

University of Warsaw
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Doctoral thesis

**Time Delays and Structured Populations
in Evolutionary Game Theory**



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Supervisor's statements

Hereby, I confirm that the presented thesis was prepared under my supervision and that it fulfills the requirements for the PhD degree in the discipline of Mathematics.

December 2025

..... Jacek Miękisz

Author's statement

Hereby I declare that the presented thesis was prepared by me and none of its contents was obtained by means that are against the law. The thesis has never before been a subject of any procedure of obtaining an academic degree. Moreover, I declare that the present version of the thesis is identical to the attached electronic version.

December 2025

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Abstract

In this thesis, we investigate how time delays and structured populations shape evolutionary game dynamics, focusing on both deterministic and stochastic models. Our models are based on differential equations with time delays and stochastic processes, both ergodic and with absorbing states.

In the first part, we investigate the effect of strategy-dependent time delays on differential replicator equations with logistic population growth. We demonstrate that depending on the type of delay, oscillatory dynamics may emerge, leading to bifurcation phenomena. In the next step, we develop a compartmental model that divides the population into juvenile and adult classes, where the time delay is incorporated as the maturation rate, described by coupled differential equations. We then construct a stochastic version of our model, a Markov jump process. Our analyses show that while deterministic dynamics with delay can generate oscillations via Hopf bifurcations, the inclusion of stochasticity reveals new behaviors absent in the deterministic setting. In particular, delayed maturation combined with noise can promote cooperation in scenarios where deterministic models predict no such advantage, thereby highlighting delay as a potential mechanism favoring cooperation.

In the second part of the dissertation, we deal with Moran birth-death processes in finite populations represented by Markov chains with two absorbing states. We first investigate how the initial placement of a mutant arising in a population of residents in structured populations influences the fixation process. Using Monte Carlo simulations and mean-field approximations, we demonstrate that fixation probability and extinction times depend strongly on the degree of the mutant node, especially in a scale-free network with mutants starting on hubs facing markedly different prospects from those starting on low-degree nodes. We then introduce delayed payoffs into the classical Moran process in well-mixed populations, embedding time delay directly into reproduction probabilities. By combining numerical calculation with simulations, we show that delayed payoffs reshape both fixation probabilities and fixation times in a game-dependent way: delays reduce fixation in coordination and dominance games, but enhance it in coexistence games, with fixation times displaying non-monotone dependence on delay length.

In the third part, we examine cooperation in structured populations. First, we incorporate participation costs into the original Prisoner's Dilemma game on scale-free networks with the Boltzmannian stochastic update rule. Using Monte Carlo simulation for our ergodic Markov chain and a pair approximation, we show that cooperation undergoes a sharp phase transition at a critical cost, with cooperation levels dropping abruptly once the cost exceeds the threshold. Finally, we investigate strategy-dependent delays numerically in spatial Prisoner's Dilemma games on a lattice network, revealing that postponing defector payoffs can stabilize cooperation

by promoting larger cooperative clusters, whereas delaying cooperators undermines reciprocity, with moderate delays sometimes accelerating cluster growth before excessive latency destabilizes order.

Taken together, these contributions provide a systematic account of how strategy-dependent time delays, structural heterogeneity, and stochastic effects jointly determine evolutionary outcomes.

Keywords: evolutionary game theory; replicator dynamics; games on graphs; time delays; Moran process; social dilemmas

Streszczenie

W niniejszej rozprawie badamy, w jaki sposób opóźnienia czasowe oraz struktura populacji kształtują dynamikę gier ewolucyjnych, koncentrując się zarówno na modelach deterministycznych, jak i stochastycznych. Nasze modele opierają się na równaniach różniczkowych z opóźnieniami oraz procesach stochastycznych – zarówno ergodycznych, jak i ze stanami pochłaniającymi.

W pierwszej części rozprawy analizujemy wpływ zależnych od strategii opóźnień czasowych na równania replikatorowe z logistycznym wzrostem populacji. Wykazujemy, że w zależności od rodzaju opóźnienia mogą pojawiać się oscylacje prowadzące do zjawisk bifurkacyjnych. Następnie opracowujemy model przedziałowy, w którym populacja dzieli się na klasy młodocianych i dorosłych osobników, a opóźnienie czasowe wprowadzane jest jako tempo dojrzewania opisane sprzężonymi równaniami różniczkowymi. Budujemy również stochastyczną wersję tego modelu w postaci procesu skokowego Markowa. Nasze analizy pokazują, że chociaż dynamika deterministyczna z opóźnieniem może generować oscylacje poprzez bifurkacje Hopfa, to uwzględnienie stochastyczności ujawnia nowe zachowania nieobecne w przypadku deterministycznym. W szczególności, opóźnione dojrzewanie w połączeniu ze szumem może sprzyjać współpracy w sytuacjach, w których modele deterministyczne nie przewidują takiej korzyści, co wskazuje, że opóźnienie może stanowić mechanizm wspierający współpracę.

W drugiej części rozprawy zajmujemy się procesami urodzin–śmierci Morana w skończonych populacjach reprezentowanych przez łańcuchy Markowa z dwoma stanami pochłaniającymi. Najpierw badamy, jak początkowe umiejscowienie mutantu pojawiającego się w populacji rezydentów w populacjach o strukturze przestrzennej wpływa na proces fiksacji strategii. Wykorzystując symulacje Monte Carlo oraz przybliżenia pola średniego, pokazujemy, że prawdopodobieństwo fiksacji i czas wymarcia silnie zależą od stopnia wierzchołka mutantu – zwłaszcza w sieciach bezskalowych, gdzie mutanci pojawiający się w wierzchołkach–hubach mają znacznie inną siłę niż ci w wierzchołkach o małym stopniu. Następnie wprowadzamy opóźnione wypłaty do klasycznego procesu Morana w dobrze wymieszanych populacjach, włączając opóźnienie bezpośrednio do prawdopodobieństw reprodukcji. Łącząc obliczenia numeryczne z symulacjami, pokazujemy, że opóźnione wypłaty modyfikują zarówno prawdopodobieństwa, jak i czasy fiksacji w sposób zależny od typu gry: opóźnienia zmniejszają fiksację w grach koordynacyjnych, lecz zwiększają ją w grach antykoordynacyjnych, przy czym czasy fiksacji wykazują niemonotoniczną zależność od długości opóźnienia.

W trzeciej części rozprawy badamy współpracę w populacjach o strukturze przestrzennej. Najpierw wprowadzamy koszt uczestnictwa do klasycznej gry Dylematu Więźnia na sieciach Barabasi-Alberty z wykorzystaniem boltzmannowskiej stochastycznej reguły aktualizacji. Stosując symulacje Monte Carlo dla ergodycznego

łańcucha Markowa oraz przybliżenie par, wykazujemy istnienie przejścia fazowego przy krytycznym koszcie – poziom współpracy spada nagle po przekroczeniu krytycznego kosztu. Na końcu badamy numerycznie zależne od strategii opóźnienia w przestrzennych grach Dylematu Więźnia na sieci kwadratowej. Pokazujemy, że opóźnienie wypłat zdradzających może stabilizować współpracę poprzez promowanie większych klastrow kooperantów, podczas gdy opóźnianie kooperantów ją osłabia.

Podsumowując, przedstawione w pracy wyniki stanowią systematyczny opis tego, jak zależne od strategii opóźnienia czasowe, strukturalna heterogeniczność oraz efekty stochastyczne wpływają na ewolucję populacji.

Słowa kluczowe: teoria gier ewolucyjnych; dynamika replikatorowa; gry na grafach; opóźnienia czasowe; proces Morana; dylematy społeczne

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Chapter 1

Introduction

1.1 General background

Game theory is a major branch of applied mathematics that provides a rigorous framework for analyzing strategic interactions between decision-makers with potentially conflicting interests. Originating in economics, game theory is now widely used across disciplines ranging from biology to computer science. In a game-theoretic model, agents (called *players*) choose among available strategies, and the outcome of their interactions is quantified through *payoffs*. These payoffs depend not only on an individual's own strategy but also on those chosen by others. The central solution concept is the *Nash equilibrium*, introduced by John Nash in the 1950s [1, 2]. At a Nash equilibrium, no player can improve their outcome by unilaterally changing strategy, given the strategies of others.

The roots of game theory, however, date back much earlier. Cournot (1838) analyzed duopoly competition [3], while Bertrand (1883) and Borel (1921) contributed further mathematical formulations of strategic interaction [4, 5]. Von Neumann's minimax theorem in 1928 [6] provided the first rigorous foundation for two-player zero-sum games, culminating in his joint work with Morgenstern, *Theory of Games and Economic Behavior* (1944), which established the modern mathematical framework of the field [7]. Later refinements by Selten and Harsanyi expanded the theory to sequential and incomplete-information games [8, 9], for which they, together with Nash, were awarded the Economics Prize in Memory of Alfred Nobel in 1994. Since then, game theory has grown into a central methodology in the social, biological, and computational sciences.

While classical game theory is based on rational-choice principles, it has limitations. In many canonical settings, such as the Prisoner's Dilemma, Nash equilibria predict outcomes that are inefficient for all participants [10]. This tension between individual rationality and collective welfare raised the question of how real populations coordinate, adapt, and evolve over time. To address this, *evolutionary*

game theory (EGT) emerged in the 1970s through the pioneering works of Maynard Smith and Price [11, 12]. EGT shifts the perspective from perfectly rational individuals to populations of agents whose strategies evolve based on their success. Strategies associated with higher payoffs (interpreted as fitness or reproductive success) spread over time through imitation, learning, or reproduction.

A hallmark of EGT is its treatment of frequency-dependent selection, where the payoff to a strategy depends on the distribution of strategies within the population, so that each individual's success is determined by its average payoff obtained through interactions with others. This fundamental feature captures how the success of individuals is shaped not only by their inherent traits but also by interactions with others in a shared environment. Such feedback between strategy composition and payoff structure gives rise to diverse evolutionary outcomes, including stable coexistence through negative frequency dependence (e.g., Hawk–Dove games), coordination through positive frequency dependence (e.g., Stag Hunt), and cyclical dominance (e.g., Rock–Paper–Scissors) [13–15]. To characterize the persistence of strategies under such conditions, Maynard Smith introduced the concept of the *Evolutionarily Stable Strategy* (ESS)—a refinement of the Nash equilibrium that is resistant to invasion by rare mutants [12]. The ESS provides a static criterion for evolutionary stability, specifying when a resident strategy can withstand perturbations due to mutant types.

While the ESS formalism offers a static view of stability, evolutionary game theory also aims to understand the underlying dynamics that lead populations toward or away from equilibrium. The replicator equation [15–18] plays a central role in this framework by describing how the frequency of each strategy changes proportionally to its relative payoff compared to the population average. It provides a dynamic representation of selection and allows for the analysis of transient and asymptotic behaviors in large, well-mixed populations. Within this dynamic context, equilibria of the replicator equation correspond to potential rest points whose stability determines whether evolutionary perturbations dissipate or amplify. This approach establishes a natural connection between dynamic and static notions of stability—an ESS typically corresponds to a stable fixed point of the replicator dynamics—thereby linking behavioral stability with population-level evolutionary trajectories [15, 16].

Deterministic models provide analytical clarity but neglect the role of random fluctuations. In finite populations, stochastic effects can dramatically alter outcomes: even advantageous mutants may die out by chance, while disadvantageous ones may fixate. Markov chain models such as the Moran process [22, 23] are central tools for quantifying absorption probabilities and times. These quantities measure, respectively, the likelihood that a mutant strategy takes over a population (or goes

extinct) and the expected time for this to occur. They connect individual-level randomness with macroscopic evolutionary outcomes.

Another crucial ingredient of evolutionary dynamics is *population structure*. Real populations are rarely well mixed but instead interact on networks, lattices, or spatial domains. The framework of evolutionary games on graphs, introduced by [24, 25], models structured populations by assigning individuals to the vertices of a graph, with edges representing possible interactions. Each individual's payoff is determined as the sum or average of the payoffs obtained from playing the game with its neighboring individuals. This approach was later examined by Lieberman et al. [26], who introduced the concept of *Evolutionary Graph Theory* and investigated the fixation probability of mutants in various population structures. Structure can amplify or suppress natural selection, influence fixation probabilities, and alter the speed of evolution [30–32]. Spatial clustering allows cooperators to survive by forming protective groups, while heterogeneous degree distributions in scale-free networks can dramatically reshape cooperative outcomes [33, 34]. This recognition that *structure matters* has been one of the most influential developments in the study of cooperation.

A central question in both biology and the social sciences is the persistence of cooperation despite incentives for selfish behavior. Defection often provides higher individual payoffs, yet cooperation is essential for the functioning of societies, ecosystems, and collective endeavors [22, 35]. Classical games such as the Prisoner's Dilemma, the Snowdrift game, and Public Goods games model the tension between individual incentives and group benefits [15, 36, 37]. Over the decades, numerous cooperation-promoting mechanisms have been identified, including kin selection [38], reciprocity [39, 40], group selection [41], and particularly *network reciprocity*, where structured interactions enable cooperation to persist [34, 43–46]. These mechanisms show that both stochasticity and structure deeply influence the evolution of cooperation.

Beyond spatial and network effects, temporal aspects of interaction are equally important. Time lags are inherent in biological, ecological, and socio-economic processes [47–50]. Gestation and maturation defer demographic renewal; production cycles postpone material returns; and information must be collected and processed before behavioural revision occurs. Models that assume instantaneous cause and effect, therefore, omit an essential characteristic of real-world dynamics. Introducing an explicit *time delay* compensates for this omission while preserving the low dimensionality of ordinary differential systems. The present state then depends on past performance, with relevant information arriving only after a finite latency. This modification generates phenomena—such as sustained oscillations, cycles, and Hopf bifurcations—that are impossible in memory-free formulations [48, 50]

Within evolutionary game theory, the replicator equation is the canonical model of frequency-dependent selection. The delay-free version has been studied extensively, and its equilibria and stability conditions are well established [15, 16]. Empirical evidence, however, indicates that assumptions of instantaneous payoff evaluation and immediate behavioral adjustment rarely hold. Consequently, incorporating time delays into evolutionary game models has become a natural and important extension, attracting significant research attention [51–58]. Delays can be classified by type—*social* versus *biological*— [51], as well as by strategy, from uniform to strategy-dependent delays [57, 58]. These variants exhibit qualitatively distinct stability properties: social delays may destabilize equilibria once the delay exceeds a critical value, while biological delays can preserve global stability [51] but change the location of interior equilibria when we have strategy-dependent delay [57]. Because a delay differential equation maps a history function to another function over time, rather than a point to a point, the introduction of delay effectively extends the state space to an infinite-dimensional function space. Nevertheless, delayed replicator models often remain analytically manageable and conceptually transparent.

This perspective highlights that understanding evolutionary processes requires considering not only stochasticity and structure but also the role of *time delays*. It is within this triad—*delay*, *structure*, and *stochasticity*—that the subsequent chapters of this thesis are situated.

1.2 Thesis overview

This thesis is structured as a collection of published research articles, each addressing different facets of evolutionary game theory with a focus on time delays and structured populations. Together, they form a coherent narrative: starting from delayed replicator equations in infinite or density-regulated populations, moving to fixation probabilities and absorption times in finite populations with and without delays, and finally examining cooperation in spatial and network-structured systems under the influence of interaction costs and delayed payoffs. By unifying these contributions into a single framework, the thesis provides a systematic understanding of how delays, structure, and stochasticity jointly shape evolutionary dynamics. Here we provide a brief overview of these publications, while Chapter 3 presents a detailed summary of each study, followed by the full published papers.

A. Strategy-dependent time delays in replicator systems with logistic growth

This study investigates how strategy-dependent social delays affect the stability of frequency-dependent populations with density regulation [94]. Using delayed repli-

cator equations with logistic growth in the Snowdrift game, we show that symmetric delays maintain stability up to a critical threshold, beyond which a supercritical Hopf bifurcation generates oscillations. Asymmetric delays play a decisive role: postponing defectors' responses destabilises coexistence, whereas delaying cooperators preserves it. Demographic feedback thus interacts with temporal delays to create richer dynamics than in unregulated models [51, 55, 56].

B. Demographic noise and maturation delays in structured populations

The second work explores how intrinsic fluctuations and delayed maturation modify deterministic outcomes [95]. A structured replicator framework with juvenile compartments is developed, and the corresponding stochastic birth–death process is analysed using Gillespie simulations. While deterministic coexistence persists under symmetric delays, stochastic effects raise cooperation levels. Moderate asymmetries further enhance cooperation even when cooperators mature more slowly, showing that demographic noise and delayed life-history stages can favour cooperation contrary to deterministic predictions [57].

C. Network heterogeneity and fixation in structured populations

Here, we examine how the degree of an initial mutant influences fixation probability and absorption time in heterogeneous networks [96]. Simulations and mean-field analysis of Moran Birth–death dynamics on Erdős–Rényi and Barabási–Albert graphs reveal that fixation probability decreases with the mutant's degree, and extinction occurs faster on nodes with a higher degree.

D. Time-delayed fitness and fixation in finite populations

This study incorporates discrete time delays into the Moran Birth–death process to assess their impact on fixation probabilities and times in social dilemma games [97]. By enlarging the state space to include historical compositions, we compute exact absorption measures via Markov-chain methods. Results show that delays suppress fixation in coordination and dominance games but enhance it in coexistence games, with fixation times varying non-monotonically with delay length. For large delays, outcomes converge to classical constant-fitness limits.

E. Participation costs and phase transitions in networked cooperation

The fifth paper investigates how interaction costs alter cooperative dynamics on Barabási–Albert networks in Prisoner's Dilemma game [98]. Introducing a participation cost per link, we find a sharp transition from full cooperation to mixed

states. Near the critical cost, the stationary distribution becomes bimodal, indicating metastable coexistence of cooperative and defective phases. Pair approximation accurately predicts this critical transition, clarifying how structural burdens undermine hub-mediated reciprocity.

F. Strategy-dependent delays in spatial Prisoner's Dilemma game

The final study analyses spatial reciprocity on square lattices under distinct delays for cooperators and defectors in Prisoner's Dilemma game [99]. Simulations reveal that delaying defectors promotes cooperation behavior, while delaying cooperators favors defection. Moderate delays promote the formation of large cooperative clusters before excessive delays disrupt spatial order, demonstrating how heterogeneous delayed payoffs reshape cooperation patterns in structured populations.

Publications included in the thesis

- A. **Javad Mohamadichamgavi** and Marek Bodnar. Bifurcation analysis of replicator dynamics with logistic growth and strategy-dependent time delays in Snowdrift game. *Dynamic Games and Applications*: 1–29 (2025).
- B. Jacek Miękisz and **Javad Mohamadichamgavi**. Intrinsic noise in structured replicator dynamics modelling time delays. *Dynamic Games and Applications* 15: 1364–1377 (2025).
- C. **Javad Mohamadichamgavi** and Jacek Miękisz. Effect of the degree of an initial mutant in Moran processes in structured populations. *Physical Review E* 109(4): 044406 (2024).
- D. **Javad Mohamadichamgavi** and Mark Broom. The impact of time delays on mutant fixation in pairwise social dilemma games. *Proceedings of the Royal Society A* 480(2300): 20240195 (2024).
- E. Jacek Miękisz and **Javad Mohamadichamgavi**. Phase transitions in the prisoner's dilemma game on the Barabási–Albert graph with participation cost. *Physical Review E* 112(3): L032302 (2025).
- F. **Javad Mohamadichamgavi**. Strategy-dependent time delays shape cooperation in the spatial prisoner's dilemma game. *Chaos, Solitons & Fractals* 201: 117255 (2025).

Other Publications

During my doctoral research, I was also involved in several collaborative and interdisciplinary projects beyond the core topics presented in this thesis. These studies, conducted in cooperation with international research teams, explore diverse aspects of evolutionary game theory, network science, and socio-behavioral modelling. Although they are not directly included in this dissertation, they represent important extensions of my scientific activity and collaborative research experience developed during the PhD period.

1. Christo Morison, Małgorzata Fic, Thomas Marcou, **Javad Mohamadichamgavi**, Javier Redondo Antón, Golsa Sayyar, *et al.* *Public goods games in disease evolution and spread. Dynamic Games and Applications* (2025).
2. Wajid Ali, **Javad Mohamadichamgavi**, Juan Manuel Ramírez, José Aguilar, Antonio Fernández Anta, and Kenneth J. Sharkey. *A mathematical model of vaccine hesitancy: political trends and demographic interactions in the USA*. Submitted to *Nature Human Behavior*, arXiv:2509.07712 (2025).
3. **Javad Mohamadichamgavi** and Mohamadreza Zahedian. *The effect of initial placement of a mutant in a subdivided population on fixation probability and time. Advances in Complex Systems* 28(5): 1–24 (2025).
4. **Javad Mohamadichamgavi**, Mahdi Hajihashemi, and Keivan Aghababaei Samani. *An analysis of correlation and comparisons between centrality measures in network models. Journal of Social Structure* 25(1) (2024).
5. Jacek Miękisz, **Javad Mohamadichamgavi**, and Jakub Łącki. *Phase transitions in the Prisoner's Dilemma game on scale-free networks. BioPhysMath* 1: 1–9 (2024).
6. Jacek Miękisz, **Javad Mohamadichamgavi**, and Raffi Vardanyan. *Small time delay approximation in replicator dynamics. arXiv:2303.08200* (2023).

Chapter 2

Introduction to evolutionary game theory

This chapter collects the minimal mathematical foundations used throughout the thesis: (i) classical game-theoretic notions (strategies, payoffs, Nash equilibria), (ii) evolutionary population dynamics via the replicator equation and its geometric interpretation, (iii) the link between replicator stationary points and Nash equilibria with canonical two-strategy regimes, (iv) delayed replicator models (social vs. biological), (v) stochastic dynamics in finite populations via the Moran process and its Markov chain analysis, and (vi) structured populations (payoff accumulation, update rules, mutation, and network effects), together with mean-field and pair approximations.

2.1 Game theory basics

Classical game theory focuses on rational best responses in one-shot (or finitely repeated) interactions, where individuals are assumed to make optimal decisions given full knowledge of their opponents' strategies. In contrast, evolutionary game theory (EGT) shifts the perspective from individual rationality to population dynamics: strategies that yield above-average payoffs (fitness) become more common over time through processes such as reproduction, imitation, or learning [12, 15, 16, 21, 59]. This framework captures how behavioral patterns emerge and stabilize in large populations, even when individuals are boundedly rational or act based on simple adaptive rules.

2.1.1 Strategies, payoffs, and normal-form games

Definition 2.1.1 (Normal-form game). *A finite normal-form game is a triple $\mathcal{G} = (I, \{S_i\}_{i \in I}, \{U_i\}_{i \in I})$, where $I = \{1, \dots, n\}$ is the player set, $S_i = \{1, \dots, m\}$ the pure strategies of player i , and $U_i : S_1 \times \dots \times S_n \rightarrow \mathbb{R}$ the payoff of player i .*

A pure strategy profile is $x = (x_1, \dots, x_n) \in S_1 \times \dots \times S_n$, often written $x = (x_i, x_{-i})$. In symmetric two-player games, both players share $S = \{1, \dots, m\}$ and identical payoffs, represented by a matrix $U \in \mathbb{R}^{m \times m}$ with row payoff $u_{k,l}$; by symmetry the column player receives $U^\top$.

2.1.2 Mixed strategies and Nash equilibria

Definition 2.1.2 (Mixed strategy). *For $S_i = \{1, \dots, m\}$, the set of mixed strategies is the simplex $\Delta_i = \{p \in \mathbb{R}_{\geq 0}^m : \sum_{k=1}^m p_k = 1\}$.*

In a two-player symmetric game with payoff matrix U , the expected payoff to row mixing p against column mixing q is $E[p, q] = p^\top U q$. The best-response correspondence is $\text{BR}_i(x_{-i}) = \arg \max_{y_i \in \Delta_i} U_i(y_i, x_{-i})$.

Definition 2.1.3 (Nash equilibrium). *A profile $x^* \in \prod_{i \in I} \Delta_i = \Delta$ is a Nash equilibrium (NE) if, for all i , $x_i^* \in \text{BR}_i(x_{-i}^*)$, equivalently, $U_i(x_i^*, x_{-i}^*) \geq U_i(y_i, x_{-i}^*) \forall y_i \in \Delta_i$. In symmetric two-player games, a symmetric NE is $p^* \in \Delta$ with $p^* \in \arg \max_{p \in \Delta(s)} p^\top U p^*$.*

Theorem 2.1.4 (Nash [2]). *Every finite normal-form game admits at least one Nash equilibrium.*

2.1.3 Classical two-strategy games

Several well-known 2×2 symmetric games form the conceptual backbone of game theory. If we consider $s_i \in A, B$, each is defined by an ordering of the four canonical payoffs $(u_{A,A}, u_{A,B}, u_{B,A}, u_{B,B})$ as follows:

$$U = \begin{array}{c|cc} & A & B \\ \hline A & u_{A,A} & u_{A,B} \\ B & u_{B,A} & u_{B,B} \end{array} = \begin{array}{c|cc} & A & B \\ \hline A & a & b \\ B & c & d \end{array} \quad (1)$$

Prisoner's Dilemma (PD)

This paradigmatic social dilemma illustrates the conflict between individual rationality and collective welfare. Two accomplices in a crime are interrogated separately and offered sentence reductions according to the payoff matrix

$$\begin{array}{c|cc} & C & D \\ \hline C & R & S \\ D & T & P \end{array}, \quad T > R > P > S \quad \text{and} \quad 2R > T + S.$$

Here, T (“temptation”) denotes the payoff a defector receives against a cooperator, R (“reward”) is the mutual cooperation payoff, P (“punishment”) is the mutual

defection payoff, and S (“sucker’s payoff”) is obtained by a cooperator facing a defector. The inequality $T > R > P > S$ and $2R > T + S$ ensures that unilateral defection yields the highest individual gain while mutual defection is worse than mutual cooperation, capturing the essence of the dilemma: rational self-interest leads both players to defect even though they would both benefit more from cooperation. Cooperation (C) is strictly dominated by defection (D), hence (D, D) is the unique strict Nash equilibrium (NE). Yet the best possible payoff outcome (C, C) would leave both players better off [10, 36, 60].

Snowdrift (SD)

The Snowdrift game (also known as Hawk–Dove game or Chicken game) models a social dilemma in which two individuals face a common task, such as clearing a road after a snowstorm, and each can either cooperate by shoveling or defect by avoiding the work [61]. The benefit of a cleared road is v , while the cost of shoveling is c , with $v > c$ ensuring that cooperation is collectively beneficial. The resulting payoff matrix is

	A	B
A	$v - \frac{c}{2}$	$v - c$
B	v	0

The typical payoff ordering for a general payoff matrix satisfies $c > a > b > d$. Two asymmetric pure Nash equilibria, (A, B) and (B, A) , coexist with a symmetric mixed equilibrium in which each individual cooperates with probability $p^* = \frac{2(v-c)}{2v-c}$. This formulation is equivalent to the classical Chicken game [62] and aligns with the ecological interpretation of the Hawk–Dove framework [12], highlighting how partial cooperation emerges when sharing the cost of a common good is advantageous.

Stag Hunt (SH)

The *Stag Hunt* game, inspired by Rousseau’s well-known parable, highlights the conflict between collective gain and individual security [63, 64]. The Stag Hunt game captures situations where cooperation yields the highest mutual payoff, but individuals may prefer a safer, lower-payoff option if they doubt the other player’s cooperation. Whenever $a > c$ and $d > b$, two pure Nash equilibria exist: (A, A) and (B, B) . The cooperative equilibrium (A, A) is *payoff-dominant*, giving the highest total return, but strategic uncertainty often favors the safer option (B, B) . Following Harsányi and Selten, strategy B *risk-dominates* A if $a + b < c + d$, i.e. if a player expecting a uniform $(1/2, 1/2)$ opponent gains more by choosing B .

2.2 From games to populations: replicator dynamics

Population state and payoffs. In a well-mixed population with $S = \{1, \dots, m\}$, the state is $x \in \Delta_m$, a probability distribution on S . For payoff matrix U , the fitness of strategy i against state x is $f_i(x) = (Ux)_i = \sum_j u_{i,j}x_j$, and the mean fitness is $\bar{f}(x) = x^\top Ux$ [65].

2.2.1 Two-strategy replicator dynamics

The simplest setting involves two competing strategies, say A and B , in a large, mixed population. Let x denote the fraction of individuals playing A (hence $1 - x$ play B). If individuals interact randomly, the average payoff to A and B in the population are $U_A(x) = ax + b(1 - x)$, $U_B(x) = cx + d(1 - x)$, where the matrix entries a, b, c, d are the payoffs from the underlying 2×2 game shown in (1). Assuming continuous-time selection dynamics in which the growth rate of each type is proportional to its excess payoff relative to the mean [11, 13, 15, 16, 18, 66], one obtains the two-strategy replicator equation

$$\dot{x} = x(1 - x) (U_A(x) - U_B(x)). \quad (2.1)$$

This one-dimensional ordinary differential equation captures the frequency change of A : if $U_A(x) > U_B(x)$ then x increases, while if $U_A(x) < U_B(x)$ then x decreases. The factor $x(1 - x)$ ensures that both boundary states $x = 0$ and $x = 1$ are stationary points. These correspond to monomorphic populations at the vertices of the simplex. Their stability depends on whether the pure strategy is a strict Nash equilibrium: if a strategy outperforms the other against itself, then the corresponding boundary point is asymptotically stable.

Equation (2.1) allows for straightforward phase line analysis. There may exist an interior equilibrium at a mixed frequency x^* , defined by equality of payoffs to both strategies, $U_A(x) = U_B(x)$. Solving explicitly gives [67]

$$x^* = \frac{d - b}{(a - c) + (d - b)}. \quad (2.2)$$

Whenever $x^* \in (0, 1)$, this interior stationary point corresponds to a mixed Nash equilibrium in which both strategies coexist with positive probability. The stability of the interior equilibrium x^* is determined by the slope of $U_A(x) - U_B(x)$ at that point. When this slope is negative, as in the Hawk–Dove or Snowdrift game, the interior equilibrium is asymptotically stable and the population maintains a stable mixture of strategies. In contrast, a positive slope, typical of coordination scenarios such as the Stag Hunt, makes the interior point unstable so that the dynamics

converge to one of the pure equilibria. If both payoffs favor A ($a > c$ and $b > d$), it becomes globally stable, whereas the opposite inequalities make B dominant. If the payoffs are identical ($a = c$ and $b = d$), every composition $x \in [0, 1]$ remains stationary.

2.3 Replicator dynamics with time delays

Time delays are intrinsic in biological and socio-economic processes [47–50]. Delays alter stability and can induce oscillations and Hopf bifurcations. Two modeling approaches are common [51]: *social delays* and *biological delays*.

2.3.1 Social-type delay

In the social delay, individuals adjust their current frequencies using payoffs that were observed τ time units in the past. Consider a panmictic population in which, during every discrete time-step of length $\varepsilon > 0$, only an ε -fraction of individuals engages in pairwise contests. Let $p_i(t)$ be the number of individuals using strategy $i \in \{A, B\}$ at time t , $p(t) = p_A(t) + p_B(t)$ the population size, and $x(t) = p_A(t)/p(t)$ the frequency of strategy A . Following [51] we consider the symmetric payoff matrix where $c > a$, $b > d$, and unique mixed evolutionarily stable strategy $x^* = \frac{\delta_2}{\delta_1 + \delta_2}$ where $\delta_1 = c - a$, $\delta_2 = b - d$, $\delta = \delta_1 + \delta_2$. Players imitate opponents according to average (time-delayed) payoffs. Denoting the time delay by τ , reproduction is governed by

$$p_i(t + \varepsilon) = (1 - \varepsilon)p_i(t) + \varepsilon p_i(t) U_i(t - \tau), \quad i = A, B, \quad (2.3)$$

with delayed payoffs

$$U_A(t - \tau) = ax(t - \tau) + b(1 - x(t - \tau)), \quad U_B(t - \tau) = cx(t - \tau) + d(1 - x(t - \tau)).$$

Dividing (2.3) by the corresponding relation for $p(t)$ and following by some simplification gives the delay differential equation

$$\dot{x}(t) = -\delta x(t) (1 - x(t)) (x(t - \tau) - x^*) \quad (2.4)$$

Linearizing (2.4) at x^* gives the characteristic equation $\lambda + B e^{-\lambda\tau} = 0$ with $B = \delta x^*(1 - x^*)$, from which one derives the critical value $\tau^* = \frac{\delta\pi}{2\delta_1\delta_2}$. Thus, the interior equilibrium x^* is locally asymptotically stable for $\tau < \tau^*$, loses stability via a Hopf bifurcation at $\tau = \tau^*$, and is unstable for $\tau > \tau^*$ [56]. Intuitively, social delay injects outdated information into the feedback loop, and a sufficiently large lag produces self-sustained oscillations around x^* .

2.3.2 Biological-type delay

In the biological delay, individuals born at time t descend from parents who interacted τ time units earlier. In this setting, we assume that individuals born at time $t - \tau$ become mature and are able to participate in contests at time t . Equivalently, players are born τ time units after their parents engaged in interactions and received payoffs. The corresponding equations take the form

$$p_i(t + \varepsilon) = (1 - \varepsilon)p_i(t) + \varepsilon p_i(t - \tau)U_i(t - \tau), \quad i = A, B, \quad (2.5)$$

where $p_i(t)$ is the number of individuals using strategy i at time t , and $U_i(t - \tau)$ is the payoff of strategy i obtained τ units of time earlier. The natural state variables are the total population size $p(t)$ and the frequency $x(t)$ of one strategy, leading to the coupled delay system

$$\dot{p}(t) = -p(t) + p(t - \tau)\bar{U}(t - \tau), \quad (2.6a)$$

$$\dot{x}(t) = \frac{p(t - \tau)}{p(t)} \left[x(t - \tau)U_A(t - \tau)(1 - x(t)) - (1 - x(t - \tau))U_B(t - \tau)x(t) \right], \quad (2.6b)$$

where $\bar{U}(t - \tau) = x(t - \tau)U_A(t - \tau) + (1 - x(t - \tau))U_B(t - \tau)$ and $U_A(t - \tau) = ax(t - \tau) + b(1 - x(t - \tau))$, $U_B(t - \tau) = cx(t - \tau) + d(1 - x(t - \tau))$.

Here, $p(t)$ declines at a unit rate (death) and increases by births from time $t - \tau$, proportional to past population and mean payoff. Two main differences from the social delay (2.4) arise: (i) biological delay requires tracking both $p(t)$ and $x(t)$ since reproduction depends on delayed numbers of parents, and (ii) for coexistence games, the interior equilibrium x^* remains asymptotically stable for all $\tau \geq 0$, as delayed reproduction acts as a stabilizing feedback, preventing Hopf bifurcations [51].

2.4 Moran process

The classical Moran process considers a finite population of size N composed of two types: residents (R) and mutants (M). It is a discrete-time birth-death model in which, at every update, one individual is selected for reproduction with probability proportional to its fitness, and another (possibly different) individual is replaced uniformly at random [23]. The offspring inherits the parent's type, so the number of mutants (and residents) can change by at most one per step. This simple mechanism, which is a Birth-death (Bd) process, captures the interplay between stochastic drift and selection in finite populations and provides the foundation for much of evolutionary graph theory [26, 28, 68–71].

In a well-mixed population, the chain is lumpable with respect to the number of mutants $i \in \{0, \dots, N\}$. The absorbing states are $i = 0$ and $i = N$. Let ϕ_i^N be the fixation probability of mutants starting from i . The transition probabilities of the induced birth-death chain are

$$P_{i,i+1}, \quad P_{i,i-1}, \quad P_{i,i} = 1 - P_{i,i+1} - P_{i,i-1}, \quad i = 1, \dots, N-1.$$

Let $\gamma_i := P_{i,i-1}/P_{i,i+1}$ denote the local bias. Standard one-dimensional birth-death theory yields [74]

$$\phi_i^N = \frac{1 + \sum_{k=1}^{i-1} \prod_{j=1}^k \gamma_j}{1 + \sum_{k=1}^{N-1} \prod_{j=1}^k \gamma_j}. \quad (2.7)$$

Frequency-dependent selection. If payoffs with i mutants are

$$f_i = \frac{(i-1)a + (N-i)b}{N-1}, \quad g_i = \frac{ic + (N-i-1)d}{N-1},$$

and fitness satisfies $F_i^M = (1-\omega) + \omega f_i$, $F_i^R = (1-\omega) + \omega g_i$, then for the well-mixed Bd update rule,

$$P_{i,i+1} = \frac{iF_i^M}{iF_i^M + (N-i)F_i^R} \cdot \frac{N-i}{N-1}, \quad P_{i,i-1} = \frac{(N-i)F_i^R}{iF_i^M + (N-i)F_i^R} \cdot \frac{i}{N-1}.$$

Hence

$$\gamma_i = \frac{F_i^R}{F_i^M}. \quad (2.8)$$

Substituting (2.8) into (2.7) yields

$$\phi_i^N = \frac{1 + \sum_{k=1}^{i-1} \prod_{j=1}^k \frac{F_j^R}{F_j^M}}{1 + \sum_{k=1}^{N-1} \prod_{j=1}^k \frac{F_j^R}{F_j^M}}. \quad (2.9)$$

Constant relative fitness (Moran's formula). If $F_i^M = r$ and $F_i^R = 1$, then $\gamma_i = 1/r$ and

$$\phi_i^N = \begin{cases} \frac{1 - r^{-i}}{1 - r^{-N}}, & r \neq 1, \\ \frac{i}{N}, & r = 1, \end{cases} \quad (2.10)$$

with neutral fixation time of order $N \log N$ [22].

Moran process on graphs (Evolutionary graph theory) In structured populations, individuals occupy the vertices of a graph $G = (V, E)$, where each vertex carries a mutant or resident. The evolutionary dynamics is a finite Markov chain

on $\Omega = \{0, 1\}^N$ with 2^N possible states.

Under the graph-based Moran process [26, 28], reproduction and replacement are restricted to edges. For the Bd rule,

$$P_{i \rightarrow j}^{\text{Bd}} = \frac{F_i}{\sum_k F_k} \cdot \frac{A_{ij}}{k_i}, \quad k_i = \sum_j A_{ij},$$

where A_{ij} is the adjacency matrix. Symmetric structures (complete, regular, star graphs) allow analytical fixation probabilities [22, 59]. Graphs may act as *amplifiers* or *suppressors* of selection, altering the success of advantageous mutants [26, 27, 29]. Approximation schemes (mean-field, pair approximation) can reduce the full 2^N chain to an effective birth-death process, often recovering well-mixed fixation formulas [73].

2.5 Evolutionary games on graphs

In structured populations, individuals occupy the vertices of a graph $G = (V, E)$ with $|V| = N$. Each vertex hosts an individual using strategy $s_i \in \{A, B\}$ (e.g. cooperation or defection), and interactions occur along edges. Thus the evolutionary process is a finite Markov chain on the configuration space $\Omega = \{A, B\}^N$, where each configuration represents a particular arrangement of strategies on the graph.

Interactions between neighboring vertices i and j are evaluated through a 2×2 payoff matrix

$$U = \begin{pmatrix} u(A, A) & u(A, B) \\ u(B, A) & u(B, B) \end{pmatrix},$$

so that the payoff from a single encounter is simply $u(s_i, s_j)$. Let $\mathcal{N}_i$ denote the neighbor set of i with degree $k_i = |\mathcal{N}_i|$. A player's total payoff is the aggregation of its pairwise interactions, typically taken as either

$$\pi_i = \sum_{j \in \mathcal{N}_i} u(s_i, s_j) \quad (\text{accumulated payoff}),$$

or

$$\pi_i = \frac{1}{k_i} \sum_{j \in \mathcal{N}_i} u(s_i, s_j) \quad (\text{average payoff}).$$

Accumulated payoffs favor high-degree vertices in heterogeneous networks, while average payoffs remove degree dependence. These payoffs determine how strategies spread across the graph.

Strategy updating is local: individuals compare their performance with that of their neighbors and may adopt a neighbor's strategy. Common microscopic update

rules include

$$\text{(Pairwise comparison)} \quad \Pr(s_i \leftarrow s_j) = \frac{1}{2} + \frac{\pi_j - \pi_i}{2\Delta}, \quad \Delta = \max |\pi_j - \pi_i|, \quad (2.11)$$

$$\text{(Boltzmann (Fermi) imitation)} \quad \Pr(s_i \leftarrow s_j) = \frac{1}{1 + \exp[-\beta(\pi_j - \pi_i)]}, \quad \beta > 0, \quad (2.12)$$

$$\text{(Moran Bd)} \quad \Pr(i \text{ reproduces}) = \frac{\pi_i}{\sum_{\ell} \pi_{\ell}}; \text{ offspring replaces } j \in \mathcal{N}_i, \quad (2.13)$$

which are commonly used in evolutionary games on graphs. Updates may be synchronous or asynchronous; the latter, often implemented via Gillespie-type algorithms, avoids artificial synchrony and yields smoother dynamics [71].

In the absence of mutation, the process has two absorbing states, A^N and B^N , corresponding to global fixation of one strategy. A small mutation probability $\varepsilon > 0$ renders the chain irreducible, yielding a unique stationary distribution u^* . The long-run abundance of cooperation is then

$$\bar{\rho}_A = \sum_{s \in \Omega} \rho_A(s) u^*(s),$$

where $\rho_A(s)$ is the fraction of cooperators in configuration s . When mutation is absent, one instead analyzes fixation probabilities or the quasi-stationary distribution describing metastable coexistence before absorption. Overall, evolutionary games on graphs provide a unified microscopic framework for studying how spatial structure shapes cooperation, competition, and selection in finite populations.

2.6 Analytical approximations

2.6.1 Mean-field approximation

The first approximation commonly used for the analytical study of evolutionary games on networks is the Mean-Field approximation (MFA). The central assumption of MFA is the absence of correlations between individuals, so that the probability of a population configuration factorizes into the product of independent single-site probabilities [71, 77]. Under MFA assumptions, the probability of selecting a mixed pair (A–B) is proportional to $\rho_A \rho_B = \rho_A(1 - \rho_A)$. Therefore, the average rate of change of cooperator fraction $\rho_A \in [0, 1]$ is

$$\frac{d\rho_A}{dt} = \rho_A(1 - \rho_A) g(\pi_A, \pi_B), \quad (2.14)$$

where $g(\pi_A, \pi_B) = (\Pr(B \rightarrow A) - \Pr(A \rightarrow B))$ is a function of payoffs dependent on update rule. It is straightforward to demonstrate that for the Fermi rule (2.12), we have $g(\pi_A, \pi_B) = \tanh((\pi_A - \pi_B)/2)$ [71]. For social dilemmas (e.g. Prisoner's Dilemma) $\pi_A - \pi_B < 0$ generically, implying $\rho_A \rightarrow 0$ unless neutrality holds. Thus, in the MFA approximation, there is no stable coexistence of strategies, except in the weak selection case (using a map fitness $f_i = 1 - \omega + \omega\pi_i$ where $\omega \rightarrow 0$). However, simulations of this system with Monte Carlo methods reveal the coexistence of strategies in a structured population in a quasi-stationary state, a phenomenon entirely missed by the MFA.

2.6.2 Pair approximation

A classical way to approximate evolutionary processes on networks is through the pair approximation (PA) method [28, 61, 77–86]. In this approach, the frequencies of higher-order structures (such as triples of nodes connected in a line) are approximated by combinations of lower-order quantities (pairs and single nodes). This method is well-suited for networks with homogeneous degree distributions, and although it can be extended to heterogeneous structures, the number of Equations increase substantially. We restrict our attention to undirected and unweighted graphs.

PA retains nearest-neighbor correlations via pair densities $\rho_{AA}, \rho_{AB}, \rho_{BB}$ with $\rho_A = \rho_{AA} + \frac{1}{2}\rho_{AB}$. Define conditional probabilities $q_{B|A} = \rho_{AB}/\rho_A$, $q_{A|B} = \rho_{AB}/\rho_B$. Assuming locally independent neighbors, the probability that an A -node has exactly i A -neighbors on a k -regular graph is

$$\phi_A(i) = \binom{k}{i} (1 - q_{B|A})^i q_{B|A}^{k-i}. \quad (2.15)$$

Let $\Pr(A \rightarrow B|i)$, $\Pr(B \rightarrow A|i)$ be rule-dependent, neighborhood-conditioned transition probabilities. Then a closed system for (ρ_A, ρ_{AA}) reads

$$\dot{\rho}_A = \rho_B \sum_{i=0}^k \phi_B(i) \Pr(B \rightarrow A|i) - \rho_A \sum_{i=0}^k \phi_A(i) \Pr(A \rightarrow B|i), \quad (2.16)$$

$$\dot{\rho}_{AA} = \frac{2}{k} \sum_{i=1}^k i \left[\rho_B \phi_B(i) \Pr(B \rightarrow A|i) - \rho_A \phi_A(i) \Pr(A \rightarrow B|i) \right]. \quad (2.17)$$

PA captures coexistence and threshold effects absent in MFA, while remaining analytically tractable [78, 79, 87].

2.7 Network structure and promotion of cooperation

2.7.1 Lattices and cooperation clustering

On regular lattices, cooperators tend to form compact spatial clusters, within which mutual cooperation yields higher average payoffs than those obtained by neighboring defectors. These cooperative domains act as advancing fronts against defecting regions, allowing cooperators to persist and often dominate. As a result, spatial structure can sustain coexistence or maintain a high fixation probability ρ_A even in parameter regimes where the well-mixed population predicts the extinction of cooperation [28, 45, 71, 72].

2.7.2 Heterogeneous (scale-free) networks

Many empirical networks are scale-free, with a degree distribution

$$P(k) \sim k^{-\gamma}, \quad 2 < \gamma < 3,$$

so that a few high-degree hubs coexist with numerous low-degree nodes [89, 90]. This structural heterogeneity strongly modifies evolutionary dynamics. Let $\rho_A(t)$ be the global cooperator fraction and $\rho_A(k, t)$ its value in degree class k . Typical trajectories follow three stages: *Early decay*: low-degree cooperators, having a small accumulated payoff $\pi_A(k) \approx k(u_{A,A}\rho_A + u_{A,B}(1 - \rho_A))$, are quickly replaced, causing an initial drop in $\rho_A(t)$. *Hub expansion*: once a hub of degree $k \gg \langle k \rangle$ adopts cooperation, its payoff grows roughly linearly in k , giving it a strong advantage under imitation kernels such as the Fermi rule $\Pr(s_j \leftarrow s_i) = 1/[1 + \exp(-\beta(\pi_i - \pi_j))]$. It then seeds a growing cooperative cluster. *Stabilization*: the system converges to a nonzero cooperative level x_C^* sustained by persistent hub-centered clusters [33, 86, 91–93].

With accumulated payoff, hubs keep their linear advantage and cooperation flourishes. Average payoff removes this advantage and lowers ρ_C . The precise stationary value depends further on the update mechanism.

Chapter 3

Main results of papers included in the thesis

3.1 Paper A: Bifurcation analysis of replicator dynamics with logistic growth and strategy-dependent time delays in snowdrift game

Motivation: Classical replicator dynamics capture frequency-dependent selection but neglect two important aspects of real populations: ecological limits and delayed responses. In reality, population growth is bounded by resources, while payoffs are rarely translated into immediate reproductive success. To address these effects, we studied replicator dynamics with logistic growth and strategy-dependent delays in the Snowdrift game [94], asking how demographic constraints and delayed feedback jointly affect coexistence and stability.

Model and Results: Unlike the single delay equation (2.4) for the social-type delay, here, we obtain a coupled system of two delay differential equations: one for the frequency of strategy A and another for the total population size:

$$\frac{dx(t)}{dt} = x(t)(1 - x(t))\left(1 - \frac{p(t)}{K}\right) \left(U_A(t - \tau_A) - U_B(t - \tau_B)\right), \quad (3.1a)$$

$$\frac{dp(t)}{dt} = p(t)\left(1 - \frac{p(t)}{K}\right) \left(x(t)U_A(t - \tau_A) + (1 - x(t))U_B(t - \tau_B)\right) - p(t). \quad (3.1b)$$

Here K denotes the carrying capacity, while τ_A, τ_B are the strategy-specific delays. For $K \rightarrow \infty$, equation (3.1a) reduces to the simple social replicator with delay (2.4).

We considered the Snowdrift game as the focal example, and all results are based on this case. With benefit $V = h$ and cost $C = s$ ($h > s$), introduced in Section 2.1.3, the undelayed system admits five unstable equilibria and one interior

coexistence equilibrium

$$\zeta_6 = \left(\frac{1}{2}, 1 - \frac{2h-s}{2h(h-s)} \right),$$

which is locally stable. Thus, under logistic regulation, cooperators and defectors can coexist. Linearization of (3.1) around ζ_6 leads to the characteristic equation

$$\lambda - \frac{s}{2h(2h-s)} \left(se^{-\lambda\tau_A} - 2he^{-\lambda\tau_B} \right) = 0. \quad (3.2)$$

Depending on the configuration of delays, we obtain four distinct scenarios:

- **Symmetric delays** ($\tau_A = \tau_B = \tau$): The equilibrium remains stable for $\tau < \tau^* = \pi h/s$. At $\tau = \tau^*$ a supercritical Hopf bifurcation occurs, generating stable oscillations in both $x(t)$ and $p(t)$.
- **Defectors delayed** ($\tau_A = 0, \tau_B > 0$): Instability arises once

$$\tau_B^* = \frac{2h(2h-s)}{s\sqrt{4h^2-s^2}} \arccos\left(\frac{s}{2h}\right),$$

beyond which the coexistence equilibrium loses stability.

