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Review

Eco-epidemiological predator–prey models: A review of models in ordinary differential equations

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ABSTRACT

Eco-epidemiology integrates ecological and epidemiological approaches to analyze both the impact of infectious diseases on ecological communities and how interspecific interactions can alter disease dynamics. With the aim of extracting general principles of eco-epidemiological dynamics, this article presents a review of the literature focusing on predator–prey type ordinary differential equation models with disease in one of the species. We included 81 articles that were categorized according to prey growth function, disease transmission function, epidemiological model compartments, and predator functional response. The findings reveal that these models share a common mathematical lineage, which in turn facilitates the construction of models based on the general assumptions identified in this study. The most prevalent models tend to assume logistic prey growth, a bilinear incidence rate for disease transmission, an epidemiological model of the Susceptible–Infected type, and a Holling Type II predator functional response.

1. Introduction

Ecology and epidemiology are two important fields of research in mathematical biology (Mackey and Maini, 2015). Cornerstone mathematical models were established by Lotka and Volterra for studying species interactions (Smith et al., 2004), and by Kermack and McKendrick for studying the spread of diseases (Diekmann et al., 1995). Actually, it is acknowledged the need to study host-pathogen interactions at different ecological levels such as populations and communities (Su, 2015). For instance, predation can change dynamics of an epidemiological system and the presence of a pathogen in a predator–prey system can produce more complex dynamics that include changes in the conditions of extinction and coexistence (Chattopadhyay and Bairagi, 2001; Bairagi et al., 2009). The discipline that links ecology and epidemiology is termed eco-epidemiology (Bate, 2013).

One approach to study the dynamics and outcomes of eco-epidemiological systems is to use models expressed as ordinary differential equations. In the last three decades, several publications have studied different scenarios. Anderson and May published the first work in eco-epidemiology in 1986; they used Lotka–Volterra-submodel for the predator–prey system and formulated a model that included a horizontally transmitted disease in the prey and a model where they included the disease in the predator (Anderson and May, 1986). On their part, Hadeler and Freedman in 1989 modeled the phenomenon of a disease that is transmitted trophically (Hadeler and Freedman, 1989). These two publications marked the beginning of eco-epidemiological compartmental models, leaving a conceptual basis to be used in the following years. During the 1990s, Chattopadhyay and Venturino, separately, provided several articles with their respective collaborators, among which are Venturino (1994), Chattopadhyay and Arino (1999a) and it was precisely Chattopadhyay and colleagues, in 1999, who first coined used the eco-epidemiology concept, so after the year 2000, several articles are registered using this term (Xiao and Chen, 2001; Haque, 2007; Haque et al., 2009; Stiefs et al., 2009).

Classical eco-epidemiological systems are composed of two species, namely the prey and the predator. Generally, the trophic level in which the disease occurs is divided in compartments corresponding to its

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health status (usually susceptible and infected), and the other trophic level is aggregated to the system as a single compartment. Some examples of these systems can be found in Das (2016), Sharma and Samanta (2015), Das et al. (2019), Jang and Wei (2020) and Kumar et al. (2020a). These systems have three key elements: prey growth, transmission function, and predator functional response. The prey growth function determines the abundance of the resource for predators, which increases in importance if the predators are specialists (Sahoo, 2012). In particular, when the disease is present in the prey population, it is discussed whether or not the infected have offspring and how it affects growth (Haque, 2007; Zhang and Yeung, 2012). The choice of mathematical functions for modeling population growth primarily revolves around two main options: exponential and logistic functions, with the selection contingent upon the specific factors under examination. Exponential growth depends only on population size, while logistic growth apart from this, depends on intraspecific competition and resource availability (Gotelli et al., 2008).

The transmission function is a determining factor in disease dynamics (Hopkins et al., 2020). In the eco-epidemiological model, it can affect the predator–prey system (Hethcote et al., 2004; Hassan et al., 2021). In Roy et al. (2019) and Das et al. (2019), it was observed that the introduction of disease within communities leads to the destabilization of the model dynamics. In particular, in Das et al. (2019), they showed that a lethal disease in the prey and a predator population could not coexist. This very concept is explored in the article (Siekmann et al., 2010), in which it is shown that the key to understanding the dynamics of a predator–prey model with prey disease is to study the system as the competition between the predator and the infection, i.e., if the infection proves excessively lethal to the prey, it may drive specialist predators to extinction due to food scarcity. Conversely, if the predators are overly efficient at hunting, they can decimate the prey population so that the infection cannot effectively propagate.

