The Host-Pathogen Game:
an evolutionary approach to biological competitions

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Abstract

We introduce a model called Host-Pathogen game for studying biological competitions. Notably, we focus on the invasive dynamics of external agents, like bacteria, within a host organism. The former are mapped to a population of defectors, that aim to spread in the extracellular medium of the latter. In turn, the host organism is composed of cells mapped to a population of cooperators, that aim to remove pathogens. The cooperative behavior of cells allows to support the living functions of the whole organism, since each one provides an unitary amount of energy. When one or more bacteria are spatially close to a cell, the latter may use a fraction of its energy to remove them. On the other hand, when a bacterium survives an attack, it absorbs the received energy, becoming stronger and more resistant to further attacks. In addition, since bacteria play as defectors, they aim only to increase their wealth, without supporting their own kind. As in many living organisms, the host temperature plays a relevant role in host-pathogen equilibria. In particular, cooperators succeed when bacteria are completely removed, while the opposite outcome entails the host undergoes a deep invasive process, like a blood poisoning. Results of numerical simulations show that the dynamics of the proposed model allow to reach a variety of equilibria. Here it is worth noting that, on a quality level, the achieved outcomes describe some real scenarios, that can be observed in living systems. To conclude, we deem that our model might open the way to further developments, based on evolutionary game theory, for studying several complex biological phenomena.
I. INTRODUCTION

