

# **ECO-EVOLUTIONARY INTERACTIONS AND THEIR DYNAMICS**

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“Science is not a heartless pursuit of objective information. It is a creative human activity, its geniuses acting more as artists than as information processors.”

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— Stephen Jay Gould

*I dedicate this thesis to my parents, and to those who taught me that scientific temper is required for social revolution including some wonderful teachers whom I have had the privilege of learning from.*



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# Zusammenfassung

Die meisten experimentellen und theoretischen Untersuchungen zu öko-evolutionärer Dynamik basieren auf biologischen Interaktionen zwischen verschiedenen Akteuren, die entweder zur selben oder zu verschiedenen Spezies gehören. Wenn wir das gesamte Bild betrachten, sind Individuen gleichzeitig an mannigfaltigen Interaktionen beteiligt, und je nach Interaktion nehmen sie verschiedene Rollen ein oder wenden verschiedene Strategien an. Um die evolutionäre Dynamik einer Population zu untersuchen, ist es deswegen wesentlich, die kombinierten Effekte aller Interaktionen zu erfassen, an denen ein Individuum beteiligt ist. Zu diesem Zweck entwickle ich im ersten Teil dieser Arbeit einen mathematischen Rahmen innerhalb der evolutionären Spieltheorie, der es erlaubt, mannigfaltige Interaktionen zu berücksichtigen. Für unendliche Populationen finde ich, dass die Zusammensetzung einer Population, in der Individuen an einer Kombination von Spielen beteiligt sind, andere dynamische Trajektorien aufweist und andere Gleichgewichtspunkte einnimmt verglichen mit einer Population, in der Individuen nur an einzelnen Spielen in Isolation betrachtet teilnehmen. Für endliche Populationen leite ich einen Ausdruck für die Berechnung der Fixationswahrscheinlichkeit einer Strategie her, die eine wichtige stochastische Größe darstellt. Auch hier finde ich, dass sogar die Kombination eines neutralen Spiels mit einem anderen Spiel die Dynamik ändern kann. Im zweiten Teil verwende ich die im ersten Teil entwickelte Theorie, um die Dynamik in den Zahlen männlicher und weiblicher Individuen in einer Population zu untersuchen, wenn gleichzeitig mehrere Life-history traits betrachtet werden. Life-history traits können Unter-

schiede zwischen den Geschlechtern aufweisen. Unter Verwendung von evolutionärer Spieltheorie und Populationsdynamik zeige ich, dass geschlechtsspezifische Unterschiede in elterlichem Investment, in Ornamenten und in der Immunantwort zu einer ungleichen Anzahl an männlichen und weiblichen Individuen in einer Population führen. Meine Ergebnisse sind im Einklang mit empirischen Beobachtungen in einer Vielzahl von Taxa. In der vorangegangenen Untersuchung waren die geschlechtsspezifischen Unterschiede in bestimmten Life-history traits ein vorgegebener Parameter. Aber wie sind diese geschlechtsspezifischen Unterschiede in verschiedenen Merkmalen des Lebenszyklus überhaupt entstanden? Der letzte Teil dieser Arbeit stellt die Evolution des sexuellen Dimorphismus in Immunität dar mit Bezug zu elterlichem Investment. Experimentelle Ergebnisse zeigen, dass vermehrtes elterliches Investment zu besseren Immunantworten führen kann. Ich verwende eine zustandsabhängige dynamische Optimierungsmethode, wobei elterliches Investment und Immunkompetenz als zwei verschiedene Merkmale eines Individuums betrachtet werden. Jedes Individuum hat zu jedem Zeitpunkt eine bestimmte Energiereserve. Ich untersuche die Trade-offs zwischen der Verwendung der Reserven eines Individuums auf die elterlichen Aufgaben und auf Immunität, so dass sie die Fitness des Individuums maximieren. Mit dieser Studie zeige ich, dass es für die meisten Spezies optimal ist, mehr in Immunkompetenz zu investieren, wenn die Zeit elterlicher Pflege lang ist. Diese Arbeit verwendet also Populationsdynamik, evolutionäre Spieltheorie und zustandsabhängige dynamische Optimierungsmethoden, um unser Verständnis über mehrere miteinander verknüpfte Interaktionen zwischen Individuen oder Merkmalen innerhalb eines Individuums zu fördern.

# Summary

Most of the experimental and theoretical studies of eco-evolutionary dynamics are based on biological interactions between various agents from either the same species or from different species. If we look at the whole picture, individuals take part in diverse interactions simultaneously, and they adopt varied roles or strategies in each interaction. To study the evolutionary dynamics of a population, it is therefore crucial to capture the combined effects of all the interactions that an individual is a part of. To that aim, in the first part of my thesis, I construct a mathematical framework residing in evolutionary game theory that allows to account for multiple interactions. In infinite populations, I find that the composition of a population in which individuals are involved in combined games shows different dynamical trajectories and different equilibrium points as compared to a population in which individuals only take part in single games considered in isolation. For finite populations, I derive an expression for calculating the fixation probability of a strategy, which is an important stochastic property and here too, I find that combining even a neutral game with another game could change the dynamics. In the second part, I use the framework developed in the first part to study the dynamics in the numbers of male and female individuals in a population when jointly considering several life-history traits. Life-history traits may have sex-specific differences. Using evolutionary game theory and population dynamics, I show that sex-specific differences in parental investment, ornamentation, and immune response give rise to skewed adult sex ratios. My results fit with empirical observations from a wide range

of taxa. In the previous study, the sex-specific differences in certain life-history traits were a given parameter. But how did these sex-specific differences in various life-history traits emerge in the first place? The last part of this thesis presents the evolution of sexual dimorphism in immunity in relation to parental investment. Experimental results show that an increase in parental investment may give rise to better immune responses. I use a state-dependent dynamic optimization method, where parental investment and immunocompetence are considered to be two different traits of an individual. Every individual has a certain amount of energy reserves at a certain time. I study the trade-off between allocating reserves to parental effort and to immunity in such a way that they maximize an individual's fitness. Through this study, I show that for most species systems, it is optimal to invest more in immunocompetence when the time spent in parental investment is longer. Thus, this thesis uses population dynamics, evolutionary game theory, and state-dependent dynamic optimization modeling in order to advance our understanding of eco-evolutionary consequences of multiple interlinked interactions between individuals or traits within individuals.

# Chapter 1

## Multiple interspecies and intraspecies interactions

“What makes it so hard to organize the environment sensibly is that everything we touch is hooked up to everything else.”

---

— Isaac Asimov

### 1.1 Multiple intertwined interactions in nature

The theory of evolution by natural selection was first detailedly set out in the book *On the Origin of Species* in 1859 by Charles Darwin. All forms of life evolve over generations through genetic changes that correspond to the changes in the observable traits of the organisms. Biological interactions are at the core of every evolutionary process and are broadly classified as competition, predation, commensalism, mutualism, and parasitism. Theoretical and empirical studies related to cooperation, antagonism, host-pathogen co-evolution, sexual selection, gene interplay, all focus

on some of the above-mentioned class of interactions between various agents. Each interaction involves different types of individuals and the number of members or frequency of each type may vary.

Interactions may occur both within (intraspecies) and between species (interspecies), and such interactions can be found among every living organism from unicellular prokaryotes like bacteria to archaea and eukaryotes. However, no interaction happens in isolation from other interactions. One intra-species and interspecies interaction would be linked to other interactions. Gut microbiota is an excellent host-microbe system consisting of individuals taking part in multiple interactions. Within a host, one can find diverse interspecies and intra-species interactions between the gut microbiomes. The microbiomes also interact with the host, and the host's interaction with its environment might also affect the microbial interactions. (Clemente et al., 2012; Nicholson et al., 2012). Therefore, looking at merely one interaction of interest will not grasp the entire story since this interaction might be coupled with other interactions as well.

Intriguing instances of combined interactions exist in diverse milieus and modeling these scenarios will provide more knowledge to the pursuit of detailed understanding of interactions and how they affect evolution. The goal of this thesis was to construct mathematical models that investigate the dynamics of multiple interactions. The interactions could be between and within individuals, and also scenarios such as competition for resources between multiple traits within an individual. A few examples of multiple interspecies and intraspecies interactions across a wide range of taxa that highlight the importance of studying multiple interactions' dynamics are listed below.

- **Interactions between bacterial strains**

*Pseudomonas fluorescens* SBW25 bacterial strains undergo a special spontaneous mutation called the wrinkly spreader (WS) from the smooth ancestral type (SM) (Rainey and Travisano, 1998). This WS mutant has a lower mass density than the SM wildtype. Therefore,

it colonizes the air-liquid interface of liquid cultures by forming a biofilm. The WS mutants interact cooperatively to form this stable biofilm that is well-supplied with oxygen since it is situated right at the surface separating the liquid medium and air. Therefore, these mutants are advantageous over the wildtype as their colony gives them greater access to oxygen (Rainey and Rainey, 2003). WS mutants perform cooperative interactions among themselves, but they also compete with the SM ancestor, and possibly with other species, for resources. Thus one colony of a bacterial strain is involved in many interactions.

- **Hosts and pathogens**

There are multiple examples of host-pathogen interactions (Harvell, 2004). The ecology and evolution of these interactions determine the fitness of the host and pathogens. Host-pathogen interactions are in fact diverse interspecies and intraspecies interactions combined together. Consider human-tuberculosis co-evolution (Gagneux, 2012). This involves interactions within the tuberculosis-causing bacterium *Mycobacterium tuberculosis* strains and their interspecies interactions with humans. Another classic example is plant-pathogen interactions (Frank, 1992). The plant immune system undergoes symbiotic interactions with beneficial microbes as well as antagonistic interactions with pathogens (Hacquard et al., 2017). For instance, host-pathogen dynamics exhibit the 'Red Queen dynamics' (Khibnik and Kondrashov, 1997). This is a result of what is known as the arms race between the hosts and various strains of pathogens. When a resistance towards a certain pathogen strain is developed in a host species, this strain can evolve to affect the host once more. Other strains may also compete with this strain to infect the host. Again, as the host continuously adapts, there is always scope for the selection of new mutations in the pathogens to be selected for and attack the host. This process goes on and this is a reason why eliminating pathogens (especially those that mutate fast) is a major challenge. Thus, studying multiple interlinked host-pathogen interactions is crucial for medicine and agriculture which are two

important factors for the survival of humankind.

- **Mating competitions and mate choice**

Evolution of the sexes involves both intrasexual and intersexual interactions (Moore, 1990; Wiley and Poston, 1996). Members of both the sexes may also have to interact with other species for foraging, hunting and other everyday tasks. Individuals of the same sex compete for mates known as intrasexual interaction. This is one reason for the diverse morphologies between and within the sexes. For instance, the size of testis in primates was shown to be larger in polygamous mating systems as compared to mating systems with lesser or no polygamy (Harcourt et al., 1995; Kappeler, 1997). The result of these elaborate mating competitions is being chosen by members of the other sex for copulation. This is known as mate choice or intersexual interactions. These intersexual and intrasexual interactions together cause sexual selection which can be seen in sexually reproducing animals.

## 1.2 Mathematical models of multiple interactions

Since interactions across a wide range of taxa are usually associated or linked to one another, taking this into account should be pivotal for both experimental and theoretical research. This doctoral thesis presents a general framework to study the combined dynamics of multiple interactions. The dynamics of a combination of diverse multiplayer interspecies and intra-species interactions where each interaction has several strategies can be investigated using this framework. An important and commonly used mathematical model to study eco-evolutionary interactions is evolutionary game theory. *Games* are a proxy for interactions. So one game would be equal to studying an interaction. The types of members participating in a game are referred to as *players*. Each of these players has a set of *strategies* which are the different phenotypic trait values, for instance *cooperation* and *defection*. A player interacting with another player receives a *payoff*.

The average payoffs contribute to the fitness of a strategy that determines its course of evolution. Details of evolutionary game theory methods shall be discussed in Chapter 2, where I used game theory and constructed a model that can study any multi-interaction scenario involving many interactions, players, and strategies. Conditions and outcomes of combining many games were also explored using this framework. The general results that emerged out of this framework were applied to study multiple interactions between life-history traits (see Figure 1.1).

## Previous works on mathematical models of multiple interactions

Until recently, evolutionary game theory was extensively used to study two-player games i.e. they assumed pairwise interactions between the agents. However, these were limited to a single game i.e. an isolated interaction until Cressman et al. (2000) and Hashimoto (2006) investigated multiple game(s) dynamics. Cressman et al. (2000) investigated two-games dynamics while Hashimoto (2006) extended this to multiple-games. The multiple-games approach was also used in a couple of studies that investigated specific biological settings. *Dictyostelium discoideum* is a social amoeba that exhibits (plays) two cooperative behaviors (games) under starvation. One cooperative game is related to fruiting body formation and the other, macrocyst formation. Fruiting body formation is the process where a few cells differentiate into spore cells. These spore cells are the ones that end up surviving and reproduce under favorable conditions. The other cells turn themselves into stalk-cells that disperse the spore cells (Queller et al., 2014). The second cooperative game is the macrocyst formation. Here, some cells form diploid zygote cells and produce offspring. The rest of the cells that are vegetative supply energy to these zygotes (O'Day and Keszei, 2012). An evolutionary game theoretical model constructed by Shibasaki and Shimada (2018) couples these two games. The results showed that cooperation is maintained only when the amoeba play both these games. The coupling between these games occurs due to the cyclic adenosine monophosphate compound which is a complex that is a derivative of adenosine

triphosphate (ATP) and aids in intracellular signal transduction. This chemical signal is central to both these games. Therefore, a multi-games model that combines interactions connected to an organism or a niche well-explains the dynamics of that population. The application of evolutionary game theory in studying cancer has a long-established tradition. In [Kareva \(2011\)](#) it was shown that aerobic and anaerobic (glycolytic) tumor cells play a game where the former is a 'defector' and the latter is a 'cooperator'. The anaerobic cells secrete lactic acid, a byproduct of glycolysis. This acid is toxic to normal somatic cells that surround the tumor and allows the tumor cell population to rise. Another cancer game is the mobility game necessary to start the process in which cancer cells spread to distant locations in the body also known as metastasis. Phenotypic plasticity allows some cells to become motile and the remaining to be stationary. 'Motility' and 'stationary' are the two strategies in this game ([Kareva and Karev, 2019](#)). This study by [Kareva and Karev \(2019\)](#) investigated the combined dynamics of two cancer games that could be applied to the two games mentioned above. They showed that the dynamics of cooperators and defectors within the respective games do not change after combination. But, selection causes a temporal change in the distribution of these games. Thus, multiple-game dynamics are crucial to accurately study the evolutionary dynamics of unicellular organisms to tumors in multicellular organisms.

