Population dynamics of entangled species

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The laws of physics enter the doctrine of population dynamics through various interactions whereby energy and matter, or more broadly, information are exchanged between different species and between species and their natural environment. The dynamics then emerge as a continual process involving many such causal interactions. Quantum physics, on the other hand, shows that the coordination between different processes must obey certain rules. We hypothetically ask: Can this game of species benefit from unique features of quantum mechanics such as entanglement and nonlocality? Here we extend quantum game theory to analyze this question and demonstrate that in certain models describing ecological systems where several predators feed on the same prey, the strength of quantum entanglement between the various species has a profound effect on the asymptotic behavior of the system. For example, if there are sufficiently many predator species who are all equally correlated with their prey, they are all driven to extinction. However, if one predator specie is correlated with its prey to the maximal extent allowed by quantum mechanics, predator and prey may coexist. Our results highlight the fundamental differences between classical and quantum complex systems, emphasizing the important role of entanglement within the latter. These results were derived in two ways: by analyzing the asymptotic dynamics of the system, and also by modeling the system as a quantum correlation network, which enabled us to apply methods from network theory in the above scenarios. Several generalizations and applications are discussed.

Population dynamics is the study of how and why population numbers change in time and space. These dynamics emerge as a continual process involving many interactions between different species and between species and their natural environment. This work has been motivated by considering whether this game of species may benefit from unique features of quantum mechanics such as entanglement and the uncertainty principle. We demonstrate that in quantum models describing ecological systems, where several predators feed on the same prey, the strength and multipartite structure of quantum correlations between the various species has a profound effect on the long-term behavior of the system. For example, if there are sufficiently many predator species who are all equally correlated with their prey, they are all driven to extinction. However, if one predator specie is correlated with its prey to the maximal extent allowed by quantum mechanics, predator and prey may coexist. Our results highlight the fundamental differences between classical and quantum complex systems, emphasizing the important role of entanglement within the latter. These results were derived in two ways: by analyzing the dynamics of the system, and by modelling the system as a complex network.
1 Background

Population dynamics is the study of how and why population numbers change in time and space. Population dynamicists distinguish five general classes of pairwise species interactions [1], classified by the positive (+), negative (−), or no (0) effect of species on each other: interference competition (−, −), mutualism (+, +), commensalism (+, 0), amensalism (−, 0), and trophic (+, −). Although a trophic interaction is only one of five types, population ecologists have devoted a massive share of their attention to studying trophic interactions. Trophic interactions, also known as predator-prey interactions, seem to be of universal importance: all organisms are consumers of something, and most are also a resource to some other species. In fact, predator-prey theory is one of the best-developed areas in population ecology.

Maynard Smith and Price introduced game theory into the study of evolutionary population dynamics [2]. They considered games where each individual member of the population can play one of a fixed set of strategies, and the game payoff represents fitness, or reproductive success. The games usually model pairwise contests over some shared resource, and a Payoff Matrix represents the possible outcomes (in terms of fitness) to each player, depending on the players’ strategies. Thus, augmenting population dynamics models by accounting for the possible strategies for each species, allows one to study the effect of individual organisms’ properties on large-scale, long-term dynamics. Evolutionary Game Theory uses this approach to explain the emergence of some animal behaviors [3]. In this paper we are interested in modifications to these behaviors introduced by quantum mechanics, and more specifically, by quantum nonlocality (see a graphical illustration in Fig. 1).

Such a departure from a classicist mindset was not even an option when the famous EPR paradox [4] was conceived. Indeed, one of the presumptions in the EPR paper is that of locality, the other being realism – the premise that one may speak meaningfully about potential measurements, those which have not actually been carried out. Taken together, these two presumptions came to be known as local realism (or local hidden variable (LHV) models). Numerous Bell inequalities that have been devised and tested in recent decades following [5] seems to imply that quantum mechanics defies local realism, while different consequences have been studied as well [6, 7, 8].

A Bell inequality is associated with an experimental setup that involves two or more experimenters performing measurements and comparing their outcomes. Each participant will have at least two measuring instruments to choose from before carrying out a measurement. The inequality sets an upper limit on the correlations between the measurement results in a reality describable by a LHV model. The Bell-CHSH inequality [9], for instance, underlies a bipartite setting where two distant parties, named Alice and Bob, perform measurements each at her/his own site. The measurement outcomes are dichotomic taking either one of two values. In this experiment each participant chooses one out of two apparatuses with which to measure. The Bell-CHSH inequality tells us that in a classical (local) reality some linear function of the correlators may not exceed 2. The mathematical formalism of quantum mechanics, however, predicts a violation of this inequality that can reach as high as $2\sqrt{2}$ [10]. This violation can be attained if an entangled state is shared between Alice and Bob. However, quantum theory prohibits certain correlations in multipartite systems, e.g. by enforcing entanglement monogamy [11]. Such considerations are typically utilized by quantum games.