The choice of the transmission function plays a crucial role in determining the outcomes of eco-epidemiological models (Siekmann et al., 2010). Two commonly used functions are the bilinear and standard incidence rates. The critical characteristic of bilinear incidence is its dependence on population density. In other words, the more individuals are infected, the higher the probability of contracting the disease. In contrast, the standard incidence rate depends on contact frequency. In this case, the number of infected individuals does not matter; what matters is the number of contacts between susceptible and infected individuals (Han et al., 2001; De Jong et al., 1994).

The bilinear incidence rate has often been used in eco-epidemiological models because it allows for a more direct comparison of qualitative results with the classical predator–prey model (see, for example,Xiao and Chen (2001), Mukhopadhyay and Bhattacharyya (2009), Sarwardi et al. (2011b), Chakraborty et al. (2015), Sarwardi et al. (2011a)). On the other hand, some researchers opt for the standard incidence rate (Sasmal et al., 2015; Haque et al., 2009). The choice of the standard incidence rate is driven by achieving a more precise approximation under a substantial number of infected individuals. However, it is essential to note that using the standard incidence rate introduces greater complexity to qualitative analysis (Pal et al., 2006). Nevertheless, this additional complexity can lead to expanded stability regions for coexistence equilibria within eco-epidemiological models. This, in turn, allows to model a reduce the risk of species extinction, as demonstrated in the article (Chatterjee et al., 2007).

The predator functional response describes the interaction between predator and prey, playing a vital role in characterizing the dynamics of an eco-epidemiological system (Chatterjee et al., 2006). In some cases, it is assumed that disease does not affect predation, e.g., in Sasmal et al. (2015), the disease only affects the prey, but the predator does not distinguish between susceptible and infected prey. Likewise, selective predation has been studied. In some cases, predators would expressly seek out infected ones, as these are weaker individuals, while in other situations, they may discard diseased ones, as they are less palatable (Hethcote et al., 2004; Roy and Chattopadhyay, 2005; Das et al., 2009; Venturino, 2010). If the disease is present in predators, a similar situation may occur, that the predator is affected because of the disease and cannot hunt optimally (Das, 2016; Krishchenko and Starkov, 2020), or simply that predation is not related to its epidemiological status (Auger et al., 2009; Chevé et al., 2010). Thus, in the context of eco-epidemiological models, it is crucial to consider the different types of existing functional responses, as the various feeding strategies adopted by predators not only influence predator–prey interactions but also significantly impact disease dynamics. This allows us to conclude the effect of predation on the spread of a disease, which is particularly relevant in zoonotic disease research. Specifically, it can enable us to understand better what happens before a pathogen is transmitted to humans (Gómez-Hernández et al., 2023).

The most widely recognized functional responses in ecology are categorized as Holling type I, II, and III. In the case of the Holling type I functional response, the predator consumption rate rises linearly with an increase in prey density. This indicates that as the prey density grows, the predator proportionally consumes more prey, and as such, its consumption rate is constrained solely by the availability of prey. In contrast, within the Holling type II functional response, the predator's consumption rate initially climbs with rising prey density, but subsequently levels off as prey density continues to increase. This suggests that predators may ultimately reach a maximum limit in their consumption capacity. Lastly, in the Holling type III functional response, the predator consumption rate exhibits a slow initial increase, followed by a rapid acceleration as prey density rises, culminating in stabilizing the consumption rate (Smith et al., 2004; Gotelli et al., 2008; Kang et al., 2014).

In light of this, we reviewed eco-epidemiological models in ordinary differential equations that study the dynamics of a disease in a two-level food chain. We want to highlight the fundamental components necessary for embarking on the initial stages of learning about eco-epidemiological models. Therefore, the aims of this review are (i) to provide an overview of the functions used for both prey growth, disease transmission and predator functional response and (ii) to determine the analytical techniques used to explain the biological phenomena described by these mathematical models.