## **A generalized mathematical framework for multiple interactions' dynamics**

All the previous studies related to multiple-games had considered only pairwise interactions. However, in reality, interactions are among multiple agents. Studies on single evolutionary games to multiplayer games and multiple strategies expanded the domain of study to public goods games and other social dilemmas e.g [Gokhale and Traulsen \(2010\)](#). I enhanced the sphere of multiple-game dynamics that can consider multiplayer games with multiple strategies ([Venkateswaran and Gokhale, 2019](#)). My work has a generalized approach and the method that I have developed

can be used for analyzing multiple games, where each involved game could be a multiplayer (and multi-strategy) game. Thus, this approach enables us to study the entire space of multiple interactions with multiple strategies consisting of multiple players. In fact, the previous studies on multi-games dynamics (Cressman et al., 2000; Hashimoto, 2006; Shibasaki and Shimada, 2018; Kareva and Karev, 2019) can be derived from my general framework. I have also extended the multiple-games dynamics approach to study the coupled interactions or relationships between life-history traits (see Figure 1.1).

The remainder of this chapter presents an overview of the thesis.

### 1.3 Overview of the thesis

As illustrated in Figure 1.1, the next three chapters are devoted to the development of mathematical models to study multiple interspecies and intraspecies interactions that individuals take part during a period. Chapter 2 describes an evolutionary game theory based model on multiple interactions and their dynamics. This model gives us information about the dynamics of combined interactions. A crucial outcome that we obtained from the mathematical framework in Chapter 2 is that investigating combined interactions' differs from studying a single interaction in isolation. Another information that we get from this mathematical framework is that interactions influence each other. A combination of one interaction with an already existing one can change its dynamics in both deterministic (infinite population) and stochastic (finite populations) scenarios. Thus, the combined interactions' dynamics present a clearer story about the individuals. A population dynamics based model presented in Chapter 3 examined interactions between multiple life-history traits with sex-specific differences in each of them. This helped study the overall life-history of an individual. Chapter 4 contains a state-dependent dynamic model that focused on two life-history traits: parental investment and immunocompetence. These two traits interact through competition for resources i.e. there is a trade-off between the allocation of an individual's reserves

towards offspring (parental investment) or survival (immunocompetence). This model illustrates how sex-specific differences in parental investment can give rise to sexual immune dimorphism.

Synthesis of each of these chapters is listed below.

- **Chapter 2: a theoretical framework to study combined interactions**

The first phase of my thesis was to construct a mathematical framework for investigating multiple interactions (or games). The mathematical framework that I have constructed is a complete and general method to study multiple games with many strategies and players, all at once. This was done for infinite and finite populations. The results are exhibited in the paper ([Venkateswaran and Gokhale, 2019](#)) attached in Chapter 2. To summarize the results from this paper: a combination of games (interactions) is not the same as the 'sum' of the individual games. For infinite populations, we found that the combined game(s) dynamics shows different trajectories and different equilibrium points for the strategies as compared to their dynamics in the individual games. When the games have more than two strategies, we find that they cannot be separated back to their single game dynamics, in line with previous findings ([Hashimoto, 2006](#)). Interestingly, however, we find a dependency on the initial conditions i.e., the initial frequencies of each strategy. For certain initial conditions, one may still be able to capture the single game dynamics from their multi-game dynamics. Finite populations can account for natural properties like drift. Thus, for finite populations we also derived an expression for calculating an important stochastic property known as the fixation probability: when a new mutant enters a resident wild-type population, will it fixate or not? Our results show that the fixation probability of a single game changes even when a neutral game is combined with it. The details of these results are shown and discussed in the following chapter.

- **Chapter 3: an application of the theoretical combined interactions' dynamics**

### **approach to study the consequences of interacting life-history traits**

The model and its results discussed in Chapter 3 is an application of the above mentioned multiple games approach. I studied the outcome of various interactions during a reproductive lifetime of the different sexes in sexually reproducing polygamous animals. The manuscript (V.R Venkateswaran, O Roth and C.S Gokhale. *Consequences of combining life-history traits with sex-specific differences*. bioRxiv doi: <https://doi.org/10.1101/2020.01.03.892810>) is attached in Chapter 3. In this manuscript, I show that sex differences in parental investment, ornamentation and immunity (Trivers, 1972, 2002; Hedrick and Temeles, 1989; Roved et al., 2017) give rise to a bias in the adult sex ratio (Pipoly et al., 2015). As shown in many empirical studies, the sex ratio bias among adults has an impact on sex differences and roles (Liker et al., 2013, 2015; Székely et al., 2014). This model shows that the opposite is also possible (Kokko and Jennions, 2008) by allowing one to capture the diverse sex-specific interactions and traits of an individual. Details of this model and its results as illuminated in Chapter 3

- **Chapter 4: relationships between interacting life-history traits**

The model presented in Chapter 4 zooms further into interactions between multiple life-history traits. This study is aimed towards investigating the emergence of species or sex-specific differences in life-history traits. How do interactions between life-history traits influence each other? More precisely, how do interactions between life-history traits result in sex-specific differences or sexual dimorphism in certain traits? The two life-history traits we consider are parental investment and immunity. There have been studies, both experimental and theoretical to answer the evolution of sexual immune dimorphism (Stoehr and Kokko, 2006; Nunn et al., 2008; Restif and Amos, 2010). But in the theoretical models, an important factor is missing - parental investment. Parental investment has three parts: gamete production, pregnancy (in some cases only internal fertilization) and parental care.

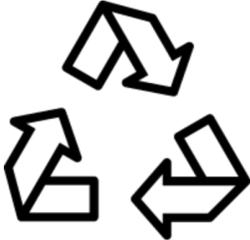
Experimental results across diverse taxa show that increase in parental investment may give rise to better immunocompetence (Roth et al., 2011; Lin et al., 2016; Peck et al., 2016; Keller et al., 2017). Chapter 4 presents my final PhD project which was pursued to study sexual dimorphism in immune response in relation to parental investment provided. The manuscript (V.R Venkateswaran, C.S Gokhale, M. Mangel and S. Eliassen. *The effect of parental investment on immunocompetence and sexual immune dimorphism*) is attached in this chapter. In this study, I used state-dependent dynamic programming where an individual allocates its resources towards maintaining the two traits: parental investment and immune response i.e. there is a competition for resources between these two traits. An optimal allocation will grant maximum fitness (reproductive success). Various situations of trade-offs in allocating resources towards parental investment and immunity follow for different species systems. This allows us to understand the effect of parental investment on immune responses. Our results (discussed in detail in Chapter 4) show that for most cases, when an individual spends more time in pregnancy or brooding period, it allocates more resources towards its own immunity. The methods and results are presented in Chapter 4.

The overarching conclusion of this thesis is that studying coupled interactions between individuals or traits within individuals is necessary to obtain more accurate and meticulous results. This is because our results show that combined interactions' dynamics differs from studying an isolated interaction. The dynamics differ in terms of trajectories of frequencies of individuals of every type and the equilibrium values for the frequencies, both. Coupling even a neutral interaction to another interaction affects their dynamics.

I also successfully applied the multiple interactions approach to interactions between life-history traits. By doing so, I was able to investigate how one trait affects another and also how the combined dynamics of the traits can result in population-level consequences.

# Thesis Overview

## Chapter 2: Many Interactions' Combined Dynamics



**Questions:** Is it possible to combine diverse multiplayer interactions and study the resulting dynamics?  
How does looking at the combined dynamics differ from looking at each interaction in isolation?

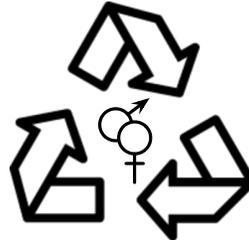
**Tools:**  
Evolutionary game theory, stochastic Moran process.

**Results:** I constructed a mathematical framework that can combine many multiple multiplayer games (interactions) with a diverse set of strategies in each game.

Combined game dynamics differs from single game dynamics in terms of trajectories of frequencies of individuals of each strategy and equilibrium values, both.

'Addition' of even a neutral game to another game, affects its dynamics.

## Chapter 3: Dynamics of Combined Interactions' between Life-History Traits



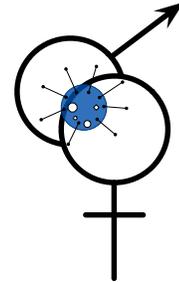
**Question:** What is the outcome of the combined dynamics of multiple life-history traits' (here, parental investment, ornamentation immune response) dynamics that have sex-specific differences.

**Tools:**  
Population genetics, evolutionary game theory, frequency dependent fitnesses models of sexual selection that measure reproductive success.

**Results:** A bias in the adult sex ratio emerges as an outcome of the combination of life-history traits with sex-specific differences.

An increase in immune heterozygotes within one sex as compared to the other can also evolve. So this sex may show a higher immune response on average (due to the heterozygous advantage i.e. having diverse immune alleles helps recognise more pathogens).

## Chapter 4: Influence on one Life-History Trait by another



**Question:** How does sex-specific differences occur in one life-history trait (here, immune response) due to its interaction with another trait (parental investment).

**Tools:**  
State-dependent dynamic modeling.

**Result:** Species or sex-specific differences in the time spent in parental investment has an effect on the optimal investment towards immunity.

For most species systems, it is optimal to invest more in immunity when the time spent in parental investment is longer.

The sex that can undergo pregnancy or brooding periods, invests more towards its immunity.

Figure 1.1: Overview of the thesis. Chapter 2 presents a mathematical model that I developed to study combined evolutionary interaction' dynamics. Two important outcomes from this model were applied to two other models which I conceived: 1) to investigate lifetime reproduction success as well as its consequences for the two sexes (Chapter 3), and 2) the emergence of sex-specific differences in life-history traits due to the interactions (here, competition for resources) between them (Chapter 4).



# Chapter 2

## Evolutionary dynamics of multiple complex interactions

“The most extraordinary thing about trying to piece together the missing links in the evolutionary story is that when you do find a missing link and put it in the story, you suddenly need all these other missing links to connect to the new discovery. The gaps and questions actually increase - it’s extraordinary.”

---

— David Attenborough

### 2.1 Introduction to evolutionary games

Evolutionary game theory or EGT (von Neumann and Morgenstern, 1944; Maynard Smith and Price, 1973) has been applied for studying phenotypes such as sex ratio, social interaction, hunting

techniques, allocation of resources between male and female functions in a hermaphrodite species. Evolutionary games are representative of interactions between individuals. In EGT, a set of players is considered where each player has a fixed strategy while they interact in a game. The strategies are the different phenotype trait values. When a player interacts with another player, that individual gets a payoff. The average payoffs contribute to fitness. To illuminate this idea, let us look at an example of a contest between Hawk (aggressive behavior) and Dove (calmer behavior and retreats when opponent's aggressiveness escalates) for a resource  $V$ . The cost of injury is  $C$ . When a Hawk meets Hawk, each shares a 50% chance of obtaining the resource and 50% chance of injury i.e. their payoff is  $(V - C)/2$ . When Hawk interacts with Dove, it gets a high payoff ( $V$ ) and when Dove meets Hawk, the payoff is 0 i.e. its fitness does not increase while interacting with the Hawk. When two Doves interact, they share the resource, leaving each with a payoff equal to  $V/2$ .

In almost every scenario, only one game is considered; i.e. individuals can play only one game at an instant or during a certain period. However, in nature is it highly likely for an individual to play more than one game with the same or different groups of individuals, not applying the same strategy in every game. An individual might perform different roles in various games. There also exist scenarios where a division of labor takes place (Wahl, 2002). Organisms have a range of tasks to execute in various social settings. Thus, the average payoffs should depend on all games. Our framework incorporates all these possibilities; to get closer to modeling the world around us more accurately. Realistic situations can be applied in this model to explain them better. Take the case of humans interacting in public goods games such as climate change issues (Milinski et al., 2006). These games usually consist of Cooperators (C) and Defectors (D). The Cooperators contribute a cost  $c$  for the public good but the defectors do not contribute anything. There are different versions of such public goods games where we could have additional strategies such as Punisher, Loner (Hauert et al., 2002). When individuals participate in many public good

games, the contribution of say, a Cooperator need not be similar in all the games. Their payoff will depend on all the games they are associated with (Santos et al., 2008).

A multiple games model could also be used for explaining how the avirulent 'cheater' trait has been evolutionarily stable among fellow subpopulations of virulent 'cooperators' in intestinal pathogen *S. typhimurium* population. It has been observed that the avirulent types are useful in the 'maintaining cooperative virulence' game (Diard et al., 2013). Likewise, in *Pseudomonas fluorescens* communities, the seemingly destructive cheating cells can promote evolution of collectives (Hammerschmidt et al., 2014). So a simple Cooperator-Defector game cannot be the only game that should be considered while modeling such situations. Over time, the individuals play other games which may also depend on the population composition and size.