Quantum games extend classical game theory to the quantum domain, often considered in the context of quantum networks [12, 13, 14, 15, 16, 17]. They allow superposed or entangled initial conditions, as well as superposed strategies. When considering game theory in the context of predator-prey population dynamics, it seems natural to ask how could these dynamics be affected if the individual predator-prey encounters are modeled as quantum games?
In this paper we analyze a novel kind of multi-agent quantum games whose population dynamics are captured by the well-known Lotka-Volterra system of coupled differential equations. According to the proposed games, there are two types of species, virus and cell, and while interacting they may get entangled. This opens up both opportunities, such as stronger-than-classical correlations, as well as limitations such as the Tsirelson bound and entanglement monogamy — both are shown to play a crucial role in the dynamics. Employing our previously developed framework [18], we describe the system as a random correlation network and use it to prove a relation between its quantum parameters and the system’s capacity to sustain coexisting species in the asymptotic time regime. We also present an analytical treatment of these predator-prey games, as well as a simulation. These complementary approaches all point to a decisive advantage for species that cleverly use entanglement.

2 Results

2.1 A cell-virus game

Pairwise inter-species interactions in ecological systems can be modeled by games. When the species differ fundamentally, e.g., “virus” and “cell” communities, then the underlying game might be a zero-sum game in which one player’s loss is the other’s gain. Here the virus may naturally become infected (loses) and the virus may reproduce (wins). Only when the virus is attenuated and the host cell is immune, their outcomes of a binding are the opposite and the virus dies (loses).

The cell and virus are each equipped with different types of receptors and ligands, respectively, on their envelopes. Binding occurs when the virus ligands attach to complementary receptors on the cell membrane. This procedure may or may not be successful depending on various physical conditions and the types of ligands and receptors used [19]. To model this behavior let $C$ be a binary random variable representing two types of receptors, −1 or 1, on the cell envelope. Similarly, let $V$ be a binary random variable representing two types of ligands, −1 or 1, on the virus envelope. Only when their receptors fit, $C = V$, may the cell and virus bind. We assume that the players do not communicate before their interaction and for that reason the type of receptor used by any of them is chosen beforehand and independently of the state of the other player, i.e., $C$ is independent of $b$, and $V$ is independent of $a$. See Fig. 2.

The outcome of a successful cell-virus bond depends on their physiological states. In the game, this is expressed by defining the cell’s payoff after a successful bond as $- (-1)^{ab}$, and for the virus as $(-1)^{ab}$. Moreover, we assume a failed bond attempt has opposite payoffs. Thus, the payoffs may concisely be written as $- (-1)^{ab} CV$ for the cell and $(-1)^{ab} CV$ for the virus. Note that the cell (virus) payoff should express the change in the cell (virus) population as a result of a single cell-virus interaction; however, for the sake of simplicity we define it in a symmetric manner. Thus, in our game the cell population increases by one when it kills an attenuated virus. One of several mechanisms could explain this phenomenon; for example, an immune cell could be “programmed” to duplicate when facing a threat, as a defense mechanism. Furthermore, a failed bond attempt results in the cell dying and the virus reproducing if $a = b = 1$, or the cell reproducing and the virus dying otherwise.

A cell/virus may have many receptors/ligands of both kinds, ±1, and so binding may occur with some probability depending on the cell and virus physiology. This probability is given by $(1 + \rho_{ab})/2$, where $\rho_{ab} \equiv E[CV | a, b]$ is the cell-virus correlator, the expected value of $CV$ conditioned on the physiological states $a, b \in \{0, 1\}$. Using the correlator, $(-1)^{ab} \rho_{ab}$ is the average payoff for the virus given that the cell is in state $a$, and the virus in state $b$. When all physiological states are equally likely the resulting total payoff is $B/4$ for the virus and $-B/4$ for the cell, where (see Fig. 2):

$$B \equiv \rho_{00} + \rho_{01} + \rho_{10} - \rho_{11}. \quad (1)$$

The binding probability defined above has an evidential flavour for it measures the chance of binding independently of the character of the binding mechanism (be it random or deterministic).
Figure 2: The pair of rows from the top illustrate four types of interactions between a cell (Alice) and a virus (Bob). The uppermost row illustrates successful bindings when the cell receptors correlate with the virus ligands, $C = V = 1$ or $C = V = -1$. The row below shows unsuccessful binding attempts when the opposite occurs, $C \neq V$. This setting is reminiscent of the Alice-Bob coordination game underlying bipartite Bell inequalities. Indeed, the spin of Alice’s quantum particle may either be correlated or anti-correlated with that of Bob’s particle. The remaining rows depict the payoff matrix in the cell-virus game. Starting from the upper-left “quadrant” and going clockwise: vulnerable cell ($a = 0$) – virulent virus ($b = 0$), immune cell ($a = 1$) – virulent virus ($b = 0$), immune cell ($a = 1$) – attenuated virus ($b = 1$), vulnerable cell ($a = 0$) – attenuated virus ($b = 1$). The respective virus and cell payoffs are $(-1)^{ab}\rho_{ab}$ and $-(1)^{ab}\rho_{ab}$.

But the same probability may be viewed from a frequentist perspective by considering populations rather than individuals. Suppose there are cell and virus colonies of which some individuals interact according to the above. If the physiological states, $a = 0$, $a = 1$, $b = 0$, and $b = 1$, all are equally likely across the cell and virus populations then $\pm B/4$ may be seen as population payoffs, the average payoff over a large number of individuals that play the game [3]. Accordingly, the payoffs of the cell and virus populations conditioned on their collective physiology are $\pm (1)^{ab}\rho_{ab}$.