2. Methods

A literature review was made to determine modeling practices in eco-epidemiology by which articles published between 1986 and 2021 were selected. To conduct our search, we utilized the Google Scholar and the search terms were as follows: "Predator–Prey" AND ("Modeling" OR "Model" OR "system") AND ("eco-epidemiological" OR "eco-epidemiology" OR "eco-epidemic") AND ("Disease" OR "Infection" OR "Parasite" OR "Infectious" OR "epidemiological" OR "epidemic").

- We employed the specific term "Predator–Prey" to focus our research on the main topic.
- We included the possibility of finding at least one of the following keywords: "Modeling", "Model", or "system". This allows us to encompass various approaches related to modeling.
- We also aimed to find at least one of the following keywords: "eco-epidemiological", "eco-epidemiology", or "eco-epidemic". This enables us to explore aspects related to ecology and epidemiology within our study context.
- Furthermore, we searched for at least one of the following keywords related to diseases and epidemics: "Disease", "Infection", "Parasite", "Infectious", "epidemiological", or "epidemic".

It is important to note that quotation marks around search terms indicate that we are looking for documents that contain precisely those words or phrases. The "OR" operator allows us to broaden our search by including at least one of the keywords within parentheses.

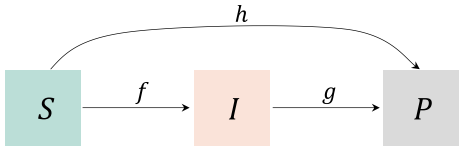

Fig. 1. Schematic diagram of a predator–prey system with disease in the prey.

In contrast, the "AND" operator connects and combines these different aspects in our search strategy.

Studies were excluded if they provided insufficient information to assess differences in mathematical modeling approaches. Additionally, editorials, case reports, review articles, and news reports were intentionally excluded. To ensure the relevance of the included studies, a quality assessment was conducted based on the following criteria:

- Do they propose a model addressing predator–prey dynamics in the presence of diseases affecting one of the populations (predator or prey)?
- Do they employ a model based on ordinary differential equations?
- Is the mathematical model derived from a suitable conceptual framework for investigating an eco-epidemiological phenomenon?

The identification, selection, eligibility, and inclusion process is summarized in Figure 1 of the Supplementary Material.

3. Results

The following results used a selection of 81 articles reviewed as follows. From the total papers, 62 papers did consider disease in the prey and 16 from this works did consider disease in the predator (Supplementary Table 1).

3.1. Disease in the prey species

Among the 62 articles that focus on a predator–prey system with disease in the prey, 4 includes the Susceptible–Infected–Susceptible (SIS) scheme, one consists of the Susceptible–Exposed–Infected (SEI) scheme, and 36 articles include a Susceptible-Infected (SI) scheme (see Supplementary Table 1). The dynamics can be represented basically by the scheme of Fig. 1, where S is the population of susceptible prey, I is the population of infected prey, P is the population of predators, the functions h and g are the functional responses of the predator and f the function of transmission of the disease. Fig. 1 is valid when the disease is type SI or SIS. In case it is type SEI, as in the situation of Venturino (2010), we must add another compartment for the exposed class.

Fig. 2 shows the number of articles as a function of the mathematical expression used in the models to describe the growth rate of the prey. The studies used linear, exponential, and logistic growth rates. Most articles used logistic growth expressed as $rS(1-(S+I)/k)$, where r is the growth rate, k is the carrying capacity, S and I are the susceptible and infected prey respectively. Here the infected do not reproduce. They only exert interspecific competition. Another mathematical expression to describe logistic growth is $r(S+I)(1-(S+I)/k)$. In this type of model, the infected do have susceptible offspring. Within these categories, we have also found articles such as Chakraborty et al. (2015), where expressions such as $rS(1-(S+pI)/k)$ are used to differentiate the competitive difference to susceptible and infected.