Previous studies on multi-game dynamics (Fig. 2.1) have shown that a combination of games with more than two strategies cannot be separated into its constituent single game dynamics (Hashimoto, 2006). However, these results are restricted to two-player games. When more players are involved, different dynamics emerge (Pacheco et al., 2009; Gokhale and Traulsen, 2010; Peña, 2012). For instance, consider lionesses who live in fusion-fission pride. These lionesses defend their territory against other invading lionesses. They need at least two individuals to form a territory for breeding, rearing cubs in crèches, protecting each other and their cubs from invading males, etc. So we would expect all the members to cooperate during an attack on their holding. However, it was observed that not all females cooperate in this territory defending game (Heinsohn and Parker, 1995). Some lionesses (the leaders) always lead the defense. The others lag behind. If we merely look at this territory protection as a simple Prisoners' Dilemma game, these laggards may end up being classified as defectors. The leaders could turn into defectors too or they could be more prone to injury/death as they are at a higher risk. Eventually, this could be deleterious for cooperation and could lead to what is commonly known as the tragedy of commons. But we do not see this happening since, at a given point in time, they are not

only players in the territory game. It might not be ideal to ‘judge’ the payoffs of an individual by looking at this game in isolation. They could also be mothers (or involved in communal rearing) or wings/centers in a hunting game (Stander, 1992) and their collective payoffs from all the games should be considered to analyze the dynamics. The territory defense by female lions is beyond a single iterated Prisoners’ Dilemma game.

We deduce that individuals have a particular role in the multiple games they play and the payoffs could be dependent on each other. Therefore, we need to look at multiplayer multi-game dynamics. A complete picture of multiple game dynamics, where many players are involved, was lacking and the aim of our project was to capture that (see Fig. 2.1) and apply it to biological scenarios. If multiple players and strategies are involved, then can the multiple-game dynamics be decomposed back into its constituent single game dynamics? If yes, the conclusions drawn from individual games are valid. If not, it will be necessary to use multi-game dynamics to obtain realistic results. To answer this question, we developed a method to study combinations of symmetric multiplayer games and provide an analytical framework for analyzing an ensemble of games in a tractable manner. We present a complete and general method to study multiple games with many strategies and players, all at once.

### 2.1.1 Single evolutionary game dynamics

Consider a  $2 \times 2$  payoff matrix (2.1). There are two players and the two types of strategies they could employ are 1 and 2. The frequencies of the individuals with strategies 1 and 2 are  $x_1$  and  $x_2$ , respectively.

$$\begin{array}{cc}
 & \begin{array}{cc} 1 & 2 \end{array} \\
 \begin{array}{c} 1 \\ 2 \end{array} & \left( \begin{array}{cc} a_{1,(1,0)} & a_{1,(0,1)} \\ a_{2,(1,0)} & a_{2,(0,1)} \end{array} \right)
 \end{array} \tag{2.1}$$

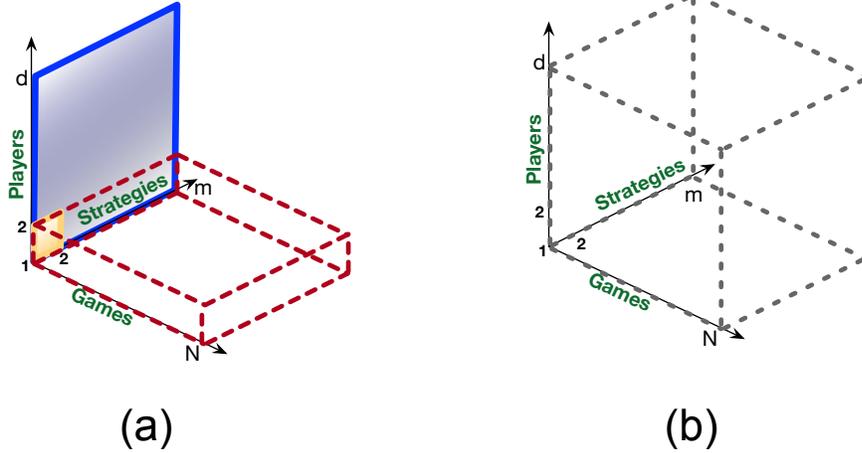


Figure 2.1: **Scope of this study.** (a) Typical evolutionary game dynamics focuses on two player games with two strategies (solid yellow cube). Extensions to multiplayer games ( $d$ ) and multiple strategies ( $m$ , solid blue rectangle) expands the domain of study to public goods games and other social dilemmas. However this is still limited to a single game. Hashimoto (2006) has extended two player-multi-strategy games in a novel direction of multiple games ( $N$ , dotted red rectangle). (b) Our work generalizes this approach and develops a method for analysing multiple games, where each involved game could be a multiplayer (and multi-strategy) game. Thus, this approach enables us to study the entire space of multiple games ( $N$ ) with multiple strategies ( $m$ ) consisting of multiple players ( $d$ ).

In matrix 2.1, we write the elements in the form  $a_{i,\alpha}$ , where  $i$  is the strategy of the focal player.  $\alpha$  (using multi-index notation) is a vector written as  $\alpha = (\alpha_1, \alpha_2)$ .  $\alpha_1$  and  $\alpha_2$  together represent the group composition i.e. the number of strategy 1 and strategy 2 players. The average payoffs of the two strategies are given by  $f_1 = a_{1,(1,0)}x_1 + a_{1,(0,1)}x_2$  and  $f_2 = a_{2,(1,0)}x_1 + a_{2,(0,1)}x_2$ . The replicator equation (2.2) (Hofbauer and Sigmund, 1998; Nowak, 2006) describes the change of a frequency  $x_i$  of strategy  $i$  over time.

$$\dot{x}_i = x_i[(f_i - \phi)] \quad (2.2)$$

where  $f_i$  is the fitness of strategy  $i$  and  $\phi$  is the average fitness. Here, in this  $2 \times 2$  game, the

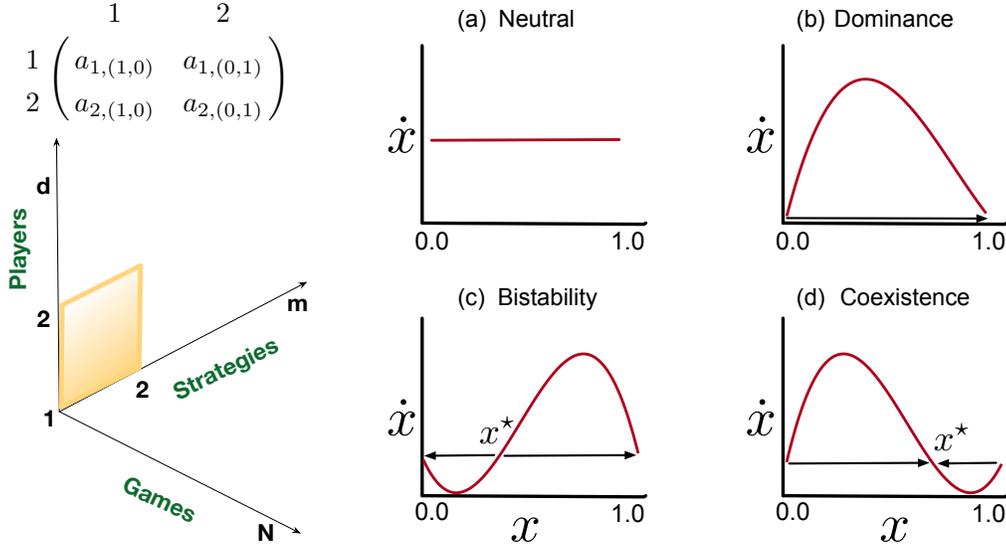


Figure 2.2: **Simplest Evolutionary Game: Two player games with two strategies.** For a payoff matrix of the form as shown in matrix (2.1), there can be four possible cases of replicator or evolutionary dynamics. This depends on the magnitude and sign of the payoff matrix elements. Plotted here is the change in frequency of strategy 1 ( $x$ ) over time i.e.  $\dot{x}$  for all  $x$ . **(a)** When  $a_{1,(1,0)} = a_{2,(1,0)}$  and  $a_{1,(0,1)} = a_{2,(0,1)}$  we see no dynamics. This means that all strategies are equally selected for as they have the same fitness. Thus we see no change in the frequencies of strategy 1 and 2 in the population. **(b)** When  $a_{1,(1,0)} > a_{2,(1,0)}$  and  $a_{1,(0,1)} > a_{2,(0,1)}$  the whole population becomes strategy 1 individuals. **(c)** When  $a_{1,(1,0)} < a_{2,(1,0)}$  and  $a_{1,(0,1)} > a_{2,(0,1)}$ , the equilibrium  $x^*$  is unstable and we see bistability. When we start from an initial condition above the value of  $x^*$ , the population becomes all strategy 1 individuals. If we start below  $x^*$ , the population will be full of strategy 2 individuals. **(d)** When  $a_{1,(1,0)} > a_{2,(1,0)}$  and  $a_{1,(0,1)} < a_{2,(0,1)}$ , the equilibrium  $x^*$  is stable and there will be coexistence of the two types in the population.

replicator equations for strategies 1 and 2 give

$$\begin{aligned} \dot{x} &= x(1-x)(f_1 - f_2) \\ &= x(1-x)[(a_{1,(1,0)} - a_{1,(0,1)} - a_{2,(1,0)} + a_{2,(0,1)})x + a_{2,(1,0)} - a_{2,(0,1)}]. \end{aligned} \quad (2.3)$$

where  $x_1 = x$ ,  $x_2 = 1 - x$ . Apart from the trivial fixed points ( $x = 0$  and  $x = 1$ ), there is an internal equilibrium given by,

$$\begin{array}{c}
R \\
P \\
S
\end{array}
\begin{pmatrix}
R & P & S \\
0 & -a_2 & b_3 \\
b_1 & 0 & -a_3 \\
-a_1 & b_2 & 0
\end{pmatrix}
\begin{array}{l}
x_1 = \text{frequency of Rock} \\
x_2 = \text{frequency of Paper} \\
x_3 = \text{frequency of Scissor}
\end{array}$$

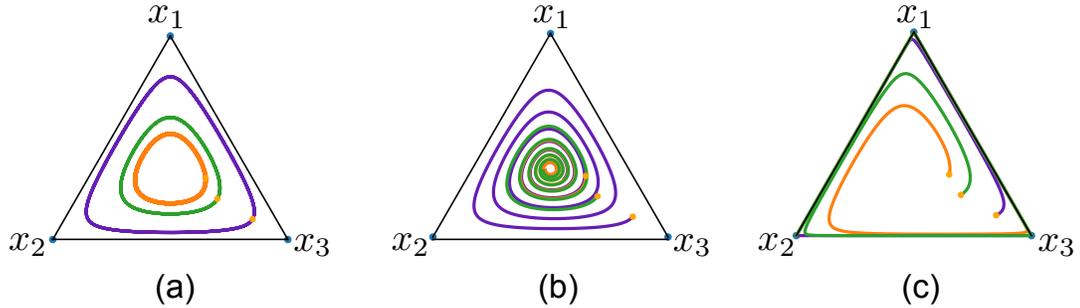


Figure 2.3: **A two player game with three strategies: Rock-Paper-Scissors game.** To illustrate a  $3 \times 3$  game or two player game with three strategies, let us consider the famous childhood game: Rock-Paper-Scissors, where Paper beats Rock, Scissor beats Paper and in turn Rock beats Scissor. A strategy against itself brings about a tie; gives no yield. Thus the general payoff matrix for R-P-S can be written as shown in the matrix. **(a)** Case 1: This is a zero-sum game as the elements of the matrix fulfill the condition  $a_{ij} = -a_{ji}$ . The unique equilibrium stable solution is given by  $(1/3, 1/3, 1/3)$ . This point is not asymptotically stable. Starting from different initial conditions will lead to different periodic orbits around this point. **(b)** Case 2 is when the determinant of the matrix is positive i.e.  $a_1a_2a_3 < b_1b_2b_3$ . Then the trajectories starting from any initial condition will converge to a unique stable equilibrium. **(c)** Case 3 is when the determinant of the matrix is negative i.e.  $a_1a_2a_3 > b_1b_2b_3$ . Then the unique interior equilibrium is unstable. All trajectories will converge to the boundary with increase in amplitude in each cycle.

$$x^* = \frac{a_{2,(0,1)} - a_{2,(1,0)}}{a_{1,(1,0)} - a_{1,(0,1)} - a_{2,(1,0)} + a_{2,(0,1)}}. \tag{2.4}$$

Depending on the payoff matrix values one can obtain different evolutionary dynamics as shown in figure 2.2.

One can extend this replicator dynamics framework for games with multiple strategies. Figure 2.3 shows an example of a three strategy game. Equation 2.2, in this case, will have to be

solved for  $i \in \{1, 2, 3\}$ . However, since the sum of the frequencies of the three strategies  $x_1 + x_2 + x_3 = 1$ , it is enough to obtain the dynamics of  $x_1$  and  $x_2$ . The value of  $x_3$  would then just be  $1 - (x_1 + x_2)$ . The dynamics can be projected into a mathematical concept called a *simplex*. The dynamics of a three strategy game can be projected by a two dimensional  $S_3$  simplex i.e. a triangle where each vertex corresponds to a strategy. The plots in figure 2.2 for a two strategy were in fact one dimensional  $S_2$  simplexes. So for a game with  $m$  strategies, the dynamics will be an  $m - 1$  dimensional  $S_m$  simplex.

