The evolution on eco-epidemiological systems theory and evidence

N. Bairagi[1] and J. Chattopadhyay[2*]

[1] Centre for Mathematical Biology and Ecology, Department of Mathematics, Jadavpur University, Kolkata, 700032, India,

E-mail: nbairagi@math.jdvu.ac.in

[2] Agricultural and Ecological Research Unit,Indian Statistical Institute, 203, B. T. Road, Kolkata 700108, India

E-mail: joydev@isical.ac.in

Abstract In this article, we have briefly reviewed the development of eco-epidemiology since its initiation about two decades before. Different studies of eco-epidemiological models reveal that a predator-prey system in presence of parasites may exhibit complex but biologically relevant dynamics including switching of stability, extinction and oscillations or even unpredictable dynamics under some parametric conditions. This article also points out some important aspects that should be addressed with great importance.

1. Introduction

Ecology and epidemiology are major fields of study in their own right, but there are some common features between these systems and merging the two may show interesting dynamics. Eco-epidemiology is a new branch in mathematical biology which considers both the ecological and epidemiological issues simultaneously. The first breakthrough in modern mathematical ecology was done by Lotka [50] and Volterra [79] for a predator-prey competiting species. On the other hand, most models for the transmission of infectious diseases originated from the classic work of Kermack and McKendrick [45]. After these pioneering works in two different fields, lots of research works have been done both in theoretical ecology and epidemiology. Anderson and May [3] were the first who merged the above two fields and formulated a predator-prey model where prey species were infected by some disease. In the subsequent time many authors proposed and studied different predator-prey models in presence of disease, and Chattopadhyay and Arino [12] coined the name eco-epidemiology for the study of such systems.

Ecologists are now taking into account the importance of parasites not only on individual hosts but also on their population dynamics and community structure [61, 63]. Understanding how parasites affect biodiversity and ecosystem dynamics is now a central question in conservation biology [23, 36, 61] as infectious disease can be a factor in regulating host population. On the other hand, successful invasion of a parasite into a host population and resulting host-parasite dynamics can depend crucially on other members of a host's community such as predators [28]. Predation can

* To whom any correspondence should be addressed.

dramatically shape community structure and ecosystem properties [71] and becomes particularly interesting in host-parasite systems because predation itself can strongly alter population dynamics of hosts and parasites [19, 38, 40, 65]. Predators may even prevent successful invasion of parasites into host population. Although, in most theoretical studies of host-parasite-predation interactions, predator behavior is simplified and isolated from an ecosystem [65].

2. Empirical and Experimental Evidences

There are many examples where the two hosts are in a predator-prey relation and where the predation itself transmit the parasite from the prey to the predator. In many cases the parasite modifies the external features or the behavior of the prey so as to make infected individuals more vulnerable to predation. In a review article Holmes and Bethel [35] have collected many examples of such modification. Lafferty and Morris [53] observed that killifish would tend to come closer to the sea surface on contracting a disease this makes them more vulnerable to predation by birds. They stimated that predation rate of infected prey may be 31 times higher than that of susceptible prey. Williams [80] observed the same behavior in the case of sticklebacks when it is infected by plerocercoids. The Asian catfish species C. batrachus and C. macrocephalus infected by cracked skull disease make them more vulnerable to predation [43]. Wolf attacks on moose are often successful if the moose is heavily infected by Echinococcus granucosus [68]. Hudson and his colleges [37, 38] showed that predators captures a disproportionately high number of red grouse when infected with parasites. In Salton Sea of California, fish population, infected from both the vibrio and botulism, swim so slowly that they become an easy catch for piscivorous birds. Predatory birds are infected in turn when they consume heavily infected fish [24, 41, 44].

3. Factors that have been considered in different eco-epidemiological models and their effects

An eco-epidemiological model is a combination of a predator-prey model and an epidemic model of any type (SI or SIS or SIRS). The prey population or predator population is assumed to be infected by some parasites (micro or macroparasitie). The infected population is subdivided into different classes viz. susceptible, infected or removed from the infection. An eco-epidemiological model is then formulated with some ecological and epidemiological assumptions which are situation specific. Most of the models so far studied for a predator-prey-parasite system consider microparasitic infection [3, 7, 12, 13, 14, 21, 22, 27, 28, 33, 64, 65, 76, 77, 81] and there are very few models that consider macroparasitic infection [18, 21, 38, 65]. There are some fundamental differences in mathematical model formulation for micro and macroparasitic infections. For macroparasitic eco-epidemiological model it is more important to model the number of parasites, since the severity of the disease depends on the total parasitic load. In contrast, for microparasitic eco-epidemiological model it is only considered whether an individual is susceptible or infected or recovered form the disease. The epidemiological parameter R0, the basic reproduction ratio or basic reproductive number of infection, is also ubiquitous in eco-epidemiological models and determines whether a disease will spread (if $R0 < 1$) or die out (if $R0 < 1$) [4, 26].