- **Cooperators delayed** ($\tau_B = 0, \tau_A > 0$): In this case no bifurcation occurs, and the coexistence equilibrium remains stable for all τ_A .
- **General case** ($\tau_A, \tau_B > 0$): Multiple stability switches are possible, with alternating regimes of oscillatory dynamics and steady coexistence.

Conclusion: The analysis shows that logistic growth, on its own, only affects the speed of convergence but does not change the stability of coexistence between cooperators and defectors. When time delays are introduced, however, the dynamics change substantially. If both strategies experience the same delay, the coexistence equilibrium remains stable up to a critical threshold, after which a Hopf bifurcation occurs and stable oscillations appear. When only defectors are delayed, the equilibrium can lose stability once the delay passes a critical value, leading to oscillatory dynamics. In contrast, delaying only cooperators does not destabilize the system: the coexistence state remains stable regardless of the delay length. When both delays vary independently, the system may switch repeatedly between stable and oscillatory regimes.

3.2 Paper B: Intrinsic noise in structured replicator dynamics modelling time delays

Motivation: Replicator dynamics are a cornerstone of evolutionary game theory, but they typically assume infinite, well-mixed populations and instantaneous effects

of interactions. In contrast, real populations are finite, bounded by environmental constraints, and subject to delays before payoffs translate into reproduction. Moreover, demographic stochasticity plays a central role in finite populations. This study [95], inspired by the work of Tao and Cressman [42], integrates these aspects by formulating a structured replicator dynamics model with strategy-dependent time delays and analyzing the corresponding stochastic birth-death process. Our focus is the Snowdrift game, a social dilemma with a stable interior equilibrium in standard replicator dynamics.

Model and Results: To incorporate delays, we introduced a two-stage life cycle: individuals are born as juveniles (kindergarten compartment) and only later mature into adults who can interact and reproduce. Let n_C^A, n_D^A denote the numbers of adult cooperators and defectors, and n_C^K, n_D^K their juvenile counterparts. Adults interact according to the Snowdrift payoff matrix presented in section 2.1.3 with $V = b = 6$, $C = c = 4$, so the mixed Nash equilibrium of the classical replicator dynamics is $x^* = 0.5$. Juveniles mature into adults with strategy-dependent rates $1/\tau_C$ and $1/\tau_D$, interpreted as inverses of average delays. To bound population size, we included background fitness terms $-\beta_A N_A$ and $-\beta_K N_K$. The resulting compartmental model reads

$$\frac{dn_C^A}{dt} = \frac{n_C^K}{\tau_C} - \beta_A N_A n_C^A, \quad (3.2a)$$

$$\frac{dn_D^A}{dt} = \frac{n_D^K}{\tau_D} - \beta_A N_A n_D^A, \quad (3.2b)$$

$$\frac{dn_C^K}{dt} = n_C^A \pi_C - \frac{n_C^K}{\tau_C} - \beta_K N_K n_C^K, \quad (3.2c)$$

$$\frac{dn_D^K}{dt} = n_D^A \pi_D - \frac{n_D^K}{\tau_D} - \beta_K N_K n_D^K, \quad (3.2d)$$

where $\pi_C = \frac{n_C^A(b-c/2) + n_D^A(b-c)}{N_A}$, $\pi_D = \frac{n_C^A b}{N_A}$ and with $N_A = n_C^A + n_D^A$, $N_K = n_C^K + n_D^K$.

For equal delays $\tau_C = \tau_D = \tau$, the deterministic structured replicator dynamics yield the same stationary frequency of cooperators $x^* = 0.5$ as the classical replicator dynamics. When $\tau_C \neq \tau_D$, stationary states shift continuously, with longer delays reducing the basin of attraction of the corresponding strategy. Numerical analysis confirmed that asymmetric delays disadvantage the delayed strategy, in line with earlier delay models. To capture intrinsic noise, we constructed a continuous-time Markov jump process corresponding to the deterministic equations. Transitions include births, maturation events, and deaths, with intensities mirroring the rates in (3.2). For instance, juvenile maturation $n_i^K \rightarrow n_i^K - 1$, $n_i^A \rightarrow n_i^A + 1$ occurs with intensity n_i^K/τ_i . This allows us to interpret τ_i as the mean of an exponentially distributed random delay. We simulated the process using the Gillespie algorithm and studied the quasi-stationary distribution (condi-

tioning on non-extinction). Individual trajectories fluctuate strongly, but ensemble averages reveal systematic shifts compared to deterministic predictions.

A key observation is that stochasticity qualitatively changes the role of delays:

- In the deterministic model, equal delays $\tau_C = \tau_D$ do not alter the stationary frequency of cooperation, which remains 0.5.
- In the stochastic model, equal delays *increase* the long-term frequency of cooperators in the quasi-stationary state. Larger delays yield higher cooperation levels, even though the adult population size decreases.
- For asymmetric delays, deterministic and stochastic dynamics both disadvantage the more delayed strategy. However, in the stochastic case, cooperation can survive at larger values of τ_C or τ_D than predicted by deterministic equations.

This bias arises because random fluctuations in finite populations can transiently increase the number of cooperators, and due to the nonlinear dependence of birth rates on state variables, such fluctuations give cooperators a slight growth advantage on average. Larger delays amplify this stochastic effect.

Conclusion: Our results demonstrate that the interplay of time delays and intrinsic noise generates qualitatively new behaviors not seen in deterministic replicator dynamics. While deterministic dynamics predict that delays reduce the attractiveness of delayed strategies, stochastic simulations reveal that strategy-independent delays can *promote* cooperation. In particular, for equal or nearly equal delays, the expected frequency of cooperators in the quasi-stationary state exceeds the classical equilibrium of 0.5. In summary, time delays in finite populations not only change basins of attraction but, in the presence of intrinsic noise, can actively favor cooperative behavior. This highlights the importance of combining stochastic and delay effects when modeling evolutionary dynamics in structured populations.

3.3 Paper C: Effect of the degree of an initial mutant in Moran processes in structured populations

Motivation: While fixation probabilities in well-mixed populations are fully understood, much less is known about how the initial placement of a mutant in a structured population affects its fate. Graph structure determines which neighbors can be replaced, and in heterogeneous networks, the degree of the initial mutant can strongly influence its success. In this work [96], we study how the degree of the initial mutant affects fixation probability, extinction time, and fixation time in Moran processes on Erdős–Rényi (ER) and Barabási–Albert (BA) graphs.

Model and Results: We consider a Birth–death Moran process on graphs of $N = 500$ nodes. At each step, an individual is selected with probability proportional to its fitness (r for mutants, 1 for residents) to reproduce, and its offspring replace a random neighbor. We focus on the degree k of the vertex where the first mutant is placed. Stochastic simulations (10,000 Monte Carlo runs for each setup) are performed for different fitness values r and degrees k . To complement simulations, we derive mean-field-type approximations for transition probabilities up to three mutants, reducing the full Markov chain to an effective chain with $N + 1$ states (number of mutants). For example, the fixation probability starting from one mutant of degree k is approximated by

$$\phi_1^N \approx \frac{\langle k \rangle^2 r^2 \left(2 - \frac{1}{k} - \frac{1}{\langle k \rangle} \right)}{(r\langle k \rangle + k) \left(r\langle k \rangle \left(2 - \frac{1}{k} - \frac{1}{\langle k \rangle} \right) + (k - 2 + \frac{\langle k^2 \rangle}{\langle k \rangle}) \right) - r\langle k \rangle (k - 2 + \frac{\langle k^2 \rangle}{\langle k \rangle})} \cdot \frac{1 - \frac{1}{r^3}}{1 - \frac{1}{r^N}}, \quad (3.3)$$

where $\langle k \rangle$ and $\langle k^2 \rangle$ denote the mean and second moment of the degree distribution.

Both simulations and analytical approximations show that the fixation probability decreases with the degree of the initial mutant. Intuitively, a highly connected mutant is more exposed to replacement by resident neighbors, while low-degree mutants have fewer invasion paths against them. - On ER graphs, fixation probabilities are narrowly distributed around the average, reflecting the homogeneous degree distribution. - On BA graphs, the heterogeneous degree distribution produces wide variability: low-degree nodes enjoy high fixation probabilities, while hubs have very low probabilities. Although the fixation probability for a given degree k is higher in ER than BA graphs, The abundance of low-degree nodes in BA networks increases the *average* fixation probability above that of ER networks.

The degree of the initial mutant has no significant effect on the expected fixation time. This is because fixation depends on the entire trajectory of the population, not only on the early stages. In both ER and BA graphs, fixation times are distributed symmetrically around the mean, with BA networks showing longer averages due to their heterogeneity. In contrast, extinction time strongly depends on the initial degree. - Mutants starting at high-degree nodes go extinct more quickly, since they are surrounded by many resident neighbors who can replace them. - Mutants at low-degree nodes persist longer before extinction. In ER graphs, extinction times are narrowly distributed, while in BA graphs they vary widely, with some low-degree nodes sustaining mutants for long times.

Conclusion: We demonstrated that the degree of the initial mutant crucially influences fixation probability and extinction time, but not fixation time. Increasing the degree of the initial mutant reduces its chance of fixation and accelerates extinc-

tion, while fixation time remains largely degree-independent. Comparing network types, mutants of the same degree have slightly higher fixation probabilities on ER than BA graphs. However, the skewed degree distribution of BA graphs produces a higher average fixation probability overall, driven by the abundance of low-degree nodes.

3.4 Paper D: The impact of time delays on mutant fixation in pairwise social dilemma games

Motivation: Traditionally, in the Moran Process, fitness depends on the current population state. However, biological and social processes often involve temporal lags: reproductive success today may depend on interactions in the past. This study [97] incorporates time delays into the Moran process to investigate how historical interactions alter fixation dynamics in three social dilemmas: the Stag-Hunt, the Snowdrift, and the Prisoner’s Dilemma.

Model and Results: We considered a well-mixed population of size N with two strategies, A (mutant) and B (resident), interacting via a general payoff matrix as shown in 2.1.3. In the classical Bd Moran process ($\tau = 0$), reproduction is based on current fitness. Here, we extend the process so that fitness is evaluated at time $t - \tau$, introducing a homogeneous delay $\tau \in \mathbb{N}$. This modification enlarges the state space: each Markov chain state is described by $\{i_\tau, i_{\tau-1}, \dots, i_0\}$, where i_j is the number of mutants j steps ago, and i_0 is the current number. Transitions allow i_0 to increase, decrease, or remain constant. We applied absorbing Markov chain methods to compute fixation probabilities and times, Complemented by Monte Carlo simulations for larger N, τ .

Our analysis revealed that the impact of time delays strongly depends on the underlying social dilemma. In the Stag-Hunt game, delays consistently reduce the fixation probability of mutants. This occurs because coordination benefits require mutants to be sufficiently abundant, yet delayed fitness calculations reference earlier states when mutants were still rare, thereby diminishing their effective advantage. The fixation time in this case increases for small delays, reaches a maximum at an intermediate value of τ , and then decreases as $\tau \rightarrow \infty$, where fitness effectively becomes constant. In contrast, in the Snowdrift game time delays enhance mutant success, leading to higher fixation probabilities. Since both strategies gain when meeting cooperators, delayed payoffs favor mutants relatively more, thereby increasing their effective fitness ratio. Here the fixation time behaves in the opposite way to the Stag-Hunt: it decreases initially, attains a minimum, and then rises again for large delays. The Prisoner’s Dilemma presents a more subtle picture. While defection remains dominant, time delays generally further reduce the fixa-

tion probability of cooperative mutants, whereas the effect on defectors depends on the specific payoff values. Fixation times display only modest non-monotonic changes, with a slight increase followed by a decline, resembling the pattern of the Stag-Hunt but with weaker intensity. Taken together, these results demonstrate that time delays are disadvantageous for mutants in coordination games such as the Stag-Hunt, beneficial in anti-coordination settings like the Snowdrift, and yield mixed outcomes in the Prisoner’s Dilemma. Hence the evolutionary consequences of delays depend not only on the length of the delay but, crucially, on the strategic structure of the game itself..

Conclusion: This study establishes that introducing time delays into the Moran process qualitatively alters fixation dynamics. While standard models assume immediate fitness determination, historical dependence reshapes evolutionary outcomes:

- In the Stag-Hunt game, delays undermine coordination benefits and reduce fixation probability.
- In the Snowdrift game, delays enhance mutant success, increasing fixation probability.
- In the Prisoner’s Dilemma, delays usually hinder cooperation, with mixed effects on defectors.

Fixation times also display non-monotonic dependence on delay: increasing, then decreasing (Stag-Hunt), or the reverse (Snowdrift). These findings highlight that delays do not simply slow down dynamics but can reverse evolutionary outcomes, sometimes favoring mutants that would otherwise vanish.

3.5 Paper E: Phase transitions in the prisoner’s dilemma game on the Barabási-Albert graph with participation cost

Motivation: Barabási–Albert (BA) scale-free networks, characterized by hubs with many connections, are known to enhance cooperation when players update strategies via pairwise imitation. However, maintaining social links may carry costs in real systems. Earlier works showed that introducing a participation cost weakens cooperation on networks, but the precise nature of this effect in heterogeneous structures was not fully understood. In this study, we investigated how link costs affect cooperative behavior on BA networks and demonstrated the existence of a sharp phase transition in cooperation levels [98].

Model and Results: We considered a PD game with cooperation (C) and defection (D), where in addition to the canonical payoff parameters, each link incurs a maintenance cost γ shared by both neighbors. The payoff matrix is

$$U = \begin{array}{c|cc} & C & D \\ \hline C & 1 - \gamma & -\gamma \\ D & T - \gamma & -\gamma \end{array},$$

with $T > 1$ ensuring that defection is the dominant strategy in the absence of structure. Players are placed on a BA network of size N , average degree $\alpha = 4$, and interact with all neighbors. Strategy updating follows the Fermi rule: a randomly chosen player x with payoff π_x compares with a random neighbor y with payoff π_y and adopts y 's strategy with probability

$$\Pr(s_i \leftarrow s_j) = \frac{1}{1 + \exp[-\beta(\pi_x - \pi_y)]},$$

where $\beta > 0$ is the intensity of selection (set to 100 in simulations). A small error rate $\varepsilon = 0.001$ ensures ergodicity.

Monte Carlo simulations were performed on networks of size $N = 10^4$. Starting from random initial conditions, we observed that the stationary level of cooperation changes abruptly when the link cost γ crosses a critical value. For small γ , almost full cooperation is sustained due to the stabilizing effect of cooperative hubs. For large γ , cooperators and defectors coexist at low cooperation levels. In the intermediate regime $[\gamma_l(T, N), \gamma_r(T, N)]$, the system exhibits metastability: the stationary distribution of cooperation frequency is bimodal, with the population oscillating between a near-full-cooperation state and a coexistence state. The critical cost $\gamma_{cr}(T, N) = (\gamma_l + \gamma_r)/2$ decreases as temptation T increases. Moreover, the width of the metastable region shrinks with N , but the expected switching time between metastable states remains independent of system size, which precludes the existence of two stationary states in the infinite-population limit.

This behavior parallels first-order phase transitions in statistical physics, such as the ferromagnetic Ising model at low temperature, but differs in a crucial way: in the Ising case, metastability vanishes as $N \rightarrow \infty$ with coexistence of stationary states, while in the BA network PD game, ergodicity is preserved even in the limit. The role of hubs is decisive: cooperation thrives when hubs remain cooperative, but if a hub switches to defection, its high payoff rapidly spreads defection until cooperators recover.

To complement simulations, we developed a pair approximation tracking the frequency of cooperators ρ_C and cooperative pairs ρ_{CC} . The system of ODEs incorporates degree distributions and payoff-dependent update probabilities. Nu-

merical solutions reveal the same sharp transition at the critical cost γ_{cr} as observed in simulations. The agreement is particularly strong for small T and moderate γ , confirming that the discontinuous change is not an artifact of finite-size stochastic effects but an intrinsic property of the dynamics.

Conclusion: We demonstrated that introducing a participation cost in the PD game on BA networks leads to an abrupt phase transition from near-complete cooperation to coexistence of cooperators and defectors. In the critical regime, cooperation is metastable and the system alternates between high and low cooperation states. The transition sharpens with population size, but ergodicity persists because switching times between metastable states remain independent of system size. Unlike in equilibrium spin systems, this precludes multiple stationary states in the infinite-population limit.

3.6 Paper F: Strategy dependent time delays shape cooperation in the spatial prisoner's dilemma game

Motivation: Spatial structure is a powerful mechanism supporting cooperation: cooperators can cluster together, shielding themselves from exploitation by defectors. However, real interactions are rarely instantaneous, and the temporal dimension of decision-making may crucially affect the outcome. Time delays arise naturally when individuals act on outdated information or when the consequences of their actions materialize after some lag. Importantly, such delays may depend on the strategy itself—for instance, cooperation often requires coordination and joint investment, while defection may yield more immediate but sometimes delayed payoffs. While previous studies analyzed delays in well-mixed or deterministic settings, the impact of strategy-dependent delays on cooperation in structured populations remained unclear. This study [99] addresses this gap by investigating the Prisoner's Dilemma game (PDG) on a square lattice with distinct delays for cooperators and defectors.

Model and Results: We considered the weak PDG with a payoff matrix

$$U = \begin{pmatrix} 1 & 0 \\ b & 0 \end{pmatrix}, \quad b > 1,$$

where b represents the temptation to defect. Individuals occupy sites of an $L \times L$ lattice with periodic boundaries and interact with their four neighbors and themselves. Each individual i adopting strategy s_i accumulates payoffs from its neighborhood, but the effective payoff at time t is evaluated from interactions τ_{s_i} steps in the past. Thus, cooperators share a common delay τ_C and defectors a delay τ_D .

Strategy updating follows the Fermi rule:

$$Pr(s_j \rightarrow s_i) = \frac{1}{1 + \exp\left[\frac{P_i(t-\tau_{s_i}) - P_j(t-\tau_{s_j})}{\kappa}\right]},$$

with noise $\kappa = 0.1$. Simulations were performed on 200×200 lattices, averaging over multiple realizations.

The analysis reveals that even small delays can profoundly reshape cooperation levels and spatial organization. When only cooperators are delayed ($\tau_D = 0, \tau_C > 0$), two transitions emerge as the temptation b increases: from full cooperation to coexistence at a threshold b_{cr1} that shifts rightward with larger τ_C , and from coexistence to full defection at b_{cr2} , which shifts leftward. Thus, moderate delays in cooperation can extend the coexistence regime but eventually hasten extinction in stronger dilemmas. In contrast, when only defectors are delayed ($\tau_C = 0, \tau_D > 0$), full cooperation cannot be maintained, but defectors never fully dominate either. Instead, coexistence persists, and for small τ_D cooperation levels decline with b , while for larger τ_D the decline becomes flatter, stabilizing cooperation at intermediate levels.

Exploring the full parameter space of (τ_C, τ_D) shows sharp phase boundaries separating regions of full cooperation, coexistence, and full defection. Remarkably, even under harsh dilemmas ($b = 2$), cooperation can emerge if cooperators experience shorter delays than defectors. Conversely, if $\tau_C < \tau_D$, the system typically settles into coexistence. These boundaries are highly sensitive: a change of one time step can flip the system between qualitatively different outcomes.

Spatial analysis revealed that strategy-dependent delays act primarily by modifying cooperator clustering. Delaying cooperators stabilizes cooperator domains by buffering against immediate exploitation at cluster boundaries, allowing clusters to expand. Defector delays slow down exploitation, enabling cooperators to merge into large stable clusters. However, excessive delays destabilize spatial organization and can undermine cooperation. Cluster statistics confirm that delay asymmetry reshapes the balance between the number and size of cooperator domains, which directly drives global cooperation levels.

Finally, results for the standard PDG confirm that the qualitative effects of delays are unchanged, although quantitative thresholds differ due to the altered payoff structure. In particular, cooperation collapses earlier in the standard game, but the role of strategy-dependent asymmetry remains the same.

Conclusion: This study demonstrates that temporal asymmetries in payoff evaluation can dramatically alter the outcomes of spatial social dilemmas. Strategy-dependent delays generate sharp transitions between full cooperation, coexistence,

and full defection, and the outcome is determined not only by the dilemma strength b but also by the relative delays of cooperators and defectors. The key mechanism is the modification of clustering dynamics: Delays reshape the stability and expansion of cooperative domains.

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Bifurcation Analysis of Replicator Dynamics with Logistic Growth and Strategy-Dependent Time Delays in Snowdrift Game

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Abstract

This study examines the stability effects of strategy-dependent time delays on replicator dynamics with logistic population growth. Using a two-player snowdrift game, we incorporate social forms of delay where individuals' payoffs are delayed, impacting offspring production. While a stable mixed-strategy equilibrium exists without delays, introducing time delays can destabilize this equilibrium through Hopf bifurcations, inducing oscillations. Employing Hopf bifurcation theory, we explore how critical delay values depend on whether delays are equal across strategies, unequal with one strategy experiencing no delay, or fully asymmetric. The analysis reveals rich phenomena, including regions where increasing a delay can transition the system between stability and instability. Our work elucidates the complex interplay between time delays, strategic dynamics, and finite population constraints.

Keywords Evolutionary game theory · Replicator dynamics · Logistic growth · Strategy dependent time delay

1 Introduction

Evolutionary game theory (EGT) provides a framework for modeling many social and biological processes as systems of interacting individuals [1–9]. Introduced in [4], EGT is founded on the concept that when different strategic behaviors are viewed through the lens of a game, the more successful strategies will eventually predominate within the population. By analyzing individual interactions, one can attempt to derive the macroscopic behavioral patterns that emerge across the population. For very large (theoretically infinite) popula-

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tions, the evolution over time can be described by differential replicator equations tracking the changing prevalence of each strategic behavior [2, 8, 10, 11]. At the core of EGT lies the evolutionarily stable strategy (ESS)—a strategy that is immune to being overtaken by any mutant strategic variant. Once a population has adopted an ESS, inferior mutational deviations cannot spread and displace the predominant strategic behavior.

The classical evolutionary game modeling approach does not explicitly analyze population size limitations. However, population ecology studies dynamic population sizes using nonlinear models accounting for environmental factors like resource constraints and competing species [1, 2, 12–19]. While populations can grow exponentially under ideal conditions, growth is ultimately limited, as described by the Malthusian Struggle for Existence. The inhomogeneous logistic equation models both free exponential growth and resource limitations, providing insights into complex evolutionary dynamics. Therefore, we need to consider the logistic term in the replicator equation, as it captures the key tension between unconstrained growth and environmental constraints that shape evolutionary outcomes in a finite world [1, 12–14, 18–21].

Evolutionary game dynamics typically assume that the growth rate of a strategy depends solely on the current frequencies of strategies in the population. However, this assumption does not always hold true, especially in practical situations arising in biology and economics. In many real-world phenomena, effects are not immediate but rather manifest after some delay. Scholars have investigated the impact of time delays in various contexts like immune systems, neural networks, epidemic models, and financial systems [22–27]. Introducing time delays into evolutionary game models is, therefore a natural extension that has attracted significant research attention.

Many researchers have investigated the replicator equation with time delays [28–48]. When delays are significant, the evolutionarily stable strategy (ESS) can lose stability, leading to persistent oscillations around the solution of the replicator dynamics. The authors in [48] examined a model of replicator dynamics with a fixed symmetric delay in a continuous-time setting, where a strategy's expected payoff at time t depends on the frequency of strategies in the population at time $t - \tau$. They proved that for sufficiently large delays, the unique mixed ESS can become unstable. In [42], the authors studied best response dynamics with symmetric delays across strategies. Alternatively, [43] looked at replicator dynamics with asymmetric delays between strategies. Both works showed that delays can cause instability in these evolutionary models. For discrete systems, Alboszta and Miękisz [28] investigated the influence of time delay on the stability of evolutionarily stable strategies in social and biological types of delays. They proved that different forms of time delays have varying effects on the stability of mixed evolutionarily stable strategies. When considering the social delay model, small delays lead to the asymptotic stability of dynamics. Utilizing Hopf bifurcation theory and calculating the critical conditions for the existence of Hopf bifurcation, Wettergren [46] studied two-person and n -person games, respectively, obtaining the boundary conditions for the stability of the replicator dynamic system. In another study examining strategy-dependent for biological time delays, authors showed that increasing the delay can result in the emergence or disappearance of an interior equilibrium point, depending on the specific game dynamics. Changes in the interior equilibrium point tend to be unfavorable for the strategy associated with the longer delay [34].

Previous studies on time delays in replicator dynamics have primarily focused on the frequencies of strategies, neglecting the finite population size inherent to real-world sys-

tems with maximum capacities. Yet in many biological, ecological, and socio-economic contexts the total population cannot grow unboundedly: bacterial colonies are constrained by nutrients, human communities by resources, and competing technologies by market size. Accounting for a carrying capacity therefore makes the model directly relevant to microbial ecology (e.g. biofilm growth under nutrient limitation), wildlife management (competition for a fixed habitat), and vaccine-adoption dynamics where the number of susceptible individuals is capped by demography. This paper aims to address this gap by considering a population with two distinct strategies playing a two-player game with logistic growth, incorporating a maximum population capacity and a social type of time delay that is strategy-dependent. By coupling realistic demographic constraints and delayed feedback, the model captures how finite resources and behavioral lags jointly shape long-term evolutionary outcomes.

Using the snowdrift game as the primary game, we employ Hopf bifurcation theory and stability analysis to investigate the possibility of critical time delays at which Hopf bifurcations occur, leading to oscillatory and cyclic evolutionary dynamics. In the rest of the paper, initially, we introduce our discrete model with logistic growth and present the results for the snowdrift game. Subsequently, we incorporate a social type of time delay into the model, where only the payoff is affected by the time delay. By considering different possibilities for time delays, we present our stability analysis and associated results.

2 Model

2.1 Replicator Dynamic with Logistic Growth

Generally, we assume our population is haploid, meaning the offspring have the same phenotypic strategies (behaviors) as their parent. Specifically, there are two pure strategies A and B that an individual can have. The offspring will inherit strategy A if the parent has A, and strategy B if the parent has B. Let $p_i(t)$, $i = A, B$ represent the number of individuals playing strategy i at time t . Furthermore, let $p(t) = p_A(t) + p_B(t)$ be the total number of players in the population at time t , and $x(t) = \frac{p_A(t)}{p(t)}$ represent the fraction of the total population playing strategy A at time t . Individuals engage in one-on-one competitions at distinct points in time. The results of these contests are determined by the following payoff matrix:

$$U = \begin{array}{c|cc} & A & B \\ \hline A & a & b \\ B & c & d \end{array}$$

We assume a symmetric game in which both players are identical and, therefore the payoffs for the column player are represented by the matrix U transposed. Therefore, the average payoff obtained by strategies A and B is $U_A(t) = ax(t) + b(1 - x(t))$ and $U_B(t) = cx(t) + d(1 - x(t))$, respectively.

Following by [8], let's assume that in a time interval of length ϵ , only an ϵ fraction of the population participates in one-on-one competitions. We set the length of the update interval equal to the fraction of population that participate in the game in that interval. This normalization fixes the expected life-span of an individual at one unit of time and does

not affect the ensuing replicator dynamics. Also, we model the population as having a carrying capacity K , representing the maximum sustainable population level. To account for this ecological constraint, we incorporate a logistic growth term into our equations. This term limits the population growth rate as the population size $p(t)$ approaches K , reflecting density-dependent feedbacks that reduce per capita growth rates at higher densities [1, 12, 16]. Fundamentally, we impose the realistic assumption that $p(t)$ cannot surpass the ecological ceiling imposed by K . The logistic model allows us to capture the dynamics of bounded population growth in a simple yet theoretically grounded way. Therefore, we propose the following equations:

$$p_i(t + \epsilon) = (1 - \epsilon)p_i(t) + \epsilon p_i(t)U_i(t) \left(1 - \frac{p(t)}{K}\right), \quad (1)$$

$$p(t + \epsilon) = (1 - \epsilon)p(t) + \epsilon \left(1 - \frac{p(t)}{K}\right) (p_A(t)U_A(t) + p_B(t)U_B(t)). \quad (2)$$

As a result, it is used to multiply the second terms in the above equations, representing the payoff from playing the game. We are assuming a parental replacement rate, or death rate, of 1. Therefore, the variables $p_i(t)$ and $p(t)$, which represent population frequencies, are multiplied by $1 - \epsilon$. This adjusts the frequencies to account for the death rate over the time period ϵ . In other words, a proportion ϵ of the population dies during each interaction, so $1 - \epsilon$ of the population remains afterward.

By dividing Eqs. (1) by (2) and considering $i = A$ we have:

$$\frac{p_A(t + \epsilon)}{p(t + \epsilon)} = \frac{(1 - \epsilon)p_A(t) + \epsilon p_A(t)U_A(t) \left(1 - \frac{p(t)}{K}\right)}{(1 - \epsilon)p(t) + \epsilon \left(1 - \frac{p(t)}{K}\right) (p_A(t)U_A(t) + p_B(t)U_B(t))}.$$

Using the fact that $x(t)$ is equal to the proportion of individuals playing strategy A, we can derive an equation for it. Then, dividing by ϵ and taking the limit as ϵ approaches 0, we obtain a differential equation describing how the frequency of strategy A changes over time as follows:

$$\frac{dx(t)}{dt} = x(t)(1 - x(t)) \left(1 - \frac{p(t)}{K}\right) (U_A(t) - U_B(t)). \quad (3a)$$

Because this equation is not closed, so we need an equation for $p(t)$ as well. Considering Eq. (2) and doing some simple mathematics as above, we have:

$$\frac{dp(t)}{dt} = p(t) \left(1 - \frac{p(t)}{K}\right) \bar{U} - p(t), \quad (3b)$$

where

$$\bar{U}(t) = x(t)U_A(t) + (1 - x(t))U_B(t). \quad (3c)$$

Equations (3a) and (3b) describe the whole system. It is clear if we take the limit K to infinity, equation (3a) would be $\dot{x} = x(1-x)(U_a - U_B)$ which is the simple replicator equation with three equilibria given by $x^* = 0$, $x^* = 1$, and $x^* = \frac{b-d}{b-d+c-a}$.

Note, that for any $K \in (0, \infty)$ we can rescale equation (3b) by K taking $\tilde{p} = \frac{p}{K}$. Then, the equation for $\tilde{p}$ is the same as (3b) but with $K = 1$. This implies that the magnitude of K does not impact the model dynamics. This contrasts with previous findings, where the carrying capacity was shown to significantly influence the evolutionary dynamics [20, 21]. Therefore, for most of the paper, we consider $K = 1$, and we interpret p as the fraction of the maximal populations. However, we keep K in a few places to compare some of the derived equations with the model for which the population growth is unbounded.

To find the equilibrium points $\zeta^*(x^*, p^*)$ of the system of Eq. (3), we set the left-hand sides of these equations equal to zero ($\frac{dx(t)}{dt} = 0$, $\frac{dp(t)}{dt} = 0$). This gives us (for $K = 1$ clearly):

$$\begin{cases} 0 = x^* (1 - x^*) (1 - p^*) (U_A^* - U_B^*), \\ 0 = p^* (1 - p^*) \bar{U}^* - p^*. \end{cases} \quad (4)$$

From the second equation, it is obvious that either $p^* = 0$ (which has a little biological sense) or $p^* = 1 - \frac{1}{\bar{U}^*}$, where U^* is an average payoff, and it does depend only on the frequency x^* and the payoff matrix. Thus, solving system (4) we get six equilibrium points $\zeta^*(x^*, p^*)$:

$$\begin{aligned} \zeta_1^* &= (0, 0), & \zeta_2^* &= (1, 0), \\ \zeta_3^* &= \left(\frac{b-d}{b-d+c-a}, 0 \right), & \zeta_4^* &= \left(0, 1 - \frac{1}{d} \right), \\ \zeta_5^* &= \left(1, 1 - \frac{1}{a} \right), & \zeta_6^* &= \left(\frac{b-d}{b-d+c-a}, \frac{bc-ad-b+d-c+a}{bc-ad} \right). \end{aligned} \quad (5)$$

Obviously, the three equilibrium points ζ_1^* , ζ_2^* , and ζ_3^* cannot have a biological interpretation as realistic end-state population sizes since they all indicate a final population of zero. An equilibrium point with zero individuals implies that both strategies have gone extinct, rather than representing any sustainable population. Moreover, the equilibrium point ζ_4^* exists and is non-negative only if $d \geq 1$ and the equilibrium point ζ_5^* exists and is non-negative only if $a \geq 1$.

2.2 Social Time Delay

Here, we assume that individuals at time t replicate due to average payoffs obtained by their strategies at time $t - \tau$ for some delay $\tau > 0$. This type of time delay in literature is called the social type of time delay [28]. Also, we consider a strategy-dependent time delay which means the delay for strategy A (τ_A) can be different from the delay for strategy B (τ_B). We propose the following equations:

$$p_i(t + \epsilon) = (1 - \epsilon)p_i(t) + \epsilon p_i(t)U_i(t - \tau_i) \left(1 - \frac{p(t)}{K}\right), \quad (6)$$

$$p(t + \epsilon) = (1 - \epsilon)p(t) + \epsilon \left(1 - \frac{p(t)}{K}\right) (p_A(t)U_A(t - \tau_A) + p_B(t)U_B(t - \tau_B)). \quad (7)$$

Similar to the previous case, if we divide Eq. (6) by Eq. (7) and attempt to find the equation for $x(t)$, and also do the same calculation for $p(t)$, we will ultimately arrive at the following two delay differential equations:

$$\frac{dx(t)}{dt} = x(t)(1 - x(t)) \left(1 - \frac{p(t)}{K}\right) (U_A(t - \tau_A) - U_B(t - \tau_B)), \quad (8a)$$

$$\frac{dp(t)}{dt} = p(t) \left(1 - \frac{p(t)}{K}\right) (x(t)U_A(t - \tau_A) + (1 - x(t))U_B(t - \tau_B)) - p(t). \quad (8b)$$

Again, as in the case without delay, we see that for any $K \in (0, \infty)$, we can rescale the second variable by dividing it by K , obtaining the same equation with $K = 1$. Thus, we conclude that the magnitude of K does not impact the model dynamics, and we assume hereafter that $K = 1$. To trace the evolution of the population, we have to solve the system of equations (8), together with initial conditions on the interval $[-\tau_M, 0]$, where $\tau_M = \max\{\tau_A, \tau_B\}$. We assume that

$$x(t) = \phi_x(t), \quad p(t) = \phi_p(t), \quad \text{for } t \in [-\tau_M, 0]. \quad (9)$$

The following proposition establishes the existence of non-negative solutions:

Proposition 1 *If the initial functions ϕ_x and ϕ_p are continuous over the interval $[-\tau_M, 0]$, $0 \leq x(0) \leq 1$, and $0 \leq p(0) \leq K$, then there exists a unique non-negative solution to system (8), subject to the initial conditions (9), that is well-defined for all times $t \geq 0$. Moreover, the set*

$$\Omega = \left\{ \phi \in C([- \tau_M, 0], \mathbb{R}^2) : 0 \leq \phi_x(\theta) \leq 1, 0 \leq \phi_p(\theta) \leq K, \theta \in [- \tau_M, 0] \right\}$$

is positively invariant under the dynamics governed by system (8).

Proof The local existence of the solution is an immediate consequence of the well-established theory of delay differential equations. Indeed, the right-hand side of System (8) is C^1 class, so existence and uniqueness of solutions follow from the standard theorem, see [49]. The non-negativity property arises from the analysis presented in [50], since the right-hand side is non-negative for any non-negative initial functions such that $\phi_x(0) = 0$ or $\phi_p(0) = 0$. To demonstrate global existence, we employ the step method over the interval $[0, \tau_m]$, where $\tau_m = \min\{\tau_A, \tau_B\}$. On the interval $[0, \tau_m]$, the delayed terms $U_A(t - \tau_A)$ and $U_B(t - \tau_B)$ depend only on the known initial functions ϕ_x and ϕ_p via $x(t - \tau_A)$ and $p(t - \tau_B)$. Thus, over $[0, \tau_m]$, the system (8) reduces to a non-autonomous system of ordinary differential equations with continuous right-hand sides. By standard ODE theory, there exists a unique solution on $[0, \tau_m]$. It remains to prove, that solution does not blow up. Note, that the form

of the first equation implies that $0 \leq x(t) \leq 1$ for any $t \in [0, \tau_m]$ as $0 \leq x(0) \leq 1$. On the other hand, if $p(\bar{t}) = K$ for some $\bar{t} \in [0, \tau_m]$, then obviously $\frac{dp}{dt}(\bar{t}) = -K < 0$. This proves that $0 \leq p(t) \leq K$ for all $t \in [0, \tau_m]$. The mathematical induction completes the proof. $\square$

Note, that the equilibrium points of system (8) are the same as of system (3), so they are given by (5).

3 Methods and Results

This paper focuses primarily on analyzing the Snowdrift Game, which models interactions between two drivers caught in a snowstorm. They can either cooperate and clear the road together or defect, waiting for the other to do the work. If both defect, neither makes it home, while if they choose different strategies, the defector benefits without incurring the cost of cooperation. This inherent asymmetry makes it advantageous for a player to choose a strategy different from their opponent's, leading to a stable coexistence of cooperation and defection. We focus on the Snowdrift game because, unlike games such as the Prisoner's Dilemma or Stag Hunt, it admits a stable interior equilibrium in the absence of delay where this equilibrium is affected by delay and the stability of that can change. In contrast, for other games, time delay typically does not change the qualitative stability of equilibria, making them less informative in this context. The equilibrium point of the replicator equation for Snowdrift game ensures that cooperation does not vanish entirely, as some level of cooperative behavior remains advantageous under selection dynamics.

The Snowdrift Game is typically parameterized as follows:

$$U_1 = \begin{array}{c|cc} & A & B \\ \hline A & h - \frac{s}{2} & h - s \\ B & h & 0 \end{array}$$

In this matrix, strategy A represents cooperation, and B represents defection. The parameter h denotes the benefit, while s is the cost paid by a cooperator, with the assumption that $h > s$ [51, 52]. For the remainder of this paper, we fix $h = 3$ and $s = 2$ for numerical calculations and plots.

3.1 The System Dynamics in the Absence of Delay

Before exploring how time delays affect the equilibrium points and their stability, it is good to first examine the stability of the equilibrium points of the replicator system (8) for the case without delays. The values of these points are given in Eq. (5).

Clearly, as $d = 0$, the equilibrium point ζ_4^* does not exist and ζ_5^* has biological sense only if $h - \frac{s}{2} > 1$. The formulas for ζ_3^* , ζ_5^* , and ζ_6^* simplify in the case of the Snowdrift game given by matrix U_1 , and they read as

$$\begin{aligned}\zeta_1^* &= (0, 0), & \zeta_2^* &= (1, 0), & \zeta_3^* &= \left(1 - \frac{s}{2h-s}, 0\right), \\ \zeta_5^* &= \left(1, 1 - \frac{2}{2h-s}\right), & \zeta_6^* &= \left(1 - \frac{s}{2h-s}, 1 - \frac{2h-s}{2h(h-s)}\right).\end{aligned}\quad (10)$$

Note, that ζ_6^* has biological meaning only if $\frac{2h(h-s)}{2h-s} > 1$. An easy calculation shows that the fraction is a decreasing function of s . Thus, it achieves its maximum that equals h for $s = 0$, and for $s = h$ it is equal to 0. Thus, if $h < 1$, then the equilibrium point ζ_6^* has no biological sense, while for $h > 1$ it has positive coordinates for $s < \frac{2h(h-1)}{2h-1}$.

In order to investigate the dynamics of the replicator system (3) we sketch the phase portrait of the system for $(x, p) \in [0, 1] \times [0, 1]$. The null-cline for x consists of four lines: $x = 0$, $x = 1$, $x = x^* = \frac{2(h-s)}{2h-s}$ and $p = 1$. It is easy to see that $x(t)$ is increasing if $x(t) \in (0, x^*)$ and it is decreasing if $x(t) \in (x^*, 1)$ (for $p(t) < 1$ of course). The null-cline for p consists of a line $p = 0$ and the curve

$$p = 1 - \frac{1}{xU_A + (1-x)U_B} = 1 - \frac{2}{(2h-s)x(2-x)}.$$

Because we are interested only in the region $0 \leq x \leq 1$, the curve is an increasing function of x , which can be negative over the entire interval if $h - \frac{s}{2} < 1$; it can be positive only for some $x \in (x^*, 1]$ when $h > 1$, $h - \frac{s}{2} > 1$ and $s > \frac{2h(h-1)}{2h-1}$. Finally, the curve intersects with the line $x = x^*$ if $h > 1$ and $s < \frac{2h(h-1)}{2h-1}$. Thus, the system (3) exhibits one of the following dynamics:

- in the region A (see Fig. 1), x is increasing and p is decreasing;
- in the region B, both variables x and p are decreasing;
- in the region C (the region exists if $h - \frac{s}{2} > 1$), x is decreasing, while p is increasing;
- in the region D (the region exists if $h > 1$ and $s < \frac{2h(h-1)}{2h-1}$), both variables x and p are increasing.

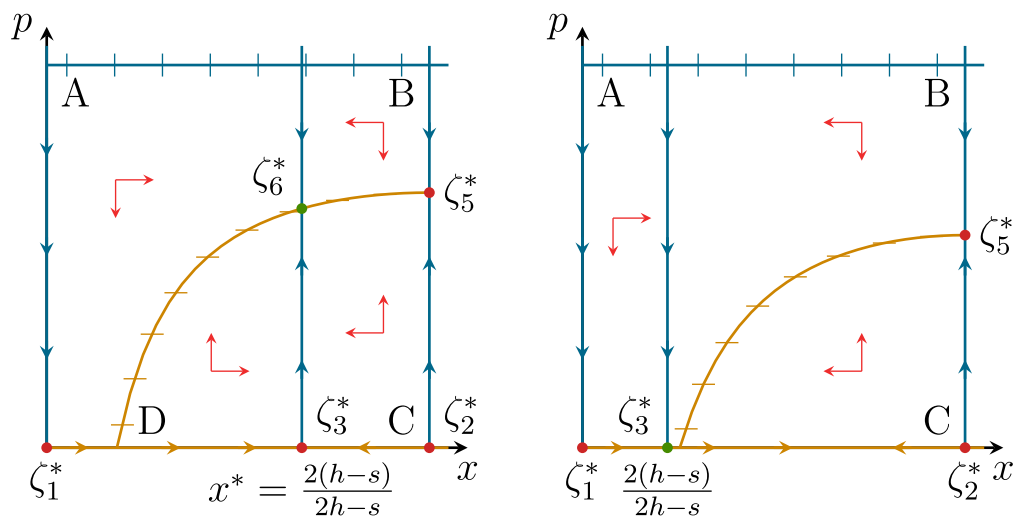


Fig. 1 A sketch of the phase portrait of the replicator system (3). In the left panel, the case when ζ_6^* exists is presented. In the right panel, the case when ζ_6^* does not exist but ζ_3^* does exist is presented. The last case, when the region C does not exist, is not presented as the system dynamics in this case can be easily deduced from the right panel

From the shape of the null-clines and uniqueness of the solutions, we deduce that regions B and D are invariant: if the solution enters one of these regions, it stays in it for all future times. From region A, the solution can only enter into D or stay in A. Similarly, solutions starting in region C can enter region B or stay in region C (this is possible only if region D exists). From this analysis, we deduce the following

Proposition 2 *For the Snowdrift game given by the matrix U_1 the stability of equilibrium points of system (3) are as follows:*

- (i) *The equilibrium point ζ_1^* is a saddle.*
- (ii) *The equilibrium point ζ_2^* is unstable—it is a node if $h - \frac{s}{2} > 1$, and a saddle for $h - \frac{s}{2} < 1$;*
- (iii) *The equilibrium point ζ_3^* is a stable node if $h < 1$ or (for $h > 1$) $\frac{2h(h-1)}{2h-1} < s < h$. Moreover it attracts all solutions such that $x(0) \neq 0$ and $x(0) \neq 1$. the equilibrium point is a saddle if $h > 1$ and $0 < s < \frac{2h(h-1)}{2h-1}$.*
- (iv) *The equilibrium point ζ_5^* is a saddle.*
- (v) *the equilibrium point ζ_6^* is a stable node if $h > 1$ and $0 < s < \frac{2h(h-1)}{2h-1}$ (otherwise it does not exist in the first quadrant). Moreover, it attracts all solutions except stationary ones and those for which $x(0) = 0$ or $x(0) = 1$.*

Corollary 3 *For $h = 3$ and $s = 2$ we have $\zeta_1^* = (0, 0)$, $\zeta_2^* = (1, 0)$, $\zeta_3^* = (\frac{1}{2}, 0)$, $\zeta_5^* = (1, \frac{1}{2})$, and $\zeta_6^* = (\frac{1}{2}, \frac{1}{2})$. Since $h > 1$ and $s = 1 < \frac{12}{5}$ only ζ_6^* is a stable node, while other equilibrium points are unstable.*

Examples of phase portraits of the replicator system (3) for two most interesting cases are shown in Fig. 2. The right panel demonstrates the case where an interior equilibrium point ζ_6^* emerges for $h = 3$ and $s = 2$, fulfilling the required constraints. In contrast, the left panel illustrates a scenario where no interior equilibrium exists for $h = 3$ and $s = 2.5$, as the conditions for its existence are not satisfied.

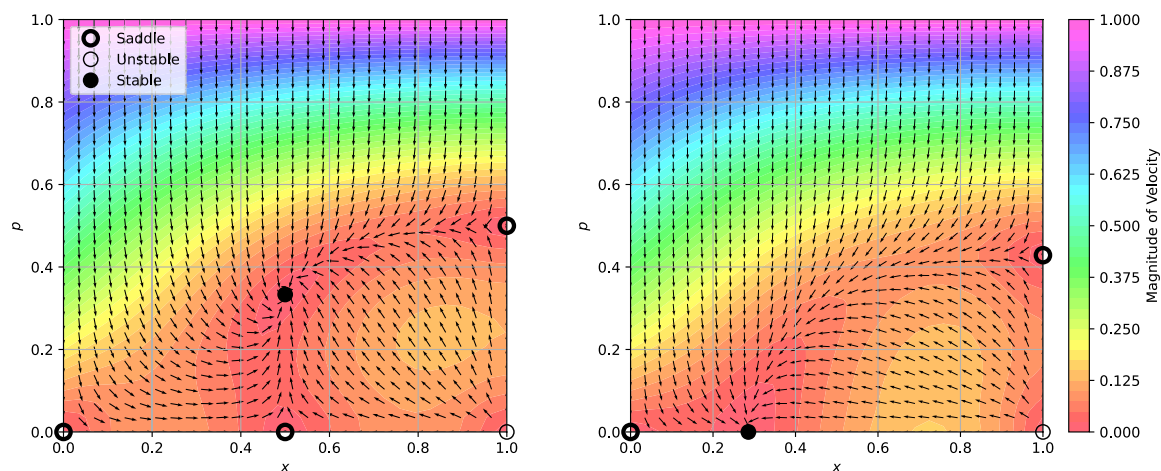


Fig. 2 Phase portrait for two different scenarios. In the right panel, the interior equilibrium point ζ_6^* exists for $h = 3$ and $s = 2$, satisfying the conditions $h > 1$ and $0 < s < \frac{2h(h-1)}{2h-1}$. In contrast, the left panel depicts the case where no interior equilibrium exists for $h = 3$ and $s = 2.5$

Note, that if payoffs are too small, that is either $h < 1$ or $h > 1$, but $\frac{2h(h-1)}{2h-1} < s < h$ (so $h - s$ is small) then the average payoff is too small to sustain the population, and the population extinctions. Thus, the only interesting case is $h > 1$ and $s < \frac{2h(h-1)}{2h-1}$.

3.2 Bifurcation Analysis

This section explores the asymptotic behavior of solutions of equations (8) by analyzing the stability of the equilibrium points. We use a standard linearization technique, see [49, 53]. We assume $x(t) = x^* + x_1(t)$ and $p(t) = p^* + p_1(t)$, where $x_1(t)$ and $p_1(t)$ represent small independent perturbations of the solution from the equilibrium point x^* and p^* . Thus, linear approximation of system (8) around the equilibrium point (x^*, p^*) reads:

$$\begin{aligned}\frac{dx_1(t)}{dt} &= \alpha_1 x_1(t) + \alpha_2 p_1(t) + \alpha_3 x_1(t - \tau_A) + \alpha_4 x_1(t - \tau_B), \\ \frac{dp_1(t)}{dt} &= \beta_1 x_1(t) + \beta_2 p_1(t) + \beta_3 x_1(t - \tau_A) + \beta_4 x_1(t - \tau_B).\end{aligned}$$

We determine the values of the necessary coefficients α_i and β_i later. First, observe that $\alpha_2 = 0$ as either $x^* = 0$ or $x^* = 1$ or $U_A^* = U_B^*$, so the characteristic function of system (11) reads

$$W(\lambda) = \det \left(\begin{pmatrix} \alpha_1 + \alpha_3 e^{-\lambda \tau_A} + \alpha_4 e^{-\lambda \tau_B} & 0 \\ \beta_1 + \beta_3 e^{-\lambda \tau_A} + \beta_4 e^{-\lambda \tau_B} & \beta_2 \end{pmatrix} - \lambda \text{Id} \right).$$

We see that the matrix is lower-triangular, and thus

$$W(\lambda) = (\lambda - \beta_2) (\lambda - \alpha_1 - \alpha_3 e^{-\lambda \tau_A} - \alpha_4 e^{-\lambda \tau_B}). \quad (11)$$

For the Snowdrift game and the equilibrium point (x^*, p^*) we have

$$\begin{aligned}\alpha_1 &= (1 - 2x^*)(1 - p^*)(U_A^* - u_B^*), \\ \alpha_3 &= \frac{s}{2} x^*(1 - x^*)(1 - p^*), \\ \alpha_4 &= -h x^*(1 - x^*)(1 - p^*), \\ \beta_2 &= (1 - 2p^*)(x^* U_A^* + (1 - x^*) U_B^*) - 1.\end{aligned}$$

Values of β_1 , β_3 , and β_4 are irrelevant for our analysis.