### Multiplayer games with multiple strategies

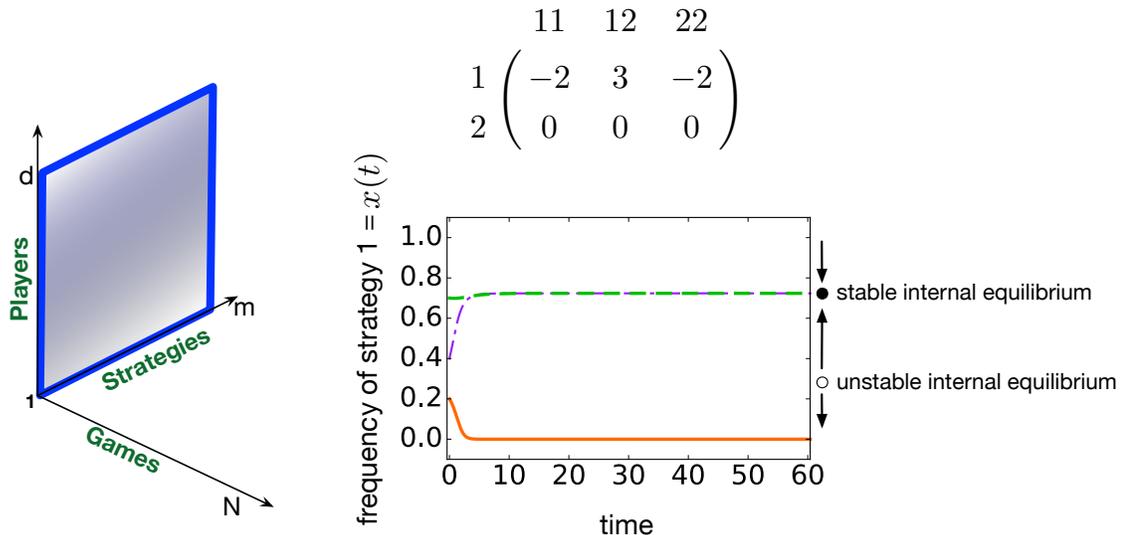


Figure 2.4: **Illustration of a multiplayer game with two strategies.** The matrix shown in this figure is an example of a three player two strategy game. If the focal (row) player adopts strategy 1, then it can interact with two other players having strategy 1 each, strategy 2 each, or one opposing player can adopt strategy 1 and the other can adopt strategy 2. The same applies when the focal player adopts strategy 2. Since we have three players, the number of internal fixed points (non-trivial solutions) have increased to two as shown in Gokhale and Traulsen (2010). One of these is a stable equilibrium solution and other is unstable.

Previously, we looked at pairwise interactions. One individual interacts with another by adopting one of the two strategies. We now extend the dynamics to multiplayer games where we can

have more than two players. The payoff matrix (2.5) represents a three player ( $d = 3$ ), two strategy ( $n = 2$ ) game; a  $2 \times 2 \times 2$  game.

$$\begin{array}{ccc} & 11 & 12 & 22 \\ \begin{array}{l} 1 \\ 2 \end{array} & \left( \begin{array}{ccc} a_{1,(2,0)} & a_{1,(1,1)} & a_{1,(0,2)} \\ a_{2,(2,0)} & a_{2,(1,1)} & a_{2,(0,2)} \end{array} \right) & \end{array} \quad (2.5)$$

The rows correspond to the focal player. Focal player with strategy 1 interacting with two other players with strategies 1 and 1 will receive a payoff  $a_{1,(2,0)}$ . While interacting with strategy 1 and strategy 2 players, he will get  $a_{1,(1,1)}$ . When he interacts with two other individuals both playing strategy 2, he obtains a payoff equal to  $a_{1,(0,2)}$ . Similarly, we obtain payoffs for a focal strategy 2. The average payoffs (or in this case, the fitnesses) will be

$$\begin{aligned} f_1 &= x^2 a_{1,(2,0)} + 2x(1-x)a_{1,(1,1)} + (1-x)^2 a_{1,(0,2)} \\ f_2 &= x^2 a_{2,(2,0)} + 2x(1-x)a_{2,(1,1)} + (1-x)^2 a_{2,(0,2)}. \end{aligned} \quad (2.6)$$

The replicator equations give

$$\begin{aligned} \dot{x} &= x(1-x)((a_{1,(0,2)} - 2a_{1,(1,1)} + a_{1,(2,0)} - a_{2,(0,2)} + 2a_{2,(1,1)} - a_{2,(2,0)})x^2 \\ &\quad + (-a_{1,(0,2)} + a_{1,(1,1)} + a_{2,(0,2)} - a_{2,(1,1)})2x + a_{1,(0,2)} - a_{2,(0,2)}. \end{aligned} \quad (2.7)$$

Here for a  $d = 3$  or three player game, the quadratic  $x^2$  term (in equation 2.7) will give rise to a maximum of two interior fixed points (non-trivial solutions) as shown in figure 2.4.

In general, for a  $d$ -player two strategy game, the replicator equation will not give just one but  $d - 1$  maximum interior fixed points, since the fitnesses  $f_1$  and  $f_2$  are polynomials of maximum  $d - 1$  degree. The number of interior fixed points will depend on the magnitudes of the payoff matrix elements.

For an  $n$  strategy  $d$ -player game, the maximum number of internal equilibria is  $(d-1)^{(n-1)}$  (Gokhale and Traulsen, 2010).

## 2.2 Manuscript: Multiple evolutionary game(s) dynamics

To study multiple evolutionary games dynamics, I constructed a mathematical framework that is a complete and general method to study multiple games with many strategies and players, all at once (Fig. 2.1) for infinite and finite populations. The results are exhibited in our paper (Venkateswaran and Gokhale, 2019).

To summarize the results from this paper, we found out that a combination of games (interactions) is not the same as the 'sum' of the individual games. For infinite populations, we found that the combined game(s) dynamics show different trajectories and different equilibrium points for the strategies as compared to their dynamics in the individual games. When the games have more than two strategies, we find that they cannot be separated back to their single game dynamics (SGDs), in line with previous findings. Interestingly, however, I found dependency on the initial conditions i.e., the initial frequencies of each strategy. For certain initial conditions, one may still be able to capture the SGDs from their multi-game dynamics (MGD).

This paper also has an analysis of the lionesses' territory defense question discussed in the introduction of this chapter, where a pride of lionesses is involved in many games. The combined game dynamics method explains the dynamics of an individual lioness and her pride more accurately. For finite populations, I derived an expression for calculating the fixation probability which is the probability with which a new mutant strategy can fixate in the population. I showed that the fixation probability is different for scenarios where fitness is calculated using payoffs at a certain time and temporal fitness i.e. fitness accumulated over time. I also show that combining even a neutral game to an existing game can lead to different combined dynamics (see Figure 2.5). The evolutionary many-player multi-game method and details of its results are presented in the following paper. The electronic supplementary material (ESM) of this paper is in Appendix A.

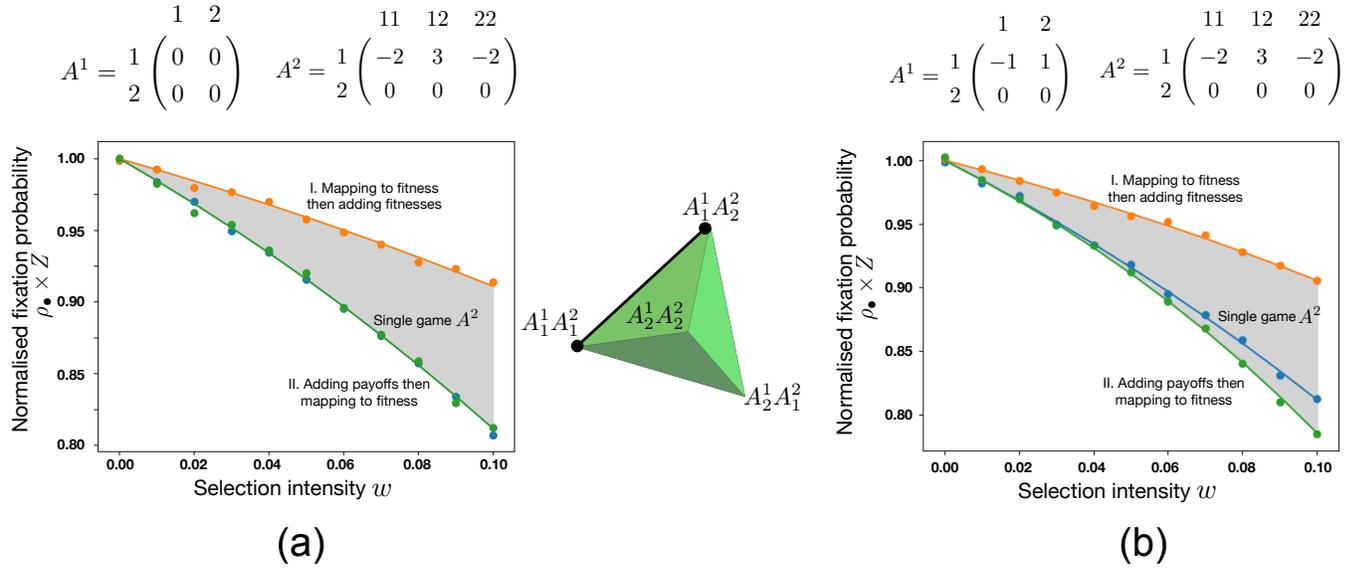


Figure 2.5: **Fixation probability of a single individual playing  $A_1^1 A_1^2$  strategy on the edge  $A_1^1 A_1^2 \rightleftharpoons A_1^1 A_2^2$  varying with selection intensity for a combination of two games having two strategies each.** The fixation probability is the probability with which a mutant strategy invades over the entire population. Here, we have a population that is divided into four types: playing strategy 1 in game  $A^1$  and game  $A^2$ , strategy 1 in  $A^1$  and 2 in  $A^2$ , strategy 2 in  $A^1$  and 1 in  $A^2$ . And finally, strategy 2 in  $A^1$  and  $A^2$ . Thus, we have four types of strategies,  $A_1^1 A_1^2$ ,  $A_1^1 A_2^2$ ,  $A_2^1 A_1^2$  and  $A_2^1 A_2^2$ . Since there are four ‘categorical types’, the dynamics is shown in an  $S_4$  simplex which is a tetrahedron. Here, we look at the edge  $A_1^1 A_1^2 \rightleftharpoons A_1^1 A_2^2$  (highlighted by a black line in the green tetrahedron) where  $A^1$  is the same for both vertices i.e. neutral in both the vertices, and  $A^2$  is what matters. The line labeled ‘single game’ corresponds to single game dynamics of  $A^2$ . The plots from Method I (mapping payoffs to fitnesses and then adding the fitnesses) and Method II (adding the payoffs first, and then mapping to fitness) for a combination of the two games  $A^1$  and  $A^2$ . The results from analytics and stochastic simulations are plotted as solid lines and solid circles, respectively. The simulations are averaged over  $10^6$  realizations. For a population of  $Z = 10$  the fixation probabilities are normalized according to the neutral fixation probability,  $\frac{1}{Z} = 0.1$ . **(a)** The payoffs in Game  $A^1$  are zero. Since the payoff of playing strategy 2 in  $A^2$  is greater than playing strategy 1, the fixation probability decreases. Since the payoff of playing strategy 1 and 2 in  $A^1$  are equal to zero, results from Method II and the single game dynamics (SGD) of  $A^2$  are the same. However, Method I shows a different result. Thus, adding another game  $A^1$  to  $A^2$  can modify the dynamics even when game  $A^1$  is neutral. **(b)** There are non-zero payoffs in Game  $A^1$ . For this combination of the two games  $A^1$  and  $A^2$ , the multi-game dynamics (MGD) differs from the SGD of  $A^2$  over which  $A^1$  is added. Within the MGD, the two methods of mapping from payoffs to fitness i.e. Method I and Method II differ from each other (shown by the shaded region). The difference is due to the different baseline payoffs that the different mappings produce.

Therefore, while looking at a combination of various games, there can be different methods of mapping and one needs to choose a mapping method that reflects their model best as they can bring about different results.



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# Evolutionary dynamics of complex multiple games

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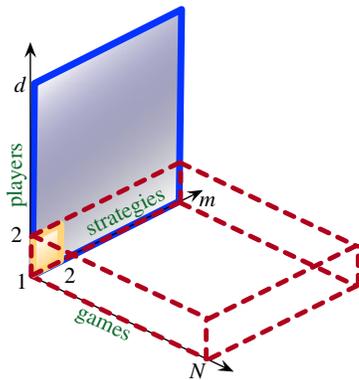
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Evolutionary game theory has been successful in describing phenomena from bacterial population dynamics to the evolution of social behaviour. However, it has typically focused on a single game describing the interactions between individuals. Organisms are simultaneously involved in many intraspecies and interspecies interactions. Therefore, there is a need to move from single games to multiple games. However, these interactions in nature involve many players. Shifting from 2-player games to multiple multiplayer games yield richer dynamics closer to natural settings. Such a complete picture of multiple game dynamics (MGD), where multiple players are involved, was lacking. For multiple multiplayer games—where each game could have an arbitrary finite number of players and strategies, we provide a replicator equation for MGD having many players and strategies. We show that if the individual games involved have more than two strategies, then the combined dynamics cannot be understood by looking only at individual games. Expected dynamics from single games is no longer valid, and trajectories can possess different limiting behaviour. In the case of finite populations, we formulate and calculate an essential and useful stochastic property, fixation probability. Our results highlight that studying a set of interactions defined by a single game can be misleading if we do not take the broader setting of the interactions into account. Through our results and analysis, we thus discuss and advocate the development of evolutionary game(s) theory, which will help us disentangle the complexity of multiple interactions.