In the last years, scientists coming from different communities investigated several socio-economic systems and biological phenomena under the lens of evolutionary game theory (hereinafter EGT) [1–15]. In general, studying the evolution of a population and identifying strategies that trigger cooperation [16, 17] constitute some of the major aims in EGT. In particular, the emergence of cooperation becomes really interesting when agent interactions are based on games having a Nash equilibrium of defection. In this context, the spatial Public Goods Game (PGG hereinafter) is one of the most famous models [18], and it is based on a simple dynamics: agents play as cooperators or as defectors; at each time step cooperators contribute to a common pool with a coin; then the total pot is equally divided among all agents (no matter their strategy). In the classical PGG defection represents the Nash equilibrium, hence finding methods for driving the whole population towards an equilibrium of full cooperation is a challenging task. It is worth noting that the coin, constituting the contribution provided by each cooperator in the PGG, represents a very general form of resource, as money in an economical system or food in an ecological one. Thus, in general, the contribution can be viewed as a form of energy. The evolutionary aspect of games is due to the possibility, for each agent, of changing strategy over time. Notably, rational agents tend to imitate richer or stronger opponents in order to increase the probability to gain higher payoffs at the next time steps. Since the concept of evolution has its early roots in the investigations of nature, in this work we aim to model a complex biological phenomenon by the framework of EGT. In particular, we focus on host-pathogen interactions [19], like those we can observe between some bacteria and more complex organisms, as human beings. The underlying complexity of these processes emerges from the interactions between bacteria and the immune system of an host organism, being the latter the result of an orchestration among several entities. Nowadays, mathematical biology [20] represents one of the main attempts for describing similar scenarios both in quantitative and in analytical terms. Although a mathematical approach requires an high level of abstraction from the real scenario, a wide list of results, for instance in computational epidemiology [21, 22] and in genomics [23], suggests that the research in this area is strongly helpful and promising. Let us now focus the attention on host-pathogen interactions [24]. In general, bacteria may behave as saprophyte, as commensal, or as parasite [25]. In the proposed model, we refer to
the third case (i.e., parasite), thus bacteria aim to invade the host organism. In doing so, the host-pathogen interaction can be viewed as a two species challenge, i.e. bacteria versus cells of an host organism. Therefore, we consider this competitive biological process in an heterogeneous system. Under this perspective, there are two kinds of equilibria: the success of one species, or a co-existence between them. In principle, the optimal equilibrium for pathogens is represented by the co-existence, since if they completely succeed, the living organism quickly becomes inhospitable (i.e. dying). For instance, in biological terms a widespread invasion may lead the host organism to a severe pathological state defined ‘blood poisoning’. On the other hand, a steady-state can be reached by bacteria behaving like ‘comensal’ and ‘opportunistic’ [26], since they avoid a massive spread in the host organism. Here, using the language of statistical physics [27], the processes leading to the mentioned equilibria can be interpreted as order-disorder phase transitions, where the disordered phases correspond to the various forms of co-existence, while the ordered one to the complete prevalence of one species. Therefore, even if from a theoretical point of view, the studying of order-disorder phase transitions in these heterogeneous systems may, in principle, allow to get further insights on the considered biological phenomenon. Moreover, as showed in [28, 29], theoretical approaches to biology based on physics may constitute a fundamental ingredient for achieving a deep knowledge on the dynamics of many complex biological processes. In this scenario, the orchestrating dynamics of the immune-system and the invasive strategy of parasites, inspired us to represent their interactions as an evolutionary game between co-operators and defectors. Thus, we introduce the Host-Pathogen game (hereinafter HPG) to model this complex biological challenge. In the proposed game, the host organism is mapped to cooperative cells, while the pathogens to a population of defectors. Remarkably, cells co-operate only among cells, while try to kill bacteria that, in turn, behave as defectors (even among them). It is important to point out that agents never change strategy. However, as better explained later, some cells appear to behave as defectors due to their commitment in removing close bacteria. Moreover, the spreading of pathogens comes as result of their willingness to enforce their individual wealth (i.e. their payoff). The main dynamics of HPG entail that cells can use a fraction of the energy, corresponding to the contribution they provide to the organism, to remove close pathogens, i.e. those in their neighborhood. In turn, when a pathogen survives, it absorbs the received energy, reinforcing itself. Remarkably, surviving an attack and even taking profit from it, may be viewed as the realization of a
famous Nietzsche’s aphorism “That which does not kill us makes us stronger” \cite{30} and, in our view, it can be interpreted also as a principle of adaptation in an hostile environment. Finally, as in many living systems, the temperature is strongly relevant in HPG. Notably, in very general terms, increasing its value entails an immune-system improves its efficiency in fighting pathogens so, accordingly, in the proposed model high temperatures support the host organism. The dynamics of HPG are studied by means of numerical simulations, implemented for analyzing the final equilibria that can be reached, and to evaluate whether they can be related with some real scenarios (although at a very abstract level). The remainder of the paper is organized as follows: Section II introduces the proposed model. Section III shows results of numerical simulations. Eventually, Section IV ends the paper.

II. THE HOST-PATHOGEN GAME

The HPG represents the competitive dynamics of a spatially structured \cite{31,32} two-species population, i.e. cells of a host organism versus bacteria. The former are arranged in a square lattice with continuous boundary conditions, while the latter occupy the inner squares of the lattice —see Figure 1. In doing so, when a bacterium invades a square, the four cells (at the vertices of the infected square) try to remove it. Cells behave as cooperators, whereas bacteria as defectors. Therefore, as in the PGG, cooperators contribute