The x-axis in Fig. 3 represents the different types of predator functional response, h and g, according to the scheme in Fig. 1. These are further grouped according to the transmission function used, f in Fig. 1. The y-axis in Fig. 3 is the number of articles with the characteristics described above. Generally, the h and g functions are the same in a model. In the review, the type I functional response has the form $h(S,P) = cSP$ and $g(I,P) = cIP$, where c is the capture rate. For

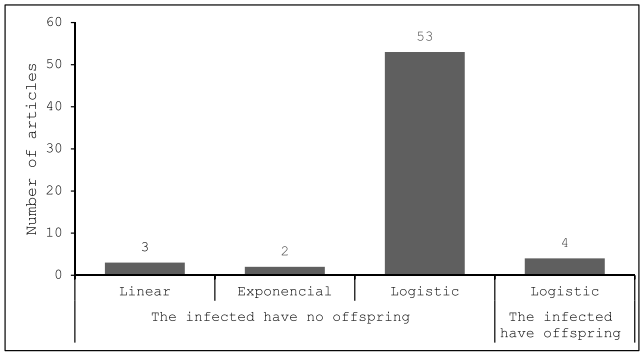

Fig. 2. The number of articles seperated according to the used function for prey growth, further distinguishing cases where infected prey have no offspring from cases with offspring from infected.

functional response type II, some authors consider the total population, i.e. $h(S,I,P) = cSP/(A+S+I)$ and $g(S,I,P) = cIP/(A+S+I)$, where A is the half saturation constant, to describe that predators can feed on both susceptible and infected prey. Other authors assume that h or g is zero, because predators feed on either susceptible or infected prey, but not both at the same time. The mathematical expressions used in this case are $h(S,P) = cSP/(A+S)$ or $g(I,P) = cIP/(A+I)$. Our results also found functional responses such as ratio-dependent, in which consumption depends on the ratio of resource abundance to consumer abundance and can be expressed as $g(S,I,P) = cSP/(nP+S+I)$, where c and n are constants. Crowley–Martin functional response $cSP/(1+n_1S)(1+n_2P)$ has been used in two articles to include mutual interference between predators. Finally, we have found one article where the interaction between predators and prey has been modeled with the Beddington–DeAngelis functional response, which is similar to Holling type II.

Four different mathematical expressions were found to model disease transmission, the function f in Fig. 1. These functions are bilinear incidence, standard incidence, saturated incidence, and nonlinear incidence, as shown in Fig. 3. In most of the models, they used bilinear incidence, i.e., $f(S,I) = \beta SI$, where the parameter β is the transmission rate. This expression is generally applicable to small populations. Disease transmission was modeled for large populations following the standard incidence law $f(S,I) = \beta SI/(S+I)$. From this results we also found disease transmission functions that take into account saturation phenomena for a large number of infected individuals, such as the saturated incidence rate, $f(S,I) = \beta SI/(1+nI)$. In addition, we find the nonlinear incidence rate expressed as $f(S,I) = \beta SI^2$, which was used to show a broader range of dynamic behaviors than the bilinear. In general, in Fig. 3, we can observe that the most frequently used combination of functions involves bilinear incidence for disease transmission and Holling Type II for predation

In general, the analysis of the models was carried out using a qualitative, analytical approach, focusing on the behavior around the equilibrium, the analysis of the models was carried out with the qualitative theory, studying the behavior around the equilibrium, except for the articles Chattopadhyay and Arino (1999b), Arino et al. (2004), and Bate and Hilker (2014). The models can have eight types of equilibria characterized as follows: $E_0(0,0,0)$ (no species), $E_1(S,0,0)$ (only susceptible prey), $E_2(S,I,0)$ (susceptible prey and infected prey), $E_3(S,0,P)$ (susceptible prey and predators), $E_4(0,I,0)$ (only infected prey), $E_5(0,0,P)$ (only predators), $E_6(0,I,P)$ (infected prey and predators), and $E_*(S,I,P)$ (all species present), which can be stable or unstable. Overall, the articles consider five equilibria of the following form E_0, E_1, E_2, E_3 and E_*, given that E_1 is unstable and the coexistence equilibrium E_* exists, E_2 and E_3 are unstable. Specifically E_4, E_5 and E_6 do not make sense biologically as there can be