## 1. Introduction

Evolutionary game theory [1–4] has been used to study phenomena ranging from the dynamics of bacterial populations to the evolution of social behaviour. In evolutionary games, individuals are cast as players that interact with each other in ‘games’, which are metaphorical summaries of interactions. For example, in the classical Prisoners’ dilemma, individuals can either cooperate or defect, and each pairwise interaction results in a payoff for the players involved [3,5]. Over time, players adopt a strategy which either performs better or worse than the average of the population and thus increases or decreases in frequency. Tracking the change in their frequencies over time, evolutionary dynamics can provide insight into the eventual fate of the strategies in a game, e.g. whether they dominate, coexist, or go extinct from the population [3].

Considerable effort has gone into making games more realistic (with interactions among multiple players and allowing players to adopt strategies from a large set [6,7]) shown by the solid blue rectangle in figure 1. As an example from the micro-scale, we discuss the interactions between microorganisms. One bacterium interacts with its neighbours. Assuming that a bacterium would interact only in a pairwise fashion is clearly an assumption. When more players are involved, dynamics can change not just quantitatively but qualitatively [9–11]. Multiplayer games in bacterial dynamics can better explain the coexistence of avirulent ‘cheaters’ and virulent ‘cooperators’ in populations



**Figure 1.** Scope of this study. Typical evolutionary game dynamics focuses on 2-player games with two strategies (solid yellow square). Extensions to multi-player games ( $d$ ) and multiple strategies ( $m$ , solid blue rectangle) expands the domain of study to public goods games and other social dilemmas. However, this is still limited to a single game. Hashimoto [8] has extended 2-player multi-strategy games in a novel direction of multiple games ( $N$ , dotted red cuboid). Our work generalizes this approach and develops a method for analysing multiple games, where each involved game could be a multiplayer (and multi-strategy) game. Thus, this approach enables us to study the entire space of multiple games ( $N$ ) with multiple strategies ( $m$ ) consisting of multiple players ( $d$ ). (Online version in colour.)

of the pathogen *Salmonella* Typhimurium [12]. Likewise, in *Pseudomonas fluorescens* communities, the seemingly destructive cheating cells can promote evolution of collectives [13], an inherently multiplayer interaction. The dynamics between the microbes constituting the microbiome are nonlinear, lending themselves to multiplayer games [14]. A constituent of the microbiome may not be playing a single multiplayer game with the other constituents but is also interacting with the host. The complete interaction in the holobiont would then be a collection of several multiplayer games [15].

Do we consider all the different games singly or as one massive game with a large number of complex strategies? The answer in short is that under certain conditions, the single games studied individually do not provide the same results as when we infer single games from the combined dynamics. Across scales of organization, single games fail to satisfactorily capture dynamics ranging from bacterial dynamics (as above) to human behaviour. Envision the interactions in public goods games such as climate change issues [16]. When nations' leaders discuss strategies to improve the global climate status, they also need to take into account the interests of the people they are representing. If the leaders agree to contribute towards achieving the goals of the climate summit, it often comes at a cost to the private interests of the nation. Using a different set of strategies, the leaders have to then appease the electorate. Thus, political leaders are playing at least two multiplayer games: one with other nations and another within their nation. Therefore, we need to shift from single game dynamics (SGD) to multiple game dynamics (MGD) as shown by the dotted red cuboid in figure 1. Previous studies on MGD have shown that a combination of games with more than two strategies is inseparable into its constituent SGD [8]. However, this result is valid only for 2-player games as shown in the figure. It ignores the complexity of multiplayer games as discussed above. We have developed a method for analysing multiplayer MGD.

Besides ecological examples, formal analysis of evolutionary games in finite populations implies the role of multiple games. The assumption of weak selection, where the game has a weak effect on an organism's fitness, typically is done not only for mathematical ease but also assuming that, the payoff differences are small, the strategies are similar, or the individuals are confused about the strategies [17]. Multiple games provide a simpler alternative where each game has a small effect on an individual's fitness.

A complete picture of MGD, where multiple players are involved, is lacking. Nonlinearity in the replicator dynamics increases with increasing number of players. As a result, multiplayer games can have multiple internal equilibria as opposed to 2-player games that have at most one internal equilibrium solution [10]. An initial condition within the MGD space can converge to another equilibrium solution than expected from the SGD. Thus, if we are aware that the dynamics are composed of a set of different games, then is the simplified use of a single bigger game justified? In other words, can the MGD be decomposed into its constituent SGDs? If yes—the conclusions drawn from individual games are valid. If not—it will be necessary to use MGD to obtain realistic results.

To answer this question, we first present a complete and general method to study multiple games with many strategies and players, all at once (figure 1). When the games have more than two strategies, we find that the MGD do not correspond to the dynamics of its constituent single games, in line with previous findings, while we also extend the analysis to finite populations. Then we discuss a specific model on how the inclusion of two different games (territorial defence and hunting) can result in the observed division of labour in lionesses [18,19]. Further, we show that for some initial conditions the MGDs and SGDs differ not only in the dynamics but the resulting equilibria as well.

## 2. Model

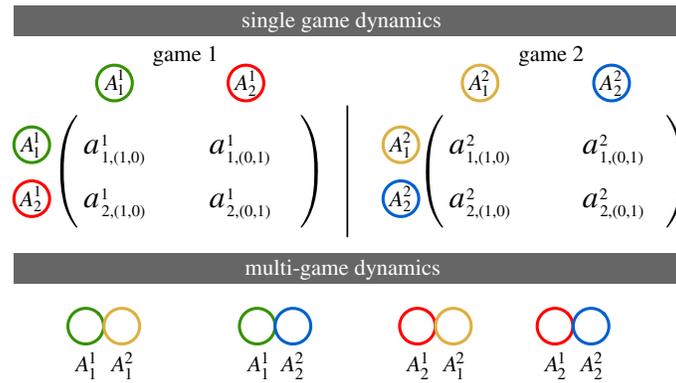
### (a) Single game dynamics

2-player games with two strategies have been studied extensively, both in infinite as well as finite populations. A game between two individuals can be represented by the following payoff matrix:

$$\begin{matrix} & \begin{matrix} 1 & 2 \end{matrix} \\ \begin{matrix} 1 \\ 2 \end{matrix} & \begin{pmatrix} a_{1,(1,0)} & a_{1,(0,1)} \\ a_{2,(1,0)} & a_{2,(0,1)} \end{pmatrix} \end{matrix} \quad (2.1)$$

The matrix represents a symmetric 2-strategy 2-player game. We do not study asymmetric games [20]. The two individuals, focal and co-player are represented by a row and a column, respectively. Each player adopts one of the two strategies, 1 or 2. We write the elements of the matrix in the form  $a_{i,\alpha}$  where  $i$  is the strategy of the focal (or row) player. The vector  $\alpha$  is written as  $\alpha = (\alpha_1, \alpha_2)$  where  $\alpha_i$  indicates the number of strategy  $i$  individuals the focal individual interacts with. For example, in a 3-player game with two strategies, the payoff entry  $a_{2,(1,1)}$  corresponds to a focal player with strategy 2 interacting with two other players with strategies 1 and 2, respectively.

The average payoff obtained from the game is the reproductive success of that strategy [21]. This analysis has been extended to interactions having *multiple* strategies [22] as well as *multiple* players [23,24]. To make our notation clear,



**Figure 2.** From SGD to MGD. The population after combination is divided into four types: playing strategy 1 in game  $A^1$  and game  $A^2$ , strategy 1 in  $A^1$  and 2 in  $A^2$ , strategy 2 in  $A^1$  and 1 in  $A^2$ . And finally, strategy 2 in  $A^1$  and  $A^2$ . Thus, we have four types of strategies,  $A_1^1 A_1^1$ ,  $A_1^1 A_2^2$ ,  $A_2^1 A_1^2$ , and  $A_2^2 A_2^2$ . Their respective frequencies are  $x_{11}$ ,  $x_{12}$ ,  $x_{21}$ , and  $x_{22}$ . Since there are four 'categorical types', the dynamics is shown in an  $S_4$  simplex. (Online version in colour.)

we illustrate a payoff matrix for a multiplayer ( $d$  player) game with two strategies as

no. of opposing strategy 1 players	$d-1$	$d-2$	...	$k$	...	0
1	$a_{1,(d-1,0)}$	$a_{1,(d-2,1)}$	...	$a_{1,(k,d-1-k)}$	...	$a_{1,(0,d-1)}$
2	$a_{2,(d-1,0)}$	$a_{2,(d-2,1)}$	...	$a_{2,(k,d-1-k)}$	...	$a_{2,(0,d-1)}$

(2.2)

Even when extending the number of strategies, the dynamics of this complicated system can still be analysed by the replicator dynamics [25,26]. For a  $d$  player game with  $m$  strategies, the replicator dynamics is given by a set of  $m$  differential equations:  $\dot{x}_i = x_i(f_i - \phi)$  where  $x_i$  is the frequency of strategy  $i$ , and  $f_i$  is the average payoff of the strategy  $i$ . The average payoff of the population is given by  $\phi = \sum_{j=1}^m x_j f_j$ . This simple evolutionary game framework has been used to describe a wide range of phenomena from chemical reactions of prebiotic elements to the evolution of social systems [27].

While this extension to multiple players and strategies is not trivially obtained [28], it still belongs to the domain of a single game. The framework lacks the ability to incorporate interactions which have differential impacts on fitness. Therefore, we now incorporate multiple games and measure their cumulative impact on individual fitness.

### (b) Multi-game dynamics

Individuals may employ different strategies in various games (e.g. division of labour scenarios [29]) and their (average) payoffs will depend on their performance in all such games. Switching between such socially driven games is realistic and not only a matter of theoretical interest but has been experimentally explored as well [30]. This section generalizes the multi-game approach by Hashimoto [8] to an arbitrary number of players. To contrast MGD with the previously discussed SGD, consider a simple example of two, 2-player games, each having two strategies:

$$A^1 = \begin{matrix} & A_1^1 & A_2^1 \\ A_1^1 & \begin{pmatrix} a_{1,1(1,0)} & a_{1,1(0,1)} \\ a_{1,2(1,0)} & a_{1,2(0,1)} \end{pmatrix} & A_2^1 \end{matrix} \quad \text{and} \quad A^2 = \begin{matrix} & A_1^2 & A_2^2 \\ A_1^2 & \begin{pmatrix} a_{1,1(1,0)} & a_{1,1(0,1)} \\ a_{2,2(1,0)} & a_{2,2(0,1)} \end{pmatrix} & A_2^2 \end{matrix}.$$

Combining the strategies from the above two games results in four categories of individuals. The frequencies of the four categories are given by  $x_{11}$ ,  $x_{12}$ ,  $x_{21}$ , and  $x_{22}$  where the first and

second positions (in the subscript) denote the strategies adopted in games 1 and 2, respectively (figure 2).

For a combination of  $N$  games, each game  $j$  can be described by a payoff matrix  $A^j$ . Each game  $j$  could be a  $d_j$  player game with  $m_j$  number of strategies. The categorical frequencies would then be given by  $x_{i_1 i_2 \dots i_N}$ , where  $i_j$  is the strategy being played in game  $j$ . The frequencies of the individual strategies for all  $N$  games can be written down as

$$p_{j i_j} = \sum_{k=1, k \neq j}^{k=N} \sum_{i_k=1}^{m_k} x_{i_1 i_2 \dots i_N} \quad (2.3)$$

which allows us to compute the fitness of strategy  $i_j$  as

$$f_{j i_j} = \sum_{|\alpha|=d_j-1} \binom{d_j-1}{\alpha} p^\alpha a_{i_j, \alpha}^j \quad (2.4)$$

As before,  $\alpha_{m_j}$  is the number of strategy  $m_j$  players. Using multi-index notation, we have  $\alpha = (\alpha_1, \alpha_2, \dots, \alpha_{m_j})$  which gives us the multinomial coefficient, with the absolute value  $|\alpha| = \alpha_1 + \alpha_2 + \dots + \alpha_{m_j}$  and the power  $p^\alpha = p_{j1}^{\alpha_1} p_{j2}^{\alpha_2} \dots p_{jm_j}^{\alpha_{m_j}}$ . The average fitness of the population is given by,  $\phi_j = (\mathbf{p}\mathbf{f})_j$ . Using this, we can write down the time evolution of all the categorical strategies as

$$\dot{x}_{i_1 i_2 \dots i_N} = x_{i_1 i_2 \dots i_N} \left( \sum_{j=1}^N (f_{j i_j} - \phi_j) \right) \quad (2.5)$$

This system of equations is reminiscent of the replicator equation for the SGD. The summation in the MGD replicator equations is due to an assumption of additive fitness effects from all games [8]. In the following sections, we will explore the use of this formulation for multiple games where each game can have a different number of players. Through the examples of specific cases, we aim to highlight the general principles of multiple games.

### 3. Results

#### (a) Multiplayer game(s) with multiple strategies

Combining multiplayer games, frequency feedback between strategies is possible. Moreover, an individual can take part in different interactions. A lioness can be part of forming the defensive line (tragedy of the commons) and hunting (stag–hunt game). Strategies in game 1 would be *Cooperator*, *Defector*, *Loner*, etc. Strategies in game 2 could be hunting positions *Wing*, *Centre*, and so on. Thus in our framework, an individual can have utterly different strategy sets for each game.