So far we have considered only two species and four kinds of correlators. This may be seen as the simplest instance of a correlation network. In a more general setting we may have many species that interact in diverse ways. For example, in a tripartite network consisting of two non-interacting types of viruses (labeled $V_1$ and $V_2$) and a cell (labeled $C$), we define two population payoffs: $B_{CV_1}$, of the cell-virus-1 games, and $B_{CV_2}$, of the cell-virus-2 games, where $B_{CV_k} \triangleq \sum_{a,b_k} (-1)^{ab_k} \rho_{ab_k}$, with,

$$\rho_{ab_k} \triangleq E \left[ CV_k \mid a, b_k \right], \quad (2)$$

where $V_k$ denotes the $k$th virus’ random variable $V$, and $b_k$ denotes the $k$th virus’ state $b$.

The reader may at that point wonder whether the above game is the authors’ sheer imagination. Should the cell and virus be taken literally? With the current lack of decisive experimental evidence we obviously cannot claim it should. We suggest the critical reader to think of them as metaphoric
roles within an interactive quantum network. That said, the potentiality of cells and viruses to employ quantum mechanical principles does not seem to be ruled out yet [20, 21].

2.2 Population dynamics over correlation networks

The average population payoff for some type of virus, previously denoted $R$, is in fact the well-known Bell-CHSH parameter. The significance of this parameter stems from the following theorem: if there is a local hidden variable model for the above experiment, then $|R| \leq 2$ [9]; and 2 is called the Bell limit for this parameter. However, if Alice and Bob’s outputs are allowed to be taken as quantum measurement outcomes over a shared entangled state, then the Bell-CHSH parameter may go up to a maximum of $2\sqrt{2}$, known as the Tsirelson bound [10]. The set of four correlators, $\rho_{ij}$, are said to be nonlocal whenever $R$ exceeds the Bell limit. Thus, if the players in the cell-virus game somehow employ a quantum mechanism for generating the $C$ and $V$ then the correlations between their decisions, the $\rho_{ij}$ which underlie the average payoffs, may defy any explanation in terms of LHVs.

However, utilizing quantum strategies has additional implications. As in the previous section, assume our network consists of one cell type $C$ and two virus types $V_1, V_2$. Then, monogamy of quantum correlations implies that the cell-virus payoffs obey [11]:

$$(R_{CV_1})^2 + (R_{CV_2})^2 \leq 8.$$  

Namely, only one set of correlators, either that of $C-V_1$ or $C-V_2$, may be nonlocal.

The population evolution of the different species may be described mathematically by a Lotka-Volterra-type dynamical model whose interaction parameters account for the species’ physiological conditions. We are about to propose a similar model in which interactions enter via the species’ average payoffs. Our model makes the following assumptions:

- The cells duplicate with time parameter $\gamma$.
- The $k$th virus type dies off with time parameter $\zeta_k$.
- When a cell interacts with a virus of the $k$th type, the outcome is determined by the population payoff matrix (Fig. 2).

Relying on these assumptions, the infinitesimal change $dc$ in cell population density $c$ in some short time $dt$, may be described by:

$$dc = \gamma c dt - \sum_{k=1}^{n} (-1)^{ab_k} \rho_{ab_k} \beta cv_k dt.$$

This is justified by noting that the probability for a $C-V_k$ interaction is proportional to these species’ population densities; thus, the number of $C-V_k$ interactions in the time interval $[t, t + dt]$ is $\beta cv_k dt$ for some parameter $\beta$ which depends only on constant system characteristics (e.g., its volume); and, as noted before, a single $C-V_k$ interaction results in the cell population changing by $-(-1)^{ab_k} \rho_{ab_k}$ for given physiological states $a, b_k$.

The change $dv_k$ in the $k$th virus’ population density $v_k$ (again, over some short time $dt$) obeys a similar equation:

$$dv_k = -\zeta_k v_k dt + (-1)^{ab_k} \rho_{ab_k} \beta cv_k dt,$$

where it is assumed, in addition to the above, that viruses of different species do not interact.

Thus, assuming $a, b_k$ are uniform and independent random variables, the population dynamics may be described using the following Lotka-Volterra system:

$$\dot{c} = \gamma c - \sum_{i=1}^{n} B_i cv_k$$

$$\dot{v}_k = -\zeta_k v_k + B_k cv_k, \quad \forall k \in [n],$$

where $B_k \overset{\text{def}}{=} \beta R_{CV_k}/4$, and $n$ the number of different virus species.
2.3 Quantum mechanical description

Let us propose a quantum model for how the cells and viruses choose their receptors and ligands: the participants of the game share some (possibly entangled) quantum state $|\psi\rangle$, where the cell type and each virus type constitute a party holding a single qubit. Each specie has two pre-determined quantum operators: $\hat{O}_C^0, \hat{O}_C^1$ for the cells and $\{\hat{O}_{V_k}^0, \hat{O}_{V_k}^1\}_{k=1}^n$ for the viruses. For each cell-virus interaction, the participants share some (possibly entangled) quantum state.

We denote the computational basis of the cell’s and virus’ qubits by $\{\hat{\rho}\}$.