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure1.png}
\caption{(Color online). Pictorial representation of the interaction topology. In particular, cooperative cells are arranged over a toroid (i.e. a), while bacteria occupy inner squares. Notably, b shows a fragment of the toroid, with green circles representing cooperative cells, and red triangles representing bacteria inside infected squares.}
\end{figure}
with an unitary amount of a resource. In this specific case, the resource corresponds to a form of energy, used by the host organism to carry out the living functions, or other tasks. Then, cells indirectly receive a payoff (e.g. oxygen) when this biological engine correctly works. Now, once a square gets infected, each cell at its vertices uses a fraction of the unitary energy contribution for trying to remove the parasite. Since each cell is surrounded by four squares (see Figure 1), it may reserve only $\frac{1}{4}$ of its energy contribution for each square. As result, the contribution of a cell decreases as the number of nearest infected squares increases. Thus, when cells try to remove one or more parasites, indirectly behave as defectors. This observation recovers a particular importance since the adopted dynamics let emerge defectors even in the cell population. Remarkably, defection among cells comes as an indirect effect, because they always provide the same amount of energy but, in this particular case (i.e. defection), it is targeted towards the parasite. Due to its relevance, this phenomenon requires a deeper description: when a cell has only one nearest infected square its energy contribution is scarcely reduced (i.e. of $\frac{1}{4}$); on the other hand, when a cell is completely surrounded infected squares, its contribution is completely used for fighting parasites. In turn, since each square has four vertices, a parasite is always attacked by a unitary amount of energy, coming from the summation of the fractions of energy provided by the four cells, each equal to $\frac{1}{4}$ (whose summation gives 1). Now, using a Glauber-like approach, we define the probability to remove a parasite from a square $p^h$, and that to fail in this task $p^b$, as follows

$$
\begin{cases}
  p^h = e^{-\beta H} \\
  p^b = 1 - p^h
\end{cases}
$$

with $H = \sum_{i=1}^{4} c_i \frac{1}{4} = 1.0$, where $c$ is the energy contribution of a single cell, and $\beta = \frac{1}{(T-T_c)\alpha}$. Here, $H$ can be viewed as a Hamiltonian representing the energy transferred to a square and with constant unitary value (i.e., 1), $\alpha$ as a Boltzmann constant (set to 0.7), $T_c$ as the minimum temperature a living organism may have to work physiologically, and $T$ the effective temperature of an organism. The equations highlight the significative role of the temperature in the HPG, as for high $T$ the $p^h \rightarrow 1$, while for low $T$ the opposite occurs (i.e. parasites are never removed). Furthermore, as briefly mentioned before, once a parasite survives an attack it can use the absorbed energy to increase its payoff. Notably, when a parasite infects a square, its payoff is still equal to zero. Then, in order to increase its payoff,
the parasite has to infect all the nearest squares (i.e. the adjacent ones). Thus when an attack fails, the parasite spreads if there is a nearest square free (i.e. not infected), otherwise it increases its own payoff. The latter has a relevant role because, in the HPG, a bacterium (or parasite) can accumulate it over time, behaving as a memory-aware agent (see [33]), in order to reduce the probability to be removed from the host organism during further attacks. In particular, every time a cell succeeds according to 1, the payoff of the attacked parasite reduces of 1, so that it is removed only once its payoff becomes negative. The possibility to conserve the payoff over time entails the parasite is able to enforce itself (e.g. creating a kind of shield). As previously observed, this phenomenon can be viewed in terms of a form of adaptation to a hostile environment, or as the formation of a 'callus' resulting from a continuous mechanical stimulus in the skin. Eventually, the HPG can be summarized as follows:

1. At \( t = 0 \) a number of squares get infected by parasites;
2. While the number of infected squares is greater than 0:
3. ______ Randomly select an infected square
4. ______ The infected square gets healthy with probability \( p^h \) otherwise:
5. ______ IF all nearest squares are infected: increase the payoff of the parasite in the selected square
6. ______ ELSE: randomly select a free (i.e. healthy) nearest square and infect it
7. repeat from (2) until the population reaches an ordered phase, or up to a (limited) number of time steps elapsed.

Before to show results of numerical simulations, we observe that if agents were not ‘memory-aware’ (i.e. they cannot save their payoffs), the amount of infected squares \( s \) could be analytically computed according to the following equation

\[
\frac{ds}{dt} = s \cdot (p^b - p^h) \tag{2}
\]

Whose solution is

\[
s(t) = s(0) \cdot e^{zt} \tag{3}
\]
with \( z = (p^b - p^h) \) and \( s(0) \) is the initial amount of infected cells in the organism. However, as before described, the complexity of the HPG entails numerical simulations are mandatory to investigate its behavior.