Although, we are interested mainly in the stability of the interior equilibrium point ζ_6^* , we determine the stability of other points for completeness. First, note that from the form of the characteristic function, it is clear that β_2 is one of its zeros. Thus, if $\beta_2 > 0$, then the equilibrium point is unstable.

The equilibrium point $\zeta_1^* = (0, 0)$. Here we have $\beta_2 = -1 < 0$, $\alpha_3 = \alpha_4 = 0$, and $\alpha_1 = h - s > 0$. Thus, the equilibrium point is unstable.

The equilibrium point $\zeta_2^* = (1, 0)$. Here we have $\alpha_3 = \alpha_4 = 0$, and $\alpha_1 = \frac{s}{2} > 0$, $\beta_2 = h - \frac{s}{2} - 1$. Thus, the equilibrium point is unstable.

The equilibrium point ζ_3^* . Here we have $U_A^* = U_B^*$, thus $\alpha_1 = 0$, $\beta_2 = \frac{2h(h-s)}{2h-s} - 1$ which is positive if $h > 1$ and $0 < s < \frac{2h(h-1)}{2h-1} < h$, and then the equilibrium point is unstable. For $h < 1$ or (for $h > 1$) $\frac{2h(h-1)}{2h-1} < s < h$. The stability of the equilibrium point is then determined by zeros of the transcendental equation

$$\lambda - \frac{s(h-s)}{(2h-s)^2} (s e^{-\lambda\tau_A} - 2h e^{-\lambda\tau_B}) = 0. \quad (12)$$

It is easy to see that for $\tau_A = \tau_B = 0$ the zero of (12) is negative. Thus, (see eg. [53]), for small τ_A and τ_B all zeros of (12) have negative real parts, and thus the equilibrium point is stable under the condition $h < 1$ or $\frac{2h(h-1)}{2h-1} < s < h$. However, as delays increase, the equilibrium point becomes unstable. If $\tau_A = \tau_B = \tau$ there exists a single critical delay τ^* such that the equilibrium point is stable for $\tau_A = \tau_B = \tau < \tau^*$ and unstable for $\tau > \tau^*$. If delays are not equal, the situation is more complex. As this case is not biologically relevant, we do not discuss it in detail.

The equilibrium point $\zeta_5^* = \left(1, 1 - \frac{2}{2h-s}\right)$. Here we have $\alpha_1 = \frac{s}{2h-s} > 0$, $\alpha_3 = \alpha_4 = 0$, and $\beta_2 = 1 - \frac{2h-s}{2} > 0$ if the coordinates of ζ_5^* are positive. Thus, the equilibrium point is unstable.

3.2.1 Stability of the Interior Equilibrium Point

For the equilibrium point ζ_6^* we have $\alpha_1 = 0$ as $U_A^* = U_B^*$, and

$$\alpha_3 = \frac{s^2}{2h(2h-s)}, \quad \alpha_4 = -\frac{sh}{h(2h-s)}, \quad \beta_2 = 1 - \frac{2h(h-s)}{2h-s}.$$

Note, that if $p^* > 0$ for the equilibrium point ζ_6^* then $\frac{2h(h-s)}{2h-s} > 1$ and therefore $\beta_2 < 0$. Thus, the stability of the equilibrium point ζ_6^* is determined by zeros of the following transcendental equation

$$\lambda - \frac{s}{2h(2h-s)} (s e^{-\lambda\tau_A} - 2h e^{-\lambda\tau_B}) = 0. \quad (13)$$

Note that the characteristic equation can be written as

$$\lambda - \frac{\frac{s}{h}}{2\left(2 - \frac{s}{h}\right)} \left(\frac{s}{h} e^{-\lambda\tau_A} - 2 e^{-\lambda\tau_B}\right) = 0.$$

Thus, the stability of the interior equilibrium point depends only on the ratio between the cost and the benefit, as well as on the magnitudes of the delays. However, for the equilibrium to exist, the benefit must be greater than 1 and the ratio between the cost and the benefit cannot exceed $1 - \frac{1}{2h-1}$.

The case $\tau_A = \tau_B = \tau > 0$. In this case, we assume that mean time delays for both strategies are the same. The transcendental equation (13) reduces to

$$\lambda + \frac{s}{2h} e^{-\lambda\tau} = 0.$$

It is well known (see e.g. [53, Th. 4.7]) that for $\tau < \tau^* = \frac{\pi h}{s}$ the equilibrium point is stable; it is unstable for $\tau > \tau^*$, and for $\tau = \tau^*$ the Hopf bifurcation occurs. We summarize this as

Theorem 4 Suppose the equilibrium point ζ_6^* exists in the first quadrant for system (8) with the initial conditions given in (9). Let $\tau^* = \frac{\pi h}{s}$. If $\tau_A = \tau_B = \tau$ then

- (i) if $\tau < \tau^*$, the interior equilibrium point ζ_6^* is locally asymptotically stable;
- (ii) if $\tau > \tau^*$, the interior equilibrium point ζ_6^* is unstable;
- (iii) at $\tau = \tau^*$ the Hopf-bifurcation around the interior equilibrium point ζ_6^* occurs.

Figure 3 illustrates the time evolution of frequency of strategy A, $x(t)$ and relative size of the population $p(t)$ obtained by solving equations (8a) and (8b) under the condition where the time delays for both strategies are identical, i.e., $\tau_A = \tau_B = \tau$. For the parameter set $h = 3$ and $s = 2$, the system, in the absence of delay, has a stable interior equilibrium point at $(0.5, 0.33)$. As we have shown, the critical delay threshold at which bifurcation occurs for these parameter values is $\tau^* = \frac{\pi h}{s} = 4.71$. When $\tau < \tau^*$, all trajectories converge to the stable equilibrium. However, once τ exceeds this threshold, the system exhibits sustained oscillations, indicating a transition from stability to periodic behavior. This transition in system behavior is more clearly illustrated in the bifurcation diagram presented in Fig. 4, which depicts how the stability of the interior equilibrium point changes as τ varies. The figure highlights the emergence of a critical delay value beyond which the system undergoes a Hopf bifurcation, leading to the onset of oscillatory dynamics.

The case $\tau_A = 0, \tau_B > 0$. In this case we assume that only strategy B is delayed. The transcendental equation (13) reads

$$\lambda - \frac{s^2}{2h(2h-s)} + \frac{sh}{h(2h-s)} e^{-\lambda\tau_B} = 0.$$

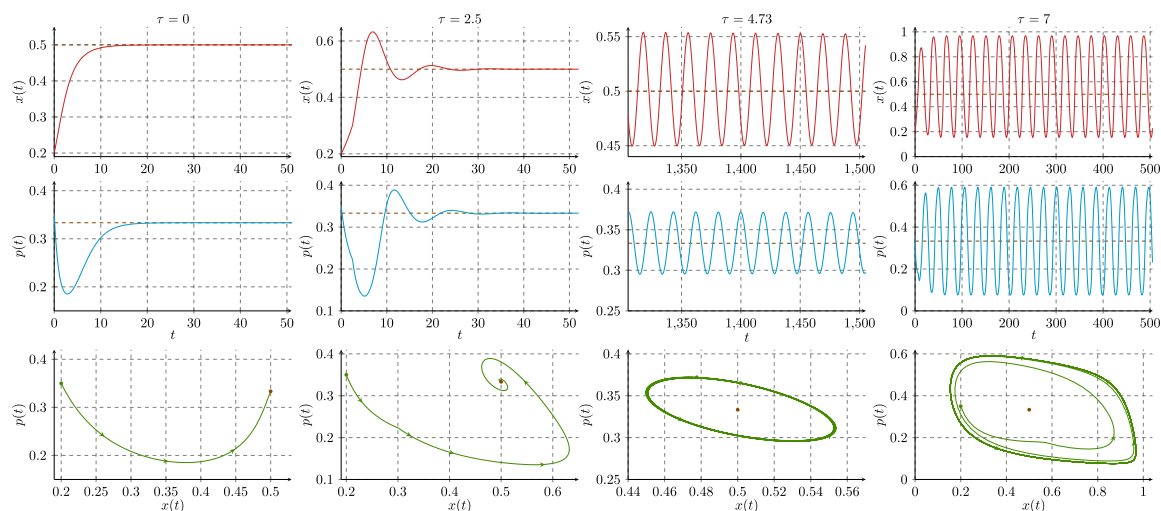


Fig. 3 The dynamics of the frequency of strategy A, $x(t)$ and relative size of the population $p(t)$ when time delays are the same for both strategies ($\tau_A = \tau_B = \tau$)

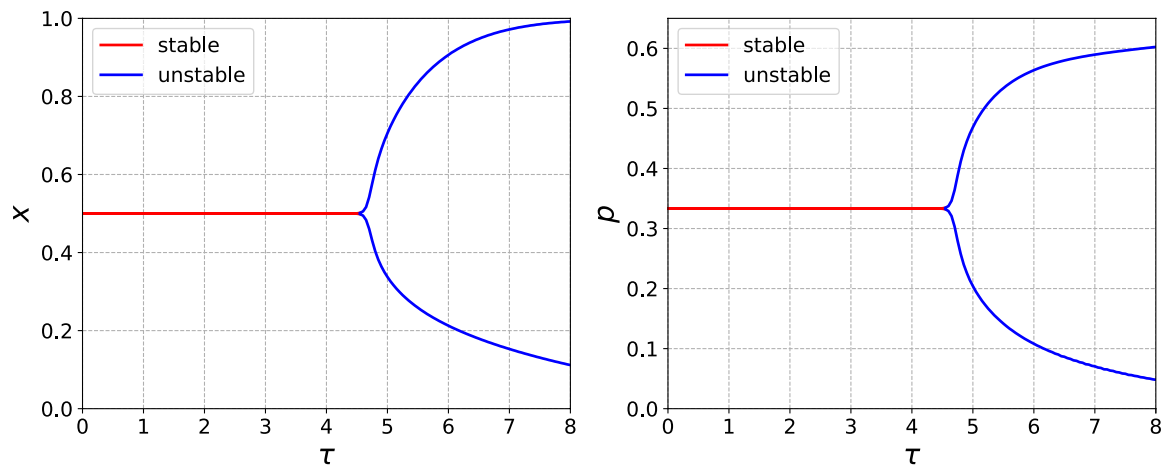


Fig. 4 Bifurcation diagram illustrating the stability of the interior equilibrium point ζ_6^* under symmetric time delays $\tau_A = \tau_B = \tau$. The diagrams highlight a critical delay value $\tau^* = 4.71$ for $h = 3$, $s = 2$ at which a Hopf bifurcation occurs, leading to the loss of stability and the emergence of stable oscillations

Again, using [53, Th. 4.7] we conclude

Theorem 5 Suppose the equilibrium point ζ_6^* exists at the first quadrant for system (8) with the initial conditions given in (9). Let

$$\tau_B^* = \frac{2h(2h-s) \arccos\left(\frac{s}{2h}\right)}{s\sqrt{4h^2-s^2}}.$$

If $\tau_A = 0$ and $\tau_B > 0$ then

- (i) if $\tau_B < \tau_B^*$, the interior equilibrium point ζ_6^* is locally asymptotically stable;
- (ii) if $\tau_B > \tau_B^*$, the interior equilibrium point ζ_6^* is unstable;
- (iii) at $\tau_B = \tau_B^*$ the Hopf-bifurcation around the interior equilibrium point ζ_6^* occurs.

Figure 5 presents representative trajectories for the strategy-A frequency $x(t)$ and the scaled population size $p(t)$. The curves were obtained by numerically solving equations (8) under the asymmetric-delay setting $\tau_A = 0$ while allowing the defector delay τ_B to vary. For the parameters $h = 3$ and $s = 2$, the system when $(\tau_B = 0)$ possesses a single asymptotically stable interior fixed point at $(x, p) = (0.5, 0.33)$. Our analysis reveals that this equilibrium loses stability through a Hopf bifurcation once the delay exceeds $\tau_B^* = \frac{2h(2h-s) \arccos(\frac{s}{2h})}{s\sqrt{4h^2-s^2}}$. When $\tau_B < \tau_B^*$, every trajectory relaxes monotonically to the fixed point; for $\tau_B > \tau_B^*$, the equilibrium becomes unstable and the system evolves toward a stable limit cycle, generating persistent oscillations in both $x(t)$ and $p(t)$. The bifurcation diagram in Fig. 6 highlights this transition by revealing how variations in τ_B affect the stability of the interior equilibrium point.

The case $\tau_A > 0$, $\tau_B = 0$. In this case we the transcendental equation (13) reads

$$\lambda - \frac{s^2}{2h(2h-s)} e^{-\lambda\tau_A} + \frac{sh}{h(2h-s)} = 0.$$

We see that in this case $\frac{s^2}{2h(2h-s)} > -\frac{sh}{h(2h-s)}$ and $\frac{s^2}{2h(2h-s)} - \frac{sh}{h(2h-s)} = -\frac{s}{2h} < 0$, so (using [53, Th. 4.7]) the equilibrium point ζ_6^* is stable for all $\tau_A > 0$.

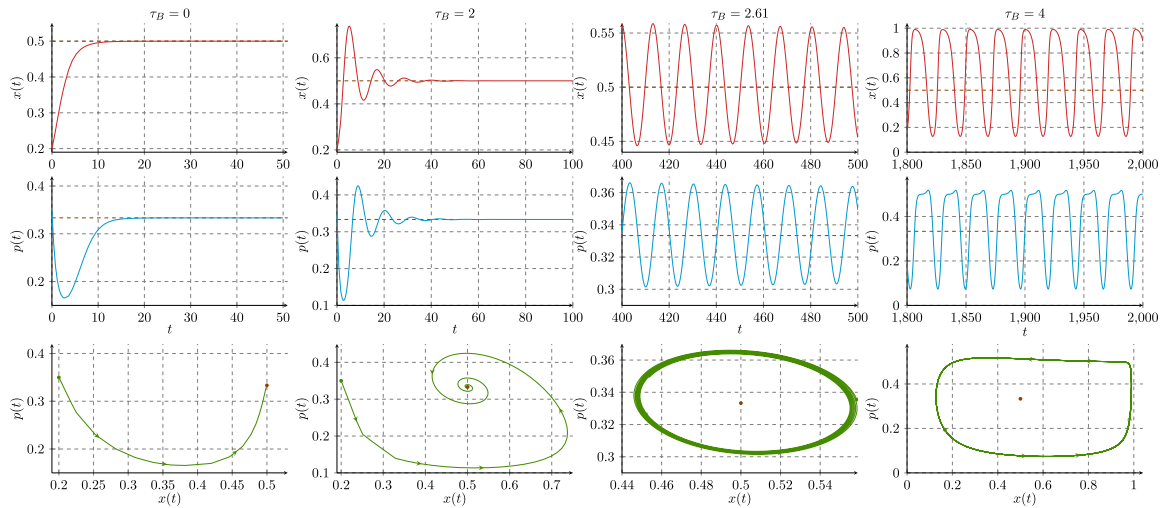


Fig. 5 The dynamics of the frequency of strategy A, $x(t)$ and relative size of the population $p(t)$ when $\tau_A = 0$

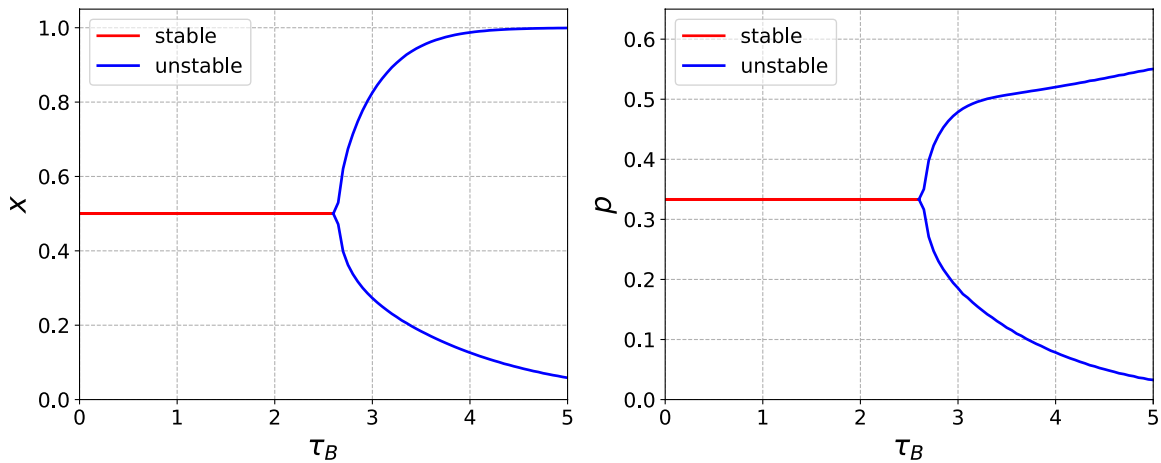


Fig. 6 Bifurcation diagram illustrating the stability of the interior equilibrium point ζ_6^* where $\tau_A = 0$ and $\tau_B > 0$. The diagrams highlight a critical delay value $\tau_B^* = 2.61$ for $h = 3, s = 2$ at which a Hopf bifurcation occurs, leading to the loss of stability and the emergence of stable oscillations

Theorem 6 Suppose the equilibrium point ζ_6^* exists in the first quadrant for system (8) with the initial conditions given in (9). For $\tau_B = 0$ the equilibrium point ζ_6^* is stable for all $\tau_A > 0$.

The general case, $\tau_A > 0$, and $\tau_B > 0$.

In this case, it is much more complicated to find zeros of a general form of the transcendental equation (13). However, we have proved that the equilibrium point is stable for $\tau_B = 0$ and any $\tau_A > 0$. Thus, the continuous dependence of zeros of (13) implies that for τ_B small enough the equilibrium point ζ_6^* is locally asymptotically stable. If it loses its stability for some $\tau_B > 0$ (and fixed $\tau_A > 0$), then there exists τ_B^* such that $\lambda = i\omega$ is a solution of (13) for some $\omega \geq 0$. Substituting $\lambda = i\omega$ into (13) and separating the real and imaginary parts we get

$$\begin{aligned} s \cos(\omega \tau_A) &= 2h \cos(\omega \tau_B), \\ \omega + \frac{s^2}{2h(2h-s)} \sin(\omega \tau_A) &= \frac{s}{2h-s} \sin(\omega \tau_B). \end{aligned} \quad (14)$$

Squaring both sides of (14) and adding equations we conclude, that $\omega \geq 0$ such that $\lambda = i\omega$ is a solution of (13) exists if and only if the function

$$G(\omega) = \omega^2 + \frac{s}{h(2h-s)} \omega \sin(\omega \tau_A) - \left(\frac{s}{2h(2h-s)} \right)^2 (2h+s)(2h-s)$$

has a positive zero. It is clear that $G(0) < 0$ and $\lim_{\omega \rightarrow \infty} G(\omega) = +\infty$. Thus, G has at least one positive zero; in the generic case, an odd number of positive zeros of G exist. Assume now, that $0 < \omega_0 < \omega_1 < \dots < \omega_{2k}$ are zeros of G and all are single. For each ω_j , $j = 0, 1, 2, \dots, 2k$, when we have a sequence of critical delays

$$\tau_{B,j,\ell}^* = \tau_{B,j}^* + \frac{2\ell\pi}{\omega_j} > 0, \quad \ell \in \mathbb{N}$$

which are solutions of (14) and such that $\lambda = i\omega_j$ is zero of (13) with $\tau_B = \tau_{B,j,\ell}^*$. From the graph of G it is clear that $G'(\omega_{2j}) > 0$ and $G'(\omega_{2j-1}) < 0$. Thus, by [54, Th. 1], we conclude that

$$\left. \frac{d \operatorname{Re}(\lambda)}{d\tau_B} \right|_{\lambda=i\omega_{2j}} > 0, \quad \left. \frac{d \operatorname{Re}(\lambda)}{d\tau_B} \right|_{\lambda=i\omega_{2j-1}} < 0.$$

Thus, when τ_B is increasing and it crosses $\tau_{B,j,\ell}^*$, zeros of (13) crosses imaginary axis from left to right for j even and from right to left for j odd. If j is even and such that the sequence of $\tau_{B,j,\ell}^*$ exists, the sequence $\tau_{B,j+1,\ell}^*$ also exists. Note also, that $\omega_j < \omega_{j+1}$, thus the points $\tau_{B,j,\ell}^*$ at which λ moves from the right of the complex half-plane to the left one are less frequent than point $\tau_{B,j+1,\ell}^*$ at which λ moves in the opposite direction. Thus, for τ_B large enough at least a pair of zeros of (13) lies in the right of the complex half-plane. Therefore, the equilibrium point ζ_6^* is unstable for sufficiently large τ_B . Although the result is not satisfactory, we have no hope for a better analytical result, as in general case we cannot find an analytical formula for zeros of G and for solutions of (14).

We proceed in a little distinct manner and we find a parametrization of the borders between stability and instability regions in the (τ_A, τ_B) plane (for $\tau_A > 0$, $\tau_B > 0$). First, note that since $G(\omega) = 0$, then have

$$\sin(\omega \tau_A) = \frac{2h+s}{4h\omega} - \frac{h(2h-s)\omega}{s^2}. \quad (15)$$

Clearly $|\sin(\omega \tau_A)| \leq 1$. As the right-hand side of (15) is a decreasing function of $\omega > 0$, solving (15) for the left-hand side equal to 1 and -1 we get bounds for ω :

$$\frac{s}{2h} \leq \omega \leq \frac{s(2h+s)}{2h(2h-s)} = \frac{s}{2h} \left(1 + \frac{2s}{2h-s} \right). \quad (16)$$

For any ω that fulfills (16) we have two solutions of the second equation of (14), namely

$$\begin{aligned}\tau_A^{1,j} &= \frac{1}{\omega} \left(\arcsin \left(\frac{\gamma^2(4h^2 - s^2) - \omega^2}{2\gamma\omega s} \right) + 2j\pi \right), \\ \tau_A^{2,j} &= \frac{1}{\omega} \left(\pi - \arcsin \left(\frac{\gamma^2(4h^2 - s^2) - \omega^2}{2\gamma\omega s} \right) + 2j\pi \right),\end{aligned}$$

for $j \in \mathbb{N}$ and $\gamma = \frac{s}{2h(2h-s)}$. Having $\tau_A^{k,j}(\omega)$ we solve (14), namely

$$\begin{aligned}\tau_B &= \frac{1}{\omega} \left(\arccos \left(\frac{s \cos(\omega\tau_A)}{h} \right) + 2\ell\pi \right), \quad \text{for } \omega + s\gamma \sin(\omega\tau_A) \geq 0, \\ \tau_B &= \frac{1}{\omega} \left(2\pi - \arccos \left(\frac{s \cos(\omega\tau_A)}{h} \right) + 2\ell\pi \right), \quad \text{for } \omega + s\gamma \sin(\omega\tau_A) < 0.\end{aligned}$$

Using these formulas we numerically draw borders of the stability region. In Fig. 7, we present the stability region for the parameters $h = 3$ and $s = 2$. As observed, fixing one delay while varying the other leads to multiple stability switches. For instance, when we fix $\tau_B = 2.9$ and vary τ_A , several critical values emerge where stability transitions occur. Specifically, at $\tau_A^* \approx 0.61$, the system transitions from an unstable to a stable state; at $\tau_A^* \approx 6.48$, oscillations reappear; at $\tau_A^* \approx 15.71$, the system regains stability; and at $\tau_A^* \approx 16.53$, stability switches back to instability. Naturally, as τ_A increases further, additional critical values exist. As another example, when we fix $\tau_A = 3$ and vary τ_B , we find a bifurcation at $\tau_B^* \approx 3.9$, where the system transitions from a stable to an unstable state. The bifurcation diagram and corresponding system dynamics for these transitions are depicted in Figs. 8 and 9.

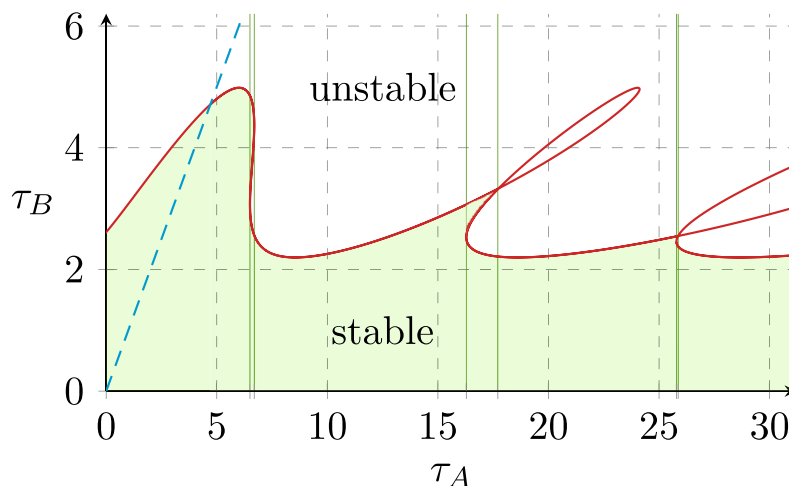


Fig. 7 The stability region in the plane (τ_A, τ_B) for $h = 3$ and $s = 2$. Note that for some τ_A (indicated by vertical green lines) multiple stability switches occur. When we increase τ_B the equilibrium point ζ_6^* is stable, then it loses stability, later, stability is regained, and it eventually becomes unstable again. The blue dashed line indicates $\tau_A = \tau_B$

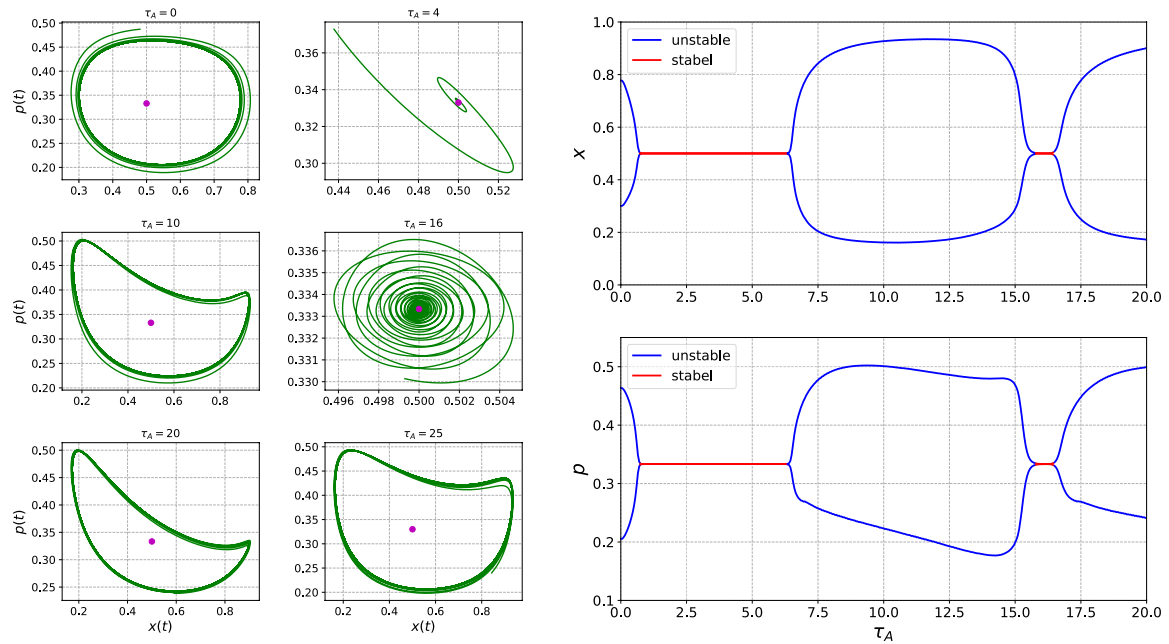


Fig. 8 Dynamics of frequency of strategy A, $x(t)$ and relative size of the population $p(t)$, along with the bifurcation diagram for the case where $\tau_B = 2.9$ and τ_A varies

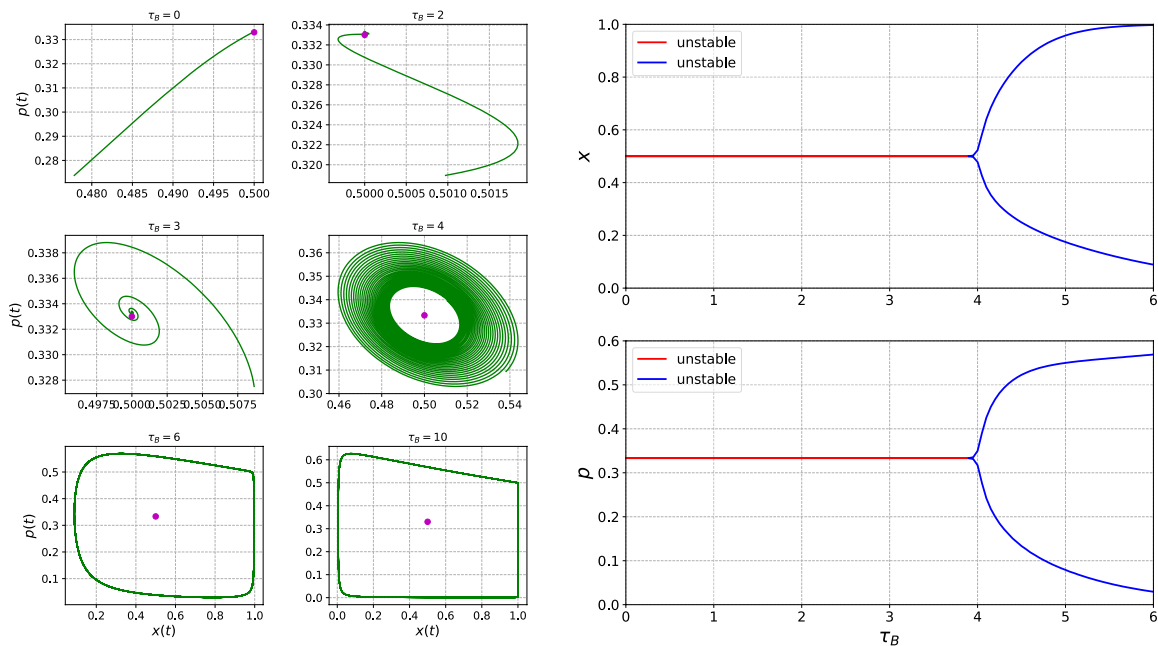


Fig. 9 Dynamics of frequency of strategy A, $x(t)$ and relative size of the population $p(t)$, along with the bifurcation diagram for the case where $\tau_A = 3.0$ and τ_B varies

4 Direction and Stability of Hopf Bifurcation

In this section, we investigate the direction and stability of the Hopf bifurcation in the delayed replicator dynamics, assuming that both strategies are subject to the same discrete time delay, i.e., $\tau_A = \tau_B = \tau$. We focus on the symmetric delay case because the analysis becomes significantly more complex when the delays differ; in such cases, the expressions for ω_0 and, consequently, for τ_0 become more intricate, making the calculations substantially

more challenging. Nevertheless, the analytical approach presented here can be extended to the asymmetric delay case following a similar methodology. We will follow the approach proposed by [55], which was successfully used to study direction of the Hopf bifurcation for some other mathematical models of biological phenomena [56–60]

In the following theorem we show that the Hopf bifurcation of the interior equilibrium arising in the system (8) with the Snowdrift payoff matrix when $\tau_A = \tau_B = \tau$ crosses critical value of $\tau_0 = \pi \frac{h}{s}$ is *supercritical*. In this case, a periodic solutions exist for delays $\tau_A = \tau_B > \tau_0$. Furthermore, as the equilibrium is stable for equal delays smaller than τ_0 , then the periodic solution is stable within the center manifold.

Theorem 7 *Assume that the interior equilibrium point ζ_6^* exists in the first quadrant. The System (8) with the Snowdrift payoff matrix and the same delays $\tau_A = \tau_B = \tau$ exhibits the supercritical Hopf bifurcation at $\tau_0 = \frac{\pi h}{s}$ around ζ_6^* .*

Proof In order to study the stability and direction of the Hopf bifurcation, we analyze the Taylor expansion of the right-hand side of System (8) around the interior equilibrium point ζ_6^* , where ζ_6^* is defined in (10). To simplify the analysis, we perform a change of variables to shift the equilibrium point to the origin, i.e., to $(0, 0)$. It would also be convenient to use $\sigma = \frac{s}{h}$ instead of s . The parameter $\sigma \in (0, 1)$ represents the fraction of the benefit that is considered as cost. Let $\tilde{x} = x - x^*$ and $\tilde{p} = p - p^*$, where x^* denotes the frequency of strategy A at equilibrium. Here, $p^* = 1 - \frac{1}{U^*}$, with $U^* = x^*U_A^* + (1 - x^*)U_B^*$ representing the average payoff. To simplify the notation, we define $\alpha^* = h(1 - \frac{\sigma}{2})$, so that $U_A^* - U_B^* = -\alpha^*x^*$. Using this notation, System (8) with $\tau_A = \tau_B = \tau$, corresponding to the Snowdrift payoff matrix, takes the form:

$$\begin{aligned} \frac{d\tilde{x}}{dt} &= -\alpha^*x(t-\tau)(\tilde{x} + x^*)(1 - \tilde{x} - x^*)\left(\frac{1}{U^*} - \tilde{p}\right), \\ \frac{d\tilde{p}}{dt} &= (p^* + \tilde{p})\left[\frac{s}{2U^*}(\tilde{x}(t-\tau) - \tilde{x}) - U^*\tilde{p} - \frac{\alpha^*}{U^*}\tilde{x}\tilde{x}(t-\tau) \right. \\ &\quad \left. + \frac{s}{2}(\tilde{x}\tilde{p} - \tilde{x}(t-\tau)\tilde{p}) + \alpha^*\tilde{x}\tilde{p}\tilde{x}(t-\tau)\right]. \end{aligned} \quad (17)$$

Now, we introduce the necessary notation. Let $\mathcal{C}$ denote the Banach space of continuous functions defined on the interval $[-\tilde{\tau}, 0]$ with values in $\mathbb{C}^2$, where $\tilde{\tau}$ is a fixed but arbitrarily chosen number greater than τ^* . The space $\mathcal{C}$ is equipped with the standard supremum norm. (The use of complex-valued functions is purely technical; see [55] for details.)

For any continuous function $z : [-\tilde{\tau}, \infty) \rightarrow \mathbb{C}^2$, $z = (z_1, z_2)$ we define $z_t \in \mathcal{C}$ by $z_t(\theta) = z(t + \theta)$ for any $\theta \in [-\tilde{\tau}, 0]$. Let $L : \mathcal{C} \rightarrow \mathbb{C}^2$ be a linear operator, and consider the linear delay differential $\dot{z} = Lz_t$, where $\dot{z}$ denotes the right-hand derivative of z . Associated with this equation is a solution operator $T(t) : \mathcal{C} \rightarrow \mathcal{C}$ defined by $T(t)\varphi = z_t$, where z is the solution to the linear DDE with initial function $\varphi \in \mathcal{C}$. The family $T(t)_{t \geq 0}$ forms a strongly continuous semigroup on $\mathcal{C}$ (see [55]). Let $\mathcal{A}$ denote the infinitesimal generator of this semigroup.

Using this notation, we decompose the right-hand side of system (17) into its linear and nonlinear components as

$$\frac{d}{dt} \begin{bmatrix} \tilde{x} \\ \tilde{p} \end{bmatrix} = L(\tilde{x}_t, \tilde{p}_t) + G(\tilde{x}_t, \tilde{p}_t),$$

where $L : \mathcal{C} \rightarrow \mathbb{C}^2$ denotes the linear operator and $G : \mathcal{C} \rightarrow \mathbb{C}^2$ is the nonlinear one and defined as follow:

$$\begin{aligned} L_1(\phi_1, \phi_2) &= -\frac{\sigma}{2}\phi_1(-\tau), \\ L_2(\phi_1, \phi_2) &= \frac{h\sigma p^*}{2U^*}(\phi_1(-\tau) - \phi_1(0)) - U^*p^*\phi_2(0), \end{aligned}$$

and

$$\begin{aligned} G_1(\phi_1, \phi_2) &= -\alpha^* \left[\frac{1-2x^*}{U^*}\phi_1(0)\phi_1(-\tau) - x^*(1-x^*)\phi_1(-\tau)\phi_2(0) \right. \\ &\quad + \frac{1}{U^*}\phi_1^2(0)\phi_1(-\tau) + (1-2x^*)\phi_1(0)\phi_1(-\tau)\phi_2(0) \\ &\quad \left. + \phi_1^2(0)\phi_1(-\tau)\phi_2(0) \right], \end{aligned} \quad (18)$$

$$\begin{aligned} G_2(\phi_1, \phi_2) &= p^* \left[\frac{-\alpha^*}{U^*}\phi_1(0)\phi_1(-\tau) + \frac{h\sigma}{2}\phi_2(0)(\phi_1(0) - \phi_1(-\tau)) + \alpha^*\phi_1(0)\phi_1(-\tau)\phi_2(0) \right] \\ &\quad + \phi_2(0) \left[\frac{h\sigma}{2U^*}(\phi_1(-\tau) - \phi_1(0)) - U^*\phi_2(0) + \frac{\alpha^*}{U^*}\phi_1(0)\phi_1(-\tau) \right. \\ &\quad \left. + \frac{h\sigma}{2}\phi_2(0)(\phi_1(0) - \phi_1(-\tau)) + \alpha^*\phi_1(0)\phi_1(-\tau)\phi_2(0) \right]. \end{aligned} \quad (19)$$

In the studied case, the characteristic matrix takes the following form:

$$\Delta(\lambda, \tau) = \begin{bmatrix} \lambda + \frac{\sigma}{2}e^{-\lambda\tau} & 0 \\ \frac{h\sigma}{2U^*}p^*(1 - e^{-\lambda\tau}) & \lambda + U^*p^* \end{bmatrix}.$$

The Hopf bifurcation occurs when the pair of eigenvalues λ crosses the imaginary axis. At this point, we have $\lambda = i\omega_0$ with $\omega_0 = \frac{s}{2h} = \frac{\sigma}{2}$, and the critical delay is given by $\tau_0 = \tau^* = \frac{\pi h}{s} = \frac{\pi}{\sigma}$. Thus, we have: $e^{-i\omega_0\tau_0} = e^{-i\pi/2} = -i$.

The eigenvector of the infinitesimal operator $\mathcal{A}$ associated with the eigenvalue $i\omega_0$ is given by $\phi(\theta) = e^{i\omega_0\theta}\vec{p}$, where $\vec{p}$ is a right eigenvector of $\Delta(i\omega_0, \tau_0)$. Because the first row of $\Delta(i\omega_0, \tau_0)$ is zero, we choose $\vec{p} = [1, 0]^T$ and therefore,

$$\phi(\theta) = e^{i\omega_0\theta} \begin{bmatrix} 1 \\ 0 \end{bmatrix}. \quad (20)$$

The eigenvector of the adjoint operator $\mathcal{A}^*$ satisfies $\psi(\theta) = \vec{q} e^{i\omega_0\theta}$, where $\vec{q}$ is left eigenvector of $\Delta(i\omega_0, \tau_0)$ and satisfies

$$\langle \psi, \phi \rangle = \vec{q} D_1 \Delta(i\omega_0, \tau_0) \vec{p} = 1,$$

where $D_1 \Delta(i\omega_0, \tau_0)$ is the derivative of $\Delta(\lambda, \tau)$ with respect to the first variable λ , and is given by

$$D_1 \Delta(\lambda, \tau) = \begin{bmatrix} 1 - \frac{\sigma}{2} \tau e^{-\lambda\tau} & 0 \\ \frac{h\sigma}{2U^*} p^* \tau e^{-\lambda\tau} & 1 \end{bmatrix} \implies D_1 \Delta(i\omega_0, \tau_0) = \begin{bmatrix} 1 - i\frac{\pi}{2} & 0 \\ -i\pi \frac{h}{2U^*} p^* & 1 \end{bmatrix}.$$

It is easy to see that the vector $\vec{q} = \left[\frac{1 - i\frac{\pi}{2}}{1 + \frac{\pi^2}{4}}, 0 \right]$ has requested properties and, thus,

$$\psi(\theta) = \vec{q} e^{i\omega_0\theta}.$$

Studying the type and stability of the bifurcating periodic solution within the center manifold, we shall determine the third term u in the Taylor expansion of this solution around the critical value τ , which is given by:

$$u = \frac{\text{Re}\{c\}}{\text{Re}\{\vec{q} D_2 \Delta(i\omega_0, \tau_0) \vec{p}\}},$$

where $c = c_1 + c_2 + c_3$ is composed of non-linear forms involving derivatives of the non-linear operator G . The terms c_i are defined as:

$$c_1 = \frac{1}{2} \vec{q} D_1^3 G(0, \tau_0)(\phi, \phi, \bar{\phi}), \quad c_2 = \vec{q} D_1^2 G(0, \tau_0)(\rho, \phi), \quad c_3 = \frac{1}{2} \vec{q} D_1^2 G(0, \tau_0)(\varsigma, \bar{\phi}) \quad (21)$$

with

$$\rho(\theta) = e^{0 \cdot \theta} (\Delta(0, \tau_0))^{-1} D_1^2 G(0, \tau_0)(\phi, \bar{\phi}), \quad (22)$$

$$\varsigma(\theta) = \exp(2i\omega_0\theta) (\Delta(2i\omega_0, \tau_0))^{-1} D_1^2 G(0, \tau_0)(\phi, \phi), \quad (23)$$

where D_k^l , $k = 1, 2$, $l = 1, 2, 3$ denotes the l -th derivative with respect to the k -th variable. We intentionally wrote $e^{0 \cdot \theta}$ to indicate that ρ is a function of θ , although it is a constant function. As usual, we need to have $u \neq 0$. Then we have two possibilities:

1. If $u > 0$, then the bifurcation is referred to as *supercritical*. In this case, a periodic solutions exist for $\tau > \tau_0$. Furthermore, if the steady state from which the periodic solution bifurcates is stable for $\tau < \tau_{cr}$, then the periodic solution is stable within the center manifold. Additionally, if the spectrum of $\mathcal{A}$ lies entirely in the left half-plane, the center manifold is attractive and therefore the periodic solution is asymptotically stable.

2. If $u < 0$, the bifurcation is referred to as *subcritical* and the bifurcating periodic solutions exist for $\tau < \tau_0$. If, as before, the steady state is stable for $\tau < \tau_0$, then the periodic solutions are unstable.

To this end, we need to calculate the derivatives of the non-linear part of the equation, that is the derivatives of $G_k, k = 1, 2$ given by (18) and (19) with respect to the first variable. For arbitrary functions $\nu, \mu, \zeta \in C_{\bar{\tau}}$, where $\nu = (\nu_1, \nu_2), \mu = (\mu_1, \mu_2)$, and $\zeta = (\zeta_1, \zeta_2)$ we have:

$$D_1^2 G_1(0, \tau_0)(\nu, \mu) = -\alpha^* \left(\frac{1 - 2x^*}{U^*} (\nu_1(0)\mu_1(-\tau) + \nu_1(-\tau)\mu_1(0)) - x^*(1 - x^*) (\nu_1(-\tau)\mu_2(0) + \nu_2(0)\mu_1(-\tau)) \right), \quad (24)$$

$$D_1^3 G_1(0, \tau_0)(\nu, \mu, \zeta) = -\alpha^* \left(\frac{-2}{U^*} \left(\nu_1(0)\mu_1(0)\zeta_1(-\tau) + \nu_1(0)\mu_1(-\tau)\zeta_1(0) + \nu_1(-\tau)\mu_1(0)\zeta_1(0) \right) + (1 - 2x^*) \left(\nu_1(0)\mu_1(-\tau)\zeta_2(0) + \nu_1(-\tau)\mu_1(0)\zeta_2(0) + \nu_1(0)\mu_2(0)\zeta_1(-\tau) + \nu_2(0)\mu_1(0)\zeta_1(-\tau) + \nu_1(-\tau)\mu_2(0)\zeta_1(0) + \nu_2(0)\mu_1(-\tau)\zeta_1(0) \right) \right), \quad (25)$$

$$D_1^2 G_2(0, \tau_0)(\nu, \mu) = p^* \left(-\frac{\alpha^*}{U^*} (\nu_1(0)\mu_1(-\tau) + \nu_1(-\tau)\mu_1(0)) + \frac{h\sigma}{2} (\nu_1(0)\mu_2(0) + \nu_2(0)\mu_1(0)) - \frac{h\sigma}{2} (\nu_2(0)\mu_1(-\tau) + \nu_1(-\tau)\mu_2(0)) \right), \quad (26)$$

$$D_1^3 G_2(0, \tau_0)(\nu, \mu, \zeta) = \left(\alpha^* p^* - \frac{\alpha^*}{U^*} \right) \left(\nu_1(0)\mu_1(-\tau)\zeta_2(0) + \nu_1(0)\mu_2(0)\zeta_1(-\tau) + \nu_2(0)\mu_1(-\tau)\zeta_1(0) + \nu_1(-\tau)\mu_1(0)\zeta_2(0) + \nu_2(0)\mu_1(0)\zeta_1(-\tau) + \nu_1(-\tau)\mu_2(0)\zeta_1(0) \right) + 2h\sigma \left(\nu_1(0)\mu_2(0)\zeta_2(0) + \nu_2(0)\mu_1(0)\zeta_2(0) + \nu_2(0)\mu_2(0)\zeta_1(0) \right) - 2h\sigma \left(\nu_1(-\tau)\mu_2(0)\zeta_2(0) + \nu_2(0)\mu_1(-\tau)\zeta_2(0) + \nu_2(0)\mu_2(0)\zeta_1(-\tau) \right). \quad (27)$$

In order to determine the sign of the coefficient u in the normal form, we analyze the sign of the real part of the numerator and denominator separately. First, we prove, that the denominator of u is negative. To this end, we compute the derivative of the characteristic matrix $\Delta(\lambda, \tau)$ with respect to τ at the Hopf bifurcation point $(\lambda = i\omega_0, \tau = \tau_0)$. We have:

$$D_2\Delta(\lambda, \tau) = \begin{bmatrix} -\lambda \frac{\sigma}{2} e^{-\lambda\tau} & 0 \\ \lambda \frac{h\sigma}{2U^*} p^* e^{-\lambda\tau} & 0 \end{bmatrix} \implies D_2\Delta(i\omega_0, \tau_0) = \begin{bmatrix} -\frac{\pi}{2} & 0 \\ -\frac{\pi h p^*}{2U^*} & 0 \end{bmatrix}.$$

Therefore, the real part of the denominator of u is:

$$\operatorname{Re}\{\vec{q} D_2\Delta(i\omega_0, \tau_0) \vec{p}\} = \operatorname{Re}\left\{\frac{1 - i\frac{\pi}{2}}{1 + \frac{\pi^2}{4}} \left(-\frac{\pi}{2}\right)\right\} = -\frac{\frac{\pi}{2}}{1 + \frac{\pi^2}{4}} < 0.$$

Now, we turn our attention to the numerator of u and prove that it is negative. We evaluate the real parts of c_1 , c_2 , and c_3 separately. Using the definition of c_1 (see (21)), the chosen vector $\vec{q}$, formula for eigenfunction ϕ (see Eq. 20, 25, and 27), we obtain:

$$c_1 = \frac{1}{2} \vec{q} D_1^3 G(0, \tau_0)(\phi, \phi, \bar{\phi}) = \frac{-1}{2(1 + \frac{\pi^2}{4})} \left(\frac{\pi \alpha^*}{U^*} + i \frac{2\alpha^*}{U^*} \right).$$

Hence,

$$\operatorname{Re}\{c_1\} = \frac{-1}{2(1 + \frac{\pi^2}{4})} \left(\frac{\pi \alpha^*}{U^*} \right). \quad (28)$$

We now show that $c_2 = 0$. To compute c_2 we evaluate the matrix $D_1^2 G(0, \tau_0)(\phi, \bar{\phi})$. Since $\phi_2(\theta) \equiv 0$, all terms involving μ_2 and ν_2 in Eqs. (24) and (26) vanish. The remaining terms take the form of a constant multiplied by $\phi_1(0)\bar{\phi}_1(-\tau) + \phi_1(-\tau)\bar{\phi}_1(0)$, which equals zero, because $\phi_1(0)$ is real and $\phi_1(-\tau)$ is purely imaginary. Therefore, $D_1^2 G(0, \tau_0)(\phi, \bar{\phi}) = [0, 0]^T$, which implies that $c_2 = 0$.