Let us propose a quantum model for how the cells and viruses choose their receptors and ligands:

We assume the cell and virus only measure normalized combinations, i.e., those are treated as inputs choosing the measurement each party performs; thus, for a $C - V_k$ interaction, the correlator is given by:

$$\rho_{ab} = E[CV_k \mid a, b] = \langle \psi | \hat{O}_C^a \otimes \hat{O}_{V_k}^b | \psi \rangle .$$

We denote the computational basis of the cell’s and virus’ qubits by $\{0\}, \{1\}$.

For simplicity, let us assume there is only one virus type, and it shares the following Bell state with the cell:

$$|\psi\rangle = \frac{1}{\sqrt{2}} (|01\rangle + |10\rangle) ,$$

i.e., the cell’s receptors and the virus’ ligands are anti-correlated when measured in the same basis.

We assume the cell and virus only measure normalized combinations,

$$\hat{O}_C^a = \cos \theta_a \hat{Z} + \sin \theta_a \hat{X}, \quad a \in \{0, 1\}$$

$$\hat{O}_{V_k}^b = \cos \varphi_b \hat{Z} + \sin \varphi_b \hat{X}, \quad b \in \{0, 1\} ,$$

where $\hat{Z} = |0\rangle \langle 0| - |1\rangle \langle 1|$ and $\hat{X} = |1\rangle \langle 1| + |0\rangle \langle 0|$ are Pauli operators, and $\theta_a, \varphi_b \in [0, \pi]$.

Using these notations, it is straightforward to derive:

$$\rho_{ab} = \cos(\theta_a - \varphi_b) ,$$

and similarly the following quantities $\eta_c, \eta_v$:

$$\eta_c \overset{\text{def}}{=} \langle \psi | \hat{O}_C^0 \hat{O}_C^1 \otimes \mathbb{1} | \psi \rangle = \cos(\theta_0 - \theta_1)$$

$$\eta_v \overset{\text{def}}{=} \langle \psi | \mathbb{1} \otimes \hat{O}_{V_k}^0 \hat{O}_{V_k}^1 | \psi \rangle = \cos(\varphi_0 - \varphi_1) .$$

Note that $\eta$ is connected with local uncertainty relations in the following manner: $\eta = 1$ implies no uncertainty, and $\eta = 0$ is the maximum uncertainty. In fact, $1 - |\eta|^2$ may be thought of as a quantifier of local uncertainty [18]. Hereon we refer to $\eta_c, \eta_v$ as the virus/cell local uncertainties, respectively.

The magnitude of the cell-virus Bell-CHSH parameter is governed by any one of the two local uncertainties according to

$$|\mathcal{R}_{CV}| \leq 2 \sqrt{2} |\sin(\theta_0 - \theta_1)| ,$$

obtained upon substituting, say, the cell’s local uncertainty.

Substitution of (12) with the respective local uncertainty into our Lotka-Volterra system yields:

$$\frac{d}{dt} \log(c) = \frac{c}{\gamma^c - \frac{\beta}{\sqrt{2}} |\sin(\theta_0 - \theta_1)| v, \quad \frac{d}{dt} \log v \leq - \zeta + \frac{\beta}{\sqrt{2}} |\sin(\varphi_0 - \varphi_1)| c,$$

implying that the specie’s local uncertainty bounds the growth rate of the log population size from below in the case of a cell, and from above in the case of a virus.

The virus (cell) obtains its maximal (minimal) possible growth rate in the scenario where the angles are chosen as follows:

$$\theta_0 = 0, \quad \theta_1 = \frac{\pi}{2}, \quad \varphi_0 = \frac{\pi}{4}, \quad \varphi_1 = - \frac{\pi}{4} .$$

This may be shown by noting that this is precisely the choice of operators saturating the Tsirelson bound for the Bell-CHSH parameter [10], i.e. $\mathcal{R}_{CV} = 2\sqrt{2}$. Equivalently, it saturates (12), i.e., $\eta_c = \eta_v = 0$. 

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6
2.4 Population dynamics over a tripartite quantum network – an example

We have conducted an agent-based simulation of the tripartite quantum network described above (i.e., \( n = 2 \)). The simulation consists of thousands of cells and viruses whose movements in the two-dimensional plane follow a random walk. An interaction takes place once agents of different species are in close proximity. Before the interaction any agent chooses its receptor’s type randomly but conditioned on its state. In addition, the cells have a natural reproduction rate \( \gamma \) and similarly the viruses have a certain death rate \( \zeta \).

In one of the cases where viruses of type \( V_1 \) demonstrated nonlocal correlations with their host cells, the other virus community of type \( V_2 \) was driven to extinction within 500 time steps. See Fig. 3 (right). On the other hand, when both types of viruses exhibited only local correlations with their host cells, the system as a whole remained stable and all communities survived for the entire time span. See Fig. 3 (left).

This phenomenon, termed here monogamy of survival, is a consequence of the monogamy property of correlations. The illustration in Fig. 4 demonstrates this concept more vividly. The pinkish and greenish spheres represent the extinction probability of the different virus communities as evaluated over 500 simulation runs with various initial population spreads. The spheres are plotted over the circle represented by (3). It shows that once the viruses of one community are coordinated with their host cells past any classical correlation the other community has a greater chance of extinction. This chance reaches a maximum when the coordination of viruses from the surviving community is the maximum allowed by quantum mechanics, the Tsirelson limit of \( 2\sqrt{2} \).