Anderson and May [3] first studied a Lotka-Volterra predator-prey model in presence of a disease.Assuming that infection causes castration and increases predation rate, they observed that predator-prey system destabilize in presence of parasite which is otherwise stable. Hadeler and Freedman [27] modified the Rosenzweig predator-prey model where the prey is infected by a parasite, and the prey in turn infects the predator with the parasite. They obtained a threshold value above which an infected equilibrium or even an infected periodic solution may appear. In another closely related problem, they showed that predators could only survive on the prey if some of the prey were caught more easily due to infection. Venturino [76] proposed a two dimensional predator-prey model and studied how the presence of the disease among the prey affects the behavior of the model. He concluded that under suitable assumptions the disease might act as a control for the system. In an another study [77], he showed that neutral stability of a Lotka-Volterra system is also carried over to the new system when a disease spreads only

among predators and prey, unaffected by the disease, grows logistically. Chattopadhyay and Arino [12] proposed a three species eco-epidemiological model and studied its local stability, extinction and hopf- bifurcation. Chattopadhyay and Bairagi [13] proposed and studied an eco-epidemiological model (SI type) of Salton Sea with the assumption that predator consumes infected prey only. They observed that the system is stable around the positive interior equilibrium if the search rate level of predator is low, but the instability sets in when search rate level increases. Han et al. [29] analyzed four eco-epidemiological models (SI and SIR types) with standard and mass action incidences. Different thresholds were identified and global stability results were proved. It was shown that disease might persist in the prey and predator populations if the basic reproduction for prey population is above some threshold and feeding efficiency of the predator population is significantly high. Chattopadhyay et al. [15] showed that there always exists a hopf bifurcation for increasing transmission rate in a classical predator-prey system with infection in prey population. Fenton and Rands [21] studied two eco-epidemiological models where prey is infected by either microparasite or macroparasite. They showed that prey manipulation by parasites can greatly alter the quantitative dynamics of the community, potentially resulting in high amplitude oscillations in abundance, but the precise outcome of the interaction depends on both, the form of manipulation and the nature of the predator's functional response. The dynamics of the macroparasite model were qualitatively similar to those seen for simpler microparsite model with only exception that predator-prey-parasite community might persist with cyclical dynamics in a larger region of parameter space. Packer et al. [65] studied eco- epidemiological models for microparasitic and macroparasitic infection with constant predator and showed that predator removal was more likely to be harmful when the parasite was highly virulent. Simulation studies of Zhdanov [83] using Monte Carlo technique revealed that predators might play a constructive role in suppression of infection among preys provided extinction of infected preys due to predator was more probable than that of healthy preys. Hethcote et al. [33] modified a predator-prey model with an SIS disease in the prey where infected prey was more vulnerable to predation. They observed that the endemicity of the disease and the greater vulnerability of the infected prey allow the predator population to persist, when it would have become extinct without the disease. Haque and Venturino [32] modified the classical Holling Tanner model allowing a disease to spread among the prey species. They showed that introduction of a disease in a pure demographic model can destabilize the otherwise stable system and sometimes the predator might act as a system preserver. How disease affect symbiotic communities was studied by Venturino [78] and showed that disease might have a positive effect on the environment. An eco-epidemiological model can also exhibit interesting dynamics when different situation specific ecological or epidemiological assumptions are considered.

When infection is also fatal to predator

It is sometimes observed that predators may have to pay a cost in terms of extra mortality in the trade-off between the easier predation and the parasitized prey acquisition, but the benefit is assumed to be greater than the cost [35, 51]. In most of the previous works it was assumed that consumed prey (both susceptible and infected) contribute positive growth to the predator population. The case, consumption of infected prey contribute negative growth to predator population, was studied by Chattopadhyay et al. [16] and observed that coexistence of all three populations (susceptible prey, infected prey and predator) was never possible. They established conditions for which the considered predator-prey system with disease in the prey species would eventually be disease free and suggested that suitable impulsive harvesting strategies might play a crucial role to achieve this goal.

Is there any chaos?

