



# Parasite-Guest Infection Modeling: Social Science Applications

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**Abstract.** In this study we argue that parasite-host infections are a major research topic because of their implications for human health, agriculture and wildlife. The evolution of infection mechanisms is a research topic in areas such as virology and ecology. Mathematical modelling has been an essential tool to obtain a better systematic and quantitative understanding of the processes of parasitic infection that are difficult to discern through strictly experimental approaches. In this article we review recent attempts using mathematical models to discriminate and quantify these infection mechanisms. We also emphasize the challenges that these models could bring to new fields of study such as social sciences and economics.

**Keywords:** Infection modeling · Parasite-host infections · Mathematical model

## 1 Introduction

Considering that the evolution of parasites and pathogens is important for human health, agricultural systems and wildlife [1, 2], there is a theory that focuses on how the mechanisms of infection can evolve. Because viruses are the most abundant and simple entities on the planet, they are often used as models to study the evolution of parasitic infections. In particular, parameters such as replication, mortality rate of the infected host, infection rate (absorption rate), among others, have been suggested as possible control parameters used by parasites to optimally infect hosts [3–6]. This paper reviewed the different mathematical models that describe the traditional and recently proposed infection mechanisms. In addition, we reviewed how these are used in the optimal dispersion of infections through susceptible host populations.

In the first section, the classic theory of the evolution of the parasite is reviewed. This theory states that natural selection maximizes the number of secondary infections

resulting from infection of a susceptible host through free channels that do not involve direct contact between infected and susceptible hosts [7]. One way of doing this is by the evolution of the infection rate, which is the probability of a parasite infecting a host after direct contact. In restricted environments, the classical theory predicts that a parasite will evolve to an infinite maximum infection rate. However, experiments using bacteria as a host and viruses as parasites show the unexpected appearance of viruses with a moderate or intermediate infection rate [8, 9]. How and under what conditions this intermediate rate evolves is still an open question. The proposed section reviews the classical and recent models that try to explain this phenomenon

It has been suggested that infection channels between infected and susceptible hosts may provide an advantage, either by allowing parasites to evade the host’s immune response [10], reducing antiviral drug activity [11], or simply having a more efficient mode of infection.

In the second section, a novel model of parasite-host interactions is proposed that accounts for transmission, both through free channels (not involving contact between infected and susceptible hosts), and through infections produced by contact between hosts. The last section examines the possible social and economic science applications that could result from this modeling.

## 2 Dynamics of Traditional Infection: The Host Free Mode of Transmission

First consider a basic model for parasite dynamics introduced by [12]. Let  $H$ ,  $I$  and  $P$  be the number of healthy and infected hosts and parasites, respectively.

$$\dot{H} = \underbrace{\lambda}_{\text{Host production}} - \underbrace{d_H H}_{\text{Host death}} - \underbrace{rHP}_{\text{Host - free infection}} \tag{1}$$

$$\dot{I} = \underbrace{rHP}_{\text{Host - free}} - \underbrace{d_I I}_{\text{infection}} \tag{2}$$

$$\dot{P} = \underbrace{Bd_I I}_{\text{Parasite Production}} - \underbrace{d_P P}_{\text{Parasite death}} \tag{3}$$

A healthy host reproduces at a rate  $\lambda$  and dies at a  $d_H$  rate. The parasite attacks hosts at a rate of  $rPH$ , where  $r$  is the rate of infection. Once the infected host dies (with latency period  $1/d_I$ ), a set of  $B$ -size parasites is released. Alternatively, the term  $Bd_I I$  in (3) can be replaced by  $BI$  in situations where infected hosts release parasites throughout their life cycle rather than dying before releasing them. Parasites that are free in the environment (outside the infected host) can die at a  $d_P$  rate. The level of parasites in the steady state system is:

$$\lim_{t \rightarrow \infty} P = \frac{\lambda r (B - 1) - d_H d_P}{d_P r} \tag{4}$$

Equation (4) can be seen as a way of measuring the parasite’s ability to infect. Note, that for the parasite to develop its maximum infective capacity, the infection rate should be infinite (the maximum population of the parasite in a stable state is  $\lambda(B - 1)/d_p$ ).