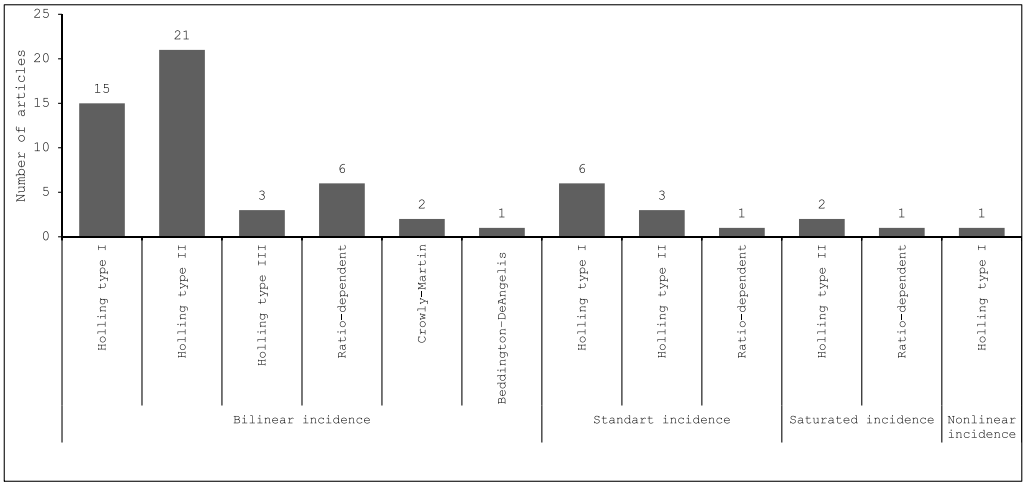

Fig. 3. The number of articles seperated according to the used functional response for predator consumption (or for predator growth) and the used transmission rate.

no infected without susceptible or no predator without prey. These equilibria therefore do not need to be considered further. Particular cases may be of interest; for example, in the models of the articles Das et al. (2009) and Das (2015) the equilibrium E_3 does not exist since in addition to the contact transmission rate, there is an external source of infection. In articles Sahoo (2015) and Wang et al. (2018), the point E_2 is not considered in the analysis. Also, in presence of a standard incidence rate, two types of host extinction rates can be distinguished: disease-induced extinction (with prevalence being nonzero as the host tends to zero) or extinction due to non-infection related reasons (with prevalence being zero as the host goes extinct).

3.2. Disease in the predator species

We found 27 articles that perform the modeling, assuming that the disease only effects the predator. Only one considers a disease SIRS type, one SEI type, and the rest consider an SI type disease. All of this models assume the prey has logistic growth (see Supplementary Table 2). Fig. 4 shows the split of this articles according to the used predator functional response and the grouped according to the transmission function used. These functions are described mathematically as in the previous subsection. It is observed that most of the articles considered saturated incidence to represent disease transmission $\beta SI/(1 + nI)$. This occurred because the authors thought that having susceptible and infected predators is similar to considering a model with a top predator. The saturated incidence rate has a mathematical expression equal to the Holling type II functional response $cN(p_1S+p_2I)/(A+N)$, here $S+I$ is the predator population, N is the prey population and c, p_1 and p_2 are constants. In fact, they assumed the Holling type II functional response to represent both predation and disease transmission.

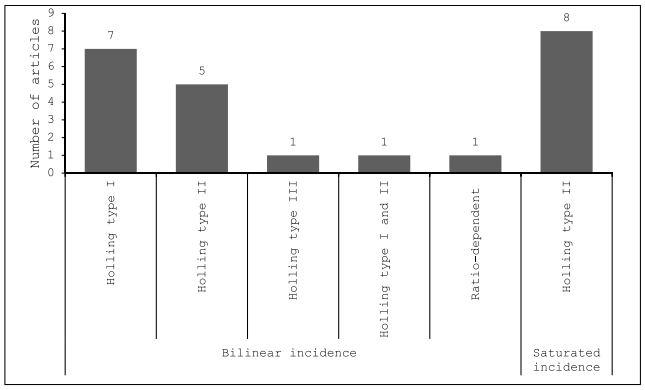

Fig. 4. Number of articles depending on the functional response of the predator grouped according to the transmission rate in the predator.