The formula for c_3 involves the function $\varsigma(\theta)$ defined in Eq. (23). First, we compute the inverse of the characteristic matrix $\Delta(2i\omega_0, \tau_0)$:

$$(\Delta(2i\omega_0, \tau_0))^{-1} = \frac{1}{\sigma(U^* p^* + i\sigma) \left(-\frac{1}{2} + i\right)} \begin{bmatrix} U^* p^* + i\sigma & 0 \\ -\frac{h\sigma p^*}{U^*} & \sigma \left(-\frac{1}{2} + i\right) \end{bmatrix}. \quad (29)$$

After some algebraic calculations we get:

$$\begin{aligned} \varsigma_1(\theta) &= \frac{(2 - \sigma)(3\sigma - 2)}{5\sigma(1 - \sigma)} (2 - i) e^{2i\omega_0\theta} \\ \varsigma_2(\theta) &= \frac{p^*}{10(1 - \sigma)\eta_m} (\eta_r + \eta_i i) e^{2i\omega_0\theta}, \end{aligned}$$

where η_r , η_i , and η_m are given by:

$$\eta_r = 4h(3\sigma - 2)(1 - \sigma) + (2 - \sigma)(4 + \sigma - 8\sigma^2), \quad (30a)$$

$$\eta_i = 2h(7 - 8\sigma)(1 - \sigma) - (2 - \sigma)(7 - 12\sigma + 6\sigma^2), \quad (30b)$$

$$\eta_m = \frac{(2h(1-\sigma) - (2-\sigma))^2 + \sigma^2(2-\sigma)^2}{(2-\sigma)^2} \quad (30c)$$

Next, we compute $D_1^2 G_1(0, \tau_0)(\varsigma, \bar{\phi})$, which is required for the evaluation of c_3 . Since $\phi_2 \equiv 0$, we have:

$$D_1^2 G_1(0, \tau_0)(\varsigma, \bar{\phi}) = -\frac{(2-\sigma)^2(3\sigma-2)^2}{20\sigma(1-\sigma)^2}(-1+3i) - \frac{h\sigma p^*}{10(2-\sigma)\eta_m}(-\eta_i + \eta_r i).$$

The value of $D_1^2 G_2(0, \tau_0)(\varsigma, \bar{\phi})$ is not important as the second coordinate of $\vec{q}$ is zero. Thus,

$$c_3 = \frac{1}{2 + \frac{\pi^4}{2}} \left(1 - \frac{\pi i}{2} \right) \left(-\frac{(2-\sigma)^2(3\sigma-2)^2}{20\sigma(1-\sigma)^2}(-1+3i) - \frac{h\sigma p^*}{10(2-\sigma)\eta_m}(-\eta_i + \eta_r i) \right),$$

and finally,

$$\operatorname{Re}\{c_3\} = \frac{2-3\pi}{4+\pi^2} \cdot \frac{(2-\sigma)^2(3\sigma-2)^2}{20\sigma(1-\sigma)^2} + \frac{2\eta_i - \eta_r \pi}{4+\pi^2} \cdot \frac{h\sigma p^*}{10(2-\sigma)\eta_m}.$$

Combining this result with the value of $\operatorname{Re} c_1$ (see (28)), the fact that $c_2 = 0$, and the identity $\frac{\alpha^*}{U^*} = \frac{(2-\sigma)^2}{4(1-\sigma)}$, we obtain after some algebraic manipulation:

$$\operatorname{Re} c = \frac{(2-\sigma)}{2(4+\pi^2)(1-\sigma)} u^*,$$

where u^* is given by:

$$u^* = \frac{\sigma p^*(2\tilde{\eta}_i - \tilde{\eta}_r \pi)}{5 \left((2h(1-\sigma) - (2-\sigma))^2 + \sigma^2(2-\sigma)^2 \right)} + (2-\sigma) \left(\frac{(2-3\pi)(3\sigma-2)^2}{10\sigma(1-\sigma)} - \pi \right). \quad (31)$$

We used here also the definition of η_m , see (30c).

The second term of u^* in (31) is negative since $2 < 3\pi$. Therefore, for $\sigma \in [0, 1]$, it can be bounded from above as

$$\left(\frac{(2-3\pi)(3\sigma-2)^2}{10\sigma(h-s)} - \pi \right) (2h-s)^2 \leq -3(2h-s)^2.$$

If the inequality $2\tilde{\eta}_i \leq \pi\tilde{\eta}_r$ holds, then the first term of u^* becomes non-positive, implying $u^* < 0$, and the assertion of the theorem is proved. Thus, we assume $2\tilde{\eta}_i > \pi\tilde{\eta}_r$. Under this condition, we can bound the first term of u^* taking the maximal value of p^* , that is, $p^* = 1$, allowing us to derive an upper bound for u^* :

$$u^* < \frac{\sigma(2\tilde{\eta}_i - \tilde{\eta}_r \pi)}{5 \left((2h(1-\sigma) - (2-\sigma))^2 + \sigma^2(2-\sigma)^2 \right)} - 3(2h-s)^2.$$

In order to prove that $u^* < 0$, it is enough to demonstrate that inequality

$$\sigma(2\tilde{\eta}_i - \tilde{\eta}_r\pi) < 15(2 - \sigma)\left(\left(2h(1 - \sigma) - (2 - \sigma)\right)^2 + \sigma^2(2 - \sigma)^2\right) \quad (32)$$

holds. We denote $b = 1 - \sigma$, and we use definitions of η_r and η_i (see (30a), (30b), respectively). Through a series of algebraic manipulations, it can be shown that inequality (32) is equivalent to

$$\begin{aligned} (1 - b)\left(4bh\left((8 + 3\pi)b - \pi - 1\right) - (b + 1)\left((12 - 8\pi)b^2 + 15\pi b + 2 - 3\pi\right)\right) < \\ < 15(b + 1)\left((b^2 - 1)^2 + 2 + 8bh(bh - 1)\right). \end{aligned} \quad (33)$$

Note, both sides of (33) are functions of bh . Let $\beta = bh > 0$. Thus, inequality (33) holds for all $b \in (0, 1)$ and $h > 1$ if and only if the inequality $g_b(\beta) < 0$ holds for all $b \in (0, 1)$, and $\beta > 0$, where

$$g_b(\beta) = a_2\beta^2 + a_1\beta + a_0,$$

with

$$\begin{aligned} a_0 &= (b + 1)\left(b\left(b(-15b - 8\pi + 12) + 23\pi + 18\right) - 18\pi + 2\right) + 3\pi - 47, \\ a_1 &= 4\left(29 - \pi - b((8 + 3\pi)b - 4\pi - 39)\right), \\ a_2 &= -120(1 + b). \end{aligned}$$

Since $a_2 < 0$ for all $b \in [0, 1]$, we prove that the discriminant of g_b is negative. A tedious calculation gives

$$a_1^2 - 4a_0a_2 = 16(b - 1)\tilde{\Delta}(b),$$

where

$$\begin{aligned} \tilde{\Delta}(b) &= 450b^5 + 30(33 + 8\pi)b^4 + (116 - 9\pi(2 + \pi))b^3 + 5(\pi(3\pi - 64) - 152)b^2 + \\ &\quad - (11 + \pi)(97 + 7\pi)b + \pi(32 + \pi) - 569 \end{aligned}$$

We prove that $\tilde{\Delta}(b) < 0$ for all $b \in (0, 1)$. The function of $\tilde{\Delta}$ is a polynomial of b . We estimate its coefficients from above. In result, we get a new polynomial that is negative on $(0, 1)$ and bounds $\tilde{\Delta}(b)$ from above.

Calculating the values of coefficients of $\Delta(\beta)$ we get

$$\begin{aligned} 30(33 + 8\pi) &\approx 1743.98 < 1800, & (116 - 9\pi(2 + \pi)) &\approx -29.38 < 0, \\ 5(\pi(3\pi - 64) - 152) &\approx -1617.27 < -1350, & -(11 + \pi)(97 + 7\pi) &\approx -1682.72 < -1350, \\ \pi(32 + \pi) - 569 &\approx -458.6 < -450. \end{aligned}$$

Thus, for all $b \in [0, 1]$, we have

$$\tilde{\Delta}(b) < 450b^5 + 1800b^4 - 1350b^2 - 1350b - 450 = 450(b^5 - 1 + b(4b^3 - 3(b+1))).$$

It is straightforward to observe that $b^5 \leq 1$ for all $b \in [0, 1]$. A direct computation shows that the polynomial $4b^3 - 3(b+1)$ is strictly negative in the interval $[0, 1]$, reaching its maximum in $b = 1$. Consequently, we have

$$b(4b^3 - 3(b+1)) < -2b \leq 0 \quad \text{for all } b \in [0, 1].$$

This implies that $\tilde{\Delta}(b) < 0$ throughout the interval $[0, 1]$, leading to the conclusion that $g_b(\beta)$ has no real zeros for $b \in (0, 1)$, and thus, it is negative for all $b \in (0, 1)$ and $\beta > 0$. This implies that inequality (32) holds, which completes the proof. $\square$

5 Discussion and Conclusion

This study investigates the stability characteristics of replicator dynamics involving logistic growth in the presence of a strategy-dependent time delay. Specifically, we focus on the social type of delay, where the payoff accrued to individuals is delayed, and new offspring arise from parents who received payoffs from the past [28]. The analysis is centered around Hopf bifurcation, which explores the transitions in stability for a two-player snowdrift game.

Extensive research has been conducted to explore the impact of time delays on evolutionary game dynamics [32, 33, 44, 45, 48]. These studies have encompassed both biological and social forms of time delays [28], accounting for various scenarios such as stochastic delays [35, 38], strategy-dependent delays [31, 34], delays influenced by costs and benefits, delays following specific distributions [29], and the presence of additional factors like penalties or multi-player games [41, 43, 46, 47]. Previous investigations have shown that introducing social time delays into the dynamics of evolutionary games can destabilize previously stable mixed-strategy equilibria. The critical value of the time delay at which the Hopf bifurcation occurs varies across different dynamics, underscoring the intricate interplay between the game's underlying mechanisms and the aspects of the system.

Most previous studies have primarily focused on scenarios where population size was not a significant factor, leaving unanswered questions about how the presence of a logistic growth with a maximum population capacity could influence the properties of the system with time delay. In the absence of time delays, the frequency of strategy A at a stable interior equilibrium point is $\frac{h-s}{h-\frac{s}{2}}$ for the Snowdrift game, which aligns with the replicator dynamic without logistic growth, where the population can theoretically increase indefinitely. As demonstrated previously and corroborated in this study, the inclusion of a logistic term in the population dynamics only affects the rate at which the system approaches the stable equilibrium point [12, 14]. However, our results show that when time delays are included, the logistic term can influence not just the speed, but also the stability and long-term behavior of the system. It can change when and how oscillations appear and disappear, and shift the conditions under which the system becomes unstable. This suggests that the combined

effect of delay and limited population size plays a much more important role than previously thought.

The stability of the equilibrium solution in the delayed version of the game depends on where the characteristic value λ falls on the plane. Specifically, as λ moves from the left side to the right side of the plane, the equilibrium goes through a stability change. This is checked by solving $\lambda = i\omega$ to find the critical (τ_A^*, τ_B^*) values where the transition happens. If a solution exists, a Hopf bifurcation takes place, causing the dynamics to switch between being asymptotically stable at the balanced strategy point and displaying unstable cyclic behavior around it [45, 47, 61]. We have shown that the delay affects only the stability of the interior equilibrium point, while the nature of stability for other equilibrium points remains unchanged. In general, The stability of the interior equilibrium depends on the cost-benefit ratio and the delays. For the equilibrium to exist, the benefit must exceed 1, and the cost-benefit ratio must be below $1 - \frac{1}{2h-1}$.

The stability effects of time delay are strategy-dependent. When the time delays are equal for both strategies, increasing the delay past a critical value that is $\tau^* = \frac{\pi h}{s}$ destabilizes the stable interior equilibrium point, causing oscillations around its equilibrium point. This critical delay value differs from the critical delay when logistic growth is ignored and the population can increase infinitely $(\frac{\pi(h - \frac{s}{2})}{s(h - s)})$ [48]. Dynamics change when one strategy's delay is zero while the other's increases. If the delay for strategy A is zero while for B increases, Hopf bifurcation eventually produces oscillations. The bifurcation delay is smaller than the equal-delay case. However, if the delay of strategy B is zero while increasing A, the stable state remains stable even for large delays. When both delays are nonzero and unequal, rich phenomena can emerge depending on the delay values. In some cases, for a fixed nonzero strategy B delay that produces oscillations when strategy A delay is zero, increasing delay for strategy A can lead to more critical values - transitioning from instability to stability, then back to instability at larger delays.

In summary, our analysis demonstrates that incorporating logistic population growth and strategy-dependent time delays significantly alters the evolutionary dynamics compared to classical replicator models. The presence of delays can both stabilize and destabilize equilibria, leading to richer bifurcation structures and new dynamical regimes. These findings highlight the importance of explicitly accounting for temporal effects in social interactions and ecological feedbacks. While our framework provides a tractable analytical setting, future work could explore stochastic extensions, heterogeneous networks, or more complex forms of delays to assess the robustness of our conclusions and their broader applicability to real-world social and biological systems.

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Data Availability No datasets were generated or analysed during the current study.

Declarations

Conflict of interest The authors declare no Conflict of interest.

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Intrinsic Noise in Structured Replicator Dynamics Modelling Time Delays

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Abstract

We construct and analyze structured replicator dynamics of the Snowdrift game. In our model, the offspring is put in juvenile compartments and then mature and join adult compartments with strategy-dependent rates. This is augmented by death rates and hence the population size is bounded. In the corresponding birth–death Markov jump process, rates of leaving juvenile compartments may be interpreted as inverses of averages of exponentially distributed time delays. We observe a novel behavior: for equal average time delays of both strategies, the frequency of cooperators in the quasi-stationary state of a stochastic dynamics is bigger than that in the corresponding stationary state of the deterministic structured replicator dynamics which is actually equal to the critical point of the original replicator equation for the Snowdrift game. In short, time delays favor cooperation in the presence of intrinsic stochastic fluctuations.

Keywords Evolutionary game theory · Structured replicator dynamics · Intrinsic noise · Random time delays · Markov jump processes

1 Introduction

The standard approach to model time evolution of populations of players is to construct appropriate replicator equations for frequencies of strategies [1–4]. One assumes that populations are infinite and well-mixed. However, real populations are finite, their growth is suppressed by an environmental carrying capacity. We would like to refer here to a paper of Tao and Cressman [5] which was an inspiration for our work presented here. They introduced a background fitness into replicator equations as a decreasing function of the population size, corresponding to the carrying capacity.

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It is usually also assumed that interactions between individuals take place instantaneously and their effects are immediate. In reality, all processes take a certain amount of time. The effects of time delays in evolutionary games were discussed in [6–15].

It is well known that time delays may cause oscillations in dynamical systems [16–19]. Recently however there were constructed models with strategy-dependent time delays with a novel behavior, namely the continuous dependence of equilibria on time delays [20–22].

In all models, incorporation of time delays led to infinite-dimensional time-delayed differential equations. In a very recent paper [23], a different approach was presented. The authors introduced a compartment for juveniles, called kindergarten, who cannot participate in games, and for adults, with appropriate rates of maturing and leaving a kindergarten to join an adult population.

Here we combine constructions of [5] and [23]. The compartment model is augmented by death rates and hence the population size is bounded.

We numerically solve our structured replicator equations for the Snowdrift game to find stationary states both for population sizes and frequencies of strategies. We show that strategy-dependent transition rates for leaving kindergartens cause continuous shifts of stationary states. We see, as in previous models, that delays are not beneficial - corresponding strategies have smaller basins of attraction.

Then we follow ideas of Tao and Cressman [5] to investigate effects of intrinsic noise. We introduce a birth and death Markov jump process corresponding to the deterministic dynamics. In such a model, an intensity of leaving a juvenile compartment may be seen as an inverse of an average of an exponentially distributed time delay.

We perform stochastic simulations to get expected values of population sizes and frequencies of strategies in the quasi-stationary state.

We report a novel behavior. For equal or almost equal average time delays, we observe that time delays are beneficial for cooperation - bigger time delay, bigger the frequency of cooperators in the quasi-stationary state of the stochastic dynamics. This is a joint effect of both time delays and stochasticity.

2 Structured Replicator Dynamics

In classical replicator dynamics one do not take into account evolution of the size of a population [1–4]. In fact it grows to infinity which is obviously not realistic.

Tao and Cressman [5] introduced a background fitness into replicator equations as a decreasing function of the population size, corresponding to an environmental carrying capacity. We extend their model in order to take into account time delays which naturally appear in every process describing evolution of systems of interacting objects, such as substrates in chemical reactions or individuals in evolutionary games. Instead of dealing with explicit time delays which leads to infinite-dimensional, not easy to analyzed, time-delayed replicator equations, as it is usually done, following [23] we introduce age compartments. Namely, the offspring is put in juvenile compartments (kindergartens) then mature and join adult compartments with strategy-dependent rates. Let us emphasize here that we do not have an explicit age structure and explicit time delays. In the corresponding birth-death Markov jump process, inverses of rates of leaving juvenile compartments may be interpreted as averages of exponentially distributed time delays.

We consider here the Snowdrift game, a two-player game with two pure strategies, cooperation (C) and defection (D), and with the following payoff matrix,

	C	D
C	$b - \frac{c}{2}$	$b - c$
D	b	0

where b is a reward of coming home and c is a cost of snow removal, $b > c$.

The game has a mixed Nash equilibrium, a globally asymptotically stable interior point in the replicator dynamics. We fix here $b = 6$ and $c = 4$, the mixed Nash equilibrium is then equal to 0.5. In the standard replicator dynamics, individuals are matched randomly into pairs, play games and receive payoffs which are interpreted as the number of offspring who inherit strategies of parents. It is assumed that offspring immediately join the population and can participate in games.

To take into account time delays, we introduce a two-stage life cycle for individuals. Specifically, juveniles born from adult parents first enter a separate juvenile compartment, called kindergarten, where they must wait for a certain random time before maturing and leaving it. Then they can engage in the game and reproduce. We construct a model with two compartments: for adults (A) and for juveniles (K).

We denote the number of individuals in each compartment as n_C^A and n_D^A for adults, and n_C^K and n_D^K for juveniles C and D players. The total number of individuals in each compartment is given by $N^A = n_C^A + n_D^A$ for adults and $N^K = n_C^K + n_D^K$ for juveniles. Since only adults can participate in games, the expected payoff of each strategy is given by $\pi_C = \frac{n_C^A(b - \frac{c}{2}) + n_D^A(b - c)}{N^A}$ and $\pi_D = \frac{n_C^A b}{N^A}$. The rate at which i players interact and consequently make the corresponding juvenile compartment to grow is given by $n_i^A \pi_i$. To follow ideas in [5] and to take into account population size effects, we assume that players share, independent of strategies the same background fitness, $-\beta^j N^j$ with a positive parameter β^j , $j = A, K$. New juveniles enter the juvenile compartment and wait for an average period τ_i , $i \in C, D$, to mature and join the adult population. Thus, the maturation rate is $\frac{n_i^K}{\tau_i}$. This leads to the following differential equations,

$$\begin{aligned}
 \frac{dn_C^A}{dt} &= \frac{n_C^K}{\tau_C} - \beta^A N^A n_C^A \\
 \frac{dn_D^A}{dt} &= \frac{n_D^K}{\tau_D} - \beta^A N^A n_D^A \\
 \frac{dn_C^K}{dt} &= n_C^A \pi_C - \beta^K N^K n_C^K - \frac{n_C^K}{\tau_C} \\
 \frac{dn_D^K}{dt} &= n_D^A \pi_D - \beta^K N^K n_D^K - \frac{n_D^K}{\tau_D}
 \end{aligned} \tag{1}$$

Let $x = \frac{n_C^A}{N^A}$ and $y = \frac{n_C^K}{N^K}$ denote frequencies of the strategy C in the adult and juvenile compartments. We can derive in the standard way the system of equations for frequencies and total populations,

$$\frac{dx}{dt} = \frac{N^K}{N^A} \left(\frac{y}{\tau_C} (1 - x) - \frac{x(1 - y)}{\tau_D} \right)$$

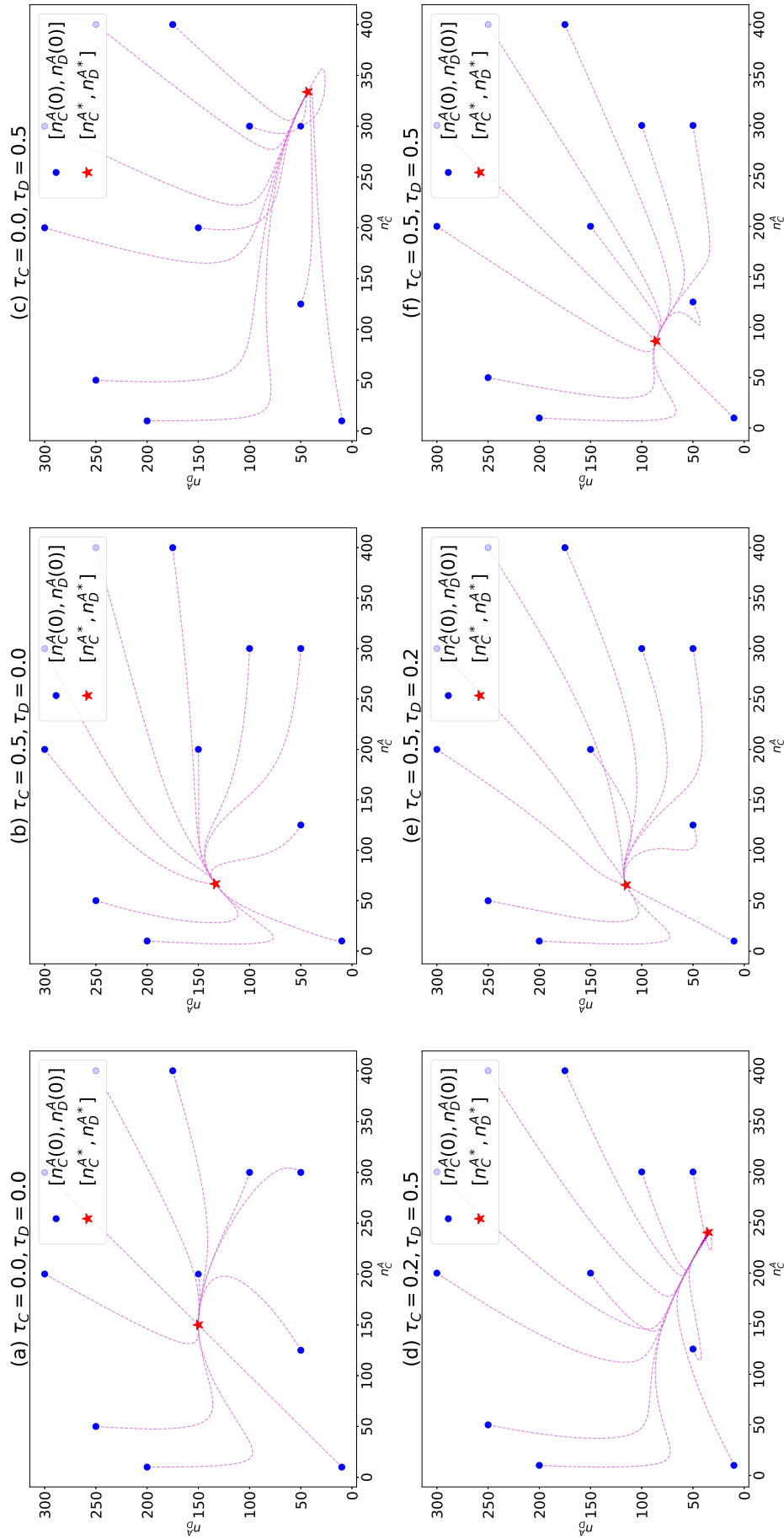


Fig. 1 Phase portraits illustrating the dynamics of (n_C^A, n_D^A) , $\beta^A = \beta^K = 0.01$, we solve Eq. (1) with 0 initial conditions in kindergartens. Interior stationary states (n_C^{A*}, n_D^{A*}) are approximately equal to **a** (150,150), **b** (67,133), **c** (334,43), **d** (240,35), **e** (66,115), **f** (86,86)

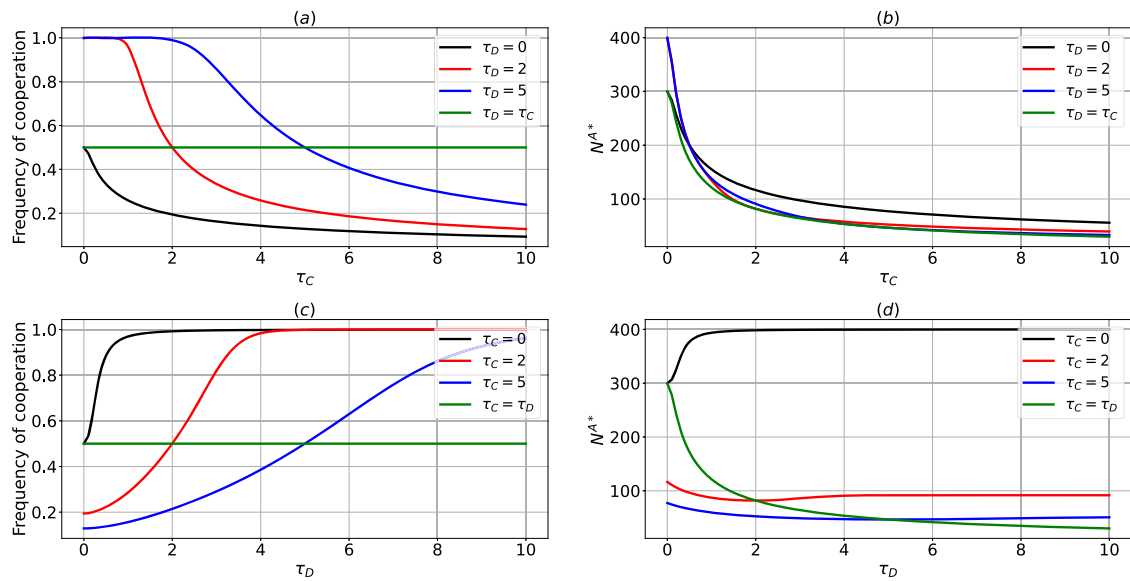


Fig. 2 Frequencies of cooperation in the adult population and total adult population sizes in the stationary state as functions of time delays, numerical solutions of (2) with left-hand sizes equal to 0

$$\begin{aligned}
 \frac{dy}{dt} &= \frac{N^A}{N^K} (x\pi_C - y\bar{\pi}) + y \left(\frac{y-1}{\tau_C} + \frac{1-y}{\tau_D} \right) \\
 \frac{dN^A}{dt} &= N^K \left(\frac{y}{\tau_C} + \frac{1-y}{\tau_D} \right) - \beta^A (N^A)^2 \\
 \frac{dN^K}{dt} &= N^A \bar{\pi} - N^K \left(\frac{y}{\tau_C} + \frac{1-y}{\tau_D} + \beta^K N^K \right)
 \end{aligned} \quad (2)$$

where $\bar{\pi} = x\pi_C + (1-x)\pi_D$. Analogous equations for cases where one time delay is equal to zero, so the corresponding rate of leaving the juvenile compartment is infinite, are presented in the Appendix A.

For equal average delays for both strategies, $\tau_C = \tau_D = \tau$, our replicator equations are particularly simple and we get that stationary values of frequencies of strategies are equal to those of classical replicator dynamics without time delays. In particular, for the Snowdrift game with $b = 6$ and $c = 4$, $x^* = y^* = 0.5$. Details of calculations are given in the Appendix A.

For strategy-dependent time delays, our structured replicator equations are highly nonlinear, this makes finding stationary states analytically impossible. Below we present numerical solutions of structured replicator dynamics and stationary states of population sizes and strategy frequencies $\{n_C^{A*}, n_D^{A*}, n_C^{K*}, n_D^{K*}, N^{A*}, N^{K*}, x^*, y^*\}$.

Figure 1 presents phase portraits illustrating the dynamics of n_C^A and n_D^A for various average time delays. Here and in all following figures, the parameters β^A and β^K are set to 0.01. As expected, when both delays are zero, the equilibrium values of n_C^{A*} and n_D^{A*} are equal to 150. However, when one delay is greater than the other one, the equilibrium point shifts, indicating a change in the system's behavior.

Figure 2 shows the effect of delays on the stable interior state (x^*, N^{A*}) (stability verified through numerical solutions of Eq. (2)). Panels (a) and (c) show the impact of τ_C and τ_D on the stationary frequency of cooperation when the other delay is fixed, while the right panels show the dependence of the size of the adult population on delays. We observe that delays are not good for strategies, bigger the strategy delay, smaller its basin of attraction. A similar result was previously demonstrated in [21] for a system of delayed differential replicator

Table 1 Intensities of transitions between states of the Markov chain

Transition	Intensities
$n_i^A \rightarrow n_i^A + 1$	$\frac{n_i^K}{\tau_i}$
$n_i^A \rightarrow n_i^A - 1$	$\beta^A N^A n_i^A$
$n_i^K \rightarrow n_i^K + 1$	$n_i^A \pi_i$
$n_i^K \rightarrow n_i^K - 1$	$\frac{n_i^K}{\tau_i} + \beta^K N^K n_i^K$

The state of the chain at any time t is given by $\{n_i^j\}$, where $i \in \{C, D\}$ and $j \in \{A, K\}$

equations and in [23] for the compartmental model. As noted earlier, for our payoff matrix, the stationary state is 0.5, and this value remains unchanged when the delays for both strategies are increased simultaneously ($\tau_C = \tau_D$). It is possible for one strategy in the adult population to disappear entirely if the delay for that strategy significantly exceeds the delay for the other one. An interesting observation is that the same time delay can reduce the size of the adult population without affecting the stationary frequency.

In the next section, we incorporate stochasticity into our model to explore how delays influence population dynamics in the presence of stochastic fluctuations.

3 Markov Jump Process of the Compartment Model

Here we study a stochastic model corresponding to replicator dynamics discussed in the previous section. We follow closely Tao and Cressman [5]. In our construction, rates present in replicator equations become now intensities in a Markov jump process, a Markov chain in continuous time and a discrete space of states.

The state of the Markov chain is described by the numbers of players of two strategies of both compartments, $\{n_i^j\}$, where $i \in \{C, D\}$ and $j \in \{A, K\}$. Intensities of jumps are given in Table 1.

Let $\Phi(n_C^A, n_D^A, n_C^K, n_D^K; t)$ be the joint probability distribution for the system to be at a given state at time t . One can write a standard Master equation,

$$\begin{aligned} \frac{d\Phi(n_C^A, n_D^A, n_C^K, n_D^K; t)}{dt} = & \sum_{i=C,D} \sum_{j=A,K} (E_i^{+,j} - 1) \beta^j N^j n_i^j \Phi(n_C^A, n_D^A, n_C^K, n_D^K; t) \\ & + \sum_{i=C,D} (E_i^{-,K} - 1) n_i^A \pi_i \Phi(n_C^A, n_D^A, n_C^K, n_D^K; t) \\ & + \sum_{i=C,D} (E_i^{-,A} E_i^{+,K} - 1) \frac{n_i^K}{\tau_i} \Phi(n_C^A, n_D^A, n_C^K, n_D^K; t), \end{aligned} \quad (3)$$

where $E_i^{\pm,j}$ is the operator given by

$$E_i^{\pm,j} F(n_C^A, n_D^A, n_C^K, n_D^K) = \begin{cases} F(n_C^A \pm 1, n_D^A, n_C^K, n_D^K) & i = C, j = A \\ F(n_C^A, n_D^A \pm 1, n_C^K, n_D^K) & i = D, j = A \\ F(n_C^A, n_D^A, n_C^K \pm 1, n_D^K) & i = C, j = K \\ F(n_C^A, n_D^A, n_C^K, n_D^K \pm 1) & i = D, j = K \end{cases}$$

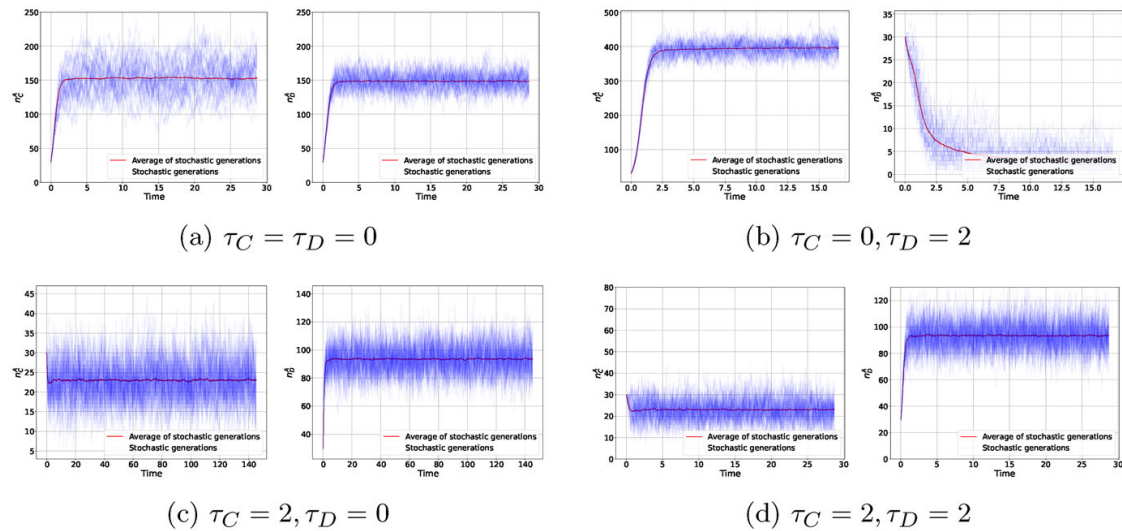


Fig. 3 Single trajectories are represented by blue lines, their averages are shown in red, $\beta^A = \beta^K = 0.01$ and the initial condition for each trajectory is set to $(30, 30, 0, 0)$

for any function F . It changes the population of strategists in adult or kindergarten by $\pm$ one individual. For example $E_C^{+,A} \beta^A N^A n_C^A \Phi(n_C^A, n_D^A, n_C^K, n_D^K; t) = \beta^A (N^A + 1)(n_C^A + 1) \Phi(n_C^A + 1, n_D^A, n_C^K, n_D^K; t)$.

One may then derive differential equations for the expected values and other moments of the number of players. However it is impossible to solve such equations. Therefore we will resort to stochastic simulations, we will implement a classic Gillespie algorithm [24].

Let us notice first that our Markov process has an absorbing state $(0, 0, 0, 0)$. Such a situation is typical in many processes in biological and social models, the Moran process being the classic example. Our focus here is on the long-term behavior of the system under the assumption that the population is not extinct, we say that the system is in the quasi-stationary state. We will estimate expected values of population sizes and strategy frequencies in the quasi-stationary state.

We simulate 1000 trajectories, with each trajectory consisting of 100,000 Monte-Carlo steps. The expected value of n_i^j is then computed as the average over the final 1000 steps of all trajectories. Similarly, we calculate $\langle x \rangle = \langle \frac{n_C^A}{N^A} \rangle$.

Figure 3 present stochastic trajectories for different delays, β^A and β^K are fixed at 0.01 and the initial condition is set to $(30, 30, 0, 0)$, single trajectories are represented by blue lines, their averages are shown in red. All trajectories remain around the quasi-stationary state for an extended period of time. Since the only absorbing state of this process is $(0, 0, 0, 0)$, the system will eventually reach extinction. However starting from a big initial conditions and small β^A and β^K , the extinction happens in a very long time.

Figure 4 shows the dependence of the expected frequency of cooperators in the adult compartment and the size of the adult population in the quasi-stationary state.

We observe fundamental differences between deterministic and stochastic evolution of our structured population. In the stochastic model, Fig. 4, panel (a), when the defector's delay is fixed at 5 and the cooperator's delay varies, we observe that cooperation takes the whole adult population for cooperator delays below approximately 3. In contrast, in the deterministic model, Fig. 2, panel (a), this threshold is lower, at around 2. This suggests that in the stochastic dynamics, cooperation can persist in the population even at higher values of the delay parameter τ_C than in the deterministic dynamics. A similar pattern is seen when

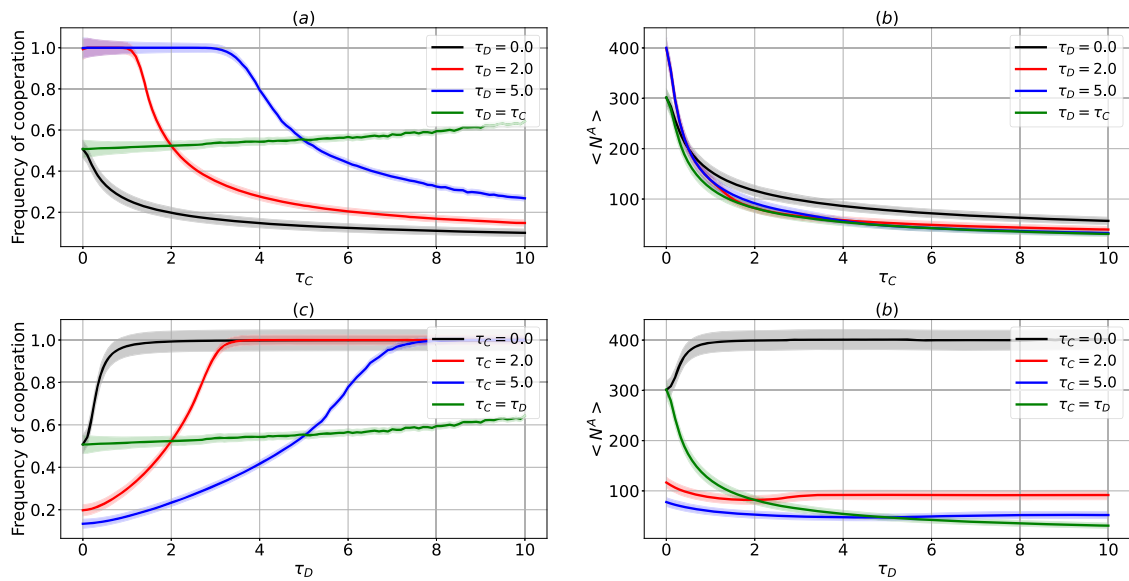


Fig. 4 Expected values of the total adult population size and the frequency of cooperation $\langle x \rangle$ in the quasi-stationary state as functions of time delays. The shaded region around each line represents the standard deviation across all trajectories of the stochastic simulation

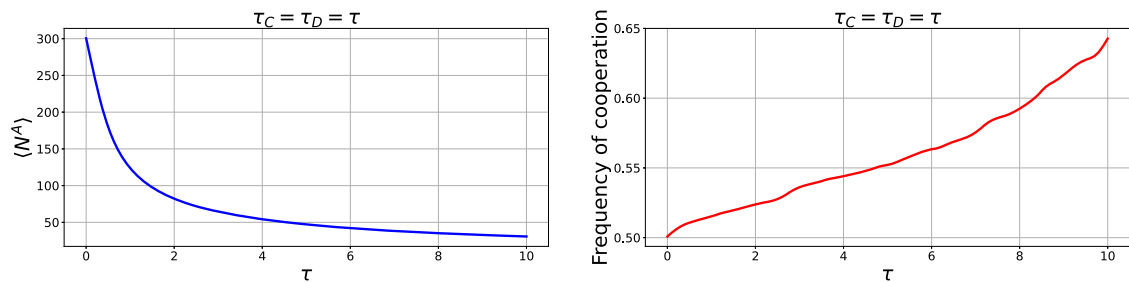


Fig. 5 Expected values, $\langle N^A \rangle$ and $\langle x \rangle$, in the quasi-stationary state as functions of delay equal for both strategies

we compare panel (c) of Fig. 4 for the stochastic model and panel (c) of Fig. 2 for the deterministic one. When the cooperator's delay τ_C is fixed and the defector's delay τ_D is varied, the stochastic model shows full cooperation in the adult population at smaller values of τ_D compared to the deterministic model.

Let us now compare both dynamics in the case of strategy-independent delays. We set $\tau_C = \tau_D = \tau$ and vary τ . The results are shown in Fig. 5. When τ increases, the size of adult populations decreases. This outcome is expected, as individuals spend more time in juvenile compartments. However, we observe a novel behavior: the frequency of cooperators $\langle x \rangle$ in adult population increases with increasing delay. This observation contrasts with previous findings in [21, 23] where for strategy-independent delays there are no changes in frequencies of strategies is the stationary state of deterministic dynamics. Here strategy-independent delays promote the cooperative behavior in our stochastic model but not in the deterministic one.

To understand why strategy-independent delays can promote cooperation, we analyze the intensity and probability of transitions in our Markov jump process. Let us assume initial conditions with equal number of cooperators and defectors in the adult population, $n_C^A = n_D^A = 30$. Because such initial conditions correspond to strategy frequencies in the mixed Nash equilibrium in our Snowdrift game, this is also a frequency stationary point of the deterministic structured dynamics, see Appendix A.

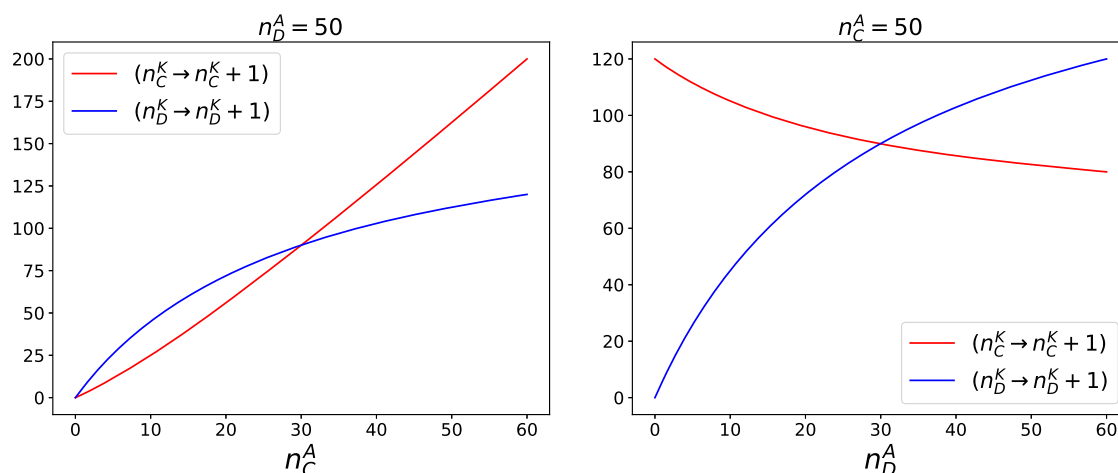


Fig. 6 The intensity of a birth of a cooperator $n_C^A \frac{4n_C^A + 2n_D^A}{n_C^A + n_D^A}$ and of a birth of a defector $n_D^A \frac{6n_C^A}{n_C^A + n_D^A}$

Let us recall that in the stochastic dynamics the intensity of the cooperator birth, $n_C^K \rightarrow n_C^K + 1$, is given by $n_C^A \frac{4n_C^A + 2n_D^A}{n_C^A + n_D^A}$, while for the defectors birth it is $n_D^A \frac{6n_C^A}{n_C^A + n_D^A}$. At the initial condition both intensities are the same. However, in our stochastic process, only one event can occur at a time which can lead to an imbalance between the number of strategies. This imbalance influences intensities. Consider a simple example of having additional cooperator or defector which happens with same probability 0.5. It can be easily calculated that the average intensity in the next step of the birth of a cooperator is 91.51, whereas for the birth of a defector is slightly lower and equal to 91.47. This suggests that, on average, cooperators have a slight advantage.

Figure 6 illustrates this behavior for scenarios where either the number of cooperators is fixed and the number of defectors varies, or vice versa. Consistent with the case of having one additional cooperator or defector, the results show that having more cooperators in the population is good for their growth. This bias toward cooperation suggests that, over time, the stochastic process will favor an increase in cooperators.

When time delays are bigger, then juveniles spend more time in kindergartens and therefore the stationary size of the adult population is smaller, as seen in Fig. 5. One can see that for smaller initial conditions the above difference between intensities is bigger hence cooperators are more favored. In Fig. 7, we present the time evolution of frequencies and transition probabilities (ratios of a particular intensity and the sum of all intensities at a given state) for two different delay values: a small delay of 1 and a larger delay of 10. The figure illustrates that for a larger time delay, the difference in birth probabilities between cooperators and defectors is more significant compared to the smaller delay. This leads to a higher prevalence of cooperators when the time delay is bigger.

Below we examine the case where delays are not equal but are very close to each other. Previous studies have shown that when the delay associated with a given strategy is larger than the delay of the other one, then it tends to disadvantage that strategy.

However, in Fig. 8, we observe that for a game with an interior stationary state at 0.5 (in the absence of delays), even a slightly larger delay for τ_C compared to τ_D promotes cooperation. In such cases, the quasi-stationary frequency of cooperators exceeds 0.5. Of course, this effect holds only when τ_C is slightly larger than τ_D . When the difference becomes significant, and τ_C is sufficiently large, similar to what is seen in the deterministic, it negatively impacts cooperation, reducing the cooperator frequency below 0.5. The value of τ_C (where $\tau_C > \tau_D$)

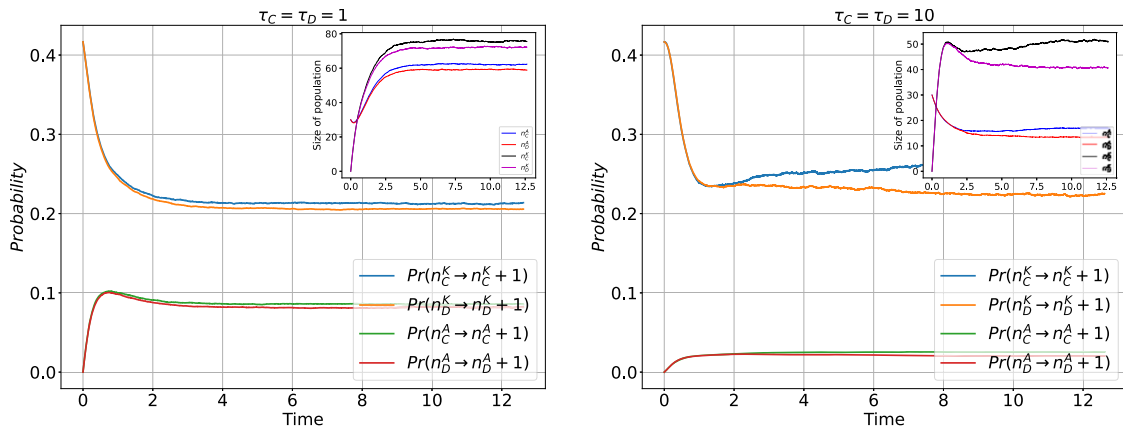


Fig. 7 Time evolution of population sizes and probabilities of transitions for strategy-independent delays. The probability of a transition is the ratio of the intensity of that transition and the sum of all intensities at a current state of the system. Each curve is the average of 1000 trajectories

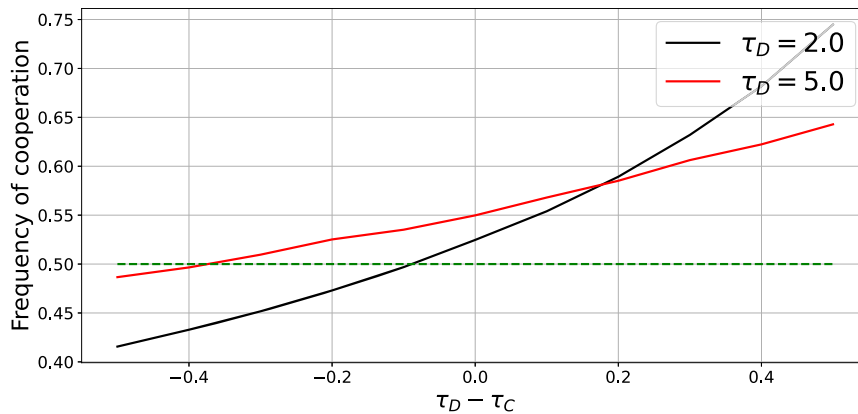


Fig. 8 Cooperation frequency in the quasi-stationary state as a function time delays difference

at which cooperation is still favored depends on the value of τ_D . For instance, in Fig. 8, when $\tau_D = 2$, cooperation is favored up to $\tau_C = 2.1$. However, when $\tau_D = 5$, cooperation can be favored up to $\tau_C = 5.35$.

Figure 9 illustrates effects of both delays on the frequency of cooperators in the quasi-stationary state. Clearly, for a fixed τ_D , increasing τ_C leads to a decrease in the level of cooperation. Conversely, for a fixed τ_C , increasing τ_D results in the opposite trend, with cooperation levels rising.