#### (i) 2-player game with 2-strategies + 3-player game with 2-strategies.

To illustrate games with two strategies, we shall use the payoff matrices shown in (3.1).

$$A^1 = \frac{1}{2} \begin{pmatrix} 1 & 2 \\ -1 & 1 \\ 0 & 0 \end{pmatrix} \quad \text{and} \quad A^2 = \frac{1}{2} \begin{pmatrix} 11 & 12 & 22 \\ -2 & 3 & -2 \\ 0 & 0 & 0 \end{pmatrix}. \quad (3.1)$$

Here,  $A^1$  is a 2-player coexistence game and  $A^2$  is a 3-player game. In  $A^2$ , the values  $a_{1,(k,d-1-k)} - a_{2,(k,d-1-k)}$  and  $a_{1,(k+1,d-k)} - a_{2,(k+1,d-k)}$  have different signs for all  $k$ . Thus solving for this scenario using our replicator-like equation (2.5), we have two interior fixed point solutions: a stable and an unstable. The equilibrium solutions for strategy 1 in the two SGDs in (3.1) are  $q_1^* = 0.5$  for  $A^1$  and  $q_2^* = (q_{2_1}^*, q_{2_2}^*) = (0.27, 0.73)$  for  $A^2$ . Since  $A^2$  is a 3-player game, it has at most two internal equilibrium solutions [10]. The result of combining these games, i.e. their MGD, is shown in electronic supplementary material, figure A.4. The first panel shows the SGD of both the games  $A^1$  and  $A^2$ . We choose three initial conditions  $ic_1$ ,  $ic_2$ , and  $ic_3$  to understand the difference between SGD and MGD by following those trajectories' dynamics in the SGDs and MGD. After combining the two games with two strategies, we obtain the MGD that has four (categorical) strategies  $x_{11}$ ,  $x_{12}$ ,  $x_{21}$ , and  $x_{22}$ . The dynamics are plotted in a three-dimensional simplex. All trajectories that start above the unstable equilibrium in  $A^2$  end up in the line given by  $E$ , the evolutionarily stable (ES) set. As shown in the third panel of electronic supplementary material, figure A.4, one can recover the SGD back from their combined game dynamics to compare the MGD and SGDs, i.e. re-obtain  $p_{11}$  ( $= x_{11} + x_{12}$ ),  $p_{12}$  ( $= x_{21} + x_{22}$ ),  $p_{21}$  ( $= x_{11} + x_{21}$ ), and  $p_{22}$  ( $= x_{12} + x_{22}$ ). As shown by the dynamics in this figure, the MGD is the

same as the separate SGDs. So the MGD can be separated back into its constituent games when both games have two strategies.

#### (ii) 2-player game with 3-strategies + 3-player game with 2-strategies.

Next, we increase the number of strategies in the 2-player game:

$$A^1 = \frac{1}{2} \begin{pmatrix} 1 & 2 & 3 \\ 0 & -1 & 2 \\ 2 & 0 & -1 \\ -1 & 2 & 0 \end{pmatrix} \quad \text{and} \quad A^2 = \frac{1}{2} \begin{pmatrix} 11 & 12 & 22 \\ 10 & 1 & 5.5 \\ 4 & 10 & 3 \end{pmatrix}. \quad (3.2)$$

Now  $A^1$  is a Rock–Paper–Scissor game. Trajectories starting from any internal initial conditions converge to a unique stable equilibrium,  $q_1^* = (1/3, 1/3, 1/3)$  [3]. For  $A^2$ , the equilibrium solutions are  $q_{2_1}^* = 0.127$  (stable) and  $q_{2_2}^* = 0.740$  (unstable). The MGD takes place in a six-dimensional space, thus to compare the MGD with their SGDs we project them in the SGD space as shown in figure 3. The SGD for  $A^1$  and  $A^2$  are shown in the first panel. Since the two games,  $A^1$  and  $A^2$  have three and two strategies; respectively, their combined MGD will have six categorical strategies. The bottom panel displays the plots that compare the SGDs recovered from the MGD (dashed lines) with the original SGDs (solid lines). The recovered dynamics do not match that of the individual games. Thus, increasing the number of strategies in at least one game shows that the MGD differs from the SGDs. Therefore, while modelling multiplayer game scenarios with more than three strategies that involve individuals participating in multiple interactions simultaneously, one must look at their combined game dynamics to study the full picture [8]. We extend the domain of such multiplayer, multiple games analysis where both games have three strategies in the next section.

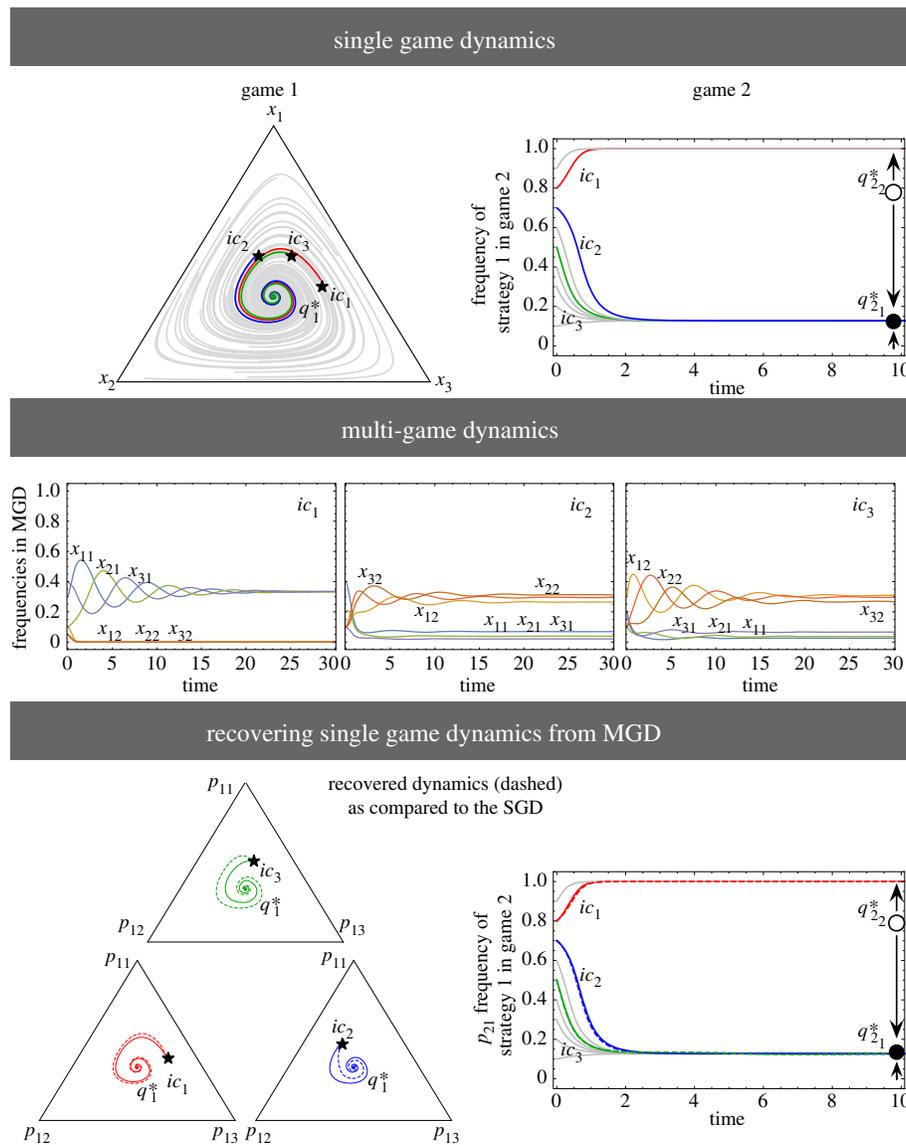
#### (iii) 2-player game with 3-strategies + 4-player game with 3-strategies.

Finally, we illustrate a case of having three strategies in both games (shown in matrices (3.3)).  $A^1$  is a Rock–Paper–Scissor game like the one discussed in the previous example.  $A^2$  is a 4-player 3-strategy game used previously in [10]. In the SGDs of the individual games,  $A^1$  has a stable equilibrium  $q_1^* = (1/3, 1/3, 1/3)$  and  $A^2$  has in total nine interior equilibrium solutions: four stable, one unstable, and four saddle points. The SGDs of  $A^1$  and  $A^2$  and their MGD are shown in figure 4.

$$A^1 = \frac{1}{2} \begin{pmatrix} 1 & 2 & 3 \\ -1 & 10 & -10 \\ -6 & -1 & 6 \\ 2 & -2 & -1 \end{pmatrix} \quad \text{and} \quad A^2 = \frac{1}{2} \begin{pmatrix} 111 & 112 & 113 & 122 & 123 & 133 & 222 & 223 & 233 & 333 \\ -9.30 & 3.83 & 3.86 & -1.03 & -1.00 & -0.96 & 0.10 & 0.33 & 0.16 & 0.20 \\ 0.10 & -1.03 & 0.13 & 3.83 & -1.00 & 0.16 & -9.30 & 4.06 & -0.96 & 0.2 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0.20 & 0 & 0 \end{pmatrix}. \quad (3.3)$$

The results show that in the MGD it is even possible for an initial condition to end up in a completely different equilibrium as opposed to the SGD.

Consider  $A^2$  which has four stable internal equilibria. In figure 4 top row, the three initial conditions go to three of the stable equilibria. After combining with  $A^1$  and then recovering

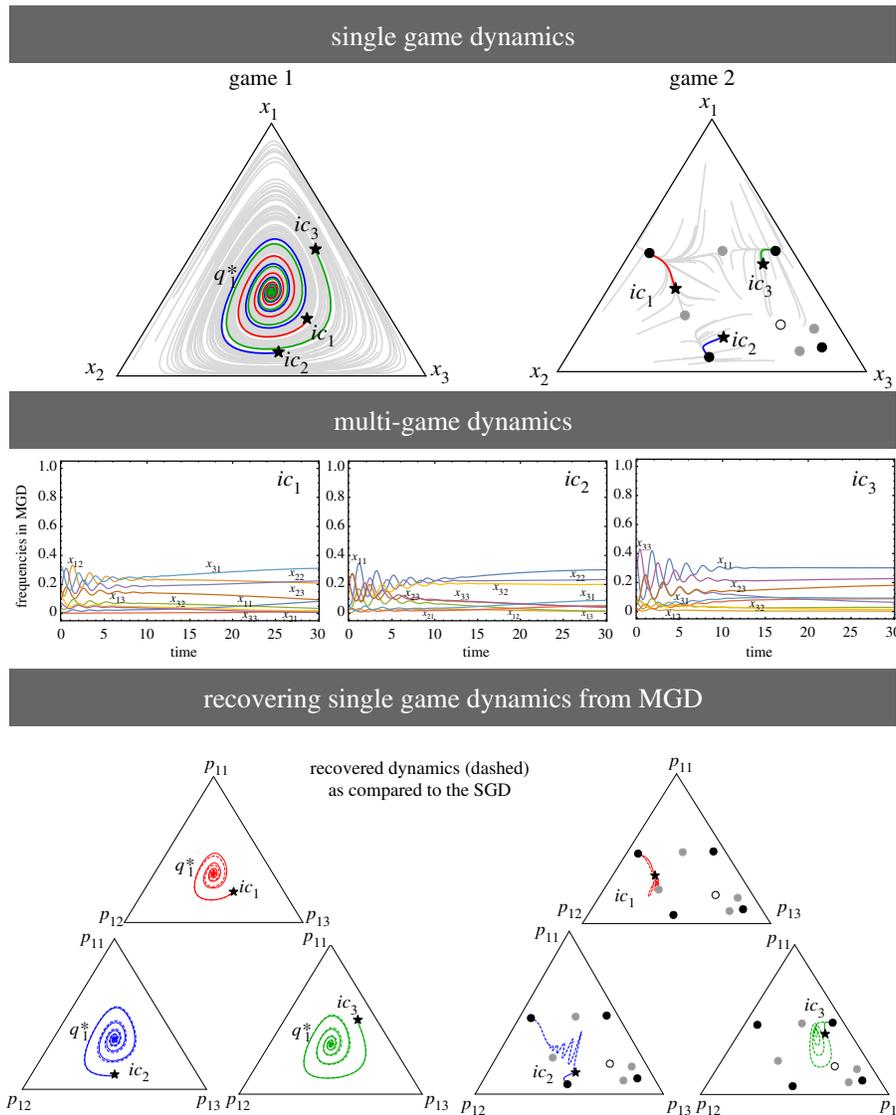


**Figure 3.** Two games, each having three and two strategies, respectively. The first row shows the SGD of the games in (3.2). Game 1 is a Rock–Paper–Scissor game with a stable internal equilibrium,  $q_1^* = (1/3, 1/3, 1/3)$ . Game 2 has two internal fixed points at  $q_{2_1}^* = 0.127$  (stable) and  $q_{2_2}^* = 0.740$  (unstable). The asterisks denote the positions from where the three trajectories  $ic_1$ ,  $ic_2$ , and  $ic_3$  begin (initial conditions). The grey trajectories are other random initial conditions. For the MGD, we have six ‘categorical types’  $x_{11}$ ,  $x_{12}$ ,  $x_{21}$ ,  $x_{22}$ ,  $x_{31}$ , and  $x_{32}$ . We plot the time evolution of the strategies for the three different initial conditions. From this MGD, we can recover the corresponding frequencies for the two SGD. These are plotted in the last row. The recovered  $p_{11}$  refers to playing strategy 1 in game 1,  $p_{21}$  refers to playing strategy 1 in game 2 and so on. All recovered trajectories (dashed) go to the same equilibria of the SGD in game  $A^1$  and in game  $A^2$  (solid). While the equilibria of the MGD correspond to that of the SGD, the dynamics can follow different routes. The initial conditions used for  $(x_{11}, x_{12}, x_{21}, x_{22}, x_{31}, x_{32})$  are:  $ic_1 = (0.3, 0.1, 0.1, 0.05, 0.4, 0.05)$ ,  $ic_2 = (0.4, 0.1, 0.2, 0.1, 0.1, 0.1)$ , and  $ic_3 = (0.2, 0.3, 0.1, 0.1, 0.2, 0.1)$ . (Online version in colour.)

the dynamics of  $A^2$ , we see that  $ic_2$  switches its long-term equilibrium behaviour (figure 4 bottom row, recovered dynamics). Multiplayer games offer the possibility of multiple internal equilibria and combined games can allow the trajectories to switch between them. Thus, the constituent games of an MGD, especially involving multiplayer games should be studied with scrutiny since their long-term evolutionary trajectory cannot be predicted by the basins of attractions of the SGD.

