Suppose that $\mathbf{f} \in C^1(E)$ and that $\mathbf{f}(\mathbf{x}_0) = \mathbf{0}$. Suppose further that there exists a real valued function $V \in C^1(E)$ satisfying $V(\mathbf{x}_0) = 0$, and $V(\mathbf{x}) > 0$ if $\mathbf{x} \neq \mathbf{x}_0$. Then (a) if $\dot{V}(\mathbf{x}) < 0$ for all $\mathbf{x} \in E$, $\mathbf{x}_0$ is stable; (b) if $\dot{V}(\mathbf{x}) < 0$ for all $\mathbf{x} \in E \sim \{\mathbf{x}_0\}$, $\mathbf{x}_0$ is asymptotically stable; (c) if $\dot{V}(\mathbf{x}) > 0$ for all $\mathbf{x} \in E \sim \{\mathbf{x}_0\}$, $\mathbf{x}_0$ is unstable.*

Lemma A.1 *(Lasalle Invariance Principle)[133] Assume that V is a Lyapunov function of the dynamical system $\dot{\mathbf{x}} = \mathbf{f}(\mathbf{x})$ on E. Define $S = \{x \in \bar{G} \cap E : \dot{V}(x) = 0\}$. Let M be the largest invariant set in S. Then every bounded trajectory (for $t \geq 0$) of $\dot{\mathbf{x}} = \mathbf{f}(\mathbf{x})$ that remains in G approaches the set M as $t \to \infty$.*

A.2 Auxiliary and standard results of the theory of Partial Differential Equations

Proposition A.1 *(Maximum Principle (Lou and Ni [147])) Suppose that $g \in C(\bar{\Omega} \times R^1)$.*

(i) Assume that $\phi \in C^2(\Omega) \cap C^1(\Omega)$ and satisfies

$$\Delta\phi(x) + g(x, \phi(x)) \geq 0, \quad x \in \Omega; \quad \partial_n \Omega \leq 0, \quad x \in \partial\Omega$$

If $\phi(x_0) = \max_{\bar{\Omega}} \phi$, then $g(x_0, \phi(x_0)) \geq 0$.

(ii) Assume that $\phi \in C^2(\Omega) \cap C^1(\Omega)$ *and satisfies*

$$\Delta\phi(x) + g(x, \phi(x)) \leq 0, \quad x \in \Omega; \quad \partial_n \Omega \geq 0, \quad x \in \partial\Omega$$

If $\phi(x_0) = \min_{\bar{\Omega}} \phi$*, then* $g(x_0, \phi(x_0)) \leq 0$.

Proposition A.2 *(Harnack inequality (Lin et.al [148])) Assume that* $c \in C(\bar{\Omega})$ *and let* $\phi \in C^2(\Omega) \cap C^1(\Omega)$ *be a positive solution to*

$$\Delta\phi(x) + c(x)\phi(x) = 0, \quad x \in \Omega; \quad \partial_n \Omega = 0, \quad x \in \partial\Omega$$

Then there exists a positive constant $C_* = C_*(\Omega, ||c||_\infty)$ *such that*

$$\max_{\bar{\Omega}} \phi \leq C_* \min_{\bar{\Omega}} \phi$$

Proposition A.3 *(Young inequality) For any* $\epsilon > 0, a, b \geq 0$ *and* $m, n > 1$

$$ab \leq \epsilon^{m/n} \frac{a^m}{m} + \frac{1}{\epsilon} \frac{b^n}{n}, \quad \frac{1}{m} + \frac{1}{n} = 1.$$

Proposition A.4 *(Poincare Inequality) [144]*

$$\alpha_1 \int_\Omega (g - \bar{g})^2 dx \leq \int_\Omega |\nabla(g - \bar{g})|^2 dx$$

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Matlab Programs used for numerical simulations

A1: Disease code 1

```
function pdot=DiseasePPSPf(t,p)
% This is the ODE program for the Disease model problem
a=0.0001787;
b=0.009525;
c=0.5;
e=0.0003;
f=0.0004;
l=0.00001705;
m=0.000027;
n=0.0000135;
q=0.0;
r=0.01342;
mu=0.0556;
nu=0.0667;
delta=0.0787;
beta=0.16;
K=200000;
pdot(1,:)=r.*p(1).*(1-p(1)./K)-a.*p(1).*p(2)./(1+e.*p(1))
-b.*p(1).*p(3)./(1+f.*p(1))-c.*b.*p(1).*p(4)./(1+f.*p(1));
pdot(2,:)=l.*p(1).*p(2)./(1+e.*p(1))-q.*p(2).*p(3)-mu.*p(2);
pdot(3,:)=m.*p(1).*p(3)./(1+f.*p(1))-beta.*p(3).*p(4)./p(5)-nu.*p(3);
pdot(4,:)=n.*p(1).*p(4)./(1+f.*p(1))+beta.*p(3).*p(4)./p(5)
-nu.*p(4)-delta.*p(4);
pdot(5,:)=m.*p(1).*p(3)./(1+f.*p(1))+n.*p(1).*p(4)./(1+f.*p(1))
-nu.*p(5)-delta.*p(4);
```