Existence of chaos in model ecological system was observed by many researchers [9, 25, 46, 57, 72]. McCane and Yodzis [62] commented that productive environment was a pre-requisite for a

system to support a chaotic dynamical behavior. On the other hand, birth rate [20], contact rate [70], age structure [11] may be considered as possible parameters to elucidate the onset of chaotic behavior in an epidemiological model. Therefore, the question is Do increased carrying capacity, infection rate or other system parameters produce unpredictable dynamics in an eco-epidemiological system? To search the possible answers of these questions, Upadhyay et al.[75] studied numerically the eco-epidemiological model of Chattopadhyay and Bairagi [13] for larger ranges of different system parameters. Their simulation results suggest that an eco-epidemiological system may observe different interesting dynamical behavior starting from stable focus/stable limit cycle to chaos. It was observed that the intrinsic growth rate of the prey population, the carrying capacity of the environment and the half-saturation constant of the predators' functional response were the key parameters responsible for the chaotic behavior of the system, whereas no chaos was found for other parameters of the considered system. The system supports stable limit cycles or stable focus in reasonably larger parameter regimes, where as chaotic behavior was exhibited, intermixed with stable limit cycles, in narrow parameter regimes. They commented that as chaos occurs in a narrow range of parameters, it might not be observed frequently in natural systems. Since higher values of intrinsic growth rate of the prey population and the carrying capacity of the environment were required to observe chaotic behavior, so harvesting of prey population and nutrient extraction might reduce the possibility of chaos in such systems.

Effect of harvesting

It is well known that harvesting dampen fluctuations by making overcompensatory dynamics undercom- pensatory and reduce the equilibrium density of hosts. The host-parasite interaction exhibits oscillations and this phenomena is also carried over to a host-parasite-predator interaction [13, 21, 27, 28, 38, 77]. Also, oscillatory population may be driven to extinction in presence of environmental stochasticity when the population density is very low [21, 42]. Therefore, the question is – how to control these oscillations if it arises in such eco-epidemiological situations? Can harvesting regulate the cyclic behavior, if it exists, of the system populations? One of the main objectives of mathematical epidemiology is possibly to de- vise the controlling strategies. Under what parametric conditions harvesting can eliminate parasites in an eco-epidemiological system, therefore, becomes a pertaining question. Bairagi et al. [6] tried to find out the possible answers of these questions. Their analytical and numerical studies reveal that harvesting has the potential significance for community structure and biodiversity, in particular their role in removing a parasite or coexistence or species exclusion and even in preventing population oscillations.

Role of delay

Models with delay are much more realistic, as in reality time delays occur in almost every biological situation [60] and assumed to be one of the reasons of regular fluctuations in population density [58]. Reproduction of predator after consuming the prey is not instantaneous, but mediated by some time lag required for gestation. Xiao and Chen [81] studied a three-dimensional eco-epidemiological system with this gestation delay. They observed that when the coefficient in conversing the prey into predator is independent of delay then the system is stable up to some critical value of the delay parameter, and unstable if it crosses the critical value. They concluded from their simulation results that time delay has both destabilizing and stabilizing effects if the conversion parameter depends on the delay. How the qualitative and quantitative behaviors of an eco-epidemiological system alter with the intracellular delay, which represents the time between the two events viz. the first effective contact between susceptible and infected prey and the newly infected prey becomes productively infectious, was studied by Bairagi et al. [8]. It was observed that for lower infection rate the system was stable for all delay, but for higher infection rate there was a threshold value of the delay above which the system was unstable and below which the system was stable leading to the persistence of all the species. They have also shown that the instability arising from the intracellular delay might be controlled if somehow the growth rate of predator population could be increased.

Does predators' response function matter?

Many biological factors ought to alter the form of predator's functional response and thereby alter the dynamics of the predator and prey populations. Since functional response encapsulates attributes of both predator and prey biology, so handling time, search efficiency, encounter rate, prey escape ability etc. should alter, in general, the functional response [2, 9]. Furthermore, structure of the prey habitat is also responsible to alter the functional response [2]. Bairagi et al. [7] proposed and analyzed a mathe-matical model of an infected predator-prey system with different predators' functional response viz. liner mass action, type II and type III. They obtained diverse array of biologically relevant behavior, including switching of stability, extinction and oscillations by varying two key parameters viz. the rate of infection (an epidemiological parameter) and the attack rate on susceptible prey (an ecological parameter). It was observed that the parameter regions for the asymptotic stability of different feasible equilibrium increased as one passed from linear to type II through III, except one where susceptible prey and predator population coexisted. In the last case type III response function has larger stability regions compare to other two for both lower and higher infection rates. They observed that in Holling type II & III species coexist in a stable form with enhanced predator population and depressed prey population levels, while in linear mass action response predator and prey population both are depressed, which contradicts the so-called biological control paradox [56]. It was also commented that the general view regarding predators' increased search rate is to increase predator population is not inevitable in an eco-epidemiological model.