In a previous study of 2-player games with two strategies [31], it was shown that the SGD can be obtained back from their MGD. The dynamics lie on the generalized invariant *manifold* [25,32] in the  $S_4$  simplex which is given by  $W_K = \{x \in S_4 | x_{11} x_{22} = K x_{12} x_{21}\}$  for  $K > 0$ . When  $K = 1$ , we have  $W = \{x \in S_4 | x_{11} x_{22} = x_{12} x_{21}\}$  which is the *Wright manifold*. On this manifold, MGD can be separated back into the SGDs of the constituent

games (see the electronic supplementary material for details). The attractor for a combination of two 2-player games having two strategies each is a line  $E$ , an ES set [31]. The point where the line  $E$  intersects the Wright manifold indicates a rest point. All the trajectories in the simplex depicting the MGD fall onto an attractor given by a line (ES set) on  $W_K$ . The dynamics on  $W_K$  and the trajectories on each  $W_K$  were analysed in the same study [31] and the conditions when they are qualitatively the same as on the Wright manifold. However, for multiple games having more than two strategies in at least one game, the MGD cannot be separated even into a linear combination of the constituent SGDs unless they are on  $W$  [8]. Increasing the number of games and strategies increases the dimension of MGD simplex and also that of the Wright manifold. Only on the Wright manifold can the MGD be separated back into its



**Figure 4.** Two games having three strategies. In the SGDs of the individual games,  $A^1$  has a stable equilibrium solution  $q_1^* = (1/3, 1/3, 1/3)$  and  $A^2$  has in total nine interior equilibrium solutions: four stable (dark circles), one unstable (open circle), and four saddle points (grey circles). The asterisks in the triangular  $S_3$  simplex denote the initial conditions ( $ic_1$ ,  $ic_2$ , and  $ic_3$ ) of interest, whereas the grey trajectories are other random initial conditions. When both games contain three strategies, nine categorical types are possible. For visualizing the MGD, we show the time evolution of the nine strategies. Retrieving the distribution of frequencies of strategies in the SGDs from the MGD, for  $A^1$  again, while the equilibrium values are all the same  $q_1^*$ , the dynamics are different. However, for  $A^2$ ,  $ic_1$  and  $ic_3$  end up in the same equilibria in the MGD as in their respective SGDs,  $ic_2$  changes equilibrium. The initials conditions used for  $(x_{11}, x_{12}, x_{13}, x_{21}, x_{22}, x_{23}, x_{31}, x_{32}, x_{33})$  are:  $ic_1 = (0.01, 0.166, 0.038, 0.002, 0.176, 0.102, 0.3251, 0.111, 0.070)$ ,  $ic_2 = (0.058, 0.005, 0.029, 0.027, 0.205, 0.212, 0.050, 0.190, 0.224)$ , and  $ic_3 = (0.176, 0.066, 0.024, 0.002, 0.176, 0.002, 0.225, 0.111, 0.218)$ . (Online version in colour.)

SGDs (see the electronic supplementary material for details). Therefore, it is important to know on which manifold the initial conditions are, for only if they start from the Wright manifold  $W$ , will the dynamics be a perfect match to the SGDs [8].

Multiple multiplayer games can give rise to numerous rest points, and they can criss-cross with the Wright manifold which for multiple strategies would be of a dimension  $\sum_{j=1}^N (m_j - 1)$ , where  $N$  is the number of games and  $m_j$  is the number of strategies in game  $j$  (see the electronic supplementary material). Future work on multiple  $d$ -player games with many strategies could involve finding traversable paths in this complex space as is shown by some unusual trajectories (figure 4). Differing from the earlier work on 2-player multiple games [8,31], we show that MGDs cannot always be trivially separated into their constituent SGDs in multiplayer games with multiple strategies.

Furthermore, including multiplayer games in combined games can lead to the SGD and the recovered dynamics differing not just in the dynamics of trajectories but also in their eventual end points. We have a generalized method that looks at a combination of many multiplayer games having diverse strategy sets.

Until now, the analysis firmly rested on the deterministic dynamics and on the derivation and analysis of the replicator-like equation. This assumes an infinitely large population. To understand combined games in realistic finite populations, we turn our attention to stochastic methods.

### (b) Finite population

Evolutionary dynamics in finite populations has the potential of having qualitatively different dynamics than their

deterministic analogues [33]. In finite populations, the size of the population controls the balance between selection and drift with smaller populations showing higher levels of stochasticity. We use a birth–death Moran process to model a finite population of size  $Z$  in our framework [33,34]. An individual is chosen (proportional to its fitness) to reproduce an identical offspring. Another individual is chosen randomly for death. Thus, the total population size remains constant. Earlier we assumed that the fitness of a strategy was its average payoff. Besides the population size, we can control the effect of the game on the fitness via a particular mapping of payoff to fitness. The mapping could be a linear function  $f = 1 - w + w\pi$  where  $w$  is the selection intensity [3]. If  $w = 0$ , selection is neutral whereas for  $w = 1$  selection is strong and the payoff determines the fitness completely. However, since negative fitnesses in this framework are meaningless, there are restrictions on the range of  $w$ . Alternatively, to avoid this restriction, we can use an exponential function  $f = e^{w\pi}$  [35]. Under any mapping scenario but weak selection, the fixation probability of strategy 1 in a population of  $Z - 1$  strategy 2 players playing a  $d$ -player game, is [10],

$$\rho_1 \approx \frac{1}{Z} + \frac{w}{Z^2} \sum_{m=1}^{Z-1} \sum_{\gamma=1}^m (\pi_1 - \pi_2), \quad (3.4)$$

where  $\pi_i$  is the fitness of strategy  $i$  and the payoffs depend on the number of mutants  $\gamma$ . We have generalized this result to multiple games. The strategies in a multiple game are categorical ones. For instance, a two game system with each game containing two strategies, has four categorical strategies as shown in figure 2. If one of the categorical strategies takes over the entire population, we term it as the fixation of the strategy defined by the category. If in a population of size  $Z$  playing  $N$  games, there is a single individual playing strategy  $A_{i_1}^1 A_{i_2}^2 \dots A_{i_N}^N$  in a population of  $Z - 1$  individuals playing strategy  $A_{j_1}^1 A_{j_2}^2 \dots A_{j_N}^N$  then we are interested in the probability that this single individual takes over the population. First we need to map the payoffs to fitness and there are two ways of implementing any kind of mapping for multiple games: *Method I*. For each game, the payoffs are mapped to fitness and then the cumulative fitness is calculated. Here, the fixation probability of a single individual of type  $A_{i_1}^1 A_{i_2}^2 \dots A_{i_N}^N$  in a population of  $A_{j_1}^1 A_{j_2}^2 \dots A_{j_N}^N$  is given by (see the electronic supplementary material for details)

$$\rho_{A_{i_1}^1 A_{i_2}^2 \dots A_{i_N}^N, A_{j_1}^1 A_{j_2}^2 \dots A_{j_N}^N} \approx \frac{1}{Z} + \frac{w}{NZ^2} \left[ \sum_{m=1}^{Z-1} \sum_{\gamma=1}^m \left( \sum_{j=1}^N (\pi_{j_i} - \pi_{j_j}) \right) \right]. \quad (3.5)$$

*Method II*. The payoffs can be added first and then mapped to fitnesses. The fixation probability through this method is (see the electronic supplementary material for details)

$$\rho_{A_{i_1}^1 A_{i_2}^2 \dots A_{i_N}^N, A_{j_1}^1 A_{j_2}^2 \dots A_{j_N}^N} \approx \frac{1}{Z} + \frac{w}{Z^2} \left[ \sum_{m=1}^{Z-1} \sum_{\gamma=1}^m \left( \sum_{j=1}^N (\pi_{j_i} - \pi_{j_j}) \right) \right]. \quad (3.6)$$

For illustration, let us consider a combination of two games with two strategies each. For instance, the games in (3.1). We make pairwise comparisons between all categorical types, i.e. all the edges of the  $S_4$  simplex in electronic

supplementary material, figure A.4. Using these comparative fixation probabilities, we can determine the flow of the dynamics over pure strategies. Let us focus on the edge  $A_1^1, A_1^2 \rightleftharpoons A_1^1, A_2^2$ , where game 1 does not change and only game 2 matters. Hence, the fixation probabilities should be the same as if only game 2 exists. The single game fixation probability of game 2 is shown in electronic supplementary material, figure A.5. As given in equations (3.5) and (3.6), when game 2 is combined with game 1, there can be two ways of mapping payoffs to fitness. The results from these two methods in multiple games in finite populations are also plotted in electronic supplementary material, figure A.5.

The fixation probabilities of a strategy in a single game changes when ‘adding’ just one more game to it. Even on the edge  $A_1^1, A_1^2 \rightleftharpoons A_1^1, A_2^2$ , where game 1 is neutral and only game 2 matters, there is an effect of game 1 on game 2. With increasing selection intensity, the fixation probability of a single individual playing  $A_1^1 A_1^2$  strategy on the edge  $A_1^1 A_1^2 \rightleftharpoons A_1^1 A_2^2$ , i.e.  $\rho_{A_1^1 A_1^2, A_1^1 A_2^2}$  is expected to decrease (electronic supplementary material, figure A.5). However, this decrease is different for the two methods and for the fixation probability of an individual with strategy 1 playing only game  $A_2^2$ , i.e.  $\rho_{A_1^1, A_2^2}$ . Method I gives a higher value of  $\rho_{A_1^1 A_1^2, A_1^1 A_2^2}$  as compared to  $\rho_{A_1^1, A_2^2}$ , whereas Method II shows that  $\rho_{A_1^1 A_1^2, A_1^1 A_2^2}$  is lower than  $\rho_{A_1^1, A_2^2}$  with increasing selection intensity. This means that while in general the fixation probabilities for the categorical type  $A_1^1 A_1^2$  decrease, it is even harder for  $A_1^1 A_1^2$  to reach fixation in the scenario where all the payoffs are first added and then converted to fitness as opposed to if the payoffs are first mapped and then added together. The difference can be explained by the difference in the baseline fitness between the two methods. The baseline fitness is provided by the game which the edge is independent of, in the case of electronic supplementary material, figure A.5, game  $A_1^1$ . In the electronic supplementary material, we calculate the difference between the two methods and show how this difference changes according to the different baseline fitness. For a large number of games, the difference is independent of the number of games.

Fixation probability is a crucial property of stochastic evolutionary game dynamics. Instead of merely looking at the fixation probabilities of certain types or strategies in a game, we have expanded the method for analysing the ‘categorical types’ in the MGD. Therefore, even for multiple games in finite populations, it might be possible to derive the long-term average dynamics [28,36] of entities playing a combination of different roles (strategies) in various interactions (games).

### (c) Territorial defence versus group hunts

We can find numerous applications of the multiple games concept not only in economics and cultural evolution [37] but also in classical ecology and evolutionary biology. As an illustration of our methodology, we choose to focus on the lioness example described in the Introduction. An explanation involving multiple games was already hypothesized in [18]. We shall consider two games: the territorial defence and a hunting game. The first game is a public goods game (PGG) with loners ( $Lo$ , not participating in the defence), leaders ( $Le$ , cooperators), and laggards ( $La$ , defectors). The cooperators patrol the territory together and thus provide an enhanced benefit of better protection via numbers. The loners can protect the territory only by themselves and get limited benefit out of it (less

than the cooperators). The defectors take part in patrolling but lag thus benefiting from the interaction without contributing. The payoffs for these strategies are

$$\pi_{Lc} = \frac{r_1 c_1 k}{d_1} - c_1; \quad \pi_{La} = \frac{r_1 c_1 k}{d_1}; \quad \pi_{Lo} = c_1 \sigma. \quad (3.7)$$

For territory defence, we set the number of individuals patrolling  $d_1$ , with the cost of cooperation  $c_1$ . The parameter  $k$  is the number of leaders (or cooperators). Here,  $r_1$  ( $1 < r_1 < d_1$ ) is the common pool's interest rate or an enhancement factor and  $\sigma$  ( $0 < \sigma < r_1 - 1$ ) is the small and fixed payoff of loners. The SGD for  $d_1 = 12$ ,  $r_1 = 3$ ,  $c_1 = 1$ , and  $\sigma = 1$  is shown in figure 5 as in [38]. The homoclinic cycles show the coexistence of all the types: leaders, laggards, and loners as discussed in the Introduction.

