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Prey, predator and super-predator model with disease in the super-predator

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ABSTRACT

The dynamics of a predator–prey model with disease in super-predator are investigated. The predator is under immense competition from the super-predator and is also facing extinction. The disease is considered as biological control to allow the predator population to regain from a low number. The results highlight that in the absence of additional mortality on predator by super-predator, the predator population survives extinction. 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 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 of 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.

1. Introduction

The sizes of species populations are important in 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 [1]. The study of transmissible diseases within an ecological setting is gaining momentum [2]. It is becoming biologically relevant to include the effects of diseases in studies on the behaviour of dynamical ecological systems [3].

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 [2]. Lu et al. [4] proposed a model for hunting strategies of predators in susceptible and infected prey. Their results show that predation is a function of the strength of the disease. When the disease is slightly endemic, predators target the susceptible prey, but switch to infected prey when the disease becomes heavily endemic. Chakraborty et al. [5] studied a prey–predator eco-epidemiological system on disease persistence and extension perspective. They outlined a criterion for describing uniformly strong persistence of the system. The disease affected the prey. Their results indicate that the disease incidence rate has a potential to

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destabilise a once stable system. Sahoo and Poria [6] studied the diseased prey–predator model with general Holling type interactions. Their model involved prey, an intermediate predator, and a top predator. They investigated different types of general Holling interactions. Their findings indicate that by properly choosing interaction functions, a diseased system can be transformed into a disease-free system, thereby controlling the disease. Hethcote et al. [7] studied a predator–prey model with Susceptible–Infective–Susceptible (SIS) disease dynamics in the prey. The infected prey was more vulnerable to predation. They showed that some parameter values increased the risk of predation of infected prey, thereby ensuring that the predator population persists. Otherwise the predators would become extinct in the absence of disease. Furthermore, they showed that the increased predation of vulnerable infected prey would allow the disease to die out, apart from the disease remaining endemic in the absence of predators.

Mukhopadhyay and Bhattacharyya [3] 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 depended 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 shaped 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, determined the dynamical behaviour of the system. Haque et al. [8] investigated an eco-epidemiological predator–prey model with disease in prey. They showed that a virulent disease in the prey may allow predators to escape extinction but destabilises a once stable system. Xiao and Chen [9] proposed a predator–prey model with disease in prey. Their model shows 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.

Sahoo and Poria [10] studied the effects of additional food on an eco-epidemic model with time delay on infection. The prey had a parasitic infection. Additional food was provided to predators as a means of controlling its population, and ultimately the parasitic infection prey. Their results show that the system becomes disease free under provision of additional food to predators. Predator population increases thereby enhancing the consumption of diseased prey. Sahoo [11] investigated the role of additional food provided to predators in an eco-epidemiological system with disease in prey. An optimal control problem was formulated and solved to determine the control of disease. The results show existence of a critical infection rate above which disease free system is not reachable without additional food. However, additional food enables a disease free system to be attained up to a certain infection level.

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 [2] studied the predator–prey model with SIS parasitic infection spreading through the predator species only. It was shown that infection in the predator species may save the prey from extinction even if the basic reproduction number for the prey to be able to invade the predator-only equilibrium, was less than one. Pal et al. [12] investigated a predator–prey model with disease present in predator species only. They showed that for some values of the predation rate all species could be saved from extinction, and that the disease did not propagate in the predator population. Han et al. [13] 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 showed that when the disease persists in the prey population and the predators feed sufficiently to survive, the disease also persists in the predator population.

In this study, the model proposed is an extension of Haque [2] and Venturino [14] models to include an infectious disease spreading through a super-predator species. The study area is Kruger National Park (KNP), South Africa's largest wildlife reserve with an area of approximately 20,000 km^2. The park consists largely of woodland savanna. 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 [15,16]. However, it is the third dominant prey species consumed by the lion after zebra and wildebeest in the KNP [17]. 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 [18]. Bovine Tuberculosis (BTB) disease, caused by *Mycobacterium bovis*, is present in the lion population of KNP [19,20]. 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 [19,21]. An 80% disease prevalence in the lion population occupying the southern part of the KNP was reported in the year 2000 [19,22]. 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 [23].

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.

2. Model formulation

2.1. Assumptions

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.

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