2.5 Random correlation networks and ecological systems

Monogamy of survival, the phenomenon where an increase in the extent of coordination in a \( C - V_1 \)-type interaction leads to a decrease in coordination in a \( C - V_2 \)-type interaction, and vice versa, may be extended to large scale networks with many species. Here, however, the superiority of one predator specie over another in terms of its coordination with the prey may not be an adequate measure of the performance of the system as a whole. It seems far more reasonable to consider the stability of the network as manifested by its capacity to sustain ecological communities – the coexistence of a number of predator and prey species, which may nevertheless be engaged in an incessant but never inharmonious struggle for survival (inharmonious in the sense of driving one or more species to extinction). In mathematical terms this translates to the appearance of large connected components in the network [23].

To analyze large scale networks, one normally assumes some generation mechanism, for example that any two nodes have a certain chance of being connected. This is the approach taken in the
Consider an Erdős-Rényi network with a total of $n + 1$ species, namely, one cell specie and $n$ virus species. The correlators in this network, designated as $\rho_{ij}$, represent interactions between the $i$th and $j$th species, and hence also $\rho_{ji} = \rho_{ij}$. Quantum mechanically,

$$\rho_{ij} = \langle \psi | 1^\otimes (i-1) \otimes \hat{O}_a \otimes 1^\otimes (j-i) \otimes \hat{O}_a \otimes 1^\otimes (n+1-j) | \psi \rangle,$$

where $1^\otimes d$ is the $d$-fold unit tensor. This definition accounts for the interactions between predators and their prey as well as between the predators themselves. In addition, we assume that one of the species, the $(n+1)$th, uses two operators, $\hat{O}_{a_{n+1}}$ and $\hat{O}_{b_{n+1}}$, and hence its local uncertainty is given by $\eta = \langle \psi | 1^\otimes n \otimes \hat{O}_{a_{n+1}} \otimes \hat{O}_{b_{n+1}} | \psi \rangle$.

We assume that an interaction between the $i$th and $j$th species, represented by $\rho_{ij} = \rho$, occurs with some probability, $p$, and otherwise, $\rho_{ij} = 0$, when no interaction is known to exist between these species. In other words, the correlator itself is a Bernoulli random variable whose mean is $p \rho$. The same applies for $\rho^a_{ij}$, with $j = n + 1$, and $a = 0, 1$, designating the operator used by the $(n+1)$th specie. This all means that the entire Erdős-Rényi network is associated with a (random) $(n+1) \times (n+1)$ quantum-mechanical correlation matrix whose off-diagonal entries are $\rho_{ij}$, $i,j = 1, \ldots, n$, and $\rho^a_{(n+1)j}$, $\rho^a_{(n+1)(n+1)}$, for $a = 0, 1$.

The assumption of a correlation matrix behind the random graph makes a departure from the classical theory of random networks. Here, the network growth and in particular the likelihood of a giant connected component is governed by both $|\rho|$ and the local uncertainty associated with any one of the species (here only one of them, the $(n+1)$th). This follows from the fact that the correlation matrix must remain positive semi-definite irrespective of the network size $n$ and the chance for interaction $p$.

An Erdős-Rényi graph having $n$ vertices and an edge probability $p$ is commonly denoted as $G(n,p)$. Here, an additional structure is imposed, that of a correlation matrix whose off-diagonal
entries each may either equal zero or \( \rho \). It follows that not all combinations of \( n \), \( p \), and \( \rho \) are feasible in this sort of structure. One may wonder what should these parameters obey when, say, the network increases unboundedly in size. This is of grave concern whenever large connected components are desirable for small binding probabilities \( p \). A large component will almost surely emerge if the network threshold parameter is greater than 1, i.e., if \( np > 1 \) [23].

The following theorem, whose proof may be found in the supplementary material, suggests a relationship between \( n \), \( p \) and \( \rho \) in large scale random correlation networks.

**Theorem 1.** Let \( \rho^* \) be a random variable, the maximum of \( \rho \) in a random correlation network described by \( G(n,p) \). In the \( n \to \infty \) limit, it follows that,

\[
\text{Prob} ( \sqrt{np} \rho^* \leq \zeta_2(g(\eta,p)) ) \to E_{\delta} \left[ W_{sc} \left( \zeta(1 + \delta) - 2 \left( \frac{1 + \delta}{\sqrt{g(\eta,p)}} \right)^3 \right) \right]
\]

where, \( W_{sc}(a) = \pi^{-1} \int_{0}^{a} \frac{1}{\sqrt{4 - x^2}} 1\{x \leq 2\} dx \), the positive half of the Wigner semicircle distribution of radius 2, and \( g(\eta,p) = \min\{1,(1-\eta)/(1-p),(1+\eta)/(1+p)\} \) for some real (local uncertainty) \( \eta \). The random variable \( \delta \) with respect to which the expectation is taken is discrete and bounded by, approximately, \((1-p)/p\). Furthermore, \( \zeta_2(g(\eta,p)) = O(\sqrt{1-\eta}) \), implying,

\[
\lim_{n \to \infty} \sqrt{np} \rho^* = O(\sqrt{1-\eta}),
\]

almost surely.