The second game is a hunting game (stag–hunt game) with cooperators and defectors. In cooperative hunting among lionesses, the ‘wings’ attack a prey and force them to move forward. The prey ends up running towards the lionesses called ‘centres’ lurking to catch it [39]. Clearly, two players are not enough for these games. For the two strategies of this multiplayer stag–hunt game, the payoffs are calculated as per [9]

$$\pi_C = \frac{r_2 c_2 j}{d_2} \theta(j - M) - c_2 \quad \text{and} \quad \pi_D = \frac{r_2 c_2 j}{d_2} \theta(j - M), \quad (3.8)$$

where  $\theta(z)$  is the Heaviside step function, i.e.  $\theta(z < 0) = 0$  and  $\theta(z \geq 0) = 1$ . The number of cooperators  $j$  each pay a cost  $c_2$ . The enhancement factor for game 2 is given by  $r_2$ . The value  $M$  is the minimum threshold number of players required to produce public good. The SGD for this scenario is depicted in figure 5. For specific parameter values,  $d_2 = 20$ ,  $M = 10$ ,  $c_2 = 1$ , and  $r_2 = 12$ , we observe two internal equilibrium solutions of the replicator dynamics [9].

Combining the stag hunt with the territorial defence game, the recovered dynamics from the MGD does not necessarily reflect the SGDs. Certain trajectories can become non-oscillatory resulting in the dominance of one of the strategies ( $ic_2$ ) or the coexistence of all but in a static equilibrium ( $ic_3$ ). For the stag–hunt game, we even see a complete switch of equilibrium ( $ic_2$ ), as in figure 4. The combination of the two games can change not just the dynamics but also the equilibria of both the games for certain initial conditions (figure 5).

From the MGD shown in figure 5, we see that judging a lioness by her action in one game does not complete the picture. An apparent cheater lioness in one game, can be a cooperator in another. For  $ic_2$ ,  $x_{Lo}$  reaches fixation but for  $ic_1$  the timing of observation matters. A lioness' entire story can only be told by looking at her ‘categorical type’ which informs us about the combined effect of playing all games as postulated by empirical observations [18]. Adding other games like cooperative breeding, nursing, or mating may also provide a better comprehension.

## 4. Conclusion

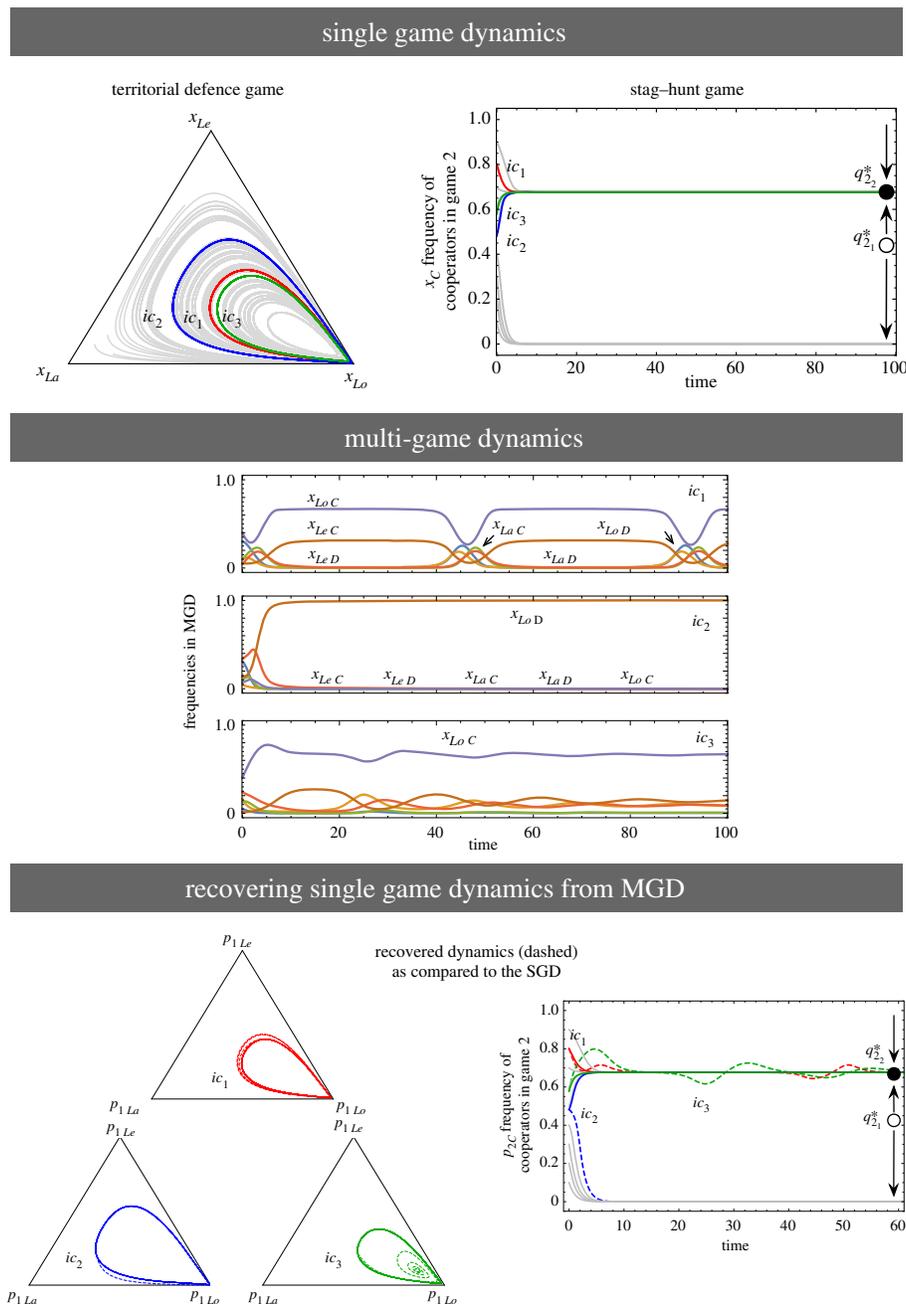
Nature is composed of many interactions in different contexts (games) [40]. The games consist of different players and strategy sets. In its lifetime, an individual plays many parts (in various games). We have devised a method to combine the various multiplayer multi-strategy games that individuals play with an aim of developing realistic evolutionary game theoretic models. For infinite populations, we provide a replicator equation which can encapsulate multiple games with multiple players and strategies. For finite populations, we

show that the fixation probabilities depend on the details of the particular model at hand and especially how the payoffs are converted to fitness.

Just as biological and social analogies of multiplayer evolutionary games can be found aplenty, the case for considering multiple multiplayer games is strong. We have discussed an application of our theory using the territorial defence and hunting behaviour of lionesses. The example highlights the fact that behaviour needs to be analysed in the light of complex multiple interaction contexts. On a smaller scale, the gut microbiota is a complex system which is capable of showing a variety of stable states, often a dynamic stability [14,41]. The different microbes within the gut community definitely interact in a variety of ways within themselves but each also interacts with the host in a unique manner. Within species and between species interactions, together, have the potential to dictate the evolutionary course of all involved species [42]. These interactions can certainly be interpreted as multiple games, each with a number of strategies and (immensely) multiplayer games. On the population genetics level, as an extension to previous work [43], multiple games and multi-strategies can be seen as multiple loci with several alleles. The case for two loci (or games) having two strategies [31], and 3-strategy games [8] has been previously investigated. Now with our inclusion of multiplayer games, we can also investigate polyploidy [44]. Considering recombination at this point would be crucial since it has been shown that under recombination the dynamics of multiple games would converge to the Wright manifold and thus to the SGD as in [45]. Deciphering the linkage between strategies used across multiple games could then be an exciting avenue for future research.

In finite populations, we have developed two methods to map the payoffs to the fitnesses. These two methods produce different fixation probability values for a particular selection intensity (electronic supplementary material, figure A.5). Both methods can have different biological justification. For example, all the actions leading to a brood produced during a season could be the culmination of all payoffs converted to fitness and then added to give the lifetime fitness—this is akin to Method I. On the other hand, in Method II, the payoffs through all breeding seasons would be summed up and then mapped to the lifetime fitness. The methods produce different results as compared with just one game (or even when the game is combined with another neutral game). Thus, even under finite populations, MGDs are different from SGDs and our formulation can be used to study multiple games in finite populations.

In a nutshell, our analysis reveals that the outcomes from multiplayer 2-strategy games are similar to previous results [31], where the MGD can be characterized by the separate analysis of the individual games. However, when the games have at least three pure strategies, different dynamics emerge [8]. This dynamical (in)consistency has already been pointed out [31,32] as ‘serious since it goes to the heart of the evolutionary approach’ [32]. With the diverse use of multiplayer games in social evolution (e.g. tragedy of the commons) and in biology, the problem is only exacerbated due to the potential existence of multiple internal steady states. For such cases, a fully comprehensive study of the initial conditions is a potential future project (as in figures 4 and 5). Even though complicated dynamics can still be captured by the relatively simple replicator-like equations and fixation



**Figure 5.** Lionesses in territory defence and stag-hunt games. The SGD of the games are plotted in the top panel. The leader  $Le$ , laggard  $La$ , and loner  $Lo$  are the strategies in the territorial defence game. Cooperation  $C$  and defection  $D$  are the strategies for the stag hunt. The grey lines are trajectories from random initial conditions to observe the SGDs. We choose three trajectories having initial conditions  $ic_1$ ,  $ic_2$ , and  $ic_3$  to track the SGDs and MGD. Homoclinic orbits that emerge from and lead to  $x_{Lo}$  can be seen in the SGD of game 1. The SGD of game 2 shows an unstable equilibrium at  $(q_{2_1}^*)$  and a stable one at  $(q_{2_2}^*)$ . The MGD consists of six categorical types  $x_{LeC}$ ,  $x_{LaC}$ ,  $x_{LoC}$ ,  $x_{LeD}$ ,  $x_{LaD}$ , and  $x_{LoD}$ . In the middle panel, the time evolution of the categorical types is plotted. For  $ic_1$ , we recover oscillatory dynamics but different dynamics as well as equilibria emerge for other initial conditions. In the last row, we show the recovered SGDs (plotted in dashed lines) from the MGD in comparison with the original SGDs (plotted with solid lines). For the recovered territorial defence game, the initial conditions  $ic_2$  and  $ic_3$  do not end up in the homoclinic cycle as in the SGDs; the equilibrium solution and dynamics in the multi-game is different from the SGD. For  $ic_1$  and  $ic_3$ , cooperation in game 2, i.e.  $p_{2C}$  does not reach a static equilibrium but oscillates. On the other hand,  $ic_2$  goes extinct; a complete switch of equilibrium as compared to the SGD. So the addition of games changes the dynamics as well as stability of both the games for certain initial conditions. (Online version in colour.)

probabilities, vast domains in the multiple games space remain unexplored.

**Data accessibility.** This article has no additional data.

**Authors' contributions.** C.S.G. conceived the model. V.R.V. and C.S.G. developed the model. V.R.V. performed the analyses. Both authors wrote the manuscript.

**Competing interests.** The authors declare no competing interests.

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## Chapter 3

# Combined dynamics of multiple life-history traits with sex specific differences

“Astronomy gave you the inorganic creation and evolution over a 15-billion-year period of time. Evolutionary logic gave you the comparable story over 4 billion years and evolutionary logic applied to *life*.”

---

— Robert Trivers, Natural Selection and  
Social Theory

## 3.1 Introduction

In the previous chapter, we looked at a mathematical framework to study multiple interlinked interactions that group(s) of individuals take part in over a period of time. This framework used evolutionary game theory (Maynard Smith, 1982) where each *game* was an interaction, and provided a concise formula to calculate the evolution of various types of individuals in a population who play different kinds of games. This formula can capture the dynamics of many games having a varying number of agents or *players* and sets of *strategies* (phenotypes, traits, decisions) they could adopt.

In this chapter, I use the idea of combined dynamics of multiple interactions to build a model that studies interactions between life-history traits and how sex-specific differences in life-history traits affect the fitness of species in the animal kingdom. Here, the focus is on sexually reproducing animals with two distinct sexes: female and male. Females provide large costly eggs and males smaller low-cost sperms. The sexes are defined by this difference in gamete size also known as anisogamy. In this model, three main inter-individual and intra-individual interactions of life-history traits during a reproductive period were considered: parental investment, ornamentation (displays used to attract potential mates) and immune response. All these traits manifest sex-specific differences. The overall life-history of a sex is affected by diverse sex-specific interactions. The model uses standard knowledge from population genetics and simple frequent dependent fitness in addition to evolutionary game theory to study the consequences of sex-specific differences in life-history traits on lifetime reproductive success.

An important outcome that emerged out of this model is the bias in adult sex ratio in many species and our results match with empirical observations (Pipoly et al., 2015). In this chapter, I present the details of the model. An important result discussed in this chapter exhibits how and when sex-specific differences affect adult sex ratios. For instance, our findings show that in species with equal parental investment, sex-specific differences in ornamentation are the major

cause of skewed sex ratios.

Before diving into the model, let us first look at the basics of sexual selection and the above mentioned life-history traits with sex-specific differences that are a crucial aspect of the reproductive lifetime of an individual or until the time of senescence i.e. period after which the individual can no longer reproduce.

## 3.2 Sexual selection

Most evolutionary biology problems boil down to studying how living organisms maximize their fitness. The fitness of an individual depends on its survivability and fertility. The first step to obtaining offspring starts with mating and this is where sexual selection occurs. Sexual selection has been discussed ever since Darwin wrote on the subject (Darwin, 1871). Darwin defined sexual selection as competition and differential choice within members of one sex for obtaining mates of the opposite sex. This means that females tend to choose some males over others and males compete with each other for females. Intrasexual selection refers to mating competition within the same sex (usually this is seen among males) whereas intersexual selection denotes mate choice; where (usually) the females choose their mates (Wong and Candolin, 2005; Andersson and Simmons, 2006). Studies on sexual selection can shed more light on the evolution of species since it is a special case of natural selection that acts on