4. Discussion

Most articles assume in their models that the prey follows logistic growth, including intraspecific competition. However, exponential growth can provide an adequate approximation in newly established populations with a low number of individuals (Tsoularis and Wallace, 2002). Hence, the selection of a growth function may be contingent upon the population size. Although logistic growth is the most widely used, it has some disadvantages, in fact, according to Sieber et al. (2014) and Saifuddin et al. (2017) they consider that logistic growth has limited applicability due to the assumption of an explicit carrying capacity. Hence, in Sieber et al. (2014) and Saifuddin et al. (2017), in contrast to the results of Fig. 3, formulated growth in terms of intraspecific coefficients and refer to this type of function as emergent carrying capacity since it is an upper limit of growth arising from processes such as reproduction and competition.

The interaction between prey and predators in eco-epidemiological models has been modeled with the different classical functional responses from the literature, such as the Holling type I, II, and III (Holling, 1959), the ratio-dependent (Kuang and Beretta, 1998), the Crowley–Martin (Crowley and Martin, 1989), and the Beddington–DeAngelis (Beddington, 1975). The most commonly used functional response of the predator is Holling type II, since it allows to model satiety when prey density is high (Gotelli et al., 2008). Still, some authors opt for the type I functional response because it is mathematically more convenient and allows identifying the effects of other components in the models (Sahoo, 2015). For its part, the ratio-dependent functional response was considered in a significant number of articles, given that in some predator–prey models, such as the one found in the article (Haque, 2009), it is considered to be more appropriate for interactions when predators involve for interactions with actively hunting predators, such as actively foraging animals.

When the disease is in the prey, disease transmission has often been assumed as in classical models of epidemiology, using mass action or standard incidence. However, when the disease is in the predator, disease transmission is modeled with a Holling type II functional response, as seen in the articles (Krishchenko and Starkov, 2020; Das, 2016). This assumption arises from an analogy between the eco-epidemiological and the tritrophic models. By tritrophic models we mean a la dinámica between prey, a predator that feeds on the prey, and a superior predator

that occupies the top of the food chain and preys on the intermediate predator. In technical terms, susceptible individuals can be likened to intermediate predators, while infected individuals can be likened to top predators. The Holling type II incidence as a transmission function is not only used in eco-epidemiological models. Also in epidemiological models, as in the case of the article Safi and Garba (2012), where analysis of SEIR Model with Holling type II incidence function. Therefore, these functions used mainly in ecology are also part of epidemiological models.

For the analysis of the eco-epidemiological systems, the qualitative analysis was usually used. This involves identifying model equilibria, linearizing the system using the Jacobian matrix around these equilibria, calculating eigenvalues, and ultimately categorizing equilibrium points in terms of stability based on the real part of the eigenvalues: positive (unstable) or negative (stable). This analysis was accompanied by numerical simulations. In the results of the models, it was observed that, in general, if the disease affects the prey then the predators reach a lower equilibrium density and the prey a higher equilibrium density compared to the densities of equilibria in the predator–prey model without disease. In the case that the disease affects the predators, a lower equilibrium density is reached for each class; and as for the transmission function, it was observed that the stability region, which depends on predation parameters, increases if the transmission of the disease follows a standard incidence law instead of the mass action law (Li et al., 2017).

While classic compartmental models such as SI, SIS, SEI, and SIR were found, most models consider an SI submodel for the diseased specie. In particular, a reduced number of articles considered the SIS model. It can be more convenient than the SI model because the infected do not reach the total population, allowing more possibilities for the eco-epidemiological dynamics. Besides that, it does not imply adding more variables to the model, allowing a similar analysis proposed in Kumar et al. (2020b). Therefore, although eco-epidemiological models have been studied with a wide variety of forms of functions for growth rate, disease transmission, and predator functional response, to the best of our knowledge, in epidemiology, there are a wide variety of compartmental models that can be studied in this type of systems. For example, one can consider the SIRS model, SEIR model, and their extensions that result from the addition of more compartments such as asymptomatic, crossed immunity, the immune of maternal origin, among others.