4 Discussion

We considered a Snowdrift game which has an asymptotically stable interior stationary state in the replicator dynamics. We combined ideas and constructions of [5] and [23] and presented a structured replicator dynamics of finite populations and the corresponding birth-death Markov jump process with two compartments: adults and juveniles. In the stochastic dynamics, inverses of rates of leaving juvenile compartments may be interpreted as averages of random exponentially distributed time delays. Our main result is that for a small difference of inverse rates (average time delays) of both strategies, the frequency of cooperators in the quasi-stationary state of the stochastic dynamics is bigger than that in the corresponding stationary state of the deterministic structured replicator dynamics; for equal average delays,

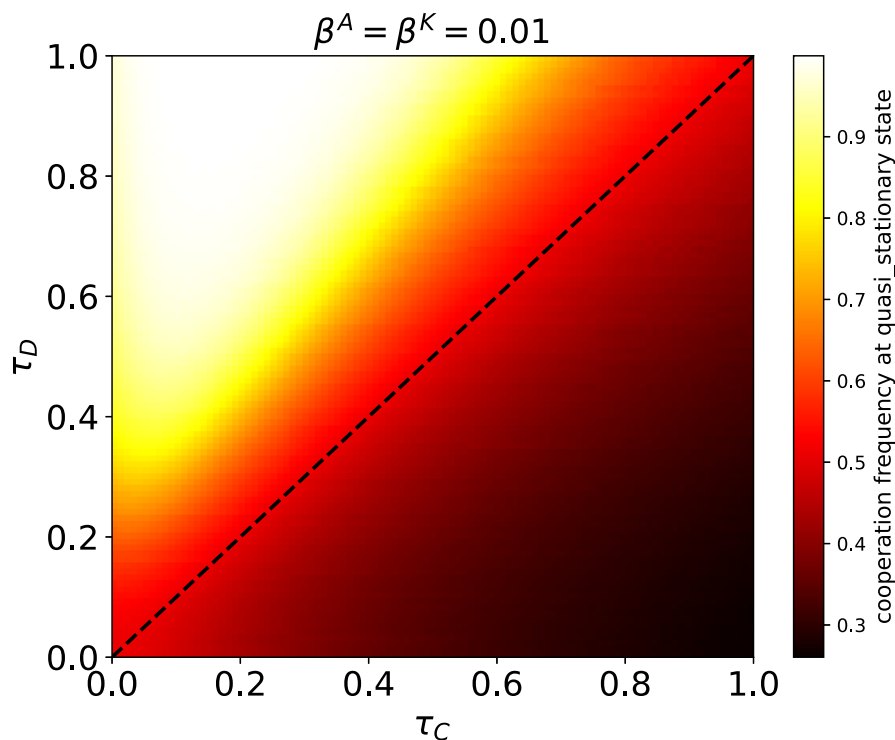


Fig. 9 Effect of τ_C and τ_D on the frequency of cooperation in the adult population in the quasi-stationary state

such stationary state is equal to the critical point of the original replicator dynamics for the Snowdrift game. In short, time delays favor cooperation in the presence of intrinsic stochastic fluctuations.

Somewhat analogous behavior has been observed recently in random walks with asymmetric time delays. It was observed there that we may reverse effects of time delays by a symmetric transformation of fitness functions and then the time delay of a given strategy increases its frequency in the stationary distribution [32].

It is worth to mention here that a logistic suppression was directly incorporated in replicator dynamics in [25–27]. It is an interesting problem to extend the authors models by adding time delays and intrinsic noise.

In general it is important to study the joint effects of time delays and stochasticity. Some results were presented in [28–33].

Appendix A

As mentioned in the main text, the deterministic equations differ when either one or both delays are zero. If the delay for a particular strategy is zero, it implies that new juveniles, born in proportion to the payoff, are immediately added to the adult population. Below, we present the equations for the various delay scenarios.

- $\tau_C = \tau_D = 0$:

$$\begin{cases} \frac{dn_C^A}{dt} = n_C^A \pi_C - \beta^A N^A n_C^A \\ \frac{dn_D^A}{dt} = n_D^A \pi_D - \beta^A N^A n_D^A \end{cases} \quad (\text{A1})$$

$$\begin{cases} \frac{dx}{dt} = x(1-x)(\pi_C - \pi_D) \\ \frac{dN^A}{dt} = N^A(\bar{\pi} - \beta^A N^A) \end{cases} \quad (\text{A2})$$

- $\tau_C = 0, \tau_D > 0$:

$$\begin{cases} \frac{dn_C^A}{dt} = n_C^A \pi_C - \beta^A N^A n_C^A \\ \frac{dn_D^A}{dt} = \frac{n_D^K}{\tau_D} - \beta^A N^A n_D^A \\ \frac{dn_D^K}{dt} = n_D^A \pi_D - \beta^K (n_D^K)^2 - \frac{n_D^K}{\tau_D} \end{cases} \quad (\text{A3})$$

$$\begin{cases} \frac{dx}{dt} = x \left((1-x) \pi_C - \frac{N^K}{\tau_D} \right) \\ \frac{dN^A}{dt} = N^A (x \pi_C - \beta^A N^A) + \frac{N^K}{\tau_D} \\ \frac{dN^K}{dt} = N^A (1-x) \pi_D - \frac{N^K}{\tau_D} - \beta^K N^K^2 \end{cases} \quad (\text{A4})$$

- $\tau_C > 0, \tau_D = 0$:

$$\begin{cases} \frac{dn_D^A}{dt} = n_D^A \pi_D - \beta^A N^A n_D^A \\ \frac{dn_C^A}{dt} = \frac{n_C^K}{\tau_C} - \beta^A N^A n_C^A \\ \frac{dn_C^K}{dt} = n_C^A \pi_C - \beta^K (n_C^K)^2 - \frac{n_C^K}{\tau_C} \end{cases} \quad (\text{A5})$$

$$\begin{cases} \frac{dx}{dt} = (1-x)^2 \pi_D - \frac{N^K x}{\tau_C} \\ \frac{dN^A}{dt} = N^A \left((1-x) \pi_D - \beta^A N^A \right) + \frac{N^K}{\tau_C} \\ \frac{dN^K}{dt} = N^A x \pi_C - \frac{N^K}{\tau_C} - \beta^K N^K^2 \end{cases} \quad (\text{A6})$$

- $\tau_C = \tau_D = \tau$:

$$\begin{cases} \frac{dn_C^A}{dt} = \frac{n_C^K}{\tau} - \beta^A N^A n_C^A \\ \frac{dn_D^A}{dt} = \frac{n_D^K}{\tau} - \beta^A N^A n_D^A \\ \frac{dn_C^K}{dt} = n_C^A \pi_C - \beta^K N^K n_C^K - \frac{n_C^K}{\tau} \\ \frac{dn_D^K}{dt} = n_D^A \pi_D - \beta^K N^K n_D^K - \frac{n_D^K}{\tau} \end{cases} \quad (\text{A7})$$

$$\begin{cases} \frac{dx}{dt} = \frac{N^K}{N^A} \left(\frac{y(1-x) - x(1-y)}{\tau} \right) \\ \frac{dy}{dt} = \frac{N^A}{N^K} (x \pi_C - y \bar{\pi}) \\ \frac{dN^A}{dt} = \frac{N^K}{\tau} - \beta^A (N^A)^2 \\ \frac{dN^K}{dt} = N^A \bar{\pi} - \frac{N^K}{\tau} - \beta^K N^K^2 \end{cases} \quad (\text{A8})$$

The key observation in our paper is that the qualitative behavior is different for deterministic and stochastic dynamics when delays for both strategies are equal. We solve the system of equations (A8) for the stationary state by setting all derivatives to zero. From the first equation we get $x^* = y^*$. Substituting this into the second one we obtain $\frac{N^A}{N^K} x(1-x)(\pi_C - \pi_D) = 0$. Hence the stationary interior state is given by $\pi_C = \pi_D$ as in the classical replicator dynamics without time delays.

We conclude that strategy-independent delays do not shift stationary states of frequencies in the deterministic dynamics.

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Author Contributions Both authors contributed equally to the manuscript.

Data Availability No datasets were generated or analysed during the current study.

Declarations

Conflict of interest The authors declare no Conflict of interest.

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Effect of the degree of an initial mutant in Moran processes in structured populations

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We study effects of the mutant's degree on the fixation probability, extinction, and fixation times in Moran processes on Erdős-Rényi and Barabási-Albert graphs. We performed stochastic simulations and used mean-field-type approximations to obtain analytical formulas. We showed that the initial placement of a mutant has a significant impact on the fixation probability and extinction time, while it has no effect on the fixation time. In both types of graphs, an increase in the degree of an initial mutant results in a decreased fixation probability and a shorter time to extinction. Our results extend previous ones to arbitrary fitness values.

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I. INTRODUCTION

Extinction and fixation of phenotypes and behaviors in animal and human populations are important features of evolutionary processes. The classical model here is the Moran process in populations of fixed finite sizes, consisting of individuals equipped with one of two phenotypes [1]. Namely, an individual is chosen proportional to its fitness and it gives birth to an offspring who inherits its phenotype and replaces a randomly chosen individual. Such a birth-death process (also called an invasion process) is a Markov chain with two absorbing states: homogeneous populations with only one phenotype present. One may also consider death-birth Moran process, where an individual is chosen with a probability inversely proportional to its fitness and is replaced by a randomly chosen individual [2–5].

Here we will study fixation probability and expected fixation and extinction times of mutants in birth-death Moran processes on graphs. In such processes, offspring replace neighbors of ancestors. The study of fixation probabilities on graphs was initiated by Lieberman, Hauert, and Nowak [6]. Since then there appeared many papers on Moran processes on graphs [7–35]. They have shown that the structure of the population can have a significant impact on the evolutionary dynamics and can affect fixation probability and fixation time.

In particular, Antal, Redner, and Sood [9] studied fixation probability on degree-heterogeneous power-law graphs. They considered various dynamics and derived approximate analytical formulas for the dependence of the fixation probability on the degree of an initial mutant. In particular, for the birth-death process (biased invasion process) in the case where fitness of a mutant is very close to that of a resident, they showed that the fixation probability of a mutant is inversely proportional to its degree. In the follow-up paper [10], they studied fixation time in the neutral case. For similar results see also Ref. [19].

The effect of the placement of an initial mutant was also studied by Broom, Rychtář, and Stadler [13]. The authors derived a useful analytical approximation of fixation probability as a function of the degree of the initial mutant for some small graphs. Their findings revealed that initial mutants with fewer connections had a greater advantage, resulting in a higher probability of fixation.

Here we present results of stochastic simulations and use a mean-field-type approximation to derive analytical formulas for the fixation probability on Erdős-Rényi (ER) [36] and Barabási-Albert (BA) [37,38] graphs which agree with stochastic simulations for any fitness values. We also derive formulas for fixation and extinction time for arbitrary fitness values.

We have extended results of Refs. [9,10,19] to models with arbitrary fitness values. In particular, we compared our approximate formula for the fixation probability to that in [9], see Appendix B. In general, we showed that the initial placement of a mutant has a significant impact on the fixation probability and extinction time, while the degree of the initial mutant has no effect on the fixation time. In both types of graphs, an increase of the degree of the initial mutant results in a decreased probability of the mutant taking over the entire population and a shorter time to extinction.

In the two graphs with the same size and average vertex degree, for an initial mutant with the same degree, the fixation probability in the Erdős-Rényi graph is higher than in the Barabási-Albert one. However, due to the presence of hubs, the average fixation probability in the Barabási-Albert graph is higher.

In Sec. II, we present our model, a Markov chain of the Moran process on graphs. In Sec. III, we present results of stochastic simulations and derive analytical results. Further analysis of extinction time is contained in Sec. IV. Discussion follows in Sec. V.

II. MODELS AND METHODS

We will study here the Moran process on two random graphs, the ER [36] and the scale-free BA one [37,38]. We

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build the ER graph by putting with probability p an edge between every pair of $N = 500$ vertices. It follows that the average degree of vertices (the average number of neighbors) is equal to $\alpha = p(N - 1)$. The BA network is built by the preferential attachment procedure. We start with m_o fully connected vertices and then we add $N - m_o$ vertices, each time connecting them with m already available vertices with probabilities proportional to their degrees. If $m_o = \alpha + 1$ and $m = \alpha/2$, then we get a graph with the average degree equal to α . It is known that such a graph is scale-free with the probability distribution of degrees given by $p(k) \sim k^{-3}$ [37–39].

Individuals—mutants with a constant fitness r and residents with a constant fitness 1—are located on the vertices of a graph. At every time step of the process, an individual is chosen with a probability proportional to its fitness and then gives birth to an offspring who inherits its phenotype and replaces a randomly chosen individual. Such a Markov chain with 2^N states has two absorbing ones: homogeneous populations with only one type of individuals present. We start our process with one mutant in the population of residents. We are interested in fixation probability, expected time to fixation and extinction of mutants, denoted respectively by ρ_1^f , e_i^f , and e_1^e . To find such quantities, we performed stochastic simulations.

We simulate the Moran process on graphs with $N = 500$ vertices. For every k , we randomly place an initial mutant on one of the nodes of the degree k and perform 10 000 Monte Carlo simulations until the population reaches one of two absorbing states. To get fixation probability, we divide the number of times the process ends in the all-mutant state by 10 000. Finally, we calculate an average fixation (extinction) time for some chosen k 's.

It is impossible to get rigorous analytical expressions for fixation and extinction probabilities and times for Moran processes on graphs. The situation is much simpler in well-mixed populations [40,41]. Then the Moran process becomes a Markov chain with $N + 1$ states corresponding to the number of mutants or more precisely, a random walk on $\{0, \dots, N\}$ with state-dependent probabilities of moving to the right, $p_{i \rightarrow i+1}$, to the left, $p_{i \rightarrow i-1}$, and not moving, $1 - p_{i \rightarrow i+1} - p_{i \rightarrow i-1}$. Such a model is well known in the context of the gambler's ruin problem. It is easy to see that here we have that $p_{i \rightarrow i+1} = r p_{i \rightarrow i-1}$ where r is the fitness of the mutant. For the fixation probability starting from i mutants, one gets that

$$\rho_i^f = \frac{1 - \frac{1}{r^i}}{1 - \frac{1}{r^N}}. \quad (1)$$

For the fixation probability on graphs, to take into account a degree of an initial mutant, we derive some approximate expressions for transition probabilities up to three mutants and then use the elementary conditional probability formula and the fixation probability ρ_3^f from (1) to obtain an analytic expression for the fixation probability as a function of a degree of the initial mutant. In that way we passed from the full Markov chain with 2^N states to the one with $N + 1$ states corresponding to the number of mutants.

In the following section, we derive analytic expressions for the fixation probability and expected fixation and

extinction times and compare them with results of stochastic simulations.

III. RESULTS

A. Fixation probability

Let ρ_i^f be the fixation probability when initially there are i mutants in the population. We denote by $P_{i \rightarrow j}$ the probability of the transition from the state i to the state j ; of course, for our Moran process, transition probabilities are nonzero only for $|i - j| \leq 1$. From the total probability formula we get

$$\rho_1^f = P_{1 \rightarrow 1} \rho_1^f + P_{1 \rightarrow 2} \rho_2^f, \quad (2)$$

$$\rho_2^f = P_{2 \rightarrow 1} \rho_1^f + P_{2 \rightarrow 2} \rho_2^f + P_{2 \rightarrow 3} \rho_3^f. \quad (3)$$

We substitute (3) into (2) and get

$$\rho_1^f = \frac{P_{1 \rightarrow 2} P_{2 \rightarrow 3}}{(P_{1 \rightarrow 2} + P_{1 \rightarrow 0})(P_{2 \rightarrow 1} + P_{2 \rightarrow 3}) - P_{1 \rightarrow 2} P_{2 \rightarrow 1}} \rho_3^f. \quad (4)$$

For the number of mutants to increase from 1 to 2, the mutant should be chosen for reproduction, and hence

$$P_{1 \rightarrow 2} = \frac{r}{N + r - 1}. \quad (5)$$

For the number of mutants to decrease from 1 to 0, a resident from the mutant's neighborhood with degree k must be selected and its offspring placed in the mutant's location so

$$P_{1 \rightarrow 0} = \sum_{k'} \frac{k}{r + N - 1} \frac{1}{k'} \rho'(k'), \quad (6)$$

where k' is the degree of the selected resident and $\rho'(k')$ is the degree distribution of neighbors of the mutant.

The distribution $\rho'(k')$ of degrees of a node which is connected to a given node is given by $\frac{k' \rho(k')}{\langle k \rangle}$, where $\rho(k')$ is the degree distribution in the graph [42,43]. Therefore

$$P_{1 \rightarrow 0} = \frac{k}{r + N - 1} \frac{1}{\langle k \rangle}. \quad (7)$$

Now we have to calculate $P_{2 \rightarrow 1}$. For this transition, one of the resident neighbors of two mutants must be chosen for the reproduction and replace the mutant. One of them is the first mutant with a degree of k , while the other is a previously mutated node with a degree of k' . We have the following formula:

$$P_{2 \rightarrow 1} = \sum_{k'} \frac{k - 1}{2r + N - 2} \frac{1}{k'} \frac{k' \rho(k')}{\langle k \rangle} + \sum_{k'} \sum_{k''} \frac{k' - 1}{2r + N - 2} \frac{1}{k''} \frac{k' \rho(k')}{\langle k \rangle} \frac{k'' \rho(k'')}{\langle k \rangle}. \quad (8)$$

In this equation, the second moment $\sum_{k'} k'^2 \rho(k')$ appears. After some simplifications we get

$$P_{2 \rightarrow 1} = \frac{1}{2r + N - 2} \frac{1}{\langle k \rangle} \left(k + \frac{\langle k^2 \rangle}{\langle k \rangle} - 2 \right). \quad (9)$$

In the the same way we obtain $P_{2 \rightarrow 3}$,

$$P_{2 \rightarrow 3} = \frac{r}{2r + N - 2} \frac{k - 1}{k} + \sum_{k'} \frac{r}{2r + N - 2} \frac{k' - 1}{k'} \frac{k' \rho(k')}{\langle k \rangle}, \quad (10)$$

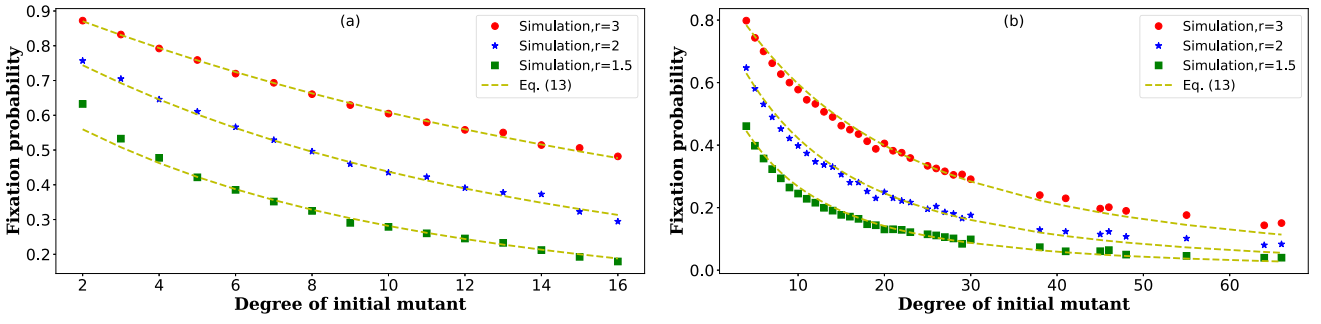


FIG. 1. Fixation probability as a function of a degree of an initial mutant for three fitness values: 3, 2, and 1.5 in (a) ER and (b) BA graphs with the size of 500 and the average degree of 8.

and after some simplifications we have

$$P_{2 \rightarrow 3} = \frac{r}{2r + N - 2} \left(\frac{k-1}{k} - \frac{1}{\langle k \rangle} + 1 \right). \quad (11)$$

This allows us to rewrite (4) as follows:

$$\rho_1^f = \frac{\langle k \rangle^2 r^2 (2 - \frac{1}{k} - \frac{1}{\langle k \rangle})}{(r\langle k \rangle + k) [r\langle k \rangle (2 - \frac{1}{k} - \frac{1}{\langle k \rangle}) + (k - 2 + \frac{\langle k^2 \rangle}{\langle k \rangle})] - r\langle k \rangle (k - 2 + \frac{\langle k^2 \rangle}{\langle k \rangle})} \rho_3^f. \quad (12)$$

Now we assume that the effect of an initial mutant is limited to transitions from one-mutant population to two-mutant population and also from two mutants to three mutants. Hence we take ρ_3^f from (1) and get

$$\rho_1^f = \frac{\langle k \rangle^2 r^2 (2 - \frac{1}{k} - \frac{1}{\langle k \rangle})}{(r\langle k \rangle + k) [r\langle k \rangle (2 - \frac{1}{k} - \frac{1}{\langle k \rangle}) + (k - 2 + \frac{\langle k^2 \rangle}{\langle k \rangle})] - r\langle k \rangle (k - 2 + \frac{\langle k^2 \rangle}{\langle k \rangle})} \frac{1 - \frac{1}{r^3}}{1 - \frac{1}{r^N}}. \quad (13)$$

To determine the fixation probability of mutants with degree k , we use (13), which requires the knowledge of the average degree $\langle k \rangle$ and the second moment $\langle k^2 \rangle$ of the graph. For the Erdős-Rényi graph with the binomial degree distribution, the average degree is equal to $\langle k \rangle = (N-1)p$ and the second moment $\langle k^2 \rangle = \langle k \rangle \{ \langle k \rangle [1 - 1/(N-1)] + 1 \}$. For the Barabási-Albert graph, the degree distribution follows a power law, resulting in an average degree of $\langle k \rangle = 2m$ and $\langle k^2 \rangle \equiv \langle k \rangle^2 \log N / 4$ [44].

Figure 1 presents the results of stochastic simulations and our analytical approximation for the fixation probability. Our results show a decrease in fixation probability as the initial mutant's degree increases, which agrees with previous findings by Antal, Redner, and Sood [9]. They demonstrated that if fitness of a mutant is close to that of a resident, then the fixation probability is inversely proportional to the degree of the initial mutant. However, comparing our approximation to theirs reveals that their model accurately calculates fixation probability for mutants with fitness close to residents, while our model maintains accuracy across all fitness levels, see Appendix B.

As the degree of the initial mutant increases, it becomes more probable that one of its resident neighbors replaces it. Hence, vertices with a lower number of connections have a higher chance of remaining and progressing toward fixation. In the BA graph, the degree distribution follows a power law, which means that there are a few nodes with very high degrees and many nodes with low degrees. Consequently, in the BA

graph, there may be initial nodes with a high probability of fixation, mainly those with low degrees, and others with very low fixation probabilities, mainly those with high degrees, in contrast to the ER graph where the probability of fixation is more uniform across nodes.

In Fig. 2, we present the distribution of fixation probabilities for a mutant with respect to all initial placements

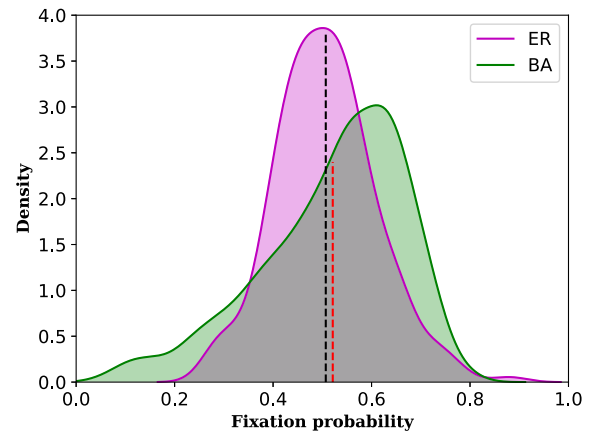


FIG. 2. Distribution of fixation probabilities of a mutant with respect to initial placements on ER and BA graphs with the size of 500 and the average degree of 8. The fitness is 2. Dash lines show the average fixation probability.

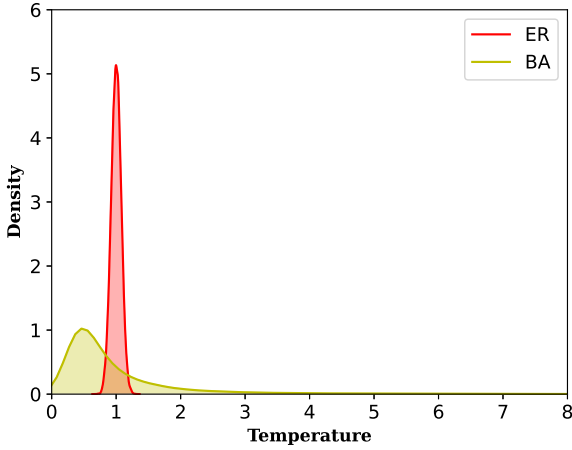


FIG. 3. The distribution of temperatures in ER and BA graphs. The node temperature is simply defined as the inverse of the degree of all nodes leading to that node.

in both graphs of the same size, average degree, and fitness. In ER graphs, fixation probabilities for all vertices are distributed approximately symmetrically around the average fixation probability. In BA graphs, the distribution is asymmetric. Moreover, we see that for $r = 2$, the average fixation probability in the ER graph is approximately 0.5 which is the fixation probability in a well-mixed population, while this value is slightly higher in the BA graph.

This can be understood on the basis of a concept of temperature vertex which is defined as the sum of the inverses of degrees of all neighbors of a given vertex. It was proven in Ref. [6] that for graphs for which all vertices have the same temperature, the so-called isothermal graphs, the average fixation probability is equal to the one in well-mixed populations.

Figure 3 displays the temperature distribution for both graphs with the size of 500 and an average degree of 8. The ER graph exhibits a peak at 1, indicating that nearly all nodes have the same temperature. In contrast to the ER graph, the BA graph's vertices show the large spectrum of temperatures. While there are many vertices with small degrees, leading to higher fixation probabilities, there are only a few vertices with large degrees, leading to low fixation probabilities. Consequently, the average fixation probability in BA graphs is higher than in well-mixed populations and ER graphs.

In Fig. 4, we present the fixation probability for an equal degree of initial placement for both graphs. As shown, the

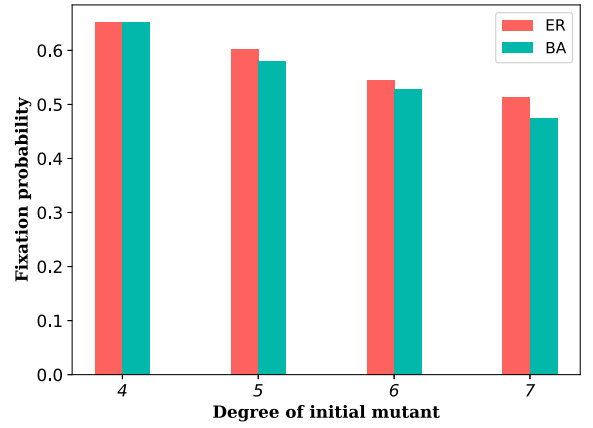


FIG. 4. Fixation probability for various initial mutants with the same degree in ER and BA graphs. The size of the graph is 500 with the average degree of 8 and $r = 2$.

fixation probability of one mutant with the the same degree in both graphs, is slightly higher for the ER graph. However, this does not contradict the observation that the average fixation probability is higher in the BA network. While it is true that the fixation probability for initial mutants with the same degree is higher in ER graphs, there are more nodes with a low degree in BA graphs, which have a higher fixation probability than in ER graphs. As a result, the average fixation probability in BA graphs is greater than that in ER graphs.

B. Fixation time

To derive an analytical expression for the expected value of the fixation time we proceed in an analogous way as for the fixation probability—we use the conditional expected value formula. We follow Antal and Sheuring [2] and obtain an equation for t_i^f , fixation time in populations with i mutants,

$$\rho_1^f t_1^f = P_{1 \rightarrow 1} \rho_1^f (t_1^f + 1) + P_{1 \rightarrow 2} \rho_2^f (t_2^f + 1) \quad (14)$$

and

$$\begin{aligned} \rho_2^f t_2^f = & P_{2 \rightarrow 1} \rho_1^f (t_1^f + 1) + P_{2 \rightarrow 2} \rho_2^f (t_2^f + 1) \\ & + P_{2 \rightarrow 3} \rho_3^f (t_3^f + 1). \end{aligned} \quad (15)$$

We have $\rho_2^f t_2^f$ from (15), we put it into (14) and after some simplifications we get

$$\begin{aligned} t_1^f = & \frac{P_{2 \rightarrow 1} + P_{2 \rightarrow 3}}{\rho_1^f [(P_{1 \rightarrow 0} + P_{1 \rightarrow 2})(P_{2 \rightarrow 1} + P_{2 \rightarrow 3}) - P_{2 \rightarrow 1} P_{1 \rightarrow 2}]} \left\{ \left[\frac{(1 - P_{1 \rightarrow 0} - P_{1 \rightarrow 2})(P_{2 \rightarrow 1} + P_{2 \rightarrow 3}) + (P_{1 \rightarrow 2} P_{2 \rightarrow 1})}{P_{2 \rightarrow 1} + P_{2 \rightarrow 3}} \right] \rho_1^f \right. \\ & \left. + \left[\frac{P_{1 \rightarrow 2}}{(P_{1 \rightarrow 0} + P_{1 \rightarrow 2})} \right] \rho_2^f + \frac{P_{1 \rightarrow 2} P_{2 \rightarrow 3}}{P_{2 \rightarrow 1} + P_{2 \rightarrow 3}} \rho_3^f (t_3^f + 1) \right\}, \end{aligned} \quad (16)$$

where ρ_1^f is given in (13), ρ_2^f in (3), and ρ_3^f in (1).

To get t_3^f we use a well-known technique described in Refs. [16,26]. The time to fixation starting from i mutant is given by the following formula:

$$t_i^f = \sum_{j=1}^{N-1} \frac{\rho_j^f}{\rho_i^f} F_{ij}, \quad (17)$$

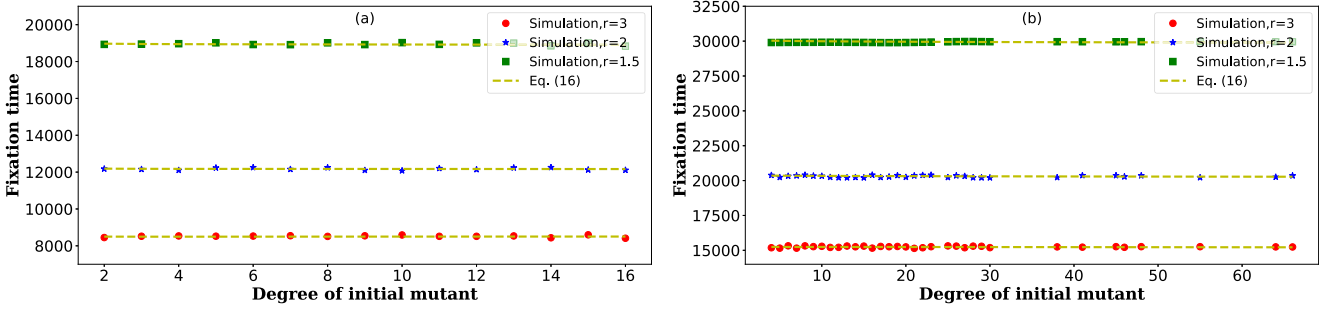


FIG. 5. Fixation time as a function of the degree of an initial mutant for three fitness values, 3, 2, and 1.5, in (a) ER and (b) BA graphs with the size of 500 and the average degree of 8.

where F_{ij} , given in (A6) in the Appendix (A), is the expected number of times, the process which started from i mutants, passes through the state with j mutants before reaching one of the absorbing states and ρ_j^f is the fixation probability starting from j mutants. Values of F_{ij} depend on graphs [see the Appendix (A) for more details].

Figure 5 shows that there is no difference in fixation times for various degrees of an initial mutant. The distribution of fixation times is shown in Fig. 6. We can see that the fixation times are distributed symmetrically around the average fixation time in both graphs. One notable point is that the average fixation time in the BA graph is higher than in the ER one.

C. Extinction time

To find an analytical expression for the extinction time we use a similar approach as we did for the fixation time. Let

$$t_1^e = \frac{P_{2 \rightarrow 1} + P_{2 \rightarrow 3}}{\rho_1^e [(P_{1 \rightarrow 0} + P_{1 \rightarrow 2})(P_{2 \rightarrow 1} + P_{2 \rightarrow 3}) - P_{2 \rightarrow 1} P_{1 \rightarrow 2}]} \left\{ P_{1 \rightarrow 0} + \left[\frac{(1 - P_{1 \rightarrow 0} - P_{1 \rightarrow 2})(P_{2 \rightarrow 1} + P_{2 \rightarrow 3}) + (P_{1 \rightarrow 2} P_{2 \rightarrow 1})}{P_{2 \rightarrow 1} + P_{2 \rightarrow 3}} \right] \rho_1^e \right. \\ \left. + \left[\frac{P_{1 \rightarrow 2}}{(P_{1 \rightarrow 0} + P_{1 \rightarrow 2})} \right] \rho_2^e + \frac{P_{1 \rightarrow 2} P_{2 \rightarrow 3}}{P_{2 \rightarrow 1} + P_{2 \rightarrow 3}} \rho_3^e (t_3^e + 1) \right\}. \quad (20)$$

Here again the idea is that we consider local information and an effect of the initial degree in states 1, 2, and 3 and then we disregard the information about the initial degree. The time to extinction, starting from $i = 3$ mutants is given by the following expression [16,26]:

$$t_3^e = \sum_{j=1}^{N-1} \frac{\rho_j^e}{\rho_3^e} F_{3j}. \quad (21)$$

We present results of stochastic simulations and our analytical approach in Fig. 7. It appears that as the initial mutant's degree increases, extinction time decreases. Essentially, as the initial mutant degree increases, it becomes more likely to select one of its neighbors to replace it. As a result, mutants with higher degrees are more likely to be wiped out in a short period of time. In the BA graph, for two initial mutants with a large difference in degrees, we have completely different extinction times due to the average degree of the network. It is worth noting that for vertices with high

$\rho_i^e = 1 - \rho_i^f$ be the extinction probability starting with i mutants and $P_{i \rightarrow j}$ the transition probability from i to j mutants. We apply the conditional expected value formula for t_i^e , the extinction time from a state with i initial mutants, and get [2]

$$\rho_1^e t_1^e = P_{1 \rightarrow 0} + P_{1 \rightarrow 1} \rho_1^e (t_1^e + 1) + P_{1 \rightarrow 2} \rho_2^e (t_2^e + 1) \quad (18)$$

and

$$\rho_2^e t_2^e = P_{2 \rightarrow 1} \rho_1^e (t_1^e + 1) + P_{2 \rightarrow 2} \rho_2^e (t_2^e + 1) + P_{2 \rightarrow 3} \rho_3^e (t_3^e + 1). \quad (19)$$

These equations for the extinction time are similar to equations we derived for the fixation time. In (18) there is an extra term $P_{1 \rightarrow 0}$ because if the number of mutants reaches zero, then the extinction probability from this state is zero $t_0^e = 0$ and the term $P_{1 \rightarrow 0} \rho_0^e (t_0^e + 1)$ drops out, leaving only the $P_{1 \rightarrow 0}$ term. As before we find $\rho_2^e t_2^e$ from (19) and substitute it into (18) and after some simplifications we get

degrees, the extinction time is almost the same for all values of r . In other words, if the process starts from a hub, then, regardless of r , it will soon become extinct. In Fig. 8, we can see the distribution of extinction times for both graphs. In the ER graph, extinction times are distributed around an average time, while in the BA one, the distribution is more extended. ER graphs exhibit lower average extinction times than BA ones.

In Fig. 9, the extinction time for various initial mutants with equal degrees is presented. Observe that for initial mutants with the same degree, the one in the BA graph exhibits a longer extinction time.

IV. FIXATION AND EXTINCTION TIMES: DISCUSSION

Here we examine in more detail the evolution of a population starting with one mutant. Figures 10 and 11 show the path to extinction and fixation of a mutant in the ER graph with a size of 500 and an average degree of 8. We derive approximate

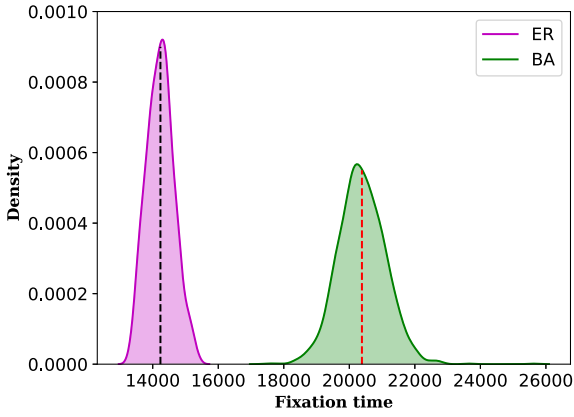


FIG. 6. Distribution of fixation times of a mutant with respect to initial placements on ER and BA graphs with the size of 500 and the average degree of 8. The fitness is 2. Dash lines show the average fixation time.

formulas for the expected number of times the process which started from i mutants, passes through the state with j mutants before reaching fixation and extinction respectively (details of derivation are given in the Appendix A),

$$t_{i,j}^f = \begin{cases} \frac{(r^j-1)^2(r^{N-i}-1)r^{j-i}}{(r-1)(r^N-1)(r^j-1)\frac{j}{N-i+rj}\frac{(N-j)(p-\frac{2}{N})}{Np}} & j \leq i \\ \frac{(r^{N-j}-1)(r^j-1)}{(r-1)(r^N-1)\frac{j}{N-i+rj}\frac{(N-j)(p-\frac{2}{N})}{Np}} & j > i \end{cases}, \quad (22)$$

$$t_{i,j}^e = \begin{cases} \frac{(r^j-1)(r^{N-i}-1)(r^N-r^j)r^{j-i}}{(r-1)(r^N-1)(r^N-r^i)\frac{j}{N-i+rj}\frac{(N-j)(p-\frac{2}{N})}{Np}} & j \leq i \\ \frac{(r^N-r^j)(r^{N-j}-1)(r^j-1)}{(r-1)(r^N-1)(r^N-r^i)\frac{j}{N-i+rj}\frac{(N-j)(p-\frac{2}{N})}{Np}} & j > i \end{cases}. \quad (23)$$

Denote by T_j^e and T_j^f the expected time it takes a single mutant to take over j nodes in a network before extinction and fixation, respectively. For a single mutant to be fixed, the Moran process must pass through the state with j mutants. Thus, T_j^f is the time needed for a single mutant to be fixed minus the time needed for j mutants to be fixed [35]. Therefore we have

$$\begin{aligned} T_j^f &= t_1^f - t_j^f = \sum_{l=1}^{N-1} t_{1,l}^f - \sum_{l=1}^{N-1} t_{j,l}^f, \\ T_j^e &= t_1^e - t_j^e = \sum_{l=1}^{N-1} t_{1,l}^e - \sum_{l=1}^{N-1} t_{j,l}^e. \end{aligned} \quad (24)$$

We have performed stochastic simulations to see how accurate these analytical formulas are. For t_1^f and t_1^e we use our analytical approximation in (16) and (20). Figures 10(a) and 11(a) show the expected number of times, the population started with one mutant passes through the state with j mutants before fixation and extinction, respectively, while Figs. 10(b) and 11(b) show the value of T_j^f and T_j^e . The results are for the ER network with a size of 500 and an average degree of 8. We present here only results for the ER network, and results for the BA network are the same. We observe that the process before extinction spends most of

its time in states with few mutants. This means that during the process of the extinction of mutants, a few vertices get mutated and if the number of mutants increases, then there is a higher chance of them to be fixed. Therefore, the initial placement of mutants plays an important role in the extinction of mutants. For fixation, unlike extinction, the beginning and the end of the process can have a significant impact on the fixation time. At the beginning of the process when few mutants are available, the probability of choosing them to reproduce is low, so it takes a long time for mutants to increase their number. Furthermore, with fewer residents in the population, it takes longer time to replace them with one of the mutants' offspring at the end of the process. Hence it does not matter where the mutant is initially placed because the start and end of the process are equally important for the fixation time.

We observe that our analytical expressions are less accurate for smaller values of r and are becoming better as r increases. We assumed that the initial mutant degree influences only the probability of transition between one to two and two to three mutants. However, there is a possibility that the number of mutants increases and then decreases again to 1. Hence, the mutant may be different from the starting mutant, leading to an error in our approximation. For large r , there is a low probability that the number of mutants will decrease to 1. Therefore, when there are more mutants, they are more likely to increase rather than decrease their number. Consequently, our approximation is better for larger r .

V. DISCUSSION

To summarize, we examined the impact of the degree of an initial mutant on the fixation probability and fixation and extinction times in Moran processes in structured populations. We performed computer simulations and developed a general mean-field approximation approach on graphs which enabled us to get analytical formulas. In this way we extended to arbitrary fitness values results of Refs. [9,10,19], where the authors provided good approximations for cases with mutant fitness close to that of a resident.

We showed that the fixation probability depends on the vertex degree at which a mutant is introduced. Increasing the degree of the initial mutant makes it more likely for the mutant to be replaced by one of its neighbors. There was no correlation between the degree of the initial mutant and the time it took for the mutant to take over the entire population. The fixation process depends on the entire population, not on the first mutant. We found that, unlike fixation time, extinction time significantly depends on the degree of the first mutant—a mutant with fewer connections needs more time to become extinct. Furthermore, we observed that the extinction time for an initial mutant with a small degree is significantly influenced by its fitness, but as the degree of the first mutant increases, extinction times become independent on the fitness value.

It is important to study the impact of the initial mutant's degree on the fixation probability and fixation and extinction times in frequency-dependent Moran processes of spatial evolutionary games, where fitness is derived from game competitions.

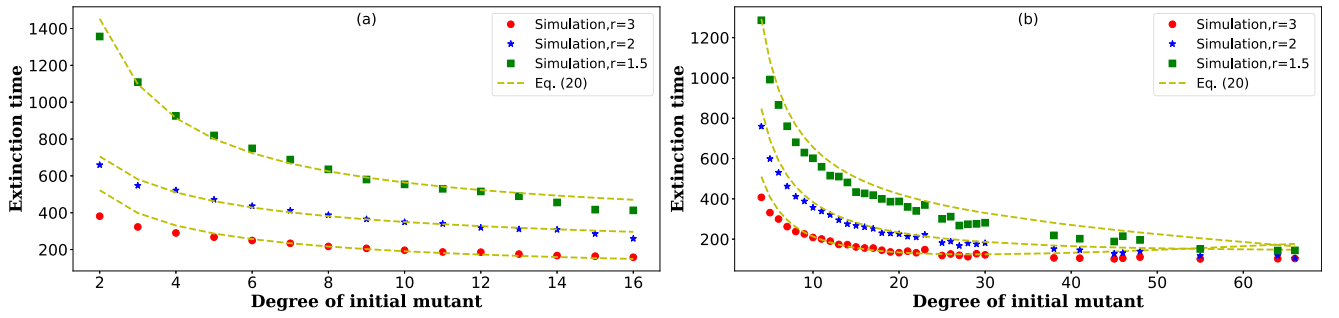


FIG. 7. Extinction time as a function of a degree of an initial mutant for three fitness values, 3, 2, and 1.5, in (a) ER and (b) BA graphs with the size of 500 and the average degree of 8.

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APPENDIX A

We consider here a reduced Markov chain for the Moran process on the graph with $N + 1$ states representing the number i of mutants in the population. The transition matrix can be then written as follows:

$$P_{ij} = p_{i \rightarrow i+1} \delta_{i+1,j} + p_{i \rightarrow i-1} \delta_{i-1,j} + q_i \delta_{i,j}, \quad (A1)$$

where $q_i = 1 - p_{i \rightarrow i+1} - p_{i \rightarrow i-1}$.

Our Markov chain has two absorbing states: $i = 0$ and $i = N$. To calculate absorbing probabilities and expected absorbing times we use the general method described in Ref. [45]. We rewrite the transition matrix as follows:

$$P = \begin{pmatrix} Q & R \\ 0 & I \end{pmatrix}, \quad (A2)$$

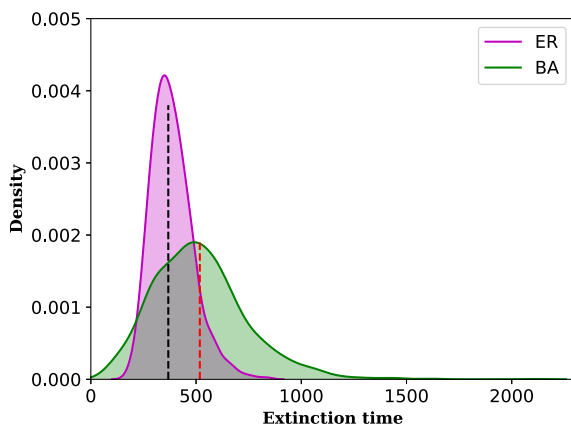


FIG. 8. Distribution of extinction times of a mutant with respect to initial placements on ER and BA graphs with the size of 500 and the average degree of 8. The fitness is 2. The dash lines show the average extinction times.

where Q contains transition probabilities between transient states and R contains transition probabilities from transient states to absorbing ones and I is the identity matrix. $F = (I - Q)^{-1}$ is called the fundamental matrix. It can be shown that F_{ij} is the expected number of times, the process which started from i mutants, passes through the state with j mutants before reaching one of the absorbing states. Then it follows that expected absorption times are given by summations,

$$t_i = \sum_{j=1}^{N-1} F_{ij}. \quad (A3)$$

Expected number of times the process which started from i mutants, passes through the state with j mutants before reaching fixation and extinction respectively can be written as follows [16]:

$$t_{i,j}^f = \frac{\rho_j^f}{\rho_i^f} F_{ij}, \quad t_{i,j}^e = \frac{\rho_j^e}{\rho_i^e} F_{ij}. \quad (A4)$$

Hence expected fixation and extinction times are given by the following expressions:

$$t_i^f = \sum_{j=1}^{N-1} \frac{\rho_j^f}{\rho_i^f} F_{ij}, \quad t_i^e = \sum_{j=1}^{N-1} \frac{\rho_j^e}{\rho_i^e} F_{ij}. \quad (A5)$$

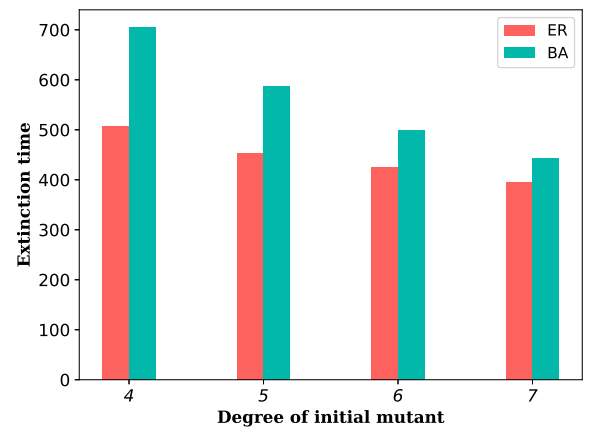


FIG. 9. Extinction time for various initial mutants with the same degree in both networks. The size of the networks is 500, the average degree 8, and $r = 2$.

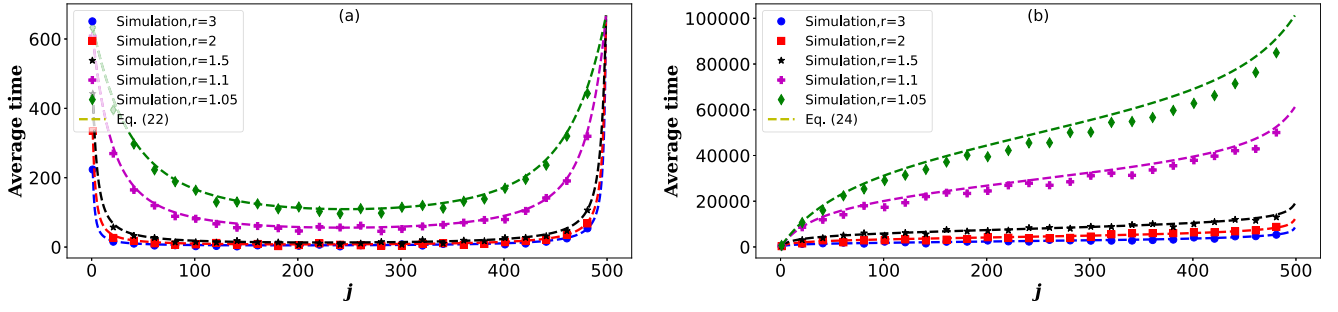


FIG. 10. (a) The expected number of times the population started with one mutant passes through the state with j mutants before fixation, $t_{1,j}^f$, Eq. (22) and (b) the expected time until the number of mutants reaches j from 1, T_j^f , Eq. (17), for various values of r in the ER graph with $N = 500$ and average degree of 8.

Now we turn our attention to graphs. It can be shown that if the Moran process on a graph satisfies the condition $p_{i \rightarrow i+1} = r p_{i \rightarrow i-1}$, then we have [26]

$$F_{ij} = \begin{cases} \frac{(r^j - 1)(r^{N-i} - 1)}{p_{j \rightarrow j-1}(r - 1)(r^N - 1)}, & j \leq i \\ \frac{r^{j-i}(r^i - 1)(r^{N-j} - 1)}{p_{j \rightarrow j-1}(r - 1)(r^N - 1)}, & i < j \end{cases}. \quad (\text{A6})$$

The authors of Ref. [26] used mean-field techniques and argue that $p_{i \rightarrow i+1} = r p_{i \rightarrow i-1}$ is a good approximation for Erdős-Rényi and Barabási-Albert graphs. We present here their arguments and results.