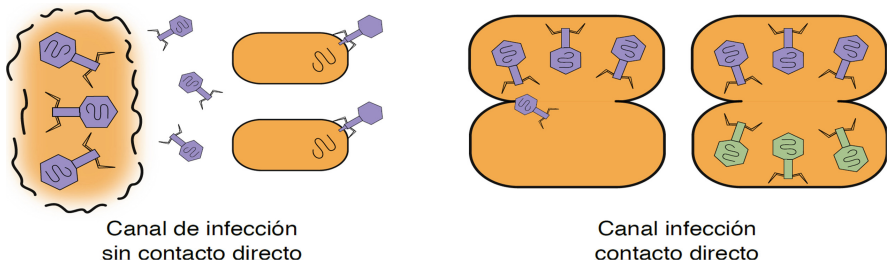
Alternatively, the number of secondary infections can be used to represent the performance of the parasite. Remember that the infection-free steady state, given by  $H = \lambda/d_H, I = 0, y P = 0$ , is an unstable point (meaning that infection will take place) if

$$R_0 = \frac{B\lambda r}{d_H d_p} > 1, \tag{5}$$

Where  $R_0$  is the number of secondary infections and can be interpreted as the number of newly infected hosts produced by an infection.  $R_0$  can be used to infer the evolutionary outcome of the system (3). For example, from (5) it is derived that the parasite should evolve towards infinite infection rates to obtain the maximum fit.

### 3 Ad Modeling of Effects Produced by Spatial Host Structures

The experiments shown in the literature challenge the theory that parasites can evolve to an infinite infection rate, suggested by the previous model. These experiments show the unexpected appearance of parasites with moderate or low infection rates [8, 9]. Intermediate infection rates can be explained by the presence of the spatial structure of the host [13]. Presumably, parasites with high infection rates tend to create a shielding effect in which the local availability of healthy hosts is reduced, resulting in more interactions with the parasite-infected host, leading to a rate of new parasites equal to zero [7]. Figure 1 shows the shielding effect. This shielding effect can be incorporated into the previous model, assuming the number of parasites released by the death of an infected host as a function of the infection rate [14].



**Fig. 1.** Screening effect. Channel of infection without direct contact Channel of infection with direct contact. Source: Own elaboration

$$B(r) = b \frac{d_f}{d_f + r\bar{X}}. \tag{6}$$

Where  $b$  is the maximum number of parasites released that can be obtained from the death of infected cells.  $d_f$  represents the ability of the newly released parasite to escape

from the harmful residues produced by the death of the infected host. A larger  $d_f$  means that the parasite has a high probability of finding a healthy host to infect and reproduce.  $\bar{X}$  represents the average amount of waste generated by the death of a single infected host.

This modification produces a new level of parasites in a stable state with an optimal finite infection rate given by (7)

$$r^* = \frac{df \sqrt{d_H d_P}}{\sqrt{b \lambda d_f \bar{X} - \bar{X} \sqrt{d_H d_P}}}. \tag{7}$$

In addition, there is an optimal number of secondary infections given this finite rate of reproduction [14].

### 4 Mode of Transmission Between Guests

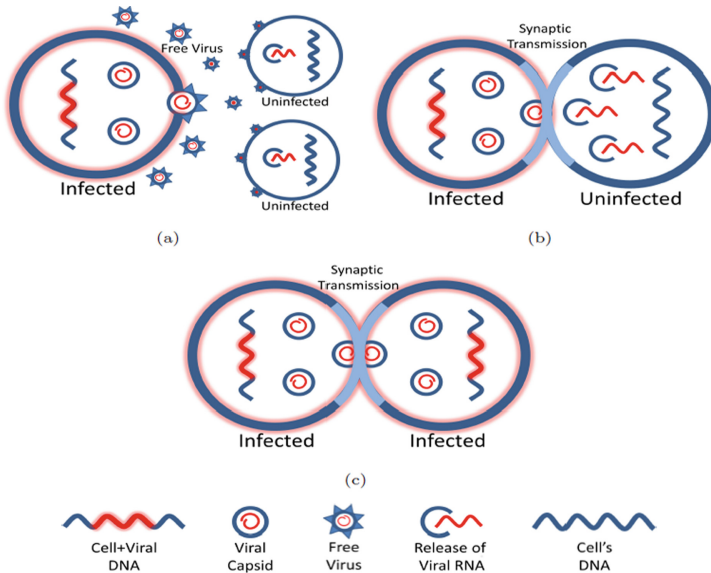
The Our system (1–3) was modified to include the ability of the parasite to carry out transmission by direct contact between infected and susceptible hosts. Let  $s$  the number of parasites sent through the channel formed between an infected and a susceptible host. In addition to the infections produced by the traditional mechanism of infection (without direct contact)  $r P H$ , we add an additional production of infections represented by  $p(s)\beta P H$ . Here  $\beta$  is the rate of interaction between infected and uninfected hosts. The  $p(s)$  function is the probability that an uninfected host will become infected by receiving  $s$  parasites through the channel formed between an infected and a susceptible host. The probability  $p(s)$  is defined as:

$$p(s) = f(s)\sigma(s), \tag{8}$$

Where  $\sigma(s)$  is the probability that the infected and uninfected host will form a host-to-host channel.  $f(s)$  is the probability that sending pathogens through a given host-to-host channel will result in an infection, and can be any monotonously increased function in  $s$ . Assuming the probability of parasites infecting a cell as a binomial distribution. If each copy of the parasite has an  $r$  probability of successful infection, then

$$f(s) = (1 - (1 - r)^s), \tag{9}$$

That is,  $f(s)$  is the probability that at least one of the parasites will have a successful infection given that there is an enabled host-to-host channel. There are two possible scenarios for host-to-host channel formation: channels between infected and uninfected hosts; and channels between infected hosts. The first scenario leads to an infection with probability  $p(s)$ . Therefore, there is a reduction in the number of  $s \sigma(s) H I$  parasites that cannot be used in other infections. The other scenario arises because there is no discrimination mechanism that causes the infected host to form channels with the uninfected host. Channels between infected hosts produce a waste of parasites  $s \sigma(s) s \sigma(s) r I^2$  that does not produce additional infections, because both cells are already infected. Figure 2 shows the three possible routes of infection from host to host: transmission of parasites without involving direct contact between hosts; transmission of parasites



**Fig. 2.** Mechanisms of infection. Source: Own elaboration (Color figure online)

through channels between infected and uninfected hosts; and transmission of parasites through channels between infected hosts.

Including the mechanism described above in the system (1–3) results in the increased. An application to the AIDS virus of this augmented system is available at work [15].

$$\dot{H} = \lambda - d_H H - rHP - \underbrace{p(s)\beta HI}_{\text{Host - to - host Infection}} \quad (10)$$

$$\dot{I} = rHP - d_I I + \underbrace{p(s)\beta HI}_{\text{Host - to - host Infection}} \quad (11)$$

$$\dot{P} = kI - \underbrace{s\sigma(s)\beta(H + I)I}_{\text{Parasite wasted}} - d_P P. \quad (12)$$

According to Fig. 2, a parasite has the ability to infect cells through (a) a free channel without direct interaction; (b) through contact. In the former, infected cells produce chains of RNA (red lines) that use information from the parasite stored in their genome (blue and red line), encapsulate them (blue and red concentric circles) and send these out of the cell. Uninfected cells absorb them and release strands of RNA parasites (blue open circle) that integrate with the cell's DNA (blue line). Interactions can occur between infected and uninfected cells (b) or between infected cells (c). Copies of the parasite in (b) sent through contact are not used in the infection of other cells.

## 5 Discussion

This paper reviewed current approaches in ecology through the use of mathematical tools such as Ordinary Differential Equations (ODEs). Extended models were presented that address issues under debate in ecology, such as optimizing parasite-host interactions and why host infection mechanisms can be beneficial to parasites.

Whether these models and their projections of infection spread can be applied to fields such as economics, business administration and public policy is relevant for future research. For example, recent studies have suggested studying crime in a region in a manner similar to an epidemic. One could, for example, predict how many additional crimes occur in a given season and design public policy using these models. In the medical field, one could determine which transmission model (host-to-host or non-host) is most effective in spreading and mitigating infections, or in agronomic science [16, 17].

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