III. RESULTS

Numerical simulations have been performed for analyzing two different cases: constant temperature and varying temperature, considering in both cases a basic temperature equal to \( T = 33 \) (previously indicated as \( T_c \)). The first case allows to study the evolution of the heterogeneous population (i.e. cells and parasites) without to vary the system temperature (i.e. that of the whole host organism). The second case allows to analyze the role of a variant temperature in this dynamics. Eventually we recall that, in both cases, we arrange cells in a bi-dimensional square lattice, with periodic boundary conditions, so that parasites can be located inside the inner squares.

**Constant Temperature**

Let us consider the first case, i.e. the evolution of the population at constant temperature. Due to the computational effort required to simulate the proposed model, we consider a population of \( N = 400 \) cells, with an initial density of bacteria corresponding to the 5% of cells. Two different temperatures are compared: \( T = 35 \) and \( T = 36 \). Here, we emphasize that, although we studied the proposed model while considering also different initial configurations (e.g. different temperatures and initial densities of bacteria), those reported are the most significant. Figures 2 and 3 show results related to the temperatures \( T = 35 \) and \( T = 36 \), respectively. Even if, in both cases, parasites are able to spread in the whole organism, the first one (i.e. Figure 2) indicates that at \( T = 35 \) parasites completely prevail, while the second (i.e. Figure 3) shows the emergence of a steady-state characterized by the co-existence between parasites and cells. Figure 4 reports the density of cooperators and defectors in the cell population, over time (plots a and b), and the payoff gained by cells (plot c) and by parasites (plot d). In all plots, we compare results achieved by setting the organism temperature to \( T = 35 \) (blue line) and to \( T = 36 \) (red line). As we can observe, Figure 4 confirms qualitative results showed in Figure 2 and Figure 3, i.e. increasing the or-
FIG. 2. (Color online). Evolution of the agent population, composed of \( N = 400 \) cells, with a constant temperature \( T = 35 \). At the beginning, the amount of parasites corresponds to the 5% of cells. Colors represent the state of cells: those green are healthy (i.e. not infected by parasites), while those cyan, blue and black are infected. The darkness of infected cells indicates the parasites’s payoff, i.e. the darkest the richest (i.e. the strongest versus cell attacks). Each subplot refers to a different time step, starting from \( t = 0 \) (i.e. the first one) up to \( t = 175000 \) (from left to right, and from the upper part to the lower part). All plots refer to a single realization of a simulation.

Remarkably, as in many biological systems, once an organism is infected by a parasite or by an organism temperature, of only one degree, the scenario radically changes. Notably, at \( T = 35 \), parasites prevail completely causing what that can be considered as a blood poisoning in living systems, while at \( T = 36 \) a steady-state of coexistence is reached, even if parasites spread in many cells.

**Varying Temperature**

Here, we show results achieved by considering a varying temperature of the host organism. Remarkably, as in many biological systems, once an organism is infected by a parasite or by an
FIG. 3. (Color online). Evolution of the agent population, composed of $N = 400$ cells, with a constant temperature $T = 36$. At the beginning, the amount of parasites corresponds to the 5% of cells. Colors represent the state of cells: those green are healthy (i.e. not infected by bacteria), while those cyan, blue and black are infected. The darkness of infected cells indicates the parasites’s payoff, i.e., the darkest the richest (i.e. the strongest versus cell attacks). Each subplot refers to a different time step, starting from $t = 0$ (i.e. the first one) up to $t = 200000$ (from left to right, and from the upper part to the lower part). All plots refer to a single realization of a simulation.