(i) Erdős-Rényi graph

In the ER graph with N vertices and j mutants, the probability of a mutant being selected for the reproduction is given by $\frac{rj}{rj + N - j}$. Although it may seem that this mutant would have $(N - j)p$ residents in its neighborhood, the population of mutants tends to form clusters, making it likely for a selected mutant to have at least two mutant neighbors. Thus, the probability of a selected mutant being connected to a resident is approximately $(p - \frac{2}{N})$, and the average number of residents connected to a selected mutant is $(N - j)(p - \frac{2}{N})$. Then

$$p_{j \rightarrow j+1} = r p_{j \rightarrow j-1} = \frac{rj}{N - i + rj} \frac{(N - j)(p - \frac{2}{N})}{(N - 1)p}. \quad (\text{A7})$$

(ii) Barabási-Albert graph

Here again, the probability for a mutant to be chosen for the reproduction is $\frac{rj}{rj + N - j}$. For a state with j mutants, I_j represents the average number of edges that connect different

species, also known as interface edges. On average, each mutant (resident) has $\langle k \rangle$ connections, and $\frac{I_j}{j} [I_j / (N - j)]$ interface edges. As a result, the probability for a mutant (resident) offspring to replace one of its resident (mutant) neighbors is $\frac{I_j}{m} [\frac{I_j}{(N - j)}]$. It follows that

$$p_{j \rightarrow j+1} = r p_{j \rightarrow j-1} = \frac{r I_j}{\langle k \rangle (N - j + r j)}. \quad (\text{A8})$$

In order to find I_j when there are i mutants, it is evident that every resident node has at least m edges and an average of $\frac{I_j}{N - j}$ interface edges. If one resident is changed to a mutant, then the number of mutants increases by 1. Therefore, we can obtain a recursive relationship for the number of interface edges as follows:

$$I_{j+1} = I_j + m - \frac{I_j}{N - j}. \quad (\text{A9})$$

Assuming $I_0 = 0$ we have

$$I_j = m \sum_{i=1}^j \frac{N - j}{N - i}. \quad (\text{A10})$$

APPENDIX B

The effect of the degree of an initial mutant on the fixation probability has been previously studied by Antal, Redner, and Sood [9]. They examined how initial placement impacts fixation probability in various dynamics. In particular, for the birth-death Moran process (biased invasion process) with the mutant fitness equal to 1 and the resident fitness set to $1 - s$,

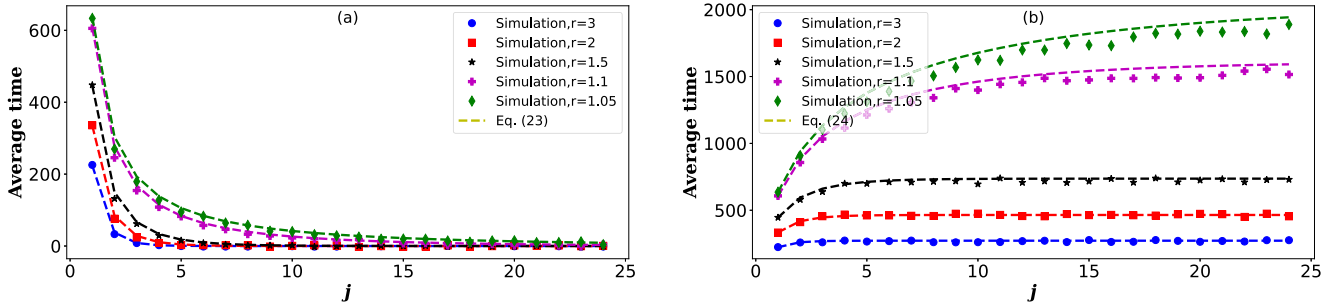


FIG. 11. (a) The expected number of times the population started with one mutant passes through the state with j mutants before extinction, $t_{1,j}^e$, Eq. (23) and (b) the expected time until the number of mutants reaches j from 1, T_j^e , Eq. (17), for various values of r in the ER graph with $N = 500$ and average degree of 8.

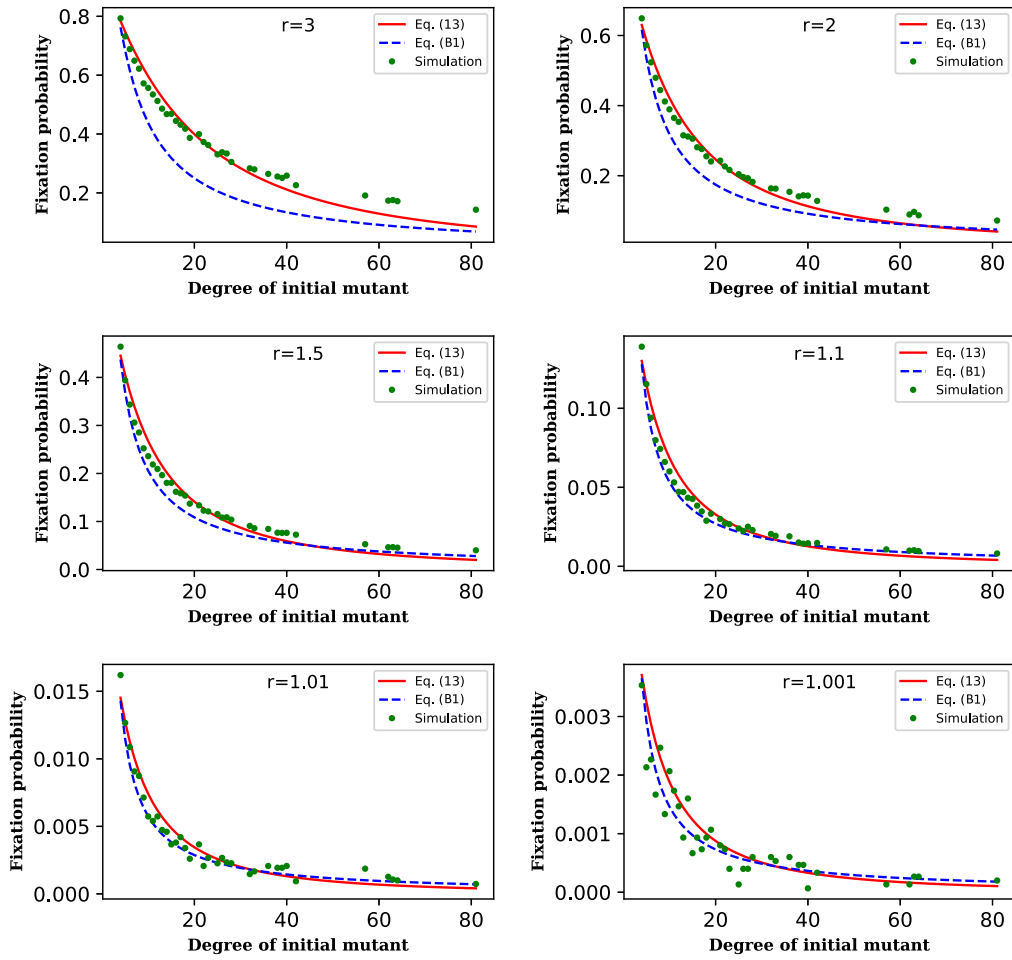


FIG. 12. Fixation probability as a function of the degree of an initial mutant for various fitness values. The red line is our approximation given by (13), the blue dashed line is an approximation given by (B1), and the green dots show the result of simulations. In Eq. (B1) $s = 1 - 1/r$.

the authors derived the following approximate formula for the fixation probability:

$$\rho_1^f(\omega_{-1}) = \frac{1 - e^{-sN\omega_{-1}/(1-s/2)}}{1 - e^{-sN/(1-s/2)}}, \quad (\text{B1})$$

where $\omega_{-1} = \frac{1}{k\mu_{-1}N}$ and $\mu_{-1} = \sum_k \frac{1}{k}\rho(k)$ is the average inverse degree [$\rho(k)$ is the degree distribution] [9].

Figure 12 compares the results of our approximation given in Eq. (13) with Eq. (B1) for various fitness values. We calculated μ_{-1} numerically. To make the connection between the fitness $r = 1 + s > 1$ for mutants and fitness 1 for residents in our paper with Ref. [9], where fitness of mutants is 1 and that of residents $1 - s$, we divide the fitnesses in (B1) by $1 - s$ (fixation probability is invariant under such a scaling).

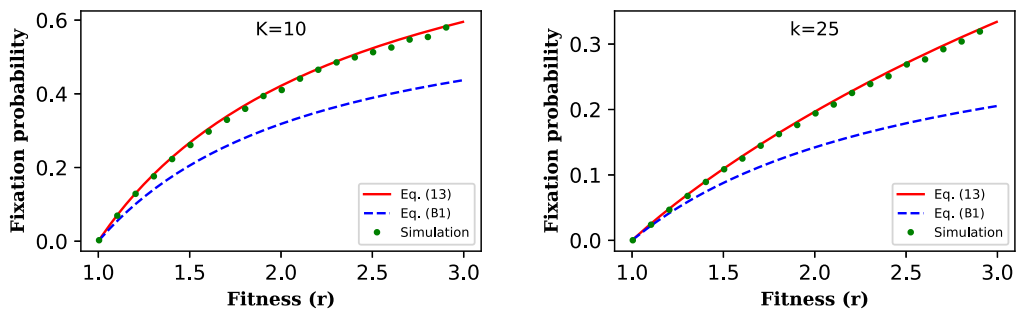


FIG. 13. The fitness effect on fixation probability for an initial mutant with degrees of 10 and 25. The red line is our approximation given by (13), the blue dashed line is an approximation given by (B1), and the green dots show the result of simulations. In Eq. (B1) $s = 1 - 1/r$.

Therefore in (B1), the fitness of mutants is $r = \frac{1}{1-s} > 1$ for $0 < s < 1$ and that of resident is 1. In this way the value of s in (B1) is $1 - \frac{1}{r}$.

We see that our approximation performs well for all values of $s > 0$, while Eq. (B1) is more accurate in cases where r is close to 1 ($s \ll 1$).

For a more direct comparison, we fixed the degree of an initial mutant and varied the fitness r from 1 to 3. Figure 13 shows the results for initial mutants with degrees of 10 and 25. For r close to 1, both approximations perform well. However, as r increases, our approximation in Eq. (13) outperforms Eq. (B1).

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The impact of time delays on mutant fixation in pairwise social dilemma games

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Evolutionary game theory examines how strategies spread and persist in populations through reproduction and imitation based on their fitness. Traditionally, models assume instantaneous dynamics where fitness depends on the current population state. However, some real-world processes unfold over time, with outcomes emerging from history. This motivates incorporating time delays into evolutionary game models, where fitness relies on the past. We study the impact of time delays on mutant fixation in a Moran Birth–death process with two strategies in a well-mixed population. At each time step of the process, an individual reproduces proportionally to fitness coming from the past. We model this as an absorbing Markov chain, allowing computational calculation of the fixation probability and time. We focus on three important games: the Stag-Hunt, Snowdrift and Prisoner’s Dilemma. We will show time delays reduce the fixation probability in the Stag-Hunt and the Prisoner’s Dilemma but increase it in the Snowdrift. For the Stag-Hunt and the Prisoner’s Dilemma, time delays lengthen the fixation time until a critical point, then reduce it. The Snowdrift exhibits the opposite trend.

1. Introduction

Evolutionary Game Theory (EGT) provides a framework for understanding how effective strategies spread through imitation or reproduction and persist in systems ranging from biology to economics [1–6]. The theory gives insights into phenomena

like cooperation, competition and diversity in populations [7–13]. It examines how strategies or types of individuals change in frequency in a population based on their fitness from interactions. Strategies that produce higher fitnesses will tend to spread and become more common as individuals reproduce or copy them. Traditional EGT models have primarily focused on well-mixed, infinitely large populations, where individuals interact at random and strategy frequencies evolve deterministically according to replicator dynamics or other differential equation models [2–4,14]. However, real-world populations are typically finite and structured. To address these limitations, recent work has incorporated stochastic processes, such as the Moran process, into EGT models to study evolutionary dynamics in finite populations [4,15,15–25].

The classical concept of an Evolutionarily Stable Strategy (ESS) does not fully capture evolutionary dynamics in finite populations. A true ESS must now satisfy two criteria, it must resist invasion by rare mutant strategies, and its probability of replacement by a rare mutant must be lower than under neutral drift [15,16,19,20]. The first requirement provides an equilibrium condition, while the second provides a stability condition. Selection opposes invasion when mutants cannot attain higher fitness than resident individuals in a pure population. It also opposes fixation when mutants have no better chance of reaching fixation than neutral mutations. Satisfying both criteria helps ensure a strategy is robust and maintains equilibrium [6]. Therefore the study of fixation probabilities became a crucial component of comprehending EGT.

The Moran process is a classic model for studying evolution in finite populations [26]. In this stochastic process, a population of N individuals with different types undergoes birth and death events over discrete time steps. At each step, one individual is selected to reproduce and its offspring replaces another chosen individual [27]. The individual's fitness can influence birth and death events in this process. In general, individuals reproduce with a probability proportional to their fitness and replace another individual at random (Birth-death (Bd) process). We note that this process can also happen in other ways, and there are a number of different dynamics used. Fitness can be constant or frequency-dependent based on game interactions between individuals [15,16]. Typically the population is assumed well-mixed so that all individuals have the chance to be replaced by others. In this process, we track the fate of any emergent mutant, assessing the probability and the time that this single novel mutant ultimately fixes in the population (fixation probability and time). Also monitoring the trajectory of the mutant through the population provides insight into the dynamics of how novel mutations establish and propagate themselves in populations.

The fixation probability and time have been extensively studied across various models [28–37]. Previous work has focused on how factors like population size [28,34–36], environmental fluctuations [38–41] and population structure [42–51] influence mutant fixation. However, most of these studies assumed that fitness is determined instantaneously based on the current population state and payoffs. This assumption may not always be realistic, as biological and social processes often involve temporal delays [52,53]. It is therefore natural for EGT models to incorporate time delays, where fitness depends on history. The effects of time delays have been explored in the context of replicator dynamics for games with an interior stable equilibrium [54–64]. In [54], the authors studied a model where individuals at time t imitate strategies that had higher average payoffs at time $t - \tau$ for some delay τ . They showed the interior fixed point is locally asymptotically stable for small τ , but becomes unstable for large τ , leading to oscillations. In [55], individuals are born after their parents play and receive payoffs. Two coupled equations governed strategy frequency and population size. Analysis showed oscillations are not possible, with the original fixed point globally asymptotically stable regardless of the time delay. In [56], the authors modified the above model by allowing time delays to vary based on the strategies used by individuals. Under this framework, they observe novel behaviour where the equilibrium states become dependent on the delays. Most previous work only considered replicator dynamics and the impact of delays on equilibrium stability.

Less attention has been given to finite populations and the effect of delays on the fixation process.

Our study looks into the influence of time delays on the fixation process in evolutionary games. We explore the impact of time delays on the fixation probability and fixation time in a well-mixed population comprising two strategies, examining a generalized Moran Bd process, where the fitness of both strategies at time step t hinges on the fitness values from τ steps previously. By constructing an absorbing Markov chain, whose states are defined by $\tau + 1$ indices, τ indices representing the history, and one the current mutant abundance, we determine the transition matrix, enabling us to analyse the fixation probability and fixation time for various game types.

The paper is structured as follows. Section 2 reviews the Moran process definition and basic properties for two-strategy games. Section 3 introduces a general framework to calculate absorption probabilities and times in Markov chains related to the process. Section 4 presents our model incorporating time delays into the Moran Bd process and defines a new associated Markov chain. Section 5 details the results of our model and the effects of time delays on the fixation probability and time across different games. The paper finishes with a discussion of the key findings and a summary of the main results.

2. Moran process

We consider the evolutionary dynamics of individuals adopting two distinct strategies, A and B within a well-mixed population, the game being played characterized by the payoff matrix [16]:

	A	B
A	a	b
B	c	d

When a player with strategy A interacts with an A player (a B player), they receive a payoff of a (b). On the other hand, a B player receives a payoff of c (d) from an A (B) player. Consequently, the average payoffs when there are i individuals with strategy A reads as follows:

$$\begin{aligned}\pi_A(i) &= \frac{(i-1)a + (N-i)b}{(N-1)}, \\ \pi_B(i) &= \frac{ic + (N-i-1)d}{(N-1)},\end{aligned}\tag{2.1}$$

where self-interactions are excluded. The corresponding average fitnesses are proportional to:

$$\begin{aligned}f_i &= 1 - w + w\pi_A(i), \\ g_i &= 1 - w + w\pi_B(i).\end{aligned}\tag{2.2}$$

The variable w represents the intensity of selection, indicating how much the payoff of individuals affects fitness [15]. When w approaches zero, each individual's payoff makes a minimal contribution to overall fitness, leading to what is known as weak selection. When $w = 0$, all individuals possess the same level of the fitness, resulting in neutral drift. Conversely, as w increases, the influence of payoffs on fitness grows and we have strong selection.

There are various update rules when individuals update their strategies during each time step of a process [15,16,29,42,65,66]. We use the Bd process, where the fitness of individuals is considered in the Birth event, which is a type of frequency-dependent Moran process [15,16]. During each time step, an individual is chosen for reproduction based on their fitness. The newly born individual then replaces a randomly selected member of the population, ensuring that the total population size, N , remains constant. At each time step, i can change by a maximum of one based on the following transition probabilities:

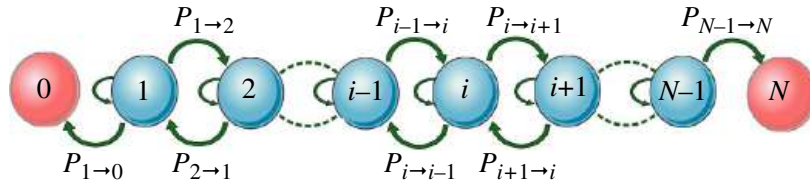


Figure 1. Absorbing Markov chain with $N + 1$ states associated with the Moran Bd process on population with size N . Each state is determined by the number of mutants in the population (i). States 0 and N are the absorbing states related to the extinction and fixation of mutants, respectively.

$$\begin{aligned} p_{i \rightarrow i+1} &= \frac{if_i}{if_i + (N-i)g_i} \frac{N-i}{N-1}, \\ p_{i \rightarrow i-1} &= \frac{(N-i)g_i}{if_i + (N-i)g_i} \frac{i}{N-1}. \end{aligned} \quad (2.3)$$

Here the objective is to examine the Bd process involving a population of residents with strategy B when introduced to a single mutant with strategy A. This process ultimately results in one of two possible outcomes: the complete replacement of the population by the mutant, known as *fixation*, or the eradication of the mutant, referred to as *extinction*. Two quantities hold significant importance in this process, the fixation probability, which indicates the probability of the mutant becoming fixed in the population, and the fixation time, which represents the number of time steps required for the fixation [34,49,67]. There are three main approaches used to analyse the fixation process: direct analytical solutions, numerical methods using transition matrices of Markov chains, and Monte Carlo simulations. Analytical solutions can be found for simple Moran processes without population structure or with regular structure, providing formulae for fixation times and probabilities. However, as models become more complex, researchers must study the dynamics using numerical techniques and Monte Carlo simulations.

In general, we can consider an absorbing Markov chain associated with the process to investigate and compute the probability and time of fixation. Figure 1 provides a visual representation of the Markov chain. The state is determined by the number of mutants present in the population (i). Essentially, the Markov chain represents a random walk on sites ranging from 0 to N . Starting from state i , the walk eventually reaches either the absorbing states of 0 (mutant extinction), or N (mutant fixation). The transition matrix is defined as follows:

$$P_{i,j} = p_{i \rightarrow i+1} \delta_{i+1,j} + p_{i \rightarrow i-1} \delta_{i-1,j} + (1 - p_{i \rightarrow i+1} - p_{i \rightarrow i-1}) \delta_{i,j}. \quad (2.4)$$

The fixation probability $\Phi_{i,N}$ from state with i initial mutants with strategy A under a Bd process can be calculated using the recursive equations:

$$\Phi_{i,N} = p_{i \rightarrow i-1} \Phi_{i-1,N} + (1 - p_{i \rightarrow i-1} - p_{i \rightarrow i+1}) \Phi_{i,N} + p_{i \rightarrow i+1} \Phi_{i+1,N}. \quad (2.5)$$

Using this recursive relationship and the boundary conditions that $\Phi_{0,N} = 0$ and $\Phi_{N,N} = 1$, the fixation probability for a single mutant is [34,67]

$$\Phi_{1,N} = \frac{1}{1 + \sum_{k=1}^{N-1} \prod_{j=1}^k \frac{g_j}{f_j}}. \quad (2.6)$$

The same recursive equation applies for the absorption time a_i and fixation time $t_{i,N}$ as follows:

$$\begin{cases} a_i = p_{i \rightarrow i-1} a_{i-1} + (1 - p_{i \rightarrow i-1} - p_{i \rightarrow i+1}) a_i + p_{i \rightarrow i+1} a_{i+1}, \\ \Phi_{i,N} t_{i,N} = p_{i \rightarrow i-1} \Phi_{i-1,N} (t_{i-1,N} + 1) + (1 - p_{i \rightarrow i-1} - p_{i \rightarrow i+1}) \Phi_{i,N} (t_{i,N} + 1) \\ \quad + p_{i \rightarrow i+1} \Phi_{i+1,N} (t_{i+1,N} + 1). \end{cases} \quad (2.7)$$

With the initial conditions $a_0 = t_{0,N} = 0$ and $a_N = t_{N,N} = 0$ we have [34,67]:

$$\begin{cases} a_1 = \Phi_{1,N} \sum_{k=1}^{N-1} \sum_{l=1}^k \frac{1}{P_{l \rightarrow l+1}} \prod_{j=l+1}^k \frac{g_j}{f_j}, \\ t_{1,N} = \sum_{k=1}^{N-1} \sum_{l=1}^k \frac{\Phi_{l,N}}{P_{l \rightarrow l+1}} \prod_{j=l+1}^k \frac{g_j}{f_j}. \end{cases} \quad (2.8)$$

3. General framework to calculate the absorbing probability and time for Markov chains

To evaluate the absorption probabilities and time for Markov chains with $n = t + a$ states (t transition state and a absorbing states), a widely employed technique in numerical analysis, as outlined in [49,68], can be employed. The transition matrix $P_{n \times n}$ can be re-expressed in a canonical form as follows:

$$P = \begin{pmatrix} Q & R \\ 0 & I \end{pmatrix}, \quad (3.1)$$

where $Q_{t \times t}$ denotes the probabilities of transitions between the transient states, $R_{t \times a}$ signifies the probabilities of transitions from the transient states to the absorbing states and $I_{a \times a}$ represents an identity matrix. The Moran process in a well-mixed population is related to a Markov chain with $N+1$ states, including two absorbing states at 0 and N . Based on the canonical transition matrix, we can construct a new matrix denoted by $F = \sum_{n=0}^{\infty} Q^n = (I - Q)^{-1}$ and called a Fundamental matrix. The element $F_{i,j}$ represents the expected number of steps that the process which started in state i spends in state j before eventually reaching one of the absorbing states (the expected *sojourn time*) [49,68]. Based on this matrix, we can derive another matrix:

$$\Phi = FR, \quad (3.2)$$

where $\Phi_{i,j}$ represents the probability that the process becomes absorbed in absorbing state j , given that it starts in state i . For a Moran process in a well-mixed population, the matrix Φ is composed of two columns, representing extinction and fixation probabilities.

The absorption time starting from state i can be obtained by summing the i_{th} row of the fundamental matrix F and is given by the following expression:

$$a_i = \sum_{j=1}^{N-1} F_{i,j}. \quad (3.3)$$

To determine the fixation time, we employ a method described in previous studies [49,69,70]. If $\Phi_{j,N}$ represents the fixation probability starting from j mutants, then the fixation time starting from state i can be calculated as follows:

$$t_{i,N} = \sum_{j=1}^{N-1} \frac{\Phi_{j,N}}{\Phi_{i,N}} F_{i,j}. \quad (3.4)$$

By identifying the Markov chain and associated transition matrix for various models, we can utilize this numerical method to determine the fixation probability and times.

4. Model and methods

A random process should have a starting point. In our model, this is the time that a single mutant appears in the population, labelled time $t = 0$. In the above, we assume that the process

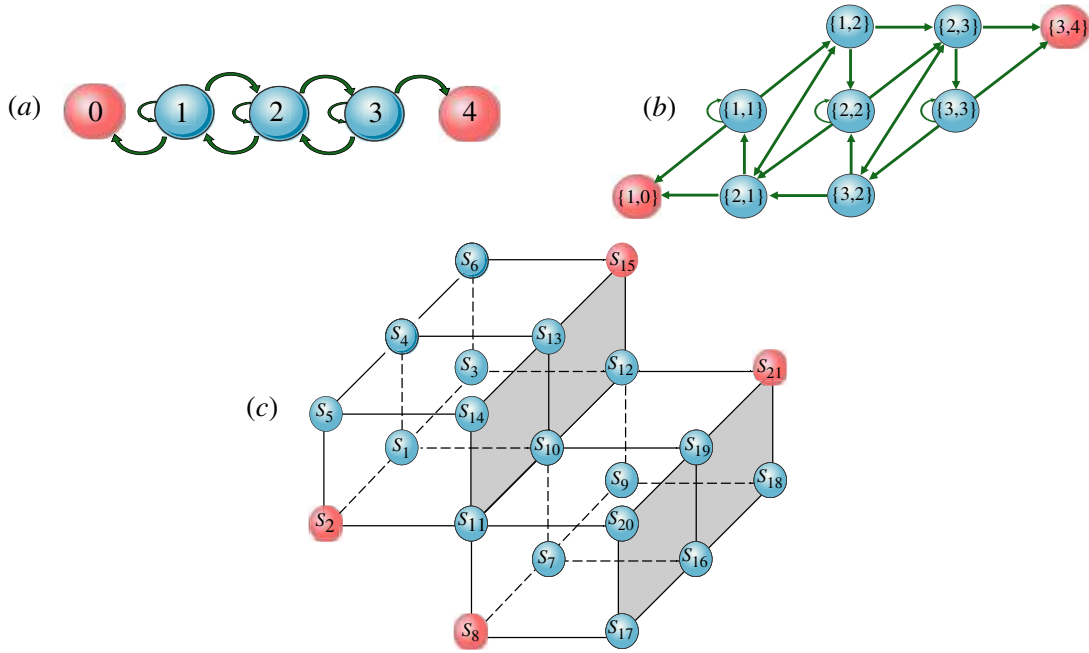


Figure 2. Three Markov chains for $\tau = 0, 1, 2$ with population size $N = 4$. The Markov chain with time delays τ has $\tau + 1$ dimensions. To generate state spaces for a Markov chain with $\tau + 1$ dimensions, we must erase the absorbing states from the τ -dimensional Markov chain and add three possible new indexes for each state $\{i_\tau, \dots, i_0\}$ like $\{i_\tau, \dots, i_0, i_{new}\}$ including $i_{new} = i_0 - 1, i_0, i_0 + 1$ (see **algorithm 1**). For instance, a population with size $N = 4$ has (a): $S_4^0 = \{0, \{1, \{2, \{3, \{4\}\}\}$; (b): $S_4^1 = \{1, 0, \{1, 1, \{1, 2, \{2, 1, \{2, 2, \{2, 3, \{3, 2, \{3, 3, \{3, 4\}\}\}\}\}$; and (c): $S_4^2 = \{s_1 = \{1, 1, 1, s_2 = \{1, 1, 0, s_3 = \{1, 1, 2, s_4 = \{1, 2, 2, s_5 = \{1, 2, 1, s_6 = \{1, 2, 3, s_7 = \{2, 1, 1, s_8 = \{2, 1, 0, s_9 = \{2, 1, 2, s_{10} = \{2, 2, 2, s_{11} = \{2, 2, 1, s_{12} = \{2, 2, 3, s_{13} = \{2, 3, 3, s_{14} = \{2, 3, 2, s_{15} = \{2, 3, 4, s_{16} = \{3, 2, 2, s_{17} = \{3, 2, 1, s_{18} = \{3, 2, 3, s_{19} = \{3, 3, 3, s_{20} = \{3, 3, 2, s_{21} = \{3, 3, 4\}\}$. The transition and absorbing states are represented by blue and red circles, respectively. When $\tau > 1$, there are more absorbing states with $i_0 = N, 0$ indicating mutant fixation and extinction, respectively.

is homogeneous, so that transition probabilities depend only upon the current state and not on the specific time that the state is reached. Thus for example in [equation \(2.5\)](#), the transition and fixation probabilities are independent of the specific time point, and so are not functions of time t . In the model developed below, we will similarly consider the process homogeneous, but develop the concept of the state of the process to include not only the population size at present, but also at the most recent times, to incorporate time delays. This still leaves the question of how to deal with times too close to the start of the process for the full required history to exist. We explain how we do this, by selecting a specific initial state, in §5a.

Now we introduce a time delay in the Bd process for a well-mixed population. In the standard Bd process, the fitness of strategies at the current (discrete) time t is important for the birth of new offspring. However, an individual's fitness can depend on conditions in the past. Thus, we incorporate a time delay τ , so that the fitness of strategies comes from time $t - \tau$, considering the same time delay for all strategies. Thus, both mutant and resident strategies receive payoffs from τ steps before. Since the Bd process proceeds in discrete time steps, τ takes integer values in the $[0, \infty)$ range. The case $\tau = 0$ corresponds to the standard Bd process described previously.

To study this process, we model it as a Markov chain and use the previous numerical approach. To fully characterize each Markov chain state, we need to know both the number of mutants at the current time t and the historical ones from time $t - \tau$ to the present. This allows us to define the state space describing the full process dynamics. Specifically, each state has $\tau + 1$ indices, with τ indices representing the number of mutants at each of the τ previous time steps, and one index for the current number at time t . When $\tau = 0$, the state reduces to a single

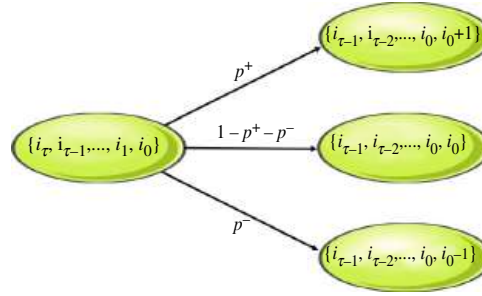


Figure 3. There are three possible transitions from state $\{i_{\tau}, i_{\tau-1}, \dots, i_1, i_0\}$ in which the number of mutants in the population represented by i_0 can rise by one, drop by one or remain unchanged. In each transition, the history will transform in such a way that $\{i_{\tau}, i_{\tau-1}, \dots, i_1, i_0\} \longrightarrow \{i_{\tau-1}, i_{\tau-2}, \dots, i_1, i_0, i_{\text{new}}\}$, $i_{\text{new}} = i_0 + 1, i_0, i_0 - 1$.

index, as originally discussed. With the general state form $\{i_{\tau}, \dots, i_1, i_0\}$, the number of mutants can only change by one per time step. This imposes $|i_n - i_{n-1}| \leq 1$ between adjacent indices. As an example, for $\tau = 1$ each state is characterized by $\{i_1, i_0\}$, with $i_0 = i_1 - 1, i_1, i_1 + 1$. Within this Markov chain, states characterized by $i_0 = 0, N$ serve as absorbing states, signifying mutant extinction and fixation.

Deriving the whole state space of the Markov chain $S_N^{\tau} = \{s_i\}$ for a time delay of τ in a population of size N as shown in figure 2, can be accomplished through the subsequent procedure.

$$\begin{aligned}
 S_N^0 &= \{i_0\}, \quad i_0 \in \{0, 1, 2, \dots, N\}, \\
 S_N^1 &= \{i_1, i_0\}, \quad i_1 \in S_N^0, \quad i_1 \neq \{0, N\}, \quad i_0 \in \{i_1 + 1, i_1, i_1 - 1\}, \\
 S_N^2 &= \{i_2, i_1, i_0\}, \quad i_2, i_1 \in S_N^1, \quad \{i_2, i_1\} \neq \{1, 0\}, \{N-1, N\}, \quad i_0 \in \{i_1 + 1, i_1, i_1 - 1\}, \\
 &\dots \\
 &\dots \\
 S_N^{\tau} &= \{i_{\tau}, i_{\tau-1}, \dots, i_1, i_0\}, \quad \{i_{\tau}, i_{\tau-1}, \dots, i_1\} \in S_N^{\tau-1}, \quad \{i_{\tau}, i_{\tau-1}, \dots, i_1\} \neq \{\text{Absorbing States}\}, \\
 &\quad i_0 \in \{i_1 + 1, i_1, i_1 - 1\}.
 \end{aligned} \tag{4.1}$$

The previous procedure allows us to find the state space of S_N^{τ} based on $S_N^{\tau-1}$. As shown in figure 2, state space S_N^{τ} can be represented as a $\tau + 1$ dimensional Markov chain. Simply we can obtain states of S_N^{τ} from $S_N^{\tau-1}$. We identify and delete the absorbing states of $S_N^{\tau-1}$ where $i_0 = 0$ or N , representing extinction or fixation. The remaining states of $S_N^{\tau-1}$ become the historical component of each state in S_N^{τ} (i_j in $S_N^{\tau-1}$ corresponding to i_{j+1} in S_N^{τ}). Since the number of mutants can change by at most one, i_0 in S_N^{τ} takes values $i_1 - 1, i_1$, or $i_1 + 1$ (i_1 in S_N^{τ} is i_0 in $S_N^{\tau-1}$). The process excludes the absorbing states corresponding to $S_N^{\tau-1}$ and adds one more dimension (one more index to each state satisfying previous conditions) to find the Markov chain of S_N^{τ} . **Algorithm 1** (described at the end of this section) can be used to find the state space for a given population size and time delay.

The next step is to find the transition matrix for the Markov chain with state space S_N^{τ} . Since we are considering the Bd process, time delays can affect the Birth part where fitness for reproduction comes from the past. If we denote the state as $\{i_{\tau}, i_{\tau-1}, \dots, i_1, i_0\}$, there are three possible transitions, as shown in figure 3. In each generation of the process, the number of mutants i_0 can either increase by one, decrease by one or remain unchanged. In each transition, the history changes such that the new i_j becomes i_{j-1} from the previous state. To find the transition probability, the fitness for the birth part comes from i_{τ} , which shows the number of mutants τ steps ago. However, for the death part, the number of current mutants i_0 is important. Considering these details, the transition probabilities are:

$$\begin{aligned}
 p^+ &= P_{\{i_\tau, i_{\tau-1}, \dots, i_1, i_0\} \rightarrow \{i_{\tau-1}, i_{\tau-2}, \dots, i_0, i_0+1\}} = \frac{i_0 f_{i_\tau}}{i_0 f_{i_\tau} + (N - i_0) g_{i_\tau}} \frac{N - i_0}{N - 1} \\
 p^- &= P_{\{i_\tau, i_{\tau-1}, \dots, i_1, i_0\} \rightarrow \{i_{\tau-1}, i_{\tau-2}, \dots, i_0, i_0-1\}} = \frac{(N - i_0) g_{i_\tau}}{i_0 f_{i_\tau} + (N - i_0) g_{i_\tau}} \frac{i_0}{N - 1}, \\
 P_{\{i_\tau, i_{\tau-1}, \dots, i_1, i_0\} \rightarrow \{i_{\tau-1}, i_{\tau-2}, \dots, i_0, i_0\}} &= 1 - p^+ - p^-
 \end{aligned} \tag{4.2}$$

where f_{i_τ} and g_{i_τ} are given in (2.2). **Algorithm 2** provides a useful way to determine the transition matrix P^τ for the time delay τ . Once P^τ is obtained, we can identify all absorbing states where $i_0 = 0, N$ and put the matrix into canonical form F^τ by finding Q^τ and R^τ . This then allows the numerical approach from the previous section to be applied to calculate the fixation probability and time. As mentioned before, there can be additional fixation states (depending on τ) with $i_0 = N$. Since the sum of each row of the absorbing probability matrix, $\Phi^\tau = F^\tau R^\tau$ is 1 (the sum of absorption probabilities starting from one state), we can find the fixation probability by summing the probabilities leading to fixation with $i_0 = N$. By dividing the state space S_N^τ into two parts $S_T^\tau \subset S_N^\tau$, $i_0 \neq 0, N$ for transient states and $S_A^\tau \subset S_N^\tau$, $i_0 = 0, N$ for absorbing states, we can define the fixation probability $\Phi_{s_m, N}^\tau$ and time $t_{s_m, N}^\tau$ starting from state s_m as:

$$\Phi^\tau = F^\tau R^\tau, \quad \Phi_{s_m, N}^\tau = \sum_{s_j = \{i_\tau, \dots, i_0\}, i_0 = N}^{S_A^\tau} \Phi_{s_m, s_j'}^\tau \tag{4.3}$$

$$t_{s_m, N}^\tau = \sum_{s_j = \{i_\tau, \dots, i_0\}}^{S_T^\tau} \frac{\Phi_{s_j, N}^\tau}{\Phi_{s_m, N}^\tau} F_{s_m, s_j'}^\tau \tag{4.4}$$

Algorithm 1. Find the state space

```

1: Input: Size N and time delay  $\tau$ 
2: state-space  $\leftarrow$  empty-array(N+1)
3: for  $l$  in  $[0, 1, 2, \dots, N]$  do
4:   state-space( $l$ )  $\leftarrow l$ 
5: end for
6: for  $t$  in  $[0, 1, 2, \dots, \tau - 1]$  do
7:   for state in state-space do
8:     if  $i_0=0$  or  $i_0=N$  then
9:       remove state from state-space
10:    end if
11:  end for
12:  new-state-space  $\leftarrow$  empty-array(size(state-space)  $\times$  3,  $t+1$ )
13:  for state in state-space do
14:    add {state,  $i_0-1$ } and {state,  $i_0$ } and {state,  $i_0+1$ }
      to new-state-space
15:  end for
16:  state-space  $\leftarrow$  new-state-space
17: end for
17: return state-space
    
```

Algorithm 2. Find the transition matrix

```

1: Input: state-space
2: transition-matrix  $\leftarrow$  zero-array(size(state-space)  $\times$  size(state-space))
3: for state in state-space do
4:   if state=absorbing-state then
5:     transition-matrix(state, state)=1
6:   else:
7:     for second-state in state-space do
8:       if  $\{i_\tau, i_{\tau-1}, \dots, i_1\} \in \text{second-state} = \{i_{\tau-1}, \dots, i_1, i_0\} \in \text{state}$  then
9:         if  $(i_0 \in \text{second-state} - i_0 \in \text{state})=1$  then
10:          transition-matrix(state, second-state)= $p^+$ 
11:        else if  $(i_0 \in \text{second-state} - i_0 \in \text{state})=-1$  then
12:          transition-matrix(state, second-state)= $p^-$ 
13:        else
14:          transition-matrix(state, second-state)= $1 - p^- - p^+$ 
15:        end if
16:      end if
17:    end for
18:  end if
19: end for
20: return transition-matrix

```

5. Results

In this section, we examine how time delays impact the fixation of mutants. Before presenting the main results, it is instructive to demonstrate some limitations of our numerical method. As figure 4 shows, the state space size grows with both N and τ . For large τ and N values, the number of states becomes enormous, necessitating more memory to store the data. Furthermore, calculating the inverse matrix has higher computational complexity as the matrix size increases. For instance, with $N = 5$ and $\tau = 6$, there are 1398 states, yielding a matrix with size of 1 954 404. This matrix would require approximately 15.6 MB of memory ($1\,954\,404 \times 8$ bytes). However, when N grows to 12 and τ to 9, the state space has 160 875 states, requiring around 207 GB of memory: a prohibitive amount. Clearly, for larger populations and time delays, our numerical method has limitations, and computer simulations become preferable.

(a) Fixation probability and time

In the following, we investigate the effect of time delays on the fixation probability and time in some well-known games: the Stag-Hunt, Snowdrift and Prisoner's Dilemma. When studying the fixation of one mutant in a population of residents, there may be multiple states with $i_0 = 1$ that differ in their historical components for a given time delay τ . For consistency, we will use the initial state $s_i = \{1, 1, \dots, 1\}$ for all our results; this is consistent with starting with a single mutant and that for times within τ of the start of our process, the pay-offs used to calculate the fitness are assumed to take the value at this starting point. We compare the results of our numerical method (as much as possible) with those from computer simulations. For the simulations, we conduct 10^7 different Monte Carlo simulations for each set of fixed parameters. The fixation probability is calculated as the ratio of fixed processes to total simulations. We determine the average fixation time only using processes that eventually undergo fixation. We constrained the pay-off matrix by setting $a = 2.1$ and $d = 0.1$. By varying the remaining two parameters, we can generate a range of distinct games. This approach reduces the total number of parameters while still allowing for diverse game scenarios.

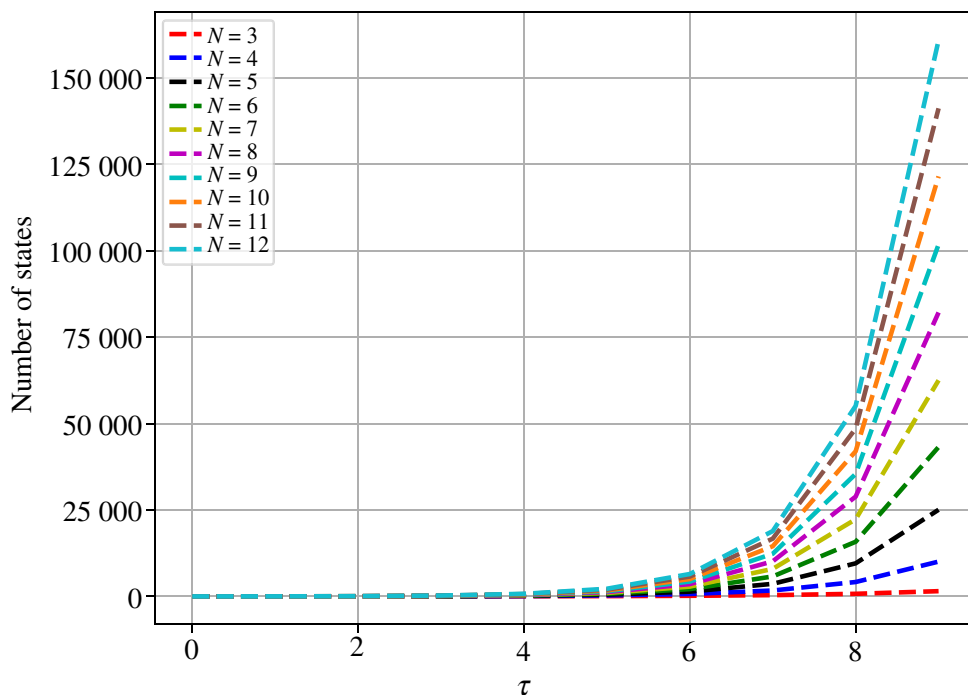


Figure 4. The number of states as a function of N and τ .

(i) Stag-Hunt game

The Stag-Hunt game is one of the earliest and most analysed coordination games [9]. This game is defined by a payoff matrix where the payoffs satisfy the relation $a > c \geq d > b$. The key feature of the Stag-Hunt is that players are incentivized to coordinate on the same strategy, represented by the payoffs a and d along the leading diagonal of the payoff matrix. However, there is also a risk, captured by the off-diagonal payoffs b and c , if the players fail to match strategies. Given that a is fixed at 2.1 and d at 0.1, the value of c must fall within the range $[0.1, 2.1)$, while b should be less than 0.1.

We first analyse how time delays impact the fixation probability when there is one mutant with strategy A (the ‘stag’ strategy) in a population of residents using strategy B. Figure 5 shows how varying the payoff matrix elements, population size and selection intensity affects the fixation probability in the presence of time delays. The lines represent numerical solutions while the dots show computer simulations. As noted previously, our numerical method is limited computationally, restricting the population size and time delay values we could examine. Thus for larger N and τ , only simulation results are presented. Regardless of the network size and payoff element values, longer time delays consistently yield lower fixation probabilities. The results demonstrate that time delays reduce the ability of a mutant to take over a population compared with the undelayed cases.

Figure 5a,b demonstrate that as τ increases, the fixation probabilities appear to converge to a constant value. Our model indicates that as τ approaches infinity, fitness remains unchanged throughout the fixation process. This is because fitness originates from the starting state with one mutant and persists across all steps until fixation. Thus, we have a Moran process with constant fitness, where the fixation probability follows equation (2.6). With $w = 1$ and the constant fitness defined as r :

$$\tau \rightarrow \infty \begin{cases} \pi_A(i) = \pi_A(1) = \frac{(N-1)b}{N-1} \rightarrow f_i = b \\ \pi_B(i) = \pi_B(1) = \frac{c + (N-2)d}{N-1} \rightarrow g_i = \frac{c + (N-2)0.1}{N-1} \end{cases} \quad r = \frac{f_i}{g_i} = \frac{b(N-1)}{c + (N-2)0.1}.$$

Therefore the fixation probability with constant fitness r is:

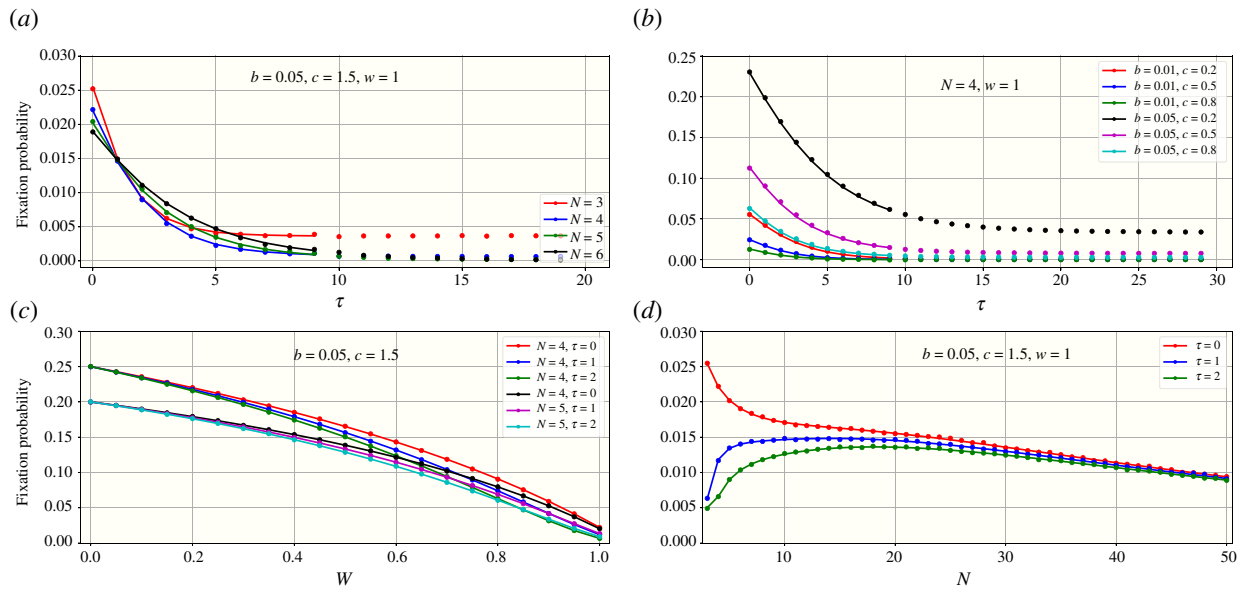


Figure 5. The effect of time delays on fixation probabilities in the Stag-Hunt game ($\alpha = 2.1$ and $d = 0.1$) according to numerical solutions (lines) and computer simulations (dots). Panel (a) shows the influence of time delays for various network sizes with $b = 0.05$, $c = 1.5$ and $w = 1$. Panel (b) illustrates the effect of time delays for different payoff matrix parameters with $N = 4$ and $w = 1$. Panel (c) depicts how selection intensity affects outcomes for $N = 4, 5$ and $\tau = 0, 1, 2$ with $b = 0.05$, $c = 1.5$. Lastly, panel (d) demonstrates the impact of network size for $\tau = 0, 1, 2$ with $b = 0.05$, $c = 1.5$ and $w = 1$.

$$\Phi_{1,N} = \frac{1}{1 + \sum_{k=1}^{N-1} \prod_{j=1}^k \frac{1}{r}} = \frac{1 - \frac{1}{r}}{1 - (\frac{1}{r})^N} = \frac{1 - \frac{b(N-1)}{c + (N-2)0.1}}{1 - (\frac{b(N-1)}{c + (N-2)0.1})^N}. \quad (5.1)$$

For sufficiently large values of τ , the fixation probability depends only on the element of the payoff matrix and N . When $b = 0.05$ and $c = 1.5$, the fixation probability is $0.05(N-1)/1.5 + (N-2)0.1$. For $N = 3, 4, 5, 6$, this gives fixation probabilities of 0.0036, 0.0006, 0.0001 and 0.00003, respectively, matching figure 5a. The same behaviour is observed in panel (b) for a large time delay for different values of the payoff matrix. The influence of the intensity of selection w is shown in panel (c) for a fixed payoff matrix with $b = 0.05$ and $c = 1.5$. When $w = 0$, the payoff has no effect on fitness, resulting in neutral selection with a fixation probability of $1/N$. As w increases, the role of the payoff and the time delay becomes more apparent. Notably, the fixation probability initially rises with increasing w before later decreasing. Panel (d) demonstrates the effect of the population size for three time delay values. For sufficiently large populations, the impact of the time delay on the fixation probability is negligible. However, for small populations, the time delay has a considerable effect on the fixation probability.

Figure 6 shows how time delays affect the fixation time in this game. Panel (a) demonstrates the impact of τ for various population sizes. Initially, increasing the time delay at a constant population size causes the fixation time to rise. However, after a critical τ value, the time delay starts to reduce fixation time until it levels off at a fixed value for very large τ . As discussed previously, a sufficiently long time delay produces a Moran Bd process with fixed fitness derived from the initial state with one mutant. Therefore, for very large τ , the fixation time can be calculated using equation (2.8) when the ratio of $r = f_i/g_i$ in (2.8) is $b(N-1)/c + (N-2)0.1$.