The theorem conveys the fact that on average the strength of correlations between any two nodes must diminish with an increase in the network threshold parameter \( np \). In particular, it must be of the order \( \sqrt{1-\eta}/\sqrt{np} \) for sufficiently large \( n \). The influence of the local uncertainty parameter \( \eta \), which is here ascribed to only one of the species, enters through \( g(\eta,p) \). It is thus clear that \( \eta \to 1 \) enforces \( \rho \to 0 \), or in words, no local uncertainty implies no correlation. This last observation is one of the characterizing features of the quantum mechanical formalism [24].

### 2.6 Dynamical analysis

Theorem 1 relates the proposed system’s ability of sustaining an ecological community, to the correlations and local uncertainty attributed to the underlying quantum state. This long-term behavior – i.e., the tendency of the system to either accommodate a coexistence between (some of) its predator and prey inhabitants, or drive them to extinction – may be viewed as its dynamical steady-state. However, it is also illuminating to study how the same quantum attributes determine the local (in the sense of configuration space) growth or decay rates, manifested in the system’s Lyapunov exponents.

To do so, we examine a specific class of states for which \( \forall k > 1 \), \( \mathcal{R}_{CV_k} = \mathcal{R} \), i.e., all cell-virus Bell parameters have the same value \( \mathcal{R} \), save perhaps the one relating to the first virus species. We call \( V_1 \) the distinguished virus specie, and \( V_{k>1} \) the homogeneous virus species. Generalizing (3), monogamy of quantum correlations implies (Eq. 52 in [18]):

\[
\sum_{k=1}^{n} (\mathcal{R}_{CV_k})^2 \leq 8, \quad \sum_{k=1}^{n} |\mathcal{R}_{CV_k}| \leq 2\sqrt{2n}.
\]  

(16)

Since we wish to utilize as much entanglement as possible, we shall take states such that the above inequalities are saturated:

\[
\mathcal{R}_{CV_1}^2 + (n-1) \mathcal{R}^2 = 8.
\]  

(17)

Recalling we have denoted \( B_k = \beta \mathcal{R}_{CV_k}/4 \), we substitute \( \mathcal{R}_{CV_1} = 4B_1/\beta \) and \( \mathcal{R} = 4B/\beta \) into (17), to obtain:

\[
B_1^2 + (n-1) B^2 = \beta^2/2,
\]  

(18)

or in polar notation, using the parameter \( 0 \leq \theta \leq \arctan \sqrt{n-1} \):

\[
B_1 = \frac{\beta}{\sqrt{2}} \cos \theta, \quad B = \frac{\beta}{\sqrt{2}(n-1)} \sin \theta.
\]  

(19)
This parameter is closely related to the local uncertainty parameter \( \eta \) defined in the previous section. The two endpoints are of particular interest: for \( \theta = \theta_{\text{max}} := \arctan \sqrt{n - 1} \) the Bell parameters all equal \( 2\sqrt{2/n} \) - this is the equally-correlated case \( (\eta = 1) \); and for \( \theta = 0 \), \( B_{CV} \), obtains its Tsirelson bound while all the other Bell parameters are zero, so we dub this the maximally-entangled case \( (\eta = 0) \). As an aside, note that for \( n = 1 \) the cases converge; if there is only one virus specie, (16) reduces to Tsirelson’s inequality, and it is saturated only for \( \theta = 0 \). Moreover, for \( n \geq 2 \) in the equally-correlated case, none of the Bell parameters violate any of their respective Bell inequalities. We further assume that all virus species have the same decay rate, i.e. \( \forall \) virus specie, 

\[ \text{On the other hand, there exists some results of our analysis follow. and examine the system’s Lyapunov exponents in the vicinity of } \theta = \theta_{\text{max}} \text{, the 'virus' Lyapunov exponent'; the dynamics of each of these populations are described using both cell and (total) virus populations. In other words, there is no 'cell’s Lyapunov exponent' or } \theta > \theta_{\text{max}}, \text{ such that there are no positive Lyapunov exponents for any } \theta > \theta_{\text{max}}. \]

\[ \text{On the other hand, there exists some } n_{c} = n (\beta, \gamma, \lambda, \theta) \text{ such that for } n \leq n_{c}, \text{ the system admits at least one non-negative Lyapunov exponent (however } n_{c} = 0 \text{ in some range of the parameters; for details refer to the paragraph following Eq. 19 in the supplementary material). Note that the dynamical modes (eigenvectors) corresponding to } \lambda_{k} \text{ are nontrivial linear combinations of the cell and (total) virus populations. In other words, there is no “cell’s Lyapunov exponent” or “virus’ Lyapunov exponent”; the dynamics of each of these populations are described using both eigenvalues.} \]