A2: Disease code 2

```
%This is a program to use the disease ODE 'DiseasePPSPf'
clear all
t0=0;
tf=300;
p0=[133000,412,1200,500,1700];
tspan=[t0 tf];
[t,p]=ode45(@DiseasePPSPf,tspan,p0);
subplot(1,2,1)
plot(t,p(:,1)/133000,t,p(:,2)/412,t,p(:,3)/1200,t,p(:,4)/500,'k-','LineWidth',2);
grid on
xlabel('Time (years)');
ylabel('Population');
legend('Impala','Cheetah','Total Lion','Infected Lion','Location','Best');
axis( [0, 300, 0, 1.2] )
subplot(1,2,2)
plot3(p(:,1)/133000,p(:,2)/412,p(:,3)/1200,'k-','LineWidth',2)
grid on
title('Phase Portrait')
xlabel('Impala')
ylabel('Cheetah')
zlabel('Total Lion')
axis( [0, 1, 0, 1, 0, 1.2] )
% plot(p(:,2)/412,p(:,3)/1200,'k-','LineWidth',2)
% grid on
% title('Phase Portrait')
% xlabel('Cheetah')
% ylabel('Susceptible Lion')
```

B: Diffusion code

```
%Solve a Reaction-Diffusion Predator Prey system of equations in 2-D space
%over time. Apply Forward Euler's Method for time integration and second-order
%central difference approximation to evaluate the spatial derivatives.

%clear all

%Grid size
Tf=10000;
a=0;                        % Right boundary
b=4;                        % Upper boundary
M=100;                      % M is the number of spaces between a and b.
dx=(b-a)/M;                 %(b-a)/M; % dx is delta x
dy=(b-a)/M;                 %(b-a)/M;
x=linspace(a,b,M+1);        % M+1 equally spaced x vectors including a and b.
y=linspace(a,b,M+1);

%Time stepping
dt=0.08;                     %100*(dx^2)/2;     % dt is delta t the time step
N=Tf/dt;                % N is the number of time steps in the interval [0,1]

%Constant Values

d2=0.01;                     % d2 is the Diffusion coefficient Dv/Du

alpha=0.0021;
mu1=320;
beta1=60;
delta1=4.97;

%pre-allocation
unp1=zeros(M+3,M+3);
vnp1=zeros(M+3,M+3);

```

```
%Initial Conditions
un=0.5+rand(M+3,M+3);       %Begin with a random point between [0,2]
vn=0.5+rand(M+3,M+3);

for n=1:N

    for i=2:M+2
        un(i,1)=un(i,3);    %Boundary conditions on bottom flux is zero
     un(i,M+3)=un(i,M+1);   %Boundary conditions on top
     vn(i,1)=vn(i,3);
     vn(i,M+3)=vn(i,M+1);
    end
    for j=2:M+2
        un(1,j)=un(3,j);    %Boundary conditions on left
     un(M+3,j)=un(M+1,j);   %Boundary conditions on right
     vn(1,j)=vn(3,j);
     vn(M+3,j)=vn(M+1,j);
    end

    for i=2:M+2
        for j=2:M+2

            %Force function for u and v
            frcu=un(i,j)*(1-un(i,j))-un(i,j)*vn(i,j)/(1+beta1*un(i,j));
            frcv=mu1*un(i,j)*vn(i,j)/(1+beta1*un(i,j))-delta1*vn(i,j);

            Lapu=(un(i-1,j)+un(i+1,j)+un(i,j-1)+un(i,j+1)-4*un(i,j))/dx^2;
%Laplacian u
            Lapv=(vn(i-1,j)+vn(i+1,j)+vn(i,j-1)+vn(i,j+1)-4*vn(i,j))/dx^2;
%Laplacian v

            unp1(i,j)=un(i,j)+dt*(alpha*Lapu+frcu);
            vnp1(i,j)=vn(i,j)+dt*(d2*alpha*Lapv+frcv);

        end
```

```
67      end
68      un=unp1;
69      vn=vnp1;
70
71      %Graphing
72      if mod(n,31250)==0
73
74          subplot(1,2,1)
75          hdl = surf(x,y,un(2:M+2,2:M+2));
76          set(hdl,'edgecolor','none');
77          axis([0, 4, 0, 4]);
78          title('u - Impala');
79          %caxis([-10,15]]);
80          view(2);
81          colorbar;
82          subplot(1,2,2)
83          hdl = surf(x,y,vn(2:M+2,2:M+2));
84          set(hdl,'edgecolor','none');
85          axis([0, 4, 0, 4]);
86          title('v - Cheetah');
87          %caxis([-10,15]]);
88          view(2);
89          colorbar;
90          fprintf('Time t =%f\n',n*dt);
91          ch = input('Hit enter to continue :','s');
92          if (strcmp(ch,'k') == 1)
93              keyboard;
94          end
95      end
96
97  end
```