The influence of the time delay on the fixation time in a population with constant size for different parameters of the payoff matrix is illustrated in panel (b) of figure 6. As the time delay increases, the fixation time rises to a maximum before decreasing. Ultimately, all fixation times converge to a constant value. Panels (c) and (d) show how the intensity of selection w

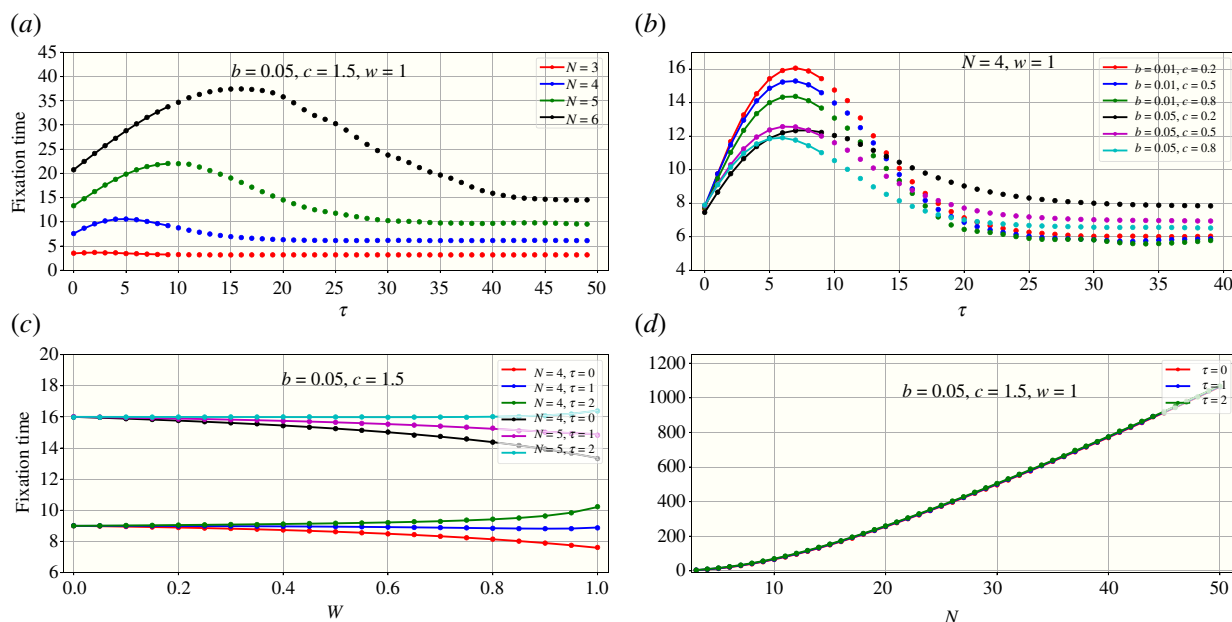


Figure 6. The effect of time delays on fixation time in the Stag-Hunt game ($\alpha = 2.1$ and $d = 0.1$) according to numerical solutions (lines) and computer simulations (dots). Panel (a) shows the influence of the time delays for various network sizes with $b = 0.05$, $c = 1.5$ and $w = 1$. Panel (b) illustrates the effect of time delays for different payoff matrix parameters with $N = 4$ and $w = 1$. Panel (c) depicts how selection intensity affects outcomes for $N = 4, 5$ and $\tau = 0, 1, 2$ with $b = 0.05$, $c = 1.5$. Lastly, panel (d) demonstrates the impact of network size for $\tau = 0, 1, 2$ with $b = 0.05$, $c = 1.5$ and $w = 1$.

and population size impact the fixation time for certain time delays. As the game becomes more influential in determining fitness and selection intensity increases, the fixation time varies with the time delay. Additionally, for a fixed time delay, increasing the population size leads to greater fixation times, with network size having a larger effect than the time delay.

(ii) Snowdrift game

Generally, a Snowdrift game is characterized by a payoff matrix for which $c > a > b > d$ [6,71]. This setting of the payoff matrix promotes cooperation (playing strategy A) by an individual when they encounter a defecting opponent (playing strategy B). The game describes two people who are stuck in a snowdrift and both want to get out of it but are not eager to share the cost of clearing the snow. To have a Snowdrift game, given that a is fixed at 2.1 and d at 0.1, the value of c must be greater than a (i.e. $c > 2.1$), while b should fall within the range (0.1, 2.1).

The time delay impacts the fixation probability in the Snowdrift game, as shown in figure 7. The top panels illustrate how increasing τ affects the fixation probability for various payoff parameters and population sizes. Unlike the Stag-Hunt game, a longer time delay boosts fixation probability in the Snowdrift game. Notably in panel (a), a small τ leads to a lower value in larger populations, but as τ grows, larger populations exhibit a greater value. In both panels, fixation probabilities plateau at constant values dependent on population size and game parameters when τ is sufficiently high. For large time delays, the fixation probability, like the Stag-Hunt game, approaches a constant value. This value corresponds to the fixation probability in a scenario where fitness is determined by the initial condition, with a single mutant in a population of residents. According to equation (5.1), when $0.1 < b < 2.1$ and $2.1 < c$, for large enough τ , $\Phi_{1,N}$ increases as N increases, as shown in panel (a). In panel (b), we see that for $N = 4$, the curves exhibit similar behaviour for a large enough delay.

Panel (c) illustrates how the population size impacts the fixation probability across three values of the time delay. Initially, increasing the population size reduces the probability of fixation regardless of the time delay. However, for sufficiently large populations, further

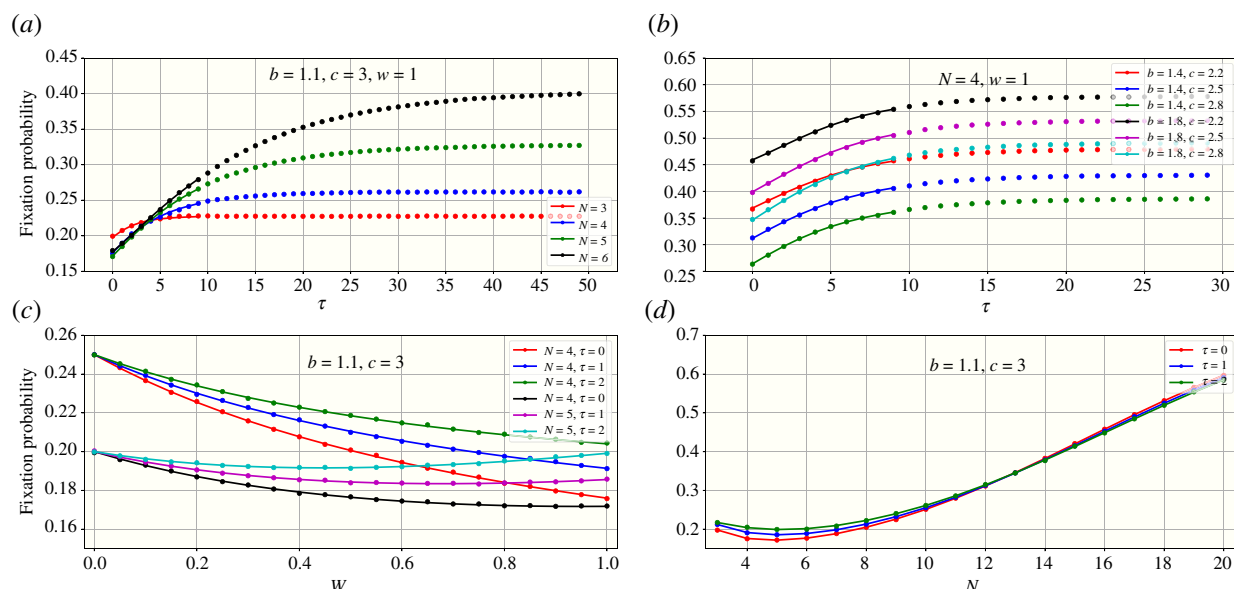


Figure 7. The effect of time delays on the fixation probabilities in the Snowdrift game ($\alpha = 2.1$ and $d = 0.1$) according to numerical solutions (lines) and computer simulations (dots). Panel (a) shows the influence of time delays for various network sizes with $b = 1.1, c = 3$ and $w = 1$. Panel (b) illustrates the effect of time delays for different payoff matrix parameters with $N = 4$ and $w = 1$. Panel (c) depicts how selection intensity affects outcomes for $N = 4, 5$ and $\tau = 0, 1, 2$ with $b = 1.1, c = 3$. Lastly, panel (d) demonstrates the impact of network size for $\tau = 0, 1, 2$ with $b = 1.1, c = 3$ and $w = 1$.

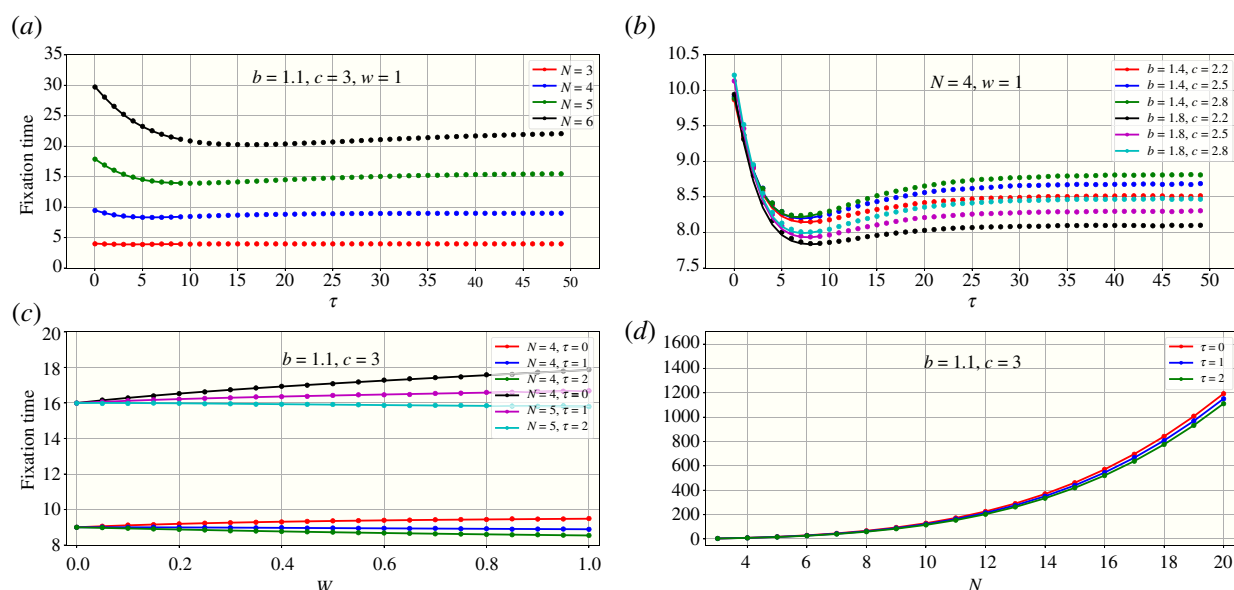


Figure 8. The effect of time delays on fixation time in the Snowdrift game ($\alpha = 2.1$ and $d = 0.1$) according to numerical solutions (lines) and computer simulations (dots). Panel (a) shows the influence of a time delay for various network sizes with $b = 1.1, c = 3$ and $w = 1$. Panel (b) illustrates the effect of a time delay for different payoff matrix parameters with $N = 4$ and $w = 1$. Panel (c) depicts how selection intensity affects outcomes for $N = 4, 5$ and $\tau = 0, 1, 2$ with $b = 1.1, c = 3$. Lastly, panel (d) demonstrates the impact of network size for $\tau = 0, 1, 2$ with $b = 1.1, c = 3$ and $w = 1$.

increases in population size increase the probability of fixation. We observe differing patterns at small versus big population sizes. In small populations, longer time delays correspond to higher fixation probabilities for mutants. Conversely, in large populations, longer delays diminish the fixation probability.

In the Snowdrift game, the impact of time delays on the fixation time is the reverse of what we saw in the Stag-Hunt game, as shown in figure 8. With a constant population size

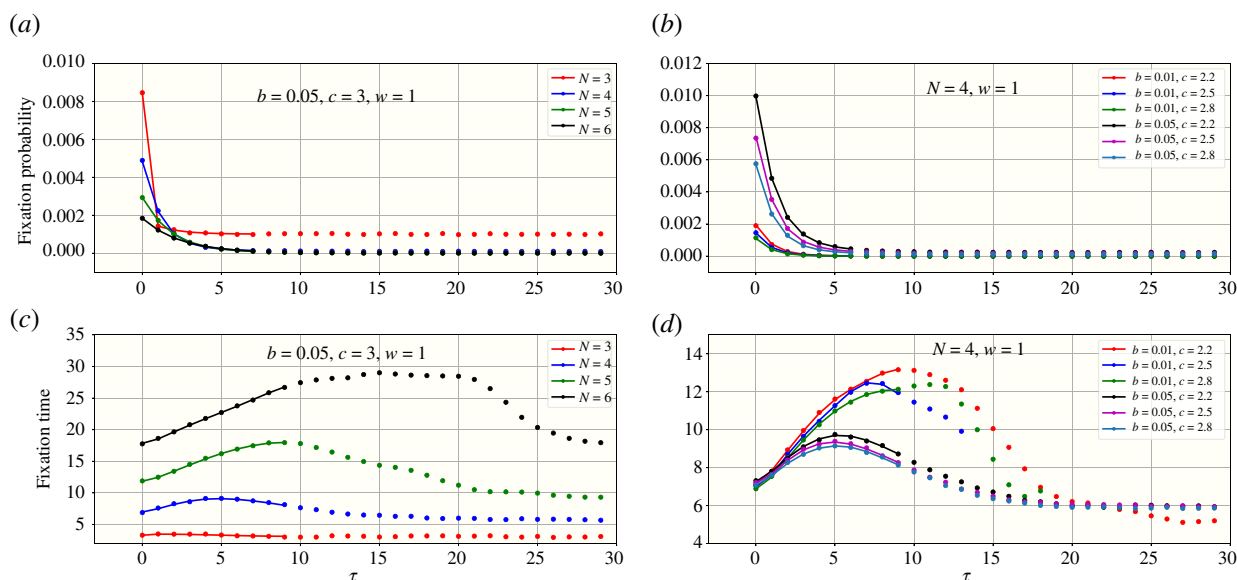


Figure 9. The effect of the time delay on the fixation probability and time in the Prisoner's Dilemma game ($\alpha = 2.1$ and $d = 0.1$) according to numerical solutions (lines) and computer simulations (dots) where the initial mutant is a cooperator. Panels (a) and (b) show the effect of time delays on the fixation probability and panels (c) and (d) show the effect of time delays on the fixation time for different network sizes and payoff values.

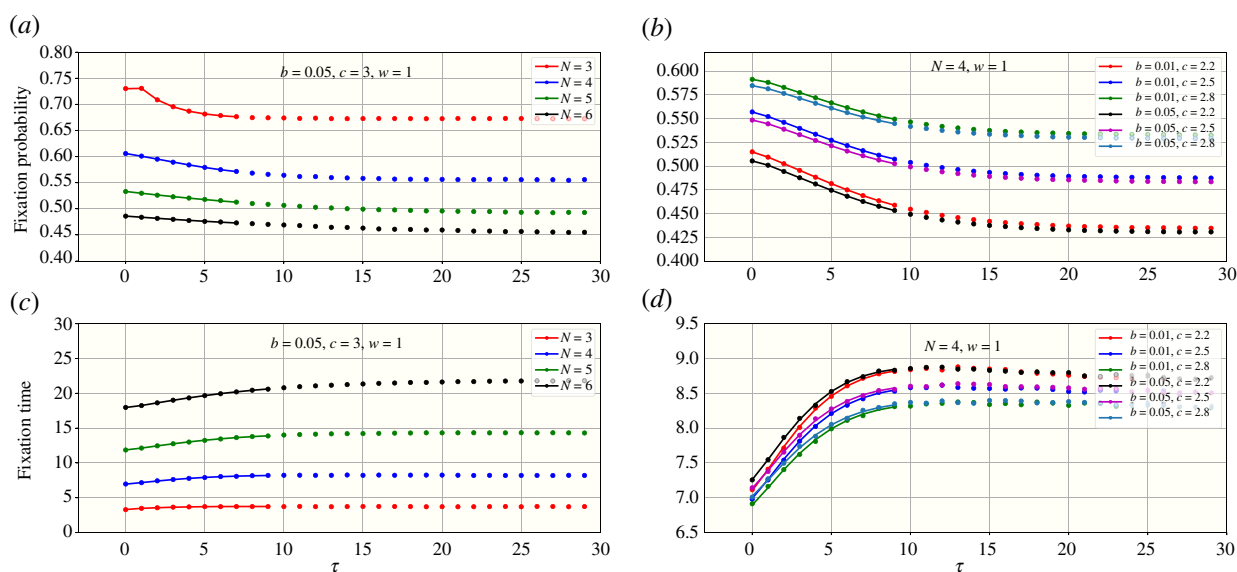


Figure 10. The effect of time delays on the fixation probability and time in the Prisoner's Dilemma game ($\alpha = 2.1$ and $d = 0.1$) according to numerical solutions (lines) and computer simulations (dots) where the initial mutant is a defector. Panels (a) and (b) show the effect of time delays on the fixation probability and panels (c) and (d) show the effect of time delays on fixation time for different network sizes and payoff values.

and payoff matrix values, increasing the time delay first lowers the fixation time, which then rises as the delay continues increasing. As before, for a large enough delay τ , the fixation time converges to a constant value that is less than the fixation time when there is no delay.

(iii) Prisoner's Dilemma game

Another interesting case is the Prisoner's Dilemma. This game is characterized by the payoff relationship $c > a > d > b$ [7,8]. In such a setting, the defect strategy D is a dominant strategy, so that for a rational player it is always optimal to play strategy D irrespective of the other player's choice. The standard Prisoner's Dilemma offers players the option of defecting or

cooperating; for mutual cooperation, two interacting players are offered a reward, $a = R$, and for mutual defection, $d = P$. In this scenario, if one player cooperates while the other defects, then the cooperator would receive the sucker's payoff $b = S$, and the defector would receive the temptation-to-defect payoff $c = T$. In this game, the value of c should be greater than $a = 2.1$, while b should be less than $d = 0.1$.

We examine two scenarios in this game. Figures 9 and 10 depict how the fixation probability and time vary with changes in the time delay, for different population sizes and payoff matrices where the initial mutant is cooperator and defector, respectively. As expected, the fixation probability is much higher when the initial mutant is a defector rather than a cooperator under the same conditions since defectors, on average, obtain higher payoffs than cooperators when playing against both cooperators and defectors. In both scenarios, increasing the time delay leads to small changes in the fixation probability and time. However, larger time delays tend to decrease the fixation probability. We also observe an increase followed by a decrease in the fixation time as the time delay grows. This observation is clearer in the case where the initial mutant is a cooperator. For sufficiently large time delays, where the fitness depends only on the initial state, the constant fitness values ($r = f_1/g_1$) are $b(N-1)/(c+(N-2)d)$ for an initial mutant with the cooperator strategy and $c(N-1)/(b+(N-2)a)$ for an initial mutant with the defector strategy (when $w = 1$).

(b) Comparison between fixation probabilities

In the preceding section, we observed that different games exhibit different patterns when incorporating a time delay into our model. To gain a more comprehensive understanding, we now compare all games by examining how changes in two parameters of the payoff matrix affect patterns influenced by a time delay. We set $a = 2.1$ and $d = 1.1$, while varying c from 1.1 to 3.1 and b from 0.1 to 2.1. Our analysis focuses solely on the fixation probability, as it holds greater significance than the time in the literature. Figures 11 and 12 illustrate the difference between fixation probability with time delay τ and fixation probability without a time delay for mutants with strategies A and B, respectively. Negative values indicate a decrease in fixation probability, while positive values signify an increase.

Our figures reveal that different parameter ranges give rise to distinct games. Among these, we observe a new game, referred to as the Harmony (H) game in the literature, which is less well known and studied compared with other games. Across the Stag-Hunt (SH) game, we observe a consistent inverse relationship between time delays and the fixation probability regardless of the strategy of the initial mutant. In the Snowdrift (SD) game, the fixation probability also decreases with a delay in both figures, suggesting that the effect of time delays is consistent regardless of the initial mutant's strategy. In the Prisoner's Dilemma (PD) game, the relationship between the time delay and the fixation probability is contingent on the game's parameters. While there are scenarios where increased time delay leads to a higher fixation probability, a broad set of parameters results in a decreased fixation probability, irrespective of the initial mutant's strategy. Notably, as we vary a single parameter while keeping others fixed (for instance, changing b from a value below 1.1, corresponding to the Prisoner's Dilemma, to a higher value associated with the Snowdrift game for $a > 2.1$), we observe a shift in behaviour. This is reflected in the change of fixation probability differences from negative to positive values, highlighting the profound impact of parameter variation on game dynamics. Introducing time delays and considering payoffs from past interactions implies that both strategies receive payoffs by engaging with a higher proportion of resident strategies. Consequently, this scenario proves unfavourable for mutant strategies in Stag-Hunt games, while being advantageous in the Snowdrift game, regardless of the mutant's strategy. This leads to a decrease in fixation probability for the Stag-Hunt game and an increase for Snowdrift game. For the Prisoner's Dilemma game, it is dependent on the value of the payoff matrix.

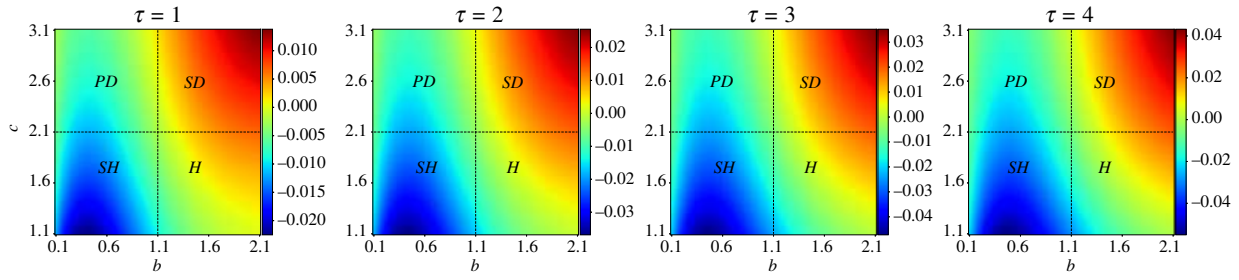


Figure 11. Fixation probability difference between delayed (τ) and without a delay for an initial mutant with strategy A. Fixed parameters: $\alpha = 2.1$, $d = 1.1$. Varying other parameters yields different game types: Prisoner's Dilemma (PD), Snowdrift (SD), Stag-Hunt (SH) and Harmony (H). The time delay is 0,1,2,3 from left to the right.

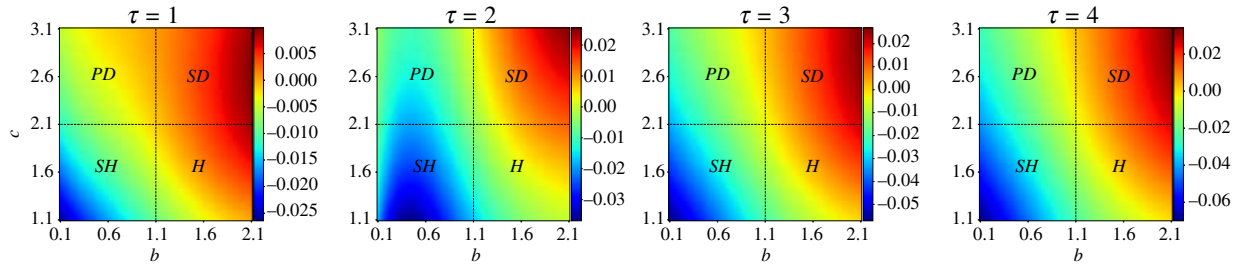


Figure 12. Fixation probability difference between delayed (τ) and without a delay for an initial mutant with strategy B. Fixed parameters: $\alpha = 2.1$, $d = 1.1$. Varying other parameters yields different game types: Prisoner's Dilemma (PD), Snowdrift (SD), Stag-Hunt (SH) and Harmony (H). The time delay is 0,1,2,3 from left to the right.

(c) Conditional sojourn time

The impact of time delays on the conditional sojourn time will be examined in this section. Conditional sojourn time refers to the duration a process spends in a particular state before fixation. The conditional sojourn time j starting from state i refers to the number of times a process is in a transition state with j mutants before fixation occurs [49,68]. Understanding conditional sojourn times is crucial for examining how traits or genotypes evolve within populations over time. When we introduce a time delay into models, we can analyse how the sojourn times in each state before fixation change. Specifically, the fixation time starts from one mutant which is the sum of the sojourn times across all states before fixation occurs. Therefore, by studying how time delays affect sojourn times, we can better comprehend their impact on overall fixation times.

For a process without time delays, when the process starts with i mutants, the conditional sojourn time in the state with j is $(\Phi_{j,N}/\Phi_{i,N})F_{ij}$. With time delays, each state with $i_0 = j$ mutants may have different historical components. To find the conditional sojourn time associated with a state having j mutants, we sum over all s_m where $i_0 = j$. Using the fundamental matrix F^τ , the conditional sojourn time for a state with $i_0 = j$ mutants starting from state s_m is defined as:

$$\text{Conditional sojourn time (j)} = \sum_{s_l = \{i_\tau, \dots, i_0\}, i_0 = j} \frac{\Phi_{s_l, N}^\tau}{\Phi_{s_m, N}^\tau} F_{s_m, s_l}^\tau. \quad (5.2)$$

Figures 13 and 14 show the mean conditional sojourn time for the Stag-Hunt and Snowdrift games, respectively, in a population of size 5. For the Stag-Hunt game, we set the payoff matrix values to $(a, b, c, d) = (2.1, 0.05, 1.5, 0.1)$. With no time delay, the smallest conditional sojourn time is when there are two mutants in the population. As the time delay increases to 10, which corresponds to the largest fixation time as seen in the right panel, the conditional sojourn time for two mutants increases. On average, the conditional sojourn time at $\tau = 12$ is

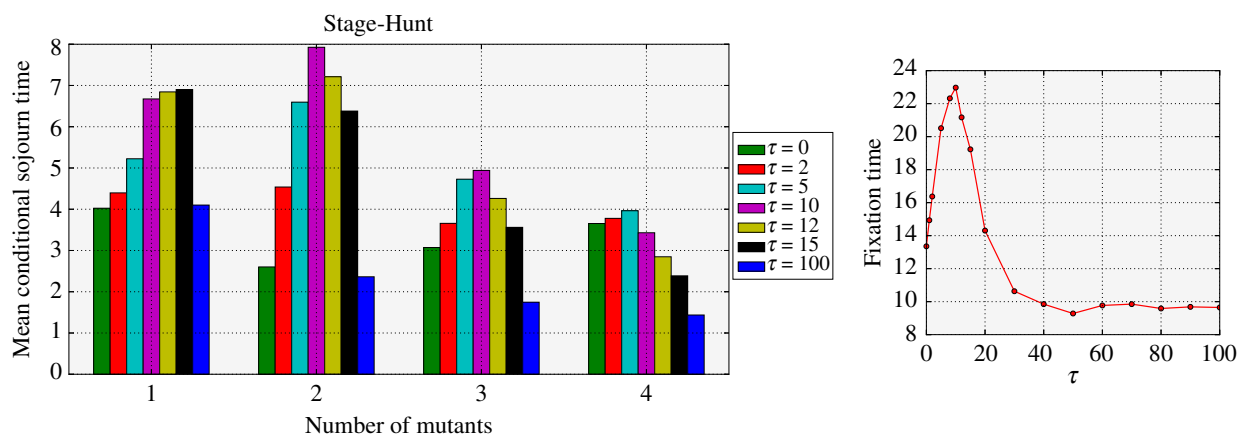


Figure 13. Conditional sojourn times for the Stag-Hunt game in a population of size 5 for different values of the time delay. The right panel shows the fixation time as the delay increases. We use $(a, b, c, d) = (2.1, 0.05, 1.5, 0.1)$ as the payoff matrix.

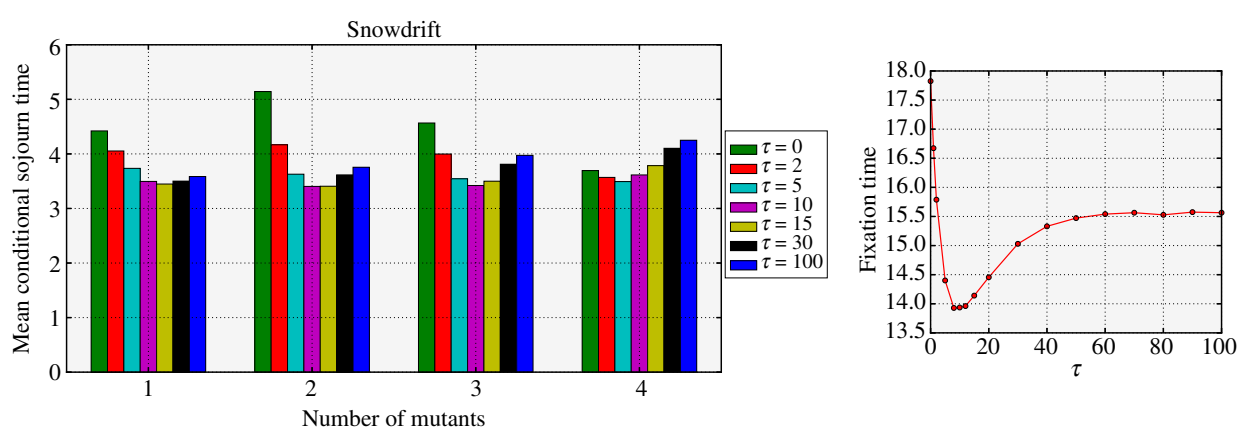


Figure 14. Conditional sojourn time for the Snowdrift game in a population of size 5 for different values of the time delay. The right panel shows the fixation time as the delay increases. We use $(a, b, c, d) = (2.1, 1.1, 3.0, 0.1)$ as the payoff matrix.

higher than for other time delays, matching the highest fixation time. This suggests $\tau = 10$ is a critical value where the process spends more time in transition states for $N = 5$. For a sufficiently large delay (here $\tau = 100$), where the fixation time converges to a constant, the conditional sojourn time decreases as the number of mutants increases, so that the process spends a small fraction of time in states with more mutants. Compared with the case without time delays, $\tau = 100$ has a much smaller conditional sojourn time in states with four mutants. In other states, the conditional sojourn times are almost the same for $\tau = 0$ and $\tau = 100$. This leads to a lower fixation time for $\tau = 100$ compared with $\tau = 0$.

In the Snowdrift game, the results are the reverse of those for the Stag-Hunt. Using the payoff matrix $(a, b, c, d) = (2.1, 1.1, 3.0, 0.1)$, the largest conditional sojourn time on average occurs when there are two mutants. This time decreases as the time delay increases from 0 until the critical delay of 10 and then increases again. On average, $\tau = 10$ has the smallest conditional sojourn time in all states, matching the lowest fixation time. In contrast to the Stag-Hunt game, for a sufficiently large delay, the conditional sojourn time increases as the number of mutants increases.

6. Discussion

Understanding fixation processes is vital for analysing evolutionary games in finite populations. Two important metrics—fixation probability and time—have received significant research attention across various evolutionary game models [28–37]. Most previous models frequently

assume that fitness instantaneously adjusts according to the current population state and payoffs [28–51]. This assumption of immediate fitness determination may not reflect reality and most of real phenomena exhibit temporal delays. Therefore we have investigated the intricate influence of time delays on mutant fixation in evolutionary games.

Previously, researchers have studied the effect of time delays in deterministic replicator equation models [54–64]. They have been incorporated in two different ways: first, by considering only the payoffs at time t coming from time $t - \tau$ in the past [54,55]; and second, by allowing individuals born in the past to replicate now based on past payoffs [56]. These studies have focused on how time delays impact the existence and stability of interior stationary states. Under the first approach, sufficiently long delays can generate limit cycle oscillations after a supercritical Hopf bifurcation [54,55]. Under the second approach, strategy-dependent delays can shift the locations of stationary states to disfavour delayed strategies [56]. However, the impact of time delays on fixation dynamics has remained an open question.

Our study extends the investigation of time delay effects from replicator dynamics to the Moran Bd process, which represents a shift in perspective. While both models are used to study evolutionary dynamics, the Moran process operates on finite populations and captures stochastic effects that are particularly relevant in small populations, which are common in real-world scenarios. This individual-based approach allows us to examine phenomena such as fixation probabilities and times, which are not directly accessible in the deterministic replicator dynamics framework [28–32]. The Moran process has widespread applications, particularly in biology, where understanding the fate of mutations (such as in cancer) is of paramount importance.

This study provides a more realistic model of how historical interactions influence current fitness and strategy adoption in social dilemmas. By examining how lags affect the spread and fixation of strategies in populations, the research offers deeper insights into the emergence and stabilization of cooperative and competitive behaviours. This approach not only enriches the theoretical framework of evolutionary dynamics but also has potential applications across various fields, from biology to economics, where time-dependent interactions significantly impact outcomes.

Here we employed a well-mixed population model where individuals adhere to one of two strategies, with pay-offs contingent upon their strategic choice and the prevalence of strategies [16]. Initially, a solitary mutant individual with strategy A emerges amidst a population of residents employing strategy B. At each discrete time step, an individual reproduces in proportion to its pay-off, randomly replacing another individual [15,16]. In the absence of a time delay, reproductive fitness is determined solely by the number of mutants in the current state [15,16,29,42,65,66]. However, when a time delay τ is introduced, reproductive fitness becomes contingent upon the population state τ steps back in time.

Generally, both strategies' fitness depends on the frequencies of the strategies. When a mutant has relatively higher fitness in the population, it has a greater chance of becoming fixed. When we add a time delay to the model, it changes the relative fitness of mutants and residents because looking at past strategy frequencies can raise or lower fitness based on the payoff matrix. In the Stag-Hunt game, a mutant adopting strategy A experiences a greater reward when collaborating with fellow A mutants compared with interacting with B residents [9]. On the contrary, residents sticking to strategy B receive the same payoff regardless of whether they play with A or B strategies. As the A mutant population expands, they reap enhanced benefits by coordinating with each other, boosting their payoff (and fitness), consequently favouring mutant fixation. However, introducing a time delay disrupts this advantage by basing mutant reproduction on a past state where A mutants were less prevalent. This lagged payoff is lower than the real-time payoff, hindering A mutants' ability to reproduce and thrive. Notably, this time delay does not affect B residents' payoff. As a result, the effective payoff for A mutants diminishes, leading to a reduced fixation probability.

In contrast to the Stag-Hunt game, in the Snowdrift game, both A mutants and B residents attain higher payoffs from coordinating with A mutants [6,71]. As the time delay increases, both strategies interact more with B residents, reducing their payoffs. However, A mutants maintain a superior effective payoff compared with B residents, enlarging the fitness ratio (f_i/g_i) beneficial to mutants. Thus, unlike the Stag-Hunt, lengthening the time delay in the Snowdrift game enhances A mutants' fixation prospects. In the Prisoner's Dilemma game [7,8], the cooperator and defector strategies obtain higher payoffs when playing with a cooperator. When the initial mutant is a cooperator, increasing the time delay leads to a decrease in the effective payoff of both strategies for reproduction. However, the effective payoff for the cooperator from the past is smaller than for the defector, so the fixation probability decreases. When the initial mutant is a defector, although both strategies get more payoff when fitness comes from the past due to playing with more cooperators, the ratio of f_i/g_i can be smaller or bigger based upon the elements of the payoff matrix. Thus, the time delay can decrease or increase the fixation probability according to how much better it is to play with more cooperators for mutants and residents.

In general, introducing a time delay alters the effective payoffs that both mutant and resident obtain for reproduction, impacting the fixation probability. A time delay tends to reduce the fixation probability if it reduces the relative payoff advantage of the mutant over the resident (f_i/g_i). It tends to increase the fixation probability if it enhances this comparative advantage. The key factor is the relative change in payoffs for mutant versus resident resulting from the delay. In summary, there are three scenarios where the time delay reduces the fixation probability:

- If it decreases the mutant's payoff but increases the resident's payoff
- If it decreases both payoffs but the resident's proportionately less than the mutant's
- If it increases both payoffs but the resident's proportionately more than the mutant's

Conversely, there are scenarios where the time delay increases the fixation probability:

- If it increases the mutant's payoff but decreases the resident's payoff
- If it increases both payoffs but the resident's proportionately less than the mutant's
- If it decreases both payoffs but the resident's proportionately more than the mutant's

We also have examined the joint effect of a time delay and population size on fixation probability in the Stag-Hunt and Snowdrift games. Previous work showed that population size can increase fixation probability in some games [36]. Here, in Stag-Hunt games, increased population size decreases fixation probability regardless of time delays, although time delays affect smaller populations more. By contrast, in Snowdrift games, increased population size increases fixation probability. However, time delays affect small and large populations differently: bigger delays help mutant fixation in small populations, while smaller delays help in large populations.

Fixation time has been less studied in past literature. This measurement can vary substantially depending on the model used and the structure of the population [34,49,67]. In some cases, fascinating behavioural patterns may emerge related to fixation time. The effect of time delays on fixation times varies across the three studied games. In the Stag-Hunt game, as time delays initially increase, fixation times grow longer, peaking and then decreasing after a critical time delay point. Analysing the conditional sojourn time (the duration the process stays in each state before fixation), we see the process lingers mostly in states with few or many mutants in this game. However, the process spends more time in intermediate states as delays rise until reaching a peak, then declining. By contrast, the Snowdrift game demonstrates the opposite pattern: fixation times decrease and then increase again after a critical delay point. Here, conditional sojourn times in intermediate states dramatically fall with longer delays until the minimum fixation time is reached. Meanwhile, time delays have little influence on fixation

times in the Prisoner's Dilemma game, although there is a slight increase then a decrease similar to the Stag-Hunt game.

Our findings suggest that incorporating time delays into evolutionary game dynamics introduces novel and potentially significant effects. While our study focused on scenarios with uniform time delays for both mutant and resident strategies, further investigations are needed to explore cases where the time delay is strategy-dependent. Additionally, our exploration of the Bd update rule, where birth is influenced by fitness and a time delay, highlights the potential of alternative update rules, such as birth–Death, death–Birth, Death–birth, and imitation, to unveil distinct time delay effects. Moreover, our analysis of well-mixed populations without structure provides a foundation for future studies in structured populations, including line graphs, star graphs and more complex graphs such as scale-free and random graphs. Examining strategy-dependent time delays across structured populations represents a promising direction for future research on time delays in evolutionary games.

Data accessibility. This article has no additional data.

Declaration of AI use. We have not used AI-assisted technologies in creating this article.

Authors' contributions. J.M.: conceptualization, formal analysis, investigation, visualization, writing—original draft, writing—review and editing; M.B.: conceptualization, formal analysis, funding acquisition, supervision, writing—review and editing.

Both authors gave final approval for publication and agreed to be held accountable for the work performed therein.

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Phase transitions in the prisoner's dilemma game on the Barabási-Albert graph with participation cost

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We examine the impact of the maintenance cost of social links on cooperative behavior in the prisoner's dilemma game on the Barabási-Albert scale-free network with a pairwise stochastic imitation. We show by means of Monte Carlo simulations and pair approximation that the cooperation frequency changes abruptly from an almost full cooperation to a much smaller value when we increase the cost of maintaining links. In the critical region, the stationary distribution is bimodal and the system oscillates between two states: the state with almost full cooperation and one with coexisting strategies. We show that the critical region shrinks with the increasing size of the population. However, the expected time the system spends in a metastable state before switching to the other one does not change as a function of the system's size, which precludes the existence of two stationary states in the thermodynamic limit of the infinite population.

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Introduction. One of the fundamental problems of evolutionary biology is understanding the origins of the prevalence of altruistic behavior in the world governed by the Darwinian rule of survival of the fittest [1]. One can discuss it within the framework of evolutionary game theory which describes the behavior of systems of many interacting individuals [2]. In evolutionary games, players have at their disposal various strategies, and receive payoffs which can be interpreted as the number of offspring who inherit their strategies. The evolution of such populations can be modeled by deterministic replicator dynamics or stochastic processes of games on graphs which describe the time changes of fractions of populations playing given strategies.

The famous prisoner's dilemma game provides us with an archetypal case of the social dilemma where individual rationality is at odds with the well being of the population [3–5]. In such a two-player game with two strategies, cooperation and defection, a player is tempted to defect, and hence both players defect but they would be better off if they both cooperated. We say that defection is a dominant strategy (it gives the highest payoff regardless of the opponent's strategy). As a result, full defection is the globally asymptotically stable state of the replicator dynamics of the prisoner's dilemma game. However, it was shown that in games on graphs (where the payoff of any player is a sum of payoffs resulting from games with its neighbors), cooperative players may form clusters which are resilient against defectors and this leads to the coexistence of both strategies in the stationary state [6,7]. In particular, a scale-free Barabási-Albert random graph [8,9], built by a preferential attachment rule, enhances cooperation. In Ref. [10] it was shown that for a certain region of game parameters, stochastic imitation of a better strategy leads to the

stationary state of the population with almost all cooperators. An important role of maintaining cooperation is played here by hubs (vertices with high degrees).

It was shown independently in Refs. [11,12] (the expanded version of Ref. [11] was later published in Ref. [13]) that when we introduce the cost of maintaining a link between neighbors, the level of cooperation in the stationary state decreases. Let us notice that in the above models and in the current one, the network is fixed, and we do not allow the deletion or creation of links. Here, we construct a stochastic process (an ergodic Markov chain) on the Barabási-Albert graph where agents follow the Boltzmann updating (called a Fermi rule in the game literature)—they imitate their neighbors with a probability proportional to the exponent of a payoff difference divided by the noise factor, the “temperature” of the system. We perform stochastic Monte Carlo simulations and show that the cooperation level abruptly changes from an almost full cooperation to a much smaller value when we increase the cost of maintaining links. We examine closely what happens around a critical value of the cost. The unique stationary probability distribution of our Markov chain is bimodal in a small interval around the critical cost (the bigger the size of the population, the smaller is the interval)—the population oscillates between peaks of the distribution.

The situation is analogous to the behavior of the ferromagnetic Ising model on a square lattice at zero magnetic field and low temperatures, where in the finite volume the system alternates between almost spin-up and spin-down configurations.

However, the situation is different in our case. We inferred from simulations that the expected time the system spends in a metastable state before switching to the other one does not change as a function of the system's size. This precludes the existence of two stationary states in the thermodynamic limit of the infinite population, as is the case in the Ising model.

We construct a pair approximation, a system of differential equations for the frequency of cooperators and the

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frequency of pairs of cooperators. We solve numerically our equations and show that the convergence of solutions changes abruptly at the same critical cost as is present in stochastic simulations.

Prisoner's dilemma on the Barabási-Albert graph. We are concerned here with the two-player prisoner's dilemma game with two strategies, cooperation (C) and defection (D), with a canonical parametrization and in addition a cost γ of maintaining a link paid by both connected players [11–13]. Our payoff matrix reads as follows,

$$U = \begin{array}{cc} & \begin{array}{cc} C & D \end{array} \\ \begin{array}{c} C \\ D \end{array} & \begin{array}{cc} 1 - \gamma & -\gamma \\ T - \gamma & -\gamma \end{array} \end{array}$$

where the entry U_{xy} is the payoff of the row player using the x strategy while the column player uses the y one.

We set $T > 1$ hence D is a dominant strategy—it gives the highest payoff regardless of the strategy of the opponent—hence a pair (D, D) is the unique Nash equilibrium of the game and the population consisting of just defectors is the globally asymptotically stable state of the replicator dynamics [14–16].

Here, we put players on vertices of the Barabási-Albert graph. It is built by the preferential attachment procedure. We start with m_o fully connected vertices and then we add $N - m_o$ vertices, each time connecting them with m already available vertices with probabilities proportional to their degrees. If $m_o = \alpha + 1$ and $m = \alpha/2$, then we get a graph with an average degree equal to α . It is known that such a graph in the limit of an infinite number of vertices, N , is scale free with the probability distribution of degrees given by $p(k) \sim k^{-3}$ [8,9].

Now we present a stochastic dynamics of a spatial game. At discrete moments of time, all individuals interact with their neighbors and receive payoffs which are sums with respect to individual games. Then the imitation process takes place. A randomly chosen player who has a strategy x and a total payoff π_x chooses randomly one of its neighbors. Assume that a chosen neighbor has a strategy y and a total payoff π_y . The first individual then with the probability $1 - \epsilon$ imitates the strategy of the second individual with the probability $w(x \rightarrow y)$, given by the Boltzmann expression

$$w(x \rightarrow y) = \frac{e^{\beta\pi_y}}{e^{\beta\pi_x} + e^{\beta\pi_y}}, \quad (1)$$

and with the probability ϵ adopts the other one.

Let us notice that the Barabási-Albert network is heterogeneous and hence the cost of maintaining links for any player depends on the number of its neighbors. Therefore the unit cost γ does not cancel in the difference of payoffs in the update rule as it would be the case for regular networks, the square lattice for example.

The parameter β represents a bias toward higher payoffs, where better-performing individuals are more likely to be imitated. It also quantifies a noise in the update process, in the limit $\beta \rightarrow 0$, where individuals are making random updates, and in the limit $\beta \rightarrow \infty$, the process becomes deterministic imitation. We set $\epsilon = 0.001$, $\beta = 100$, and the average vertex

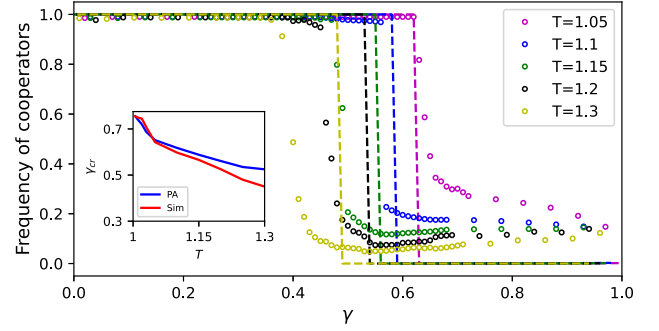


FIG. 1. Frequency of cooperators for various values of T as a function of γ —the cost of maintaining a link. Dots show the results of simulations and dashed lines indicate stationary states of pair approximation in Eqs. (7) and (8). In the inset, the critical γ_{cr} is shown as a function of T .

degree, $\alpha = \langle k \rangle = 4$. For all results, the size of population is 10 000, except where it is varied and explicitly stated.

In this way we constructed a Markov chain with 2^N states (assignments of strategies to graph vertices) and the above described transition probabilities. Our Markov chain is irreducible (one can get from any state to any other state in a finite number of steps) and aperiodic which follows from the fact that there is a nonzero probability to stay at any state, and therefore it is ergodic and hence it possesses a unique stationary probability distribution.

Phase transition—Monte Carlo simulations. We performed stochastic Monte Carlo simulations to estimate the frequency of cooperation in the stationary state. As an initial condition we assign randomly, that is, with a probability 1/2, cooperators and defectors to N vertices. To arrive reasonably close to the stationary state, we run simulations for 10^5 Monte Carlo rounds (each round consists of N steps so each vertex can be on average updated), followed by 10^3 rounds to compute an average of the cooperation level. We performed 10^3 runs per every parameter set, averaging over different random initial conditions and network realizations.

Figure 1 presents the cooperation level in the stationary state as a function of the link cost for various T values. A sharp transition occurs on an interval $[\gamma_l(T, N), \gamma_r(T, N)]$ where the system shifts from the full cooperation to the coexistence of cooperation and defection. For low costs we observe a full cooperation which is in agreement with the results in Ref. [10] for the zero cost. Critical γ defined as $\gamma_{cr}(T, N) = [\gamma_l(T, N) + \gamma_r(T, N)]/2$ is a decreasing function of T (see the inset in Fig. 1). We see that the critical value of the cost stabilizes as N increases (see Fig. 2). We conjecture that $\lim_{N \rightarrow \infty} \gamma_l(T, N) = \lim_{N \rightarrow \infty} \gamma_r(T, N) = \gamma_{cr}(T)$.

This may be compared to the behavior of the ferromagnetic Ising model on the Barabási-Albert graph investigated in Refs. [17,18]. It was shown there that the effective critical temperature (below which the system is magnetized) diverges as the logarithm of the size of the graph, hence the system does not exhibit a phase transition at the thermodynamic limit.

Figure 3 shows the time evolution of cooperation for $T = 1.1$ and 1.3 for three γ values: before, during, and after the phase transition. For $\gamma < \gamma_l(T, N)$ cooperation dominates. For $\gamma > \gamma_r(T, N)$ cooperation and defection coexist, while for

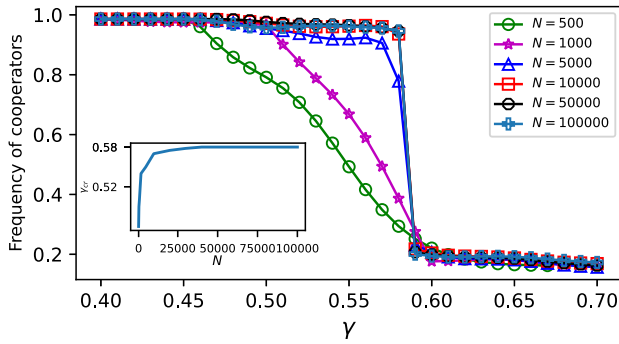


FIG. 2. The effect of γ on the frequency of cooperators for various population sizes, $T = 1.1$. In the inset we present the critical cost as a function of N .

$\gamma \in [\gamma_l(T, N), \gamma_r(T, N)]$, the system oscillates between these states.