Conversely, as \( \theta \) is decreased (i.e. the strength of entanglement is increased), at some point the real parts of two Lyapunov exponents increase as well. In the maximally-entangled case \( (\theta = 0) \), again the third eigenvalue degenerates to \( \lambda_{h} \), corresponding to all dynamical modes of the entire homogeneous virus population; this is unsurprising, since in this case the homogeneous viruses do not interact with the cells, and therefore die off by their static death rate \( \zeta \). Whether the remaining two Lyapunov exponents form a complex conjugate pair, a single value with multiplicity 2 or two distinct real numbers, depends on whether \( \delta \) is smaller, equal or larger than \( \delta_{c} = 2\sqrt{2/\beta} \), respectively. This behavior may be observed in Fig. 5, and an analytical treatment appears in the supplementary material. Here the largest Lyapunov exponent may be positive, depending on the relations between \( \gamma, \zeta \) and \( \beta \); specifically, for \( \delta \leq \delta_{c} \), the Lyapunov exponent has a positive real part iff \( \gamma > \zeta \) - i.e., the cells procreate faster than the viruses die off.
3 Discussion

Our aim here was to study the effects of quantum mechanical features on population dynamics. Specifically, we have constructed a model for an ecological system with trophic interactions, where the consequences of each pairwise interaction depend on the outcome of a quantum game. This has allowed for quantum-informational quantities to enter the Lotka-Volterra equations via the interaction parameters.

A natural description for this ecological system has been obtained by defining a random quantum correlation network, which is an Erdős-Renyi graph with the added structure of a quantum correlation matrix. This description has allowed a derivation of our main result, Theorem 1, which alludes to how the attributes of the quantum system (i.e., the correlations and counterfactual definiteness) determine the steady-state behavior of the system, that is, whether its inhabitant species will coexist or go extinct.

Moreover, we studied the system’s local behavior using a second approach: by treating it as a dynamical system and computing its Lyapunov exponents. Our in-depth analytical study demonstrates a striking impact of the strength of entanglement utilized in the quantum game, over the asymptotic population dynamics. This is illustrated best when considering the two extremes: in an equally-correlated system with a large enough number of virus species, the largest Lyapunov exponent approaches \(-\zeta\), where \(\zeta\) is the virus’ “static” decay rate (i.e. without interactions). Conversely, in a maximally-entangled system, the behavior of the two largest Lyapunov exponents depends on whether \(\delta = \gamma + \zeta\), the cells’ static procreation rate added to the virus’ static decay rate, exceeds some critical value proportional to the interaction strength parameter \(\beta\). Those two cases can also be defined in terms of \(\eta\), the cell’s local uncertainty parameter: in the equally-correlated case \(\eta = 1\), while \(\eta\) vanishes in the maximally-entangled case. This allows us to compare our two approaches: the steady-state behavior from the network approach, and the local behavior from the dynamical system approach. To summarize:

- **The equally-correlated case with many virus species** \((\eta = 1, n > n_c)\): the steady-state analysis predicts the ecological system will be driven to extinction, while the dynamical analysis predicts population decay (negative Lyapunov exponents).
- **The equally-correlated case with few virus species** \((\eta = 1, n < n_c)\): the dynamical analysis predicts possibility of coexistence (there exists a positive Lyapunov exponent); the populations may either grow or decay, depending on the relations between the parameters \(\beta, \gamma, \zeta\).
- **The maximally-entangled case** \((\eta = 0)\): the steady-state analysis predicts the species may coexist, while the dynamical analysis predicts the populations may either grow or decay.

Our simulation further supports these findings, illustrating the stability of the system in an equally-correlated scenario with \(n = 2 < n_c\), and its potential for instability in the maximally-entangled one. Moreover, the simulation suggests that this instability manifests itself in the extinction of the homogeneous virus species, and survival of the distinguished virus specie.

Follow-up studies may wish to utilize variants of the Lotka-Volterra equations, such as competitive Lotka-Volterra systems, or any other system describing population dynamics where the inter-species interactions are not necessarily of the predator-prey type; for instance, one may wish to consider collaborating species. Other types of quantum games can be used as well to describe the interactions, e.g. games involving several measurements on each side, multiple (rather than binary) measurement outcomes, or hyperentangled states. Another intriguing direction is finding a deeper connection between the dynamics and the network-theoretic description of the system. Such a connection could allow to study scenarios where the quantum features of the network affect the dynamics in a more refined manner, such as considering the time evolution of the underlying quantum state as well. Alternatively, one may wish to study how noise and decoherence affect the dynamics.

Furthermore, it would be interesting to find or develop experimental setups (which could be comprised of physical/chemical systems, biological environments or other setups) which correspond to some variant of our model. This would not only allow to test the theory developed here, but could also lead to several far-reaching applications. For example, if we wish for the cell population
(or its respective equivalent in the actual setup) to be immune to malevolent viruses, one could consider adding a new type of “virtual” (harmless) virus, and entangling it with the cells to the maximal extent possible; then, monogamy of entanglement would disallow any interactions between the malevolent viruses and the cells. Additionally, since the Lotka-Volterra equations can be used to model chemical reactions, it may be possible to use some variation of our system to study the effects of entanglement on kinetics; i.e., could one somehow use entanglement as a catalyst?

4 Methods

This section presents some of the mathematical details underlying the dynamical analysis of the proposed system, discussed previously on the subsection titled “Dynamical analysis”. A fully-detailed treatment appears in the supplementary material.