An eco-epidemiological model with ratio-dependent functional response shows richer boundary dynamics, makes the behavior of epidemic models more complex [82] and can also send all population to extinction in a deterministic way [31, 39, 47, 48, 82]. All topological structures near the origin starting from any position of the domain of interest were studied by Arino et al. [5]. They showed that introduction of infected population in the classical ratio-dependent predator-prey model might act as a biological control to save the population to extinction.

Linear vs. non-linear incidence rate

A admittedly debatable phenomena in eco-epidemiology lies in the incidence rate. In standard eco- epidemiological models, the incidence rate or the rate of new infection is linear mass action law, which assumes homogeneous mixing of susceptibles and infectives, and is given by $\lambda I S$, where S and I are respectively the numbers of susceptible and infected populations per unit area, and $\lambda > 0$ is the transmission rate or force of infection. When non-linear incidence rate $\lambda I^p S^q$, where p and q are positive parameters and are interpreted to represent how the densities of susceptible and infected hosts may affect the per capita transmission efficiency of the parasite, is considered then the system can have a much wider range of dynamical behaviors [34, 55, 73]. In contrary, the result of Bertheir et al. [10] shows that mass action incidence is more appropriate when transmission occurs through direct contact, and De Jong et al. [17] showed that there were no change in the qualitative properties upon the contact process, whether it fol- lows the law of mass action or proportional mixing rate. The effect of non-linear incidence rate on the eco-epidemiological model of Chattopadhyay and Bairagi [13] was studied by Haque and Chattopadhyay [30]. They determined different conditions on p and q so that the system enters into hopf bifurcation and determined the stability and direction of the bifurcating branches. Venturino [76, 78], Han et al. [29], Chattopadhyay and Pal [14] and Pal et al. [66] studied eco-epidemiological models with standard incidence and obtained some distinguishable results. In particular, Chattopadhyay and Pal [14] observed that there is a possibility for the coexistence of the species when the contact rate follows the law of mass action. But if the contact rate follows the law of standard incidence a minute amount of infection can destabilize the system. They concluded that the behavior of such systems is very much model dependent.

3. Concluding remarks

In this article, we have briefly reviewed the development of eco-epidemiology. Some behaviors of an eco- epidemiological system under different biological conditions have become known in the last few years of studies. However, the understanding is far from complete and there are many cavities that should be considered in depth. No previous work has considered the evolutionary aspects in an eco-epidemiological system. Though evolution theories suggest that evolution is capable to significant evolutionary change in a predator-prey interaction [1], virulence and transmission rates [59]. So the pertaining questions is How does prey evolution or predator evolution or coevolution affect a predator-prey-parasite system? Can evolution produce (or dampen) cycles when it does not occur (or does occur) in absence of evolutionary change? Does evolutionary stabilization or destabilization occur in such situations? If so, how and to what extent? Many environmental factors such as eutrophication and thermal effluent often raise infection rate because the associated increased productivity can increase the abundance of host population [52]. Pollutants may also affect adversely by increasing host susceptibility and fecundity [26]. Periodic changes in temperature, rainfall, humidity are also natural phenomena of environment. These variations may have significant effects on epidemiological parameters viz. infection rate, mortality rate of infected hosts etc. and may perturb the system dynamics. So the pertaining question is How an eco-epidemiological system will response to periodic environmental change? What are the other important scenarios that may arise from environmental changes? It has been suggested that spatial heterogeneity (e.g. age structure, immigration or diffusion) may address many of the deficiencies of epidemic models [49, 54] and therefore can be incorporated into a basic eco-epidemiological model. Thus, the future study of eco-epidemiological models with spatial heterogeneity, environmental stochasticity, seasonality may be exciting and very challenging.

Although parasites are biologically and ecologically important in ecosystem and significantly affect the trophic interaction, productivity and stability of the ecosystem, few quantitative data are available on this topic [74]. In order to better understanding of various eco-epidemiological phenomena associated with the living world extensive field studies and laboratory experiments are essential along with realistic mathematical model analysis.

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