Although we have focused on predator–prey models with a disease, either in the prey or in the predator, it is also relevant to mention models in which the disease affects both the prey and the predator. In the article Freedman (1990), the author proposes a model that assumes all predators are infected, and the prey population can act as a primary or main host. In this approach, the infection in the prey does not result from the interaction between susceptible and infected prey, but rather from the reintroduction of parasites by the predators. In contrast, in the articles Han et al. (2001) and Hsieh and Hsiao (2008), it is assumed that the predator acquires the disease only during the predation process, and the infection in the prey occurs through contact between susceptible and infected individuals. A different approach is reflected in models where both the prey and the predator are infected with different diseases that do not cross the species barrier. This is the case in the articles Kant and Kumar (2017) and Gómez-Hernández et al. (2023), where transmission is modeled using bilinear incidence, and predators consume both infected and susceptible prey without affecting their epidemiological status.

Additionally, there are simplified eco-epidemiological models in which predation is modeled as a parameter indicating the intensity of predation. In Hall et al. (2005), they study the dynamics between hosts and parasites when influenced by the presence of predators within the host community. The model is of the susceptible–infected type, with predation modeled as a parameter. They focus on discussing selectivity concerning the density of susceptible and infected hosts, as well as the parasites ability to invade a host population. In Holt and Roy (2007) and Packer et al. (2003), they propose an SIR-type model where individual mortality is a function of predator abundance. Their aim is to demonstrate that in some scenarios, predators can lead to an increase in pathogen prevalence.

The articles considered in our review also do not take into account the spatial configuration of the habitat. However, there are several articles in the literature that include space through diffusion models (Zhang et al., 2014; Li et al., 2017; Wen et al., 2016). These articles generally consider a one-dimensional space closely related to the notion of habitat. In the same vein, there are articles Hilker et al. (2006), Malchow et al. (2004) and Malchow et al. (2005) in which they employ two-dimensional diffusion models in aquatic ecosystems, providing the mathematical foundations for modeling while considering space.

5. Conclusion

This review analyzes the literature on eco-epidemiological models in ordinary differential equations. We focus on the dynamics corresponding to the Lotka–Volterra and Rosenzweig–MacArthur type predator–prey models with a disease in the prey or the predator. Our objective was to provide the functions for prey growth, disease transmission, predator functional response, and the analytical techniques used to analyze these mathematical models. Our study indicated greater interest in studying systems with a disease in the prey rather than a disease in the predator. In most models, the disease was included by dividing the population into two compartments, susceptible and infected.

In models with a disease in the prey, prey growth has been modeled with linear, exponential, and logistic functions. Disease transmission with bilinear incidence rate, standard incidence rate, Holling type II, and Beddington–DeAngelis. The predator functional response has been modeled with Holling type I, Holling type II, Holling type III, ratio-dependent, Crowley–Martin, and Beddington–DeAngelis. We find that models assuming logistic growth of the prey where the infected have no offspring, bilinear incidence rate for disease transmission, and Holling type II for the predator functional are more frequent.

In models with a disease in the predator, we found that prey growth is modeled with logistic growth, standard incidence rate, and Holling type II were used for disease transmission. We found Holling type I, Holling type II, Holling type III, and ratio-dependent in the predator functional response. In this case, it was more common to find models that considered Holling type II for both disease transmission and predator functional response. This result was generated by an analogy between this type of models with a tritrophic network.

In the articles, the authors generally used the qualitative theory of dynamic systems to analyze the models, through which they were able to determine the stability of the equilibria. Regarding the results, we could generally observe that when the disease is in the prey, and there is death by disease, the predators stabilize in equilibrium with a lower numerical value. Moreover, if the disease transmission rate is modeled with a standard incidence rate instead of a linear incidence rate, the stability region of the coexistence equilibrium increases. We can also say that although we classified the models with three key functions: the growth function, the disease transmission, and the functional response of the predator, which results in a base structure of the models. It has the same model structure does not imply qualitatively similar results because the authors considered other parameters that depend on the specific question that each researcher wanted to answer.

Knowing the different mathematical ways of expressing the growth of the prey, the transmission of the disease, and the functional response of the predator, we can conclude that, when modeling an eco-epidemiological phenomenon, the mathematical expression chosen should maximize the ability to explain the mechanisms, express hypotheses and identify critical variables and parameters. But in addition, the choice of the mathematical expressions highlighted here or others that are considered pertinent should also allow us to use the tools to obtain results that enable us to understand the problem.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

No data was used for the research described in the article.

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Appendix A. Supplementary data

Supplementary material related to this article can be found online at https://doi.org/10.1016/j.ecocom.2023.101071.

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