In Fig. 4, we present the stationary distribution of the cooperation frequency for $\gamma \in [\gamma_l(T, N), \gamma_r(T, N)]$. It is concentrated respectively on full cooperation and coexistence of cooperators and defectors at the segment ends and it is bimodal in between. This behavior is analogous to that in the two-dimensional ferromagnetic Ising model at zero magnetic field, where in a finite volume we observe switching between two ensembles of mostly up and mostly down spins, that is almost 1 and almost -1 magnetization per lattice site.

These oscillations and the previously discussed stabilization of the critical cost with respect to the size of the system

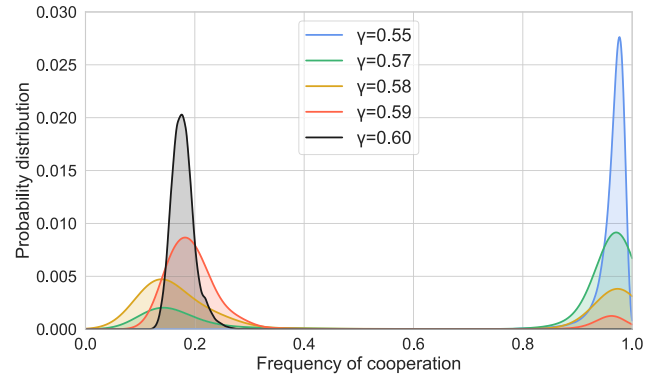


FIG. 4. Histogram of the stationary probability distribution of cooperators frequency for various values of γ and $T = 1.1$.

may suggest that in the thermodynamic limit we could have two stationary states as in the Ising model. However, it is not the case here. We inferred from simulations that the expected time the system spends in a metastable state before switching to the other one does not change as a function of the system's size. This precludes the existence of two stationary states. It is caused by the existence of hubs (vertices with a very high degree) in our model.

Notably, for low γ cooperation initially declines. If a cooperator in a highly connected hub becomes a defector, its high payoff accelerates its strategy spread, triggering a temporary cooperation decline. As defection expands, the payoff advantage fades, enabling cooperators to reclaim dominance.

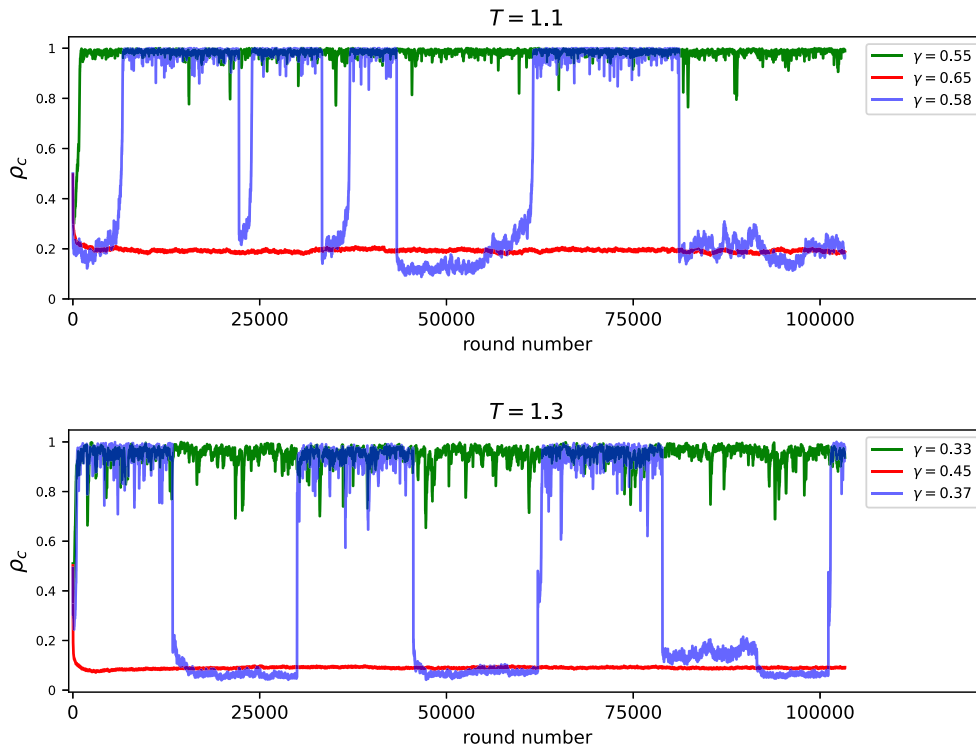


FIG. 3. Frequency of cooperators after each round in exemplary simulations for various values of γ with $T = 1.1$ and 1.3 .

For γ in the above described interval, cooperation exhibits metastability, fluctuating between high and low cooperation states. Hubs play a crucial role, noting that if a hub remains cooperative, cooperation thrives, but with a probability ϵ , it may switch to defection, tipping the balance. This leads to metastable states, where transitions occur as hubs alter their strategies.

Phase transition—pair approximation. In pair approximations, a state of a population of players on graphs is represented by frequencies of strategies and pairs of nearest-neighbor strategies. We denote by ρ_C and ρ_D a frequency of cooperators (C) and defectors (D), respectively and by ρ_{CC} , ρ_{CD} , and ρ_{DD} , frequencies of pairs of players. These variables satisfy the following relations: $\rho_C + \rho_D = 1$, $\rho_{CC} + \rho_{CD} + \rho_{DD} = 1$, and $\rho_C = \rho_{CC} + (\rho_{CD})/2$. Hence the system can be described by just two variables, say ρ_C and ρ_{CC} . Below we derive differential equations for these variables.

Consider a randomly chosen individual with strategy x , having k neighbors. It is connected to k_x individuals with the same strategy with probability $P_{x|x}$, and to $k - k_x$ individuals with the opposite strategy with probability $1 - P_{x|x}$. The probability of such a local configuration is described by a binomial distribution,

$$\Phi(k, k_x) = \binom{k}{k_x} P_{x|x}^{k_x} (1 - P_{x|x})^{k-k_x}. \quad (2)$$

In each imitation step, we first select with probability ρ_x an individual with a strategy x . Next, we choose for imitation a connected individual with the other strategy y with a probability $\frac{k-k_x}{k}$, where k is the number of connections of the first individual. The probability of this specific configuration is given by $\Phi(k, k_x)$.

The payoff of the first focal individual with strategy x is equal to

$$\pi_x(k, k_x) = k_x U_{xx} + (k - k_x) U_{xy}, \quad (3)$$

where we have taken into account that it is connected to the k_x individual with the same strategy with probability given in (2).

To compute the payoff of the second model individual near the focal individual, denoted as π'_y , we account for the fact that the degree distribution of a randomly chosen neighbor follows $k p(k)$ rather than $p(k)$ [19]. When an individual with degree k adopts strategy y and is a neighbor of a focal individual with strategy x , they must have at least one x neighbor, while the remaining $k - 1$ neighbors follow the distribution $\Phi(k - 1, k_y)$. Thus the average payoff of a neighbor of the focal individual is given by the following expressions for $y \in \{D, C\}$,

$$\begin{aligned} \pi'_C &= \sum_{k=1}^N \frac{k p(k)}{\langle k \rangle} \left(\sum_{k_C=0}^{k-1} \Phi(k-1, k_C) [k_C(1-\gamma) - (k-1-k_C)\gamma] - \gamma \right) \\ &= \frac{\langle k^2 \rangle}{\langle k \rangle} (P_{C|C} - \gamma) - P_{C|C}, \\ \pi'_D &= \sum_{k=1}^N \frac{k p(k)}{\langle k \rangle} \left(\sum_{k_D=0}^{k-1} \Phi(k-1, k_D) [k-1-k_D)(T-\gamma) - k_D\gamma] + (T-\gamma) \right) \\ &= \frac{\langle k^2 \rangle}{\langle k \rangle} [T(1 - P_{D|D}) - \gamma] + T P_{D|D}, \end{aligned} \quad (4)$$

where $\langle k^2 \rangle = \sum_{k=1}^N k^2 p(k)$ is the second moment of the degree distribution. Let us note that the first terms in the above expressions are the average degree multiplied by appropriate probabilities. Extra terms are effects of taking into account that a focal and a model player are not using the same strategy.

The first individual, with a payoff of $\pi_x(k, k_x)$, then imitates the strategy of its neighbor, who has an average payoff of π'_y , based on the probability w given in Eq. (1), denoted as $P_{y \rightarrow x}(k, k_x)$. For $x, y \in \{C, D\}$, we have

$$P_{D \rightarrow C}(k, k_D) = \frac{(1 - \epsilon) \rho_D \frac{k-k_D}{k} \Phi(k, k_D)}{1 + e^{\beta[\pi_D(k, k_D) - \pi'_C]}} + \epsilon \rho_D, \quad (5a)$$

$$P_{C \rightarrow D}(k, k_C) = \frac{(1 - \epsilon) \rho_C \frac{k-k_C}{k} \Phi(k, k_C)}{1 + e^{\beta[\pi_C(k, k_C) - \pi'_D]}} + \epsilon \rho_C. \quad (5b)$$

To determine the frequency of CC pairs over time, consider a cooperator with k neighbors, k_C of whom are cooperators. If replaced by a defector (probability $P_{C \rightarrow D}$), CC pairs decrease by k_C . If a defector with k_D defector neighbors switches to cooperation ($P_{D \rightarrow C}$), CC pairs increase by $k - k_D$. These changes occur with the following rates:

$$P_{CD \rightarrow CC}(k, k_D) = P_{D \rightarrow C}(k, k_D)(k - k_D), \quad (6a)$$

$$P_{CC \rightarrow CD}(k, k_C) = P_{C \rightarrow D}(k, k_C)(k_C). \quad (6b)$$

Now we can write differential equations for the frequency of cooperators ρ_C and pairs of cooperators ρ_{CC} ,

$$\frac{d\rho_C}{dt} = \sum_{k=1}^{\infty} p(k) \left(\sum_{k_D=0}^k P_{D \rightarrow C}(k, k_D) - \sum_{k_C=0}^k P_{C \rightarrow D}(k, k_C) \right), \quad (7)$$

$$\frac{d\rho_{CC}}{dt} = \sum_{k=1}^{\infty} p(k) \left(\sum_{k_D=0}^k P_{CD \rightarrow CC}(k, k_D) - \sum_{k_C=0}^k P_{CC \rightarrow CD}(k, k_C) \right). \quad (8)$$

We would like to see how good the pair approximation is with respect to Monte Carlo simulations. To compare it with the behavior of a system with N players we constrain summations in (7) and (8) by the maximal degree and we use $p(k)$ obtained in simulations. We set initial condition as $\rho_C = 0.5$ and $\rho_{CC} = 0.25$ (based on random distribution of strategies) and solve the system of differential equations numerically (we used the fourth-order Runge-Kutta method) to find approximate stationary states, when both ρ_C and ρ_{CC} almost do not change.

Results are presented in Fig. 1. We observe a sharp phase transition in the cooperation level.

We define the critical value of γ predicted by the pair approximation as the average of the two γ values—one corresponding to almost full cooperation and the other to almost total defection—measured just before and after the phase transition. For example, when $T = 1.1$, the system exhibits full cooperation at $\gamma = 0.5901$, but a slight increase to $\gamma = 0.5902$ leads to almost total defection. Therefore, we define the critical value as $\gamma_{cr} = 0.59015$, which is in good agreement with the simulation $\gamma_{cr} = 0.58$.

We see that our pair approximation works better for small T which is consistent with previous findings. Additionally, the pair approximation performs better for smaller γ 's.

Discussion. We analyzed the stochastic imitation dynamics of the prisoner's dilemma game on the Barabási-Albert graph with a participation cost introduced in Refs. [11–13]. We showed by means of Monte Carlo simulations the existence of a critical cost region with an abrupt decrease of the cooperation level in the stationary state. In this region, the stationary distribution is bimodal and the system oscillates between two states: the state with almost full cooperation and the state with coexisting strategies. We show that the critical region shrinks with the increasing size of the population.

We designed a pair approximation—the system of two differential equations for the frequency of cooperators and nearest-neighbor pair of cooperators. The stationary state of such a system shows an abrupt change from full cooperation to full defection.

Such a behavior is reminiscent of the discontinuous first-order phase transition in the two-dimensional ferromagnetic Ising model on a square lattice. However, we inferred from simulations that the expected time the system spends in a metastable state before switching to the other one does not change as a function of the system's size, which precludes the existence of two stationary states, that is, breaking of ergodicity in the infinite population limit. This is caused by the existence of hubs in the Barabási-Albert scale-free graphs. Nevertheless, we observe a sharp phase transition for any finite size of the graph. This seems to be a novel behavior in systems of many interacting entities.

It is worth mentioning here that the ferromagnetic Ising model on the Barabási-Albert graph was discussed in Refs. [17,18]. It was shown both by stochastic simulations and the mean-field approximation that the effective critical temperature diverges as the logarithm of the size of the population and hence in the infinite-population thermodynamic limit the system does not exhibit a phase transition. We would like to point out that this model satisfies the detailed balance, so it is time reversible. In contrast, our imitation dynamics is time irreversible.

Let us note that in our model there is no possibility of deleting or creating links, the network is fixed, and it is the Barabási-Albert graph. We simply change the payoff matrix. Allowing networks of connections to evolve would be an interesting modification of the model, which deserves some future work.

It is a fundamental problem to investigate what are the necessary and sufficient conditions of the underlying graph and dynamics to imply the existence of phase transitions.

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Data availability. The data that support the findings of this article are openly available [20].

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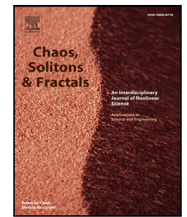
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Strategy dependent time delays shape cooperation in the spatial prisoner's dilemma game

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ABSTRACT

Understanding how temporal aspects of decision-making influence cooperation in structured populations is crucial for explaining the emergence of cooperative behavior in nature and society. While spatial reciprocity has long been known to support cooperation by allowing cooperators to form protective clusters, the role of time delays, particularly those dependent on strategy choice, remains underexplored. Here, we investigate the effects of strategy-dependent time delays on cooperation dynamics in the weak Prisoner's Dilemma game on square lattices. Using Monte Carlo simulations, we show that time delays can profoundly alter cooperation levels and spatial patterns. Our results reveal that asymmetries in delays can induce transitions between full cooperation, coexistence, and full defection. Also we show that strategy-dependent delays primarily alter the clustering dynamics of cooperators, which in turn drives the changes in overall cooperation behavior. We further show that the type of Prisoner's Dilemma (weak or standard) influences only the quantitative level of cooperation, while the qualitative effect of delays remains unchanged.

1. Introduction

Understanding the emergence and maintenance of cooperative behavior among self-interested individuals remains one of the central challenges in evolutionary biology and social science [1,2]. Cooperation, which involves individuals incurring personal costs to benefit others, significantly influences the evolutionary trajectory and stability of biological systems and human societies [1]. Evolutionary game theory has provided a robust framework to analyze and elucidate mechanisms that can foster and sustain cooperation [3–7]. Among various models, the Prisoner's Dilemma game (PDG) has emerged as a paradigmatic framework for studying cooperation [8–10]. In its classic formulation, two players simultaneously choose between cooperation (C) and defection (D), with payoffs structured such that mutual defection is the rational equilibrium, despite mutual cooperation yielding the highest collective benefit. This fundamental paradox encapsulates the conflict between individual rationality and collective welfare, motivating extensive research to identify mechanisms that promote cooperative outcomes [8,11].

Over recent decades, numerous cooperation-promoting mechanisms have been identified, such as kin selection [12], direct and indirect reciprocity [13,14], network reciprocity [11,15–19], and group selection [20]. Among these, spatial or network reciprocity has received substantial attention [11,15,16,21–26]. In structured populations, particularly on lattice and complex networks, cooperators can cluster together, effectively resisting invasion by defectors [11,15–34]. Clusters enable cooperators at their boundaries to compensate losses from interactions with defectors through mutual cooperation within the cluster, thus preserving cooperation [11,27]. Seminal work by Nowak and May illustrated that spatial structures, such as two-dimensional lattices, facilitate the coexistence and stability of cooperators and defectors, generating intricate, dynamic spatial patterns [11,35]. Subsequent studies

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further demonstrated that various structural complexities, including network topology, co-evolutionary dynamics, and multi-layered interdependent networks, significantly influence cooperation levels [29–45].

While spatial structures provide a robust mechanism for promoting cooperation, the temporal dimension of decision-making, particularly delays and memory effects, also plays a crucial role in realistic scenarios [46–53]. Time delays naturally arise when individuals use outdated information due to communication lags, processing times, or delayed feedback. Although memory and delay have frequently been conflated in the literature, these concepts differ fundamentally: memory refers to individuals actively retaining and utilizing past information, which could be short-term, long-term, or weighted, whereas delay represents a passive lag in receiving information. Several studies have explored the effects of memory, revealing that incorporating memory into evolutionary games generally promotes cooperation [46–51,54–64]. In contrast, studies examining delay effects remain relatively sparse, with limited exploration of how delayed information impacts strategy dynamics, particularly regarding strategy-dependent delays.

In reality, decision-making processes often exhibit strategy-dependent delays. For instance, cooperation or defection decisions might be based on varying response times or processing periods linked to the strategy being executed. Previous investigations of delays and strategy-dependent delays have predominantly focused on deterministic evolutionary game models, such as replicator dynamics, where delays can alter equilibrium points or affect their stability depending on the specific model [52,53,65–67]. Additionally, some stochastic models have incorporated delays [68,69]. For example, in [69], the authors investigated how delays impact the fixation process in well-mixed populations for various pairwise social dilemma games. Despite the realism and potential importance of strategy-dependent delays, their effect on cooperation behavior in structured populations remains an open question. Addressing this gap, our study investigates how strategy-dependent time delays affect cooperation dynamics in the weak Prisoner's Dilemma game on a lattice-structured population. We specifically examine how cooperation patterns evolve when individuals adopt imitation strategies with strategy-dependent information delays.

This paper is structured as follows. First, we introduce our evolutionary PDG on a lattice population incorporating strategy-dependent time delays into the imitation dynamics. In the subsequent section, we present detailed simulation results and discussions, highlighting the impact of strategy-dependent delays on the emergence of cooperation behavior and cooperator clusters. Finally, we summarize the main findings and provide concluding remarks on the implications of our study for understanding cooperative dynamics in structured populations.

2. Model

We consider the evolution of cooperation in a structured population using a Monte Carlo simulation of the weak PDG on a square lattice of size $L \times L$ with periodic boundary conditions. Each site on the lattice is occupied by a single individual, leading to a total population size of $N = L^2$. Each individual adopts one of two strategies: cooperation (C) or defection (D). At the initial time, each individual is randomly assigned to be a cooperator or a defector with equal probability.

Every individual plays the PDG with its four nearest neighbors (Von Neumann neighborhood) as well as itself. The total payoff is the sum of the payoffs from these five interactions. The payoff matrix corresponds to the weak Prisoner's Dilemma:

$$U = \begin{pmatrix} R & S \\ T & P \end{pmatrix} = \begin{pmatrix} 1 & 0 \\ b & 0 \end{pmatrix}, \quad \text{with } b > 1.$$

where b represents the temptation payoff of defectors over cooperators (dilemma strength). A central feature of our model is the incorporation of *strategy-dependent time delays* in the payoff evaluation. Specifically, if an individual adopts strategy $s \in \{C, D\}$, then its effective payoff at step $t \in \mathbb{Z}$ is computed from interactions that occurred $\tau_s \in \mathbb{Z}_{\geq 0}$ steps earlier. This reflects realistic situations in which the consequences of cooperation or defection are not felt immediately but are subject to delays that depend on the chosen strategy.

Strategy updates occur synchronously, where at each step, all individuals update their strategies simultaneously. Each individual i randomly selects one neighbor j from its neighborhood and adopts the neighbor's strategy with a probability governed by the Fermi update rule [27]:

$$W(s_j \rightarrow s_i) = \frac{1}{1 + \exp \left[\frac{P_i(t - \tau_{s_i}) - P_j(t - \tau_{s_j})}{\kappa} \right]},$$

where $P_i(t - \tau_{s_i})$ and $P_j(t - \tau_{s_j})$ denote the delayed payoffs of individuals i and j , respectively, and $\kappa > 0$ quantifies the noise level in the imitation process. A small value of κ corresponds to nearly deterministic imitation, while larger values introduce more randomness. In our simulations, we set $\kappa = 0.1$.

In our formulation, the time delay is assigned at the strategy level, meaning that all cooperators share a common delay τ_C and all defectors share a common delay τ_D . This modeling choice follows earlier works on memory and delay effects in evolutionary games, where delays are linked to types rather than to individual heterogeneity [46–48]. The rationale is that the temporal lag reflects properties of the interaction mechanism where cooperative acts often require coordination, joint investment, or accumulation of benefits, which naturally leads to delayed payoff realization, whereas defection tends to yield more immediate returns. Or in many real-world contexts, defection may also involve delayed consequences (e.g., time to realize exploitative gains, risks of being sanctioned, or long-term costs of resource depletion). Thus, delays represent the social process underlying the strategy rather than idiosyncratic attributes of individuals.

The main objective of this study is to investigate how strategy-dependent time delays influence the evolution of cooperation in structured populations. Unlike standard models where payoffs are immediately realized, we introduce distinct delays for cooperators

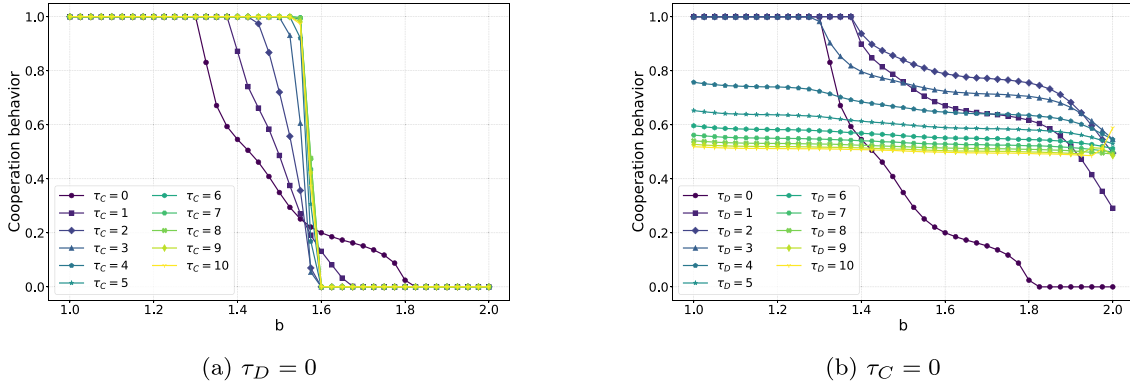


Fig. 1. Cooperation frequency as a function of the temptation to defect (the dilemma strength) b , for various delay settings. Each curve corresponds to a different delay value panel(a): $\tau_D = 0$, $\tau_C \in [0, 10]$ and panel (b): $\tau_C = 0$, $\tau_D \in [0, 10]$.

and defectors, reflecting scenarios where the benefits or costs of a strategy are not instantaneous. By systematically varying the delays τ_C and τ_D , we aim to understand how these temporal asymmetries affect the prevalence, and spatial distribution of cooperative behavior under the weak PDG.

3. Results and discussion

In this section, we investigate how strategy-dependent time delays influence the evolution of cooperation. The results are obtained from extensive Monte Carlo simulations on a lattice of size 200×200 , which we have verified to be sufficiently large: additional tests on 100×100 and 400×400 systems yielded qualitatively and quantitatively similar outcomes. Each data point represents an average over 100 independent simulations, each initialized with equal proportions of cooperators and defectors. For delayed payoffs, the initial values are fixed from the starting configuration:

$$P_C(t - \tau_C) = P_C(0), \quad P_D(t - \tau_D) = P_D(0), \quad \text{for } t < \max(\tau_C, \tau_D).$$

A simulation is considered to have reached equilibrium once the fluctuation in cooperator density $\rho_C(t)$ over the last 1000 steps is below 10^{-2} ,

$$\max_{t-999 \leq t^* \leq t} |\rho_C(t^*) - \bar{\rho}_C| < 10^{-2}, \quad (1)$$

with $\bar{\rho}_C$ denoting the average density in $[t - 999, t]$. After this criterion is satisfied, the system is run for an additional 1000 steps, and the steady-state cooperation level is measured as the mean ρ_C in this window.

We first examine how the dilemma strength b influences the frequency of cooperation under different strategy-dependent delay configurations (see Fig. 1). In panel (a), where only cooperators are delayed ($\tau_D = 0$, $\tau_C > 0$), we observe two distinct transitions in cooperation dynamics as the dilemma strength b increases. The first critical point, b_{cr1} , marking the shift from full cooperation to coexistence, moves to the right with increasing delay, indicating that for moderate dilemmas ($1 < b < 1.55$), larger delays can still support cooperation. However, the second threshold, b_{cr2} , where cooperation collapses entirely, shifts slightly leftward as delay increases: from $b_{cr2} \approx 1.82$ at $\tau_C = 0$ to 1.67 at $\tau_C = 2$ and 1.6 at $\tau_C \geq 3$. This shows that in stronger dilemmas ($b > 1.6$), delay harms cooperation by accelerating extinction. For example, at $b = 1.62$, cooperation survives for $\tau_C = 2$, but goes extinct for $\tau_C = 4$. Thus, delay initially supports but eventually undermines cooperation, depending on the dilemma strength.

In contrast, panel (b) shows the case where only defectors are delayed ($\tau_C = 0$, $\tau_D > 0$). This configuration leads to different and more nuanced behavior. For small delays ($\tau_D = 1, 2, 3$), the first transition point b_{cr1} shifts slightly to the right, with $\tau_D = 2, 3$ exhibiting the highest $b_{cr1} = 1.38$ among them. However, for larger delays ($\tau_D > 3$), full cooperation is no longer sustained even at low values of b , and the system always settles into a coexistence state. Regardless of the delay, there is no second threshold b_{cr2} beyond which cooperation goes extinct for $\tau_D > 0$. In all cases, cooperators persist, and defectors never fully dominate the population. For small delays ($\tau_D < 4$), cooperation levels decrease steadily with increasing b , while for larger delays, the decline becomes much more gradual and nearly flat. Still, the overall average cooperation level tends to drop slightly as τ_D increases.

As we observed in Fig. 1, strategy-dependent time delay can significantly influence the level of cooperation. To further explore how different combinations of delays affect cooperation dynamics, we present in Fig. 2 heatmaps of the cooperation frequency as a joint function of τ_C and τ_D for three different values of the dilemma strength $b = 1.3, 1.5$, and 2.0 . In the absence of delay ($\tau_C = \tau_D = 0$), the three panels correspond to qualitatively distinct regimes: full cooperation for $b = 1.3$, coexistence for $b = 1.5$, and full defection for $b = 2.0$. However, time delay reveals rich and nontrivial structures. Across all cases, we observe three distinct regions: full cooperation (yellow), full defection (blue), and coexistence (intermediate colors), with sharp phase boundaries between them. For all three values of b , even when $b = 2.0$ (where the baseline outcome is full defection in the absence of delay), there exist broad regions of parameter space where cooperation emerges, particularly when cooperators experience less delay than defectors

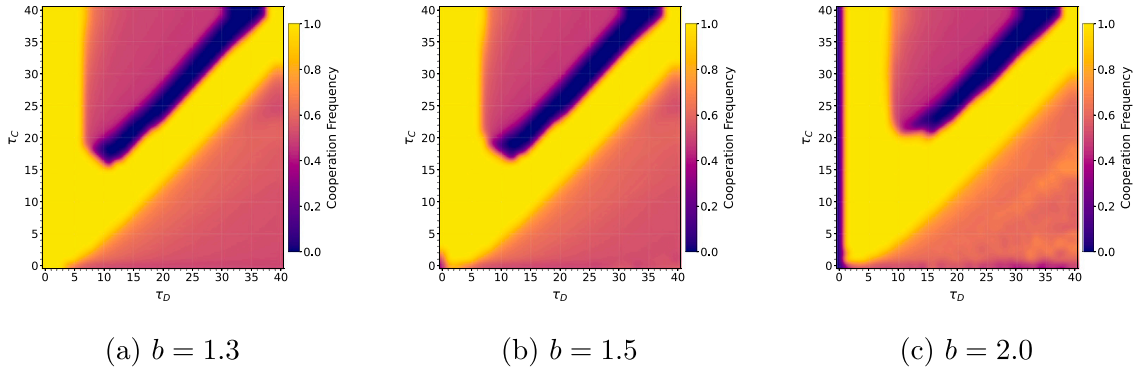


Fig. 2. Cooperation frequency as a joint function of the cooperator delay τ_C and the defector delay τ_D for three dilemma strengths: (a) $b = 1.3$, (b) $b = 1.5$ and (c) $b = 2.0$.

($\tau_C > \tau_D$). This shows that delay asymmetry can reverse the expected outcome and restore cooperation even under harsh dilemma conditions. In contrast, the condition $\tau_C < \tau_D$ leads to a stable coexistence between cooperators and defectors.

Another notable feature is that these transitions between cooperation and defection often occur abruptly: a small change in one of the delays (as little as one unit) can shift the system from full cooperation to coexistence, and immediately to full defection—or vice versa. For example, at $b = 2.0$, the system results in full defection when $\tau_C = \tau_D = 0$, but remarkably, introducing even a small delay such as $\tau_C = \tau_D = 2$ leads to full cooperation. This sensitivity highlights the strong influence of delay configuration on system outcomes. Furthermore, for values of delays ($15 < \tau_C < 40$ and $8 < \tau_D < 37$), the overall structure becomes less sensitive to b . These results show that the evolutionary outcome is not determined by delay magnitude alone, but rather by the relative timing between strategies.

To illustrate the spatial impact of strategy-dependent time delays, Fig. 3 shows typical stationary snapshots for the case $b = 1.5$. Each snapshot corresponds to an equilibrium state defined by the condition in Eq. (1), though the exact time step varies with the combination of τ_C and τ_D . In panel (a), the defector delay is fixed at $\tau_D = 0$ while τ_C increases from 0 to 5; in panel (b), $\tau_C = 0$ and τ_D varies. Blue (black) cells represent cooperators (defectors) who have maintained their strategy since $t - \tau_i$, $i \in \{C, D\}$, while yellow (red) cells indicate cooperators (defectors) who switched from the opposite strategy at $t - \tau_i$, $i \in C, D$.

Strategy-switched individuals are mostly observed at the interfaces between cooperative and defective clusters. These patterns indicate that increasing τ_C promotes cooperation by stabilizing cooperator domains through delayed payoff feedback. With such delay, occasional exploitation by defectors at the edges does not immediately reduce the apparent success of cooperation, making cooperators appear more successful than they actually are. This effect supports the persistence and growth of cooperative regions. In contrast, when $\tau_C = 0$ and τ_D increases, defectors act on outdated payoff information that may reflect earlier periods of successful exploitation. As cooperation spreads, defectors are slower to adjust to the declining returns of defection, which temporarily benefits cooperators. However, when τ_D becomes too large (e.g., $\tau_D > 4$), the delayed response turns counterproductive: defectors may continue exploiting cooperators even when the payoff conditions have changed. This inertia destabilizes the spatial structure, and excessive delay can generate disordered dynamics that ultimately undermine the formation and stability of cooperative clusters.

To further understand the temporal evolution of spatial strategy patterns under different delay settings, we present in Fig. 4 a sequence of snapshots in different time steps for three representative configurations of delay: $\tau_C = \tau_D = 0$, $\tau_C = 4, \tau_D = 0$, and $\tau_C = 0, \tau_D = 4$ for $b = 1.5$. Consistent with previous observations in spatial PDG [41,70,71], the fraction of cooperators initially declines as isolated cooperators are exploited by neighboring defectors. This early decline reflects the disadvantage cooperators face before forming protective clusters. However, as the evolution proceeds, cooperative clusters begin to emerge, and if stable enough, they can expand and dominate. Interestingly, the presence of strategy-dependent delays alters the morphology and dynamics of these clusters. In the symmetric case $\tau_C = \tau_D = 0$, the pattern follows the classical dynamics: cooperator clusters grow gradually and may stabilize. In contrast, for asymmetric delay configurations (e.g., $\tau_C = 4, \tau_D = 0$ or $\tau_C = 0, \tau_D = 4$), the spatial organization of cooperators exhibits notable differences. For instance, when cooperators experience delay, the formation of large cooperator clusters is facilitated, allowing cooperation to spread and occupy a larger portion of the population. In contrast, when defectors experience a delay, cluster formation is hindered and cooperation becomes weaker.

As we know network reciprocity suggests that spatial structures can promote cooperation by enabling cooperators to form clusters that shield them from defector exploitation [11]. To assess whether similar dynamics occur under strategy-dependent delays, we analyze the evolution of cooperation frequency, average cooperator cluster size, and the number of cooperator clusters under varying delay conditions. The results are presented in Fig. 5.

Panel(a) shows the case where the cooperator delay $\tau_C = 0$ and the defector delay τ_D varies. Initially, cooperation declines due to the vulnerability of isolated cooperators in randomly distributed populations. This decline is deeper and more prolonged for bigger delay. These settings also lead to a rapid rise in average cluster size and a sharp drop in cluster number with the maximum for $\tau_D = 2$. This suggests efficient merging of small clusters into larger ones that can resist defector invasion. Such a pattern indicates that moderate delay in defector responses enables cooperators to consolidate before being overtaken, enhancing

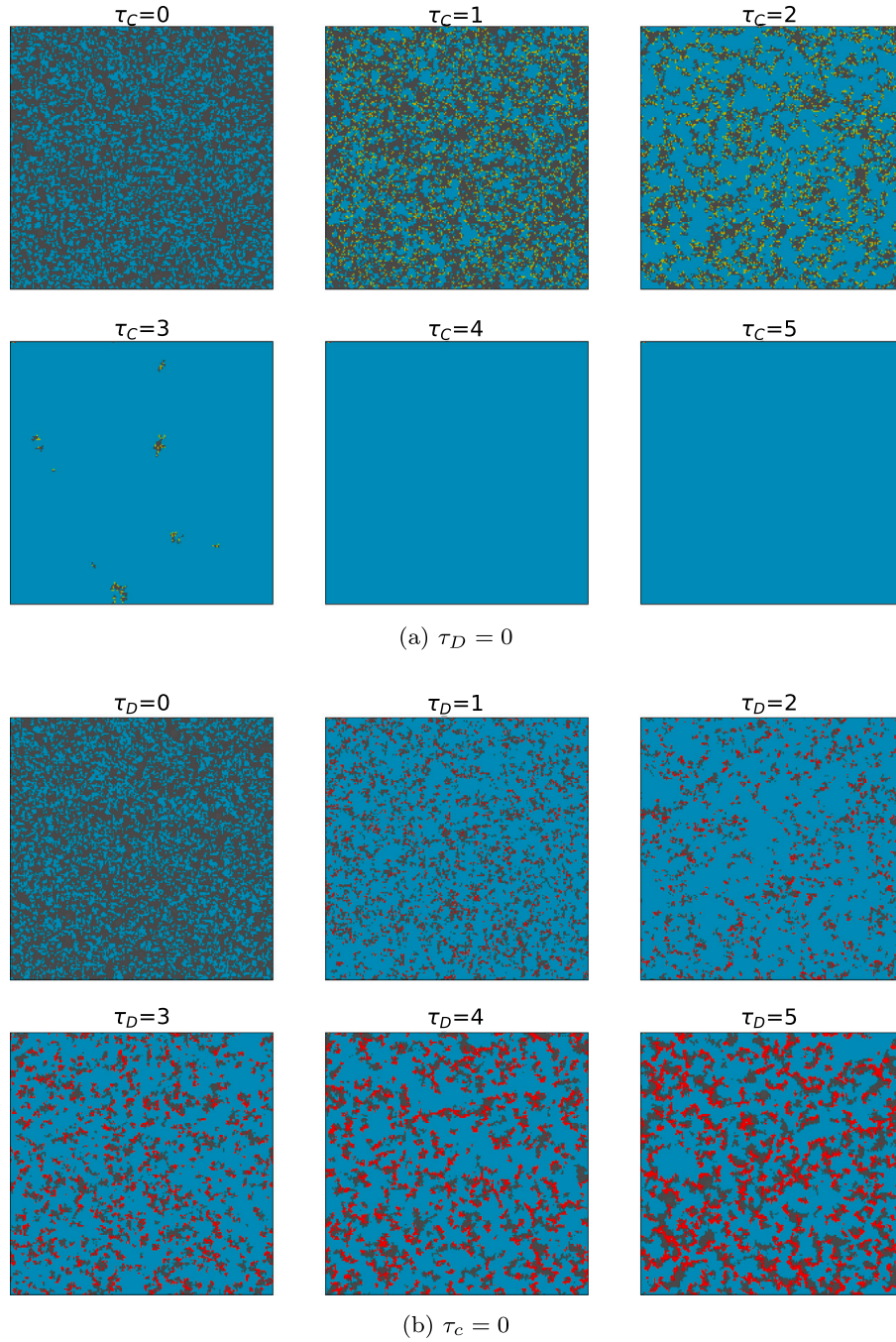


Fig. 3. Snapshots of typical equilibrium configurations for $b = 1.5$. (a) $\tau_D = 0$ and τ_C varies from 0 to 5. (b) $\tau_C = 0$ and τ_D varies from 0 to 5. Blue indicates cooperators who were cooperators at $t - \tau_C$, black shows defectors who were defectors at $t - \tau_D$, yellow marks cooperators who were defectors at $t - \tau_C$, and red shows defectors who were cooperators at $t - \tau_D$.

spatial reciprocity. The formation of fewer but larger cooperator clusters implies that delay may effectively buffer cooperators against immediate exploitation, thereby stabilizing cooperation dynamics in the populations.

In contrast, panel(b) examines scenarios where $\tau_D = 0$ and τ_C varies. Higher τ_C values delay the rebound of cooperation but facilitate the early formation of larger clusters. This is because new cooperators retain the temporarily beneficial payoffs from their prior defector state, aiding their survival and clustering. For example, at $\tau_C = 5$, average cluster size grows rapidly, and cluster number drops quickly—indicating fast and effective cluster formation.

These results suggest that the clustering process — a key mechanism for the survival and proliferation of cooperation — is highly sensitive to delay asymmetry. Therefore, the observed variation in long-term cooperation levels under different delays may, in part, be attributed to delay-induced modifications in cluster stability and expansion.

Until now, we have focused on the weak PDG, which is widely studied in the literature, and have demonstrated that strategy-dependent time delays can significantly impact the level of cooperation. A natural question that arises is whether the same effects

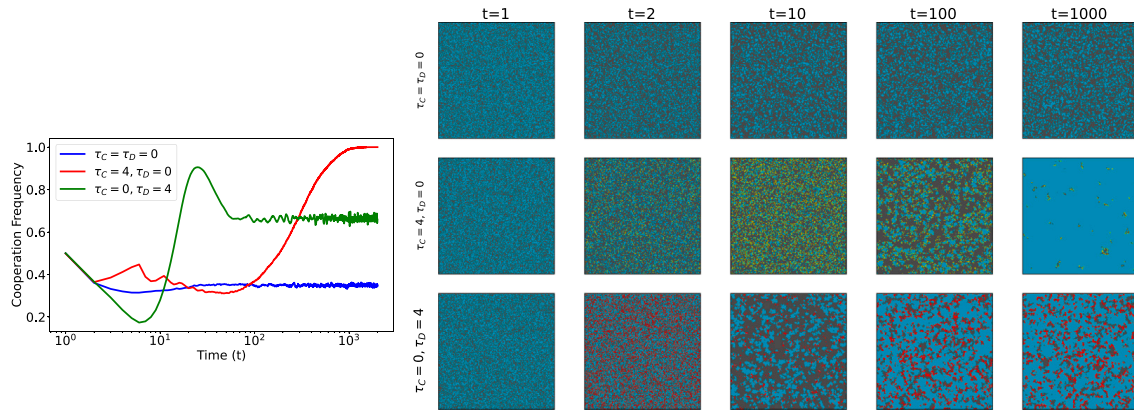


Fig. 4. Time evolution of frequency of cooperation and spatial strategy distribution for three representative delay configurations: (top row) $\tau_C = \tau_D = 0$, (middle row) $\tau_C = 4$, $\tau_D = 0$, and (bottom row) $\tau_C = 0$, $\tau_D = 4$ where $b = 1.5$. Blue indicates cooperators who were cooperators at $t - \tau_C$, black shows defectors who were defectors at $t - \tau_D$, yellow marks cooperators who were defectors at $t - \tau_C$, and red shows defectors who were cooperators at $t - \tau_D$.

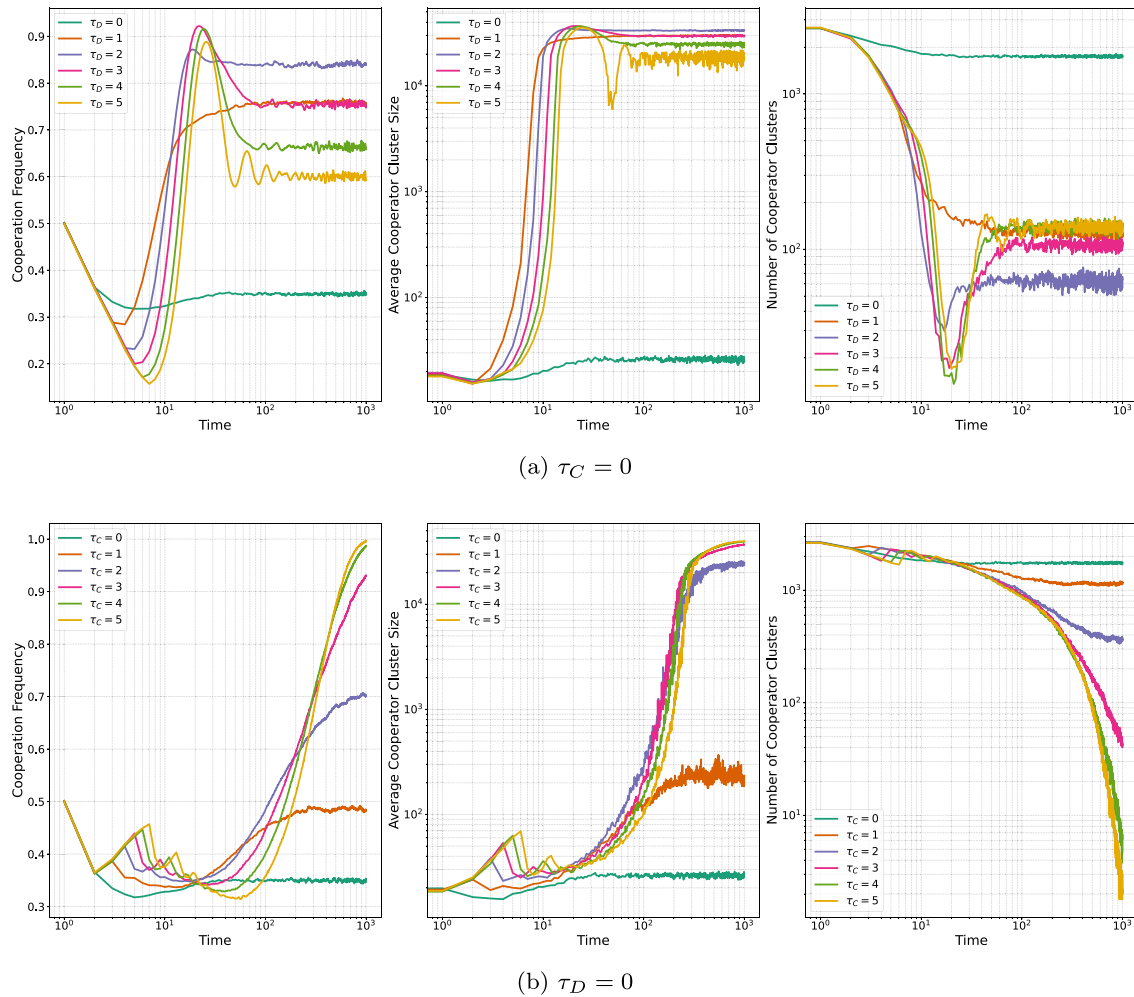


Fig. 5. Temporal evolution of cooperation frequency, average cooperator cluster size, and number of cooperator clusters under strategy-dependent delay for $b = 1.5$.

occur in the classical (standard) PDG. To investigate this, we consider the payoff matrix U with parameters $R = 1$, $S = -r$, $T = 1 + r$, and $P = 0$. Fig. 6 presents the results for the standard PDG (to be compared with Fig. 1 for the weak version). We observe that, qualitatively, the effect of time delay on the frequency of cooperation is similar in both cases: time delays shift the critical values of r ($1 + r$ corresponds to b in the weak PDG) at which transitions occur, namely from full cooperation to coexistence, and from

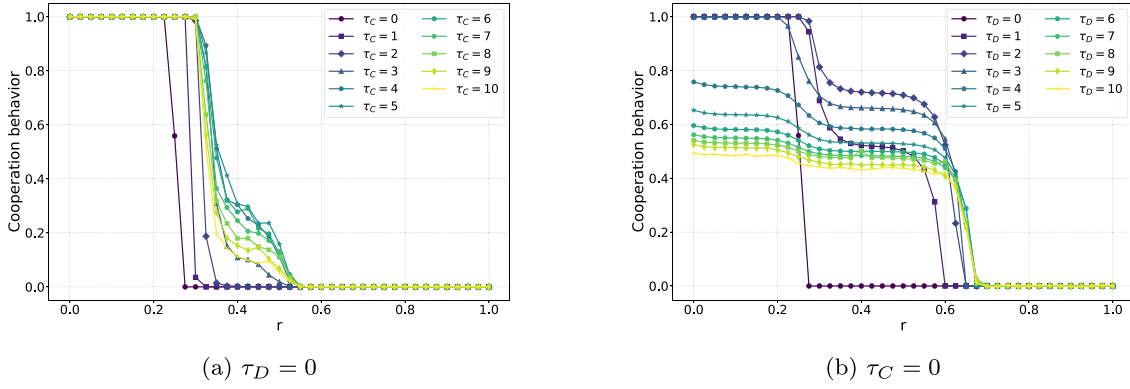


Fig. 6. Cooperation frequency in standard PDG as a function of r when $R = 1, S = -r, T = 1 + r$, and $p = 0$ in U , for various delay settings. Each curve corresponds to a different delay value panel(a): $\tau_D = 0, \tau_C \in [0, 10]$ and panel (b): $\tau_C = 0, \tau_D \in [0, 10]$.

coexistence to full defection. However, the quantitative outcomes differ. In the standard game, these critical values are smaller than in the weak case. For example, when τ_C is fixed and τ_D is varied, cooperation becomes extinct for smaller values of r .

This quantitative difference is expected, since in the standard game, a cooperator facing another cooperator receives a payoff of $-r$, which is negative. This reduction in the payoff of cooperators accelerates the decline in cooperation as r increases, leading to earlier extinction of cooperation compared to the weak Prisoner's Dilemma. Furthermore, by examining the effect of delays on the clustering behavior, as in the case of the weak PDG, we find that although the quantitative outcomes differ, the qualitative dependence on the delay remains the same.

4. Conclusion

In this study, we explored the impact of strategy-dependent time delays on the evolution of cooperation in structured populations governed by the weak Prisoner's Dilemma game. By systematically varying the delay parameters τ_C and τ_D , we demonstrated that temporal asymmetries in payoff evaluation can profoundly influence the dynamics and frequency of cooperation. Through extensive Monte Carlo simulations on a lattice network, we identified distinct regimes where cooperation is either promoted or suppressed, depending on the relative timing of cooperative and defective responses. Our findings reveal that even small delays can shift the system's behavior between full cooperation, coexistence, and full defection, underscoring the sensitivity of evolutionary outcomes to delayed feedback mechanisms.

Our results revealed that strategy-dependent delays substantially reshape the spatial dynamics of cooperation. In particular, we observed that time delays modulate the clustering process, which is a key mechanism supporting the survival of cooperators in structured populations. When defectors experience longer delays than cooperators, cooperative clusters grow more robustly and can successfully expand, even under strong dilemma conditions. Under weak dilemma conditions, increasing the defector's delay does not enhance cooperation and may even hinder it because outdated defector payoffs fail to reflect the opportunity for exploitation, thereby stabilizing defection. In contrast, for moderate dilemma strengths, we found that introducing a delay for cooperators can actually support the emergence and expansion of cooperative behavior. This occurs because the memory of higher past payoffs allows newly converted cooperators to survive longer and form stable clusters. However, in the case of strong dilemma conditions, delaying cooperation becomes detrimental: the postponed payoff feedback prevents timely reinforcement of cooperative strategies, undermining their ability to resist exploitation and sustain viable clusters. These observations highlight that the interplay between delay and dilemma strength is nontrivial and must be carefully considered when interpreting the dynamics of cooperation.

We have also considered the standard Prisoner's Dilemma game and observed that the type of game affects only the quantitative outcomes, not the qualitative behavior. In both the weak and standard versions, strategy-dependent delays influence cooperation in the same manner, while the exact thresholds for transitions differ due to the payoff structure.

A limitation of the present study is the assumption of homogeneous delays within each strategy class. While this assumption allows us to clearly isolate the impact of strategy-dependent temporal asymmetry, real populations may exhibit heterogeneous or individual-specific delays. An interesting extension would be to consider cases where delays are individual-dependent, vary with spatial location or past history, or are drawn from a probability distribution. Such generalizations would enable a more realistic description of temporal effects in structured populations and could reveal novel mechanisms by which heterogeneity in response times influences the evolution of cooperation.

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.

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Data availability

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

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