A local description of the dynamical system is obtained by linearizing it in the neighborhood of some chosen “typical” point, i.e. writing down the Jacobian matrix, and then finding its eigenvalues - these are the Lyapunov exponents of the system. As explained before, it then becomes clear that only three of those Lyapunov exponents are of interest for our purpose of observing the quantum effects. These arise as the solutions \( \lambda \) for the following cubic equation:

\[
(\gamma - [(n - 1) B + B_1] v - \lambda) (-\zeta + B c - \lambda) (-\zeta + B_1 c - \lambda) + B^2 c v (-\zeta + B c - \lambda) + (n - 1) B^2 c v (-\zeta + B_1 c - \lambda) = 0.
\]  

The next step is substituting (19) for \( B, B_1, c, v = 1 \) (this is our chosen point of interest), and denoting \( \delta := \gamma + \zeta \). Under these substitutions, the solutions of this equation (for specific choices of the parameters \( n, \beta, \delta \)) appear on the top-left part of Fig. 5.

When considering either the maximally-entangled (\( \theta = 0 \)) or the equally-correlated (\( \theta = \theta_{\text{max}} \)) cases, one of the three Lyapunov exponents degenerates to the “insignificant” value \( \lambda_h \) associated with shifts within the homogeneous virus species. Thus, only two significant Lyapunov exponents remain. In the maximally-entangled case, this happens because the homogeneous virus populations decouple from the rest of the system (they no longer interact with the cells). Thus, effectively we have a system of two first-order ODEs, corresponding to the cells and distinguished virus specie populations. It is also apparent that the parameters of this effective system do not depend on \( n \). The Lyapunov exponents, plotted as a function of \( \delta \), appear on the top-right part of Fig. 5.

Conversely, in the equally-correlated case the distinguished virus specie has the same parameters as the homogeneous species, so in fact we can treat all virus species homogeneously as a single-specie population. This is achieved by defining \( \bar{v} \) to be the average of all virus populations, \( \bar{v} := \frac{1}{n} \sum_{i=1}^{n} v_i \), and then writing down a system of two first-order ODEs depending only on \( c \) and \( \bar{v} \), i.e. the cell population and the average virus population. Clearly, all the dynamics within the virus populations - dynamical modes where \( \bar{v} \) and \( c \) are preserved and only \( v_i \) change - are “factored out” this way, and again we obtain an effective system with only two significant Lyapunov exponents. These are plotted as a function of \( n, \delta \) in the bottom-left part of Fig. 5; the bottom-right part illustrates only the \( n \)-dependence, for constant \( \delta = 4 \).
Figure 5: Top left: The real and imaginary parts (up to sign) of the three significant Lyapunov exponents, plotted as a function of $\theta$, with parameters $n = 100$, $\beta = 1$ and $\delta = 4$. Note there is a range of $\theta$ values where two of the Lyapunov exponents form a complex conjugate pair (here we only depict the one with positive imaginary part). The positivity of the Lyapunov exponents depends on the value of $\zeta$. Since $0 < \zeta < \zeta + \gamma = \delta$ ($= 4$ in this case), the answer generally depends on the relation between $\gamma$ and $\zeta$, i.e. the cell reproduction rate and virus decay rate, respectively.

Top right: The real and imaginary parts (up to sign) of the two significant Lyapunov exponents $\lambda$ as a function of $\delta$, plotted for $\beta = 1$ and $\theta = 0$; i.e., the maximally-entangled case. In this case the Lyapunov exponents no longer depend on $n$. Note the sharp transition occurring at $\delta_c = 2\sqrt{2}\beta$: for $\delta \leq \delta_c$, $\Re(\lambda) = (\gamma - \zeta)/2$, and the two Lyapunov exponents comprise a complex conjugate pair; thus, we may expect unstable behavior if $\gamma > \zeta$. However, for $\delta > \delta_c$ this is no longer true, as the upper branch exceeds the continuation of the line $\Re(\lambda) = \delta/2 - \zeta$.

Bottom left: the real parts of the two significant Lyapunov exponents $\lambda$ as a function of $n$ and $\delta$, plotted for $\theta = \theta_{\text{max}}$ and $\beta = 1$ (the equally-correlated case). The plot to the bottom right depicts the ($\delta = 4$)-cut of the left plot, and illustrates both the real and imaginary parts (up to sign) of the Lyapunov exponents. The Lyapunov exponents form a complex conjugate pair for $\delta_1 < \delta < \delta_2$, where $\delta_1 = (n+1)\beta/2n$, and the imaginary part obtains its maximal absolute value for $\delta = \sqrt{\frac{n+1}{2n}}$. Note that for small values of $n$, the plot resembles the maximally-entangled case. This is not surprising, since for $n = 1$ the two cases converge. Large $n$ behavior can be observed as well: note how the larger Lyapunov exponent approaches $-\zeta$, while the smaller one approaches $\gamma - \beta \sqrt{n/2}$. 

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Data availability
All data needed to evaluate the conclusions in the paper are present in the paper. Additional data related to this paper can be provided by the authors upon reasonable request.

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Author contributions
A.C. and E.C. conceived the project and supervised the research; A.C. and B.P. coded the simulation and generated the figures; B.P., A.T. and A.C. performed the analytical study; E.C., A.T., B.P. and A.C. co-wrote the article.

Competing interests
The authors declare that they have no competing interests.

Additional information
The online version contains supplementary material available at TBD
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