external agent (being living or not), the immune system increases the temperature in order to improve its efficiency in restoring the healthy condition. In this configuration, we considered a population of $N = 2500$ cells, beginning with two different initial temperatures: $T = 35$ and $T = 36$. As before, the basal temperature is $T_c = 33$ (i.e. the minimum physiological temperature for having a working organism). Now the starting density of parasites is equal to the 1% of cells, but the temperature can increase only once that the density of parasites is equal (or greater) than 25% of cells. The heating is slow, i.e. the temperature increases of a $\Delta T = 0.001$ at each time step as the density condition, just described, is reached. Figure 5 shows the density of cooperators and of defectors over time (plot a), and the related average magnetization (plot b). The latter is computed considering a binary variable $\sigma = \pm 1$, being +1 for cooperative cells, and −1 for defector cells. In so doing, the magnetization (see [34]) reads

$$M = \frac{1}{N} \sum_{i=1}^{N} \sigma_i,$$  (4)
FIG. 4. (Color online). Plots a and b represent the number of cooperators and that of infected cells ($I$), respectively, over time. Plots c and d represent the payoff gained by cells and by parasites over time, respectively. In all plots, the blue line represents results achieved by $T = 35$, while the red one those achieved by $T = 36$. Results are averaged over different runs.

FIG. 5. (Color online). a Density of cooperators (blue) and of defectors (red) over time. b Average magnetization of the population over time. Results are averaged over different runs.
We remind that parasites are always defectors, so they have not been considered in the computation of the average magnetization (equation 4). Now, it is worth to highlight that, from the point of view of evolutionary game theory, it is possible to observe an interesting phenomenon in the dynamics of the proposed model: once that cells are surrounded by parasites, their energy contribution is used to fight them; therefore these cells behave like defectors, since cannot provide a full contribution. In few words, indirectly the spreading of parasites lets emerge defectors among cells that, involuntarily, are no more perceived as cooperators. In Figure 6 we can observe this phenomenon with more details. In particular,

FIG. 6. (Color online). Variation of the payoff over time. a Payoff of cells (blue line) versus that of parasites (red line). b Fraction of defectors among cells. Cells are induced to defect by reducing their energy support to the organism in different fractions. Red, blue and green lines indicates partial cooperators providing, respectively, a contribution to the organism equal to: \( \frac{3}{4}, \frac{1}{2}, \frac{1}{4} \). Instead, black line indicates full defectors, i.e. cells that use the whole energy contribution to fight parasites. Payoff Results are averaged over different runs.

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\end{align*} \]
In this work we study the dynamics of a complex biological process, i.e. host-pathogen competitions, by the framework of Evolutionary Game Theory. In particular, we consider a two-species competition, i.e. cells of a host organism versus bacteria (or parasites). The proposed model, named Host-Pathogen Game, strongly simplifies real biological scenarios. However, the related dynamics, investigated by means of numerical simulations, show that different equilibria, similar on a quality level to those observable in real contexts, can be reached. For instance, both the complete removal of parasites, and the invasive spreading of these external agents, correspond to healing processes and deep infections mechanisms (like 'blood poisoning'), respectively. Remarkably, even if agents of HPG never change their strategy, the interactions between cells and parasites can lead the former to become defectors. So, we observe an evolutionary mechanism introduced by the invasion and spreading of parasites in the host organism, that entails the emergence of defectors among cells. In our view, the relevance of this observation lies in the fact that HPG introduces the possibility to become defectors while behaving as cooperators (i.e. while still paying a contribution). In particular, the strategy of an agent is not the one adopted, but that perceived by its neighbors. Therefore, we deem that this phenomenon might deserve further investigations also beyond its biological interpretation. Eventually, as in many living organisms, also in HPG the system temperature plays a key role: high values allow cells to prevail, while low ones support bacteria. Thus, this point represents a further link between the proposed model and real biological scenarios. To conclude, the simplifications here introduced, for modeling a complex process, are rewarded by results that can have a biological interpretation (e.g. 'blood poisoning'). Hence, in the light of these considerations and of the achieved outcomes, we deem relevant to point out that Evolutionary Game Theory may constitute a promising framework for studying complex biological phenomena, and we suggest that further investigations would be relevant for shedding new light on different aspects of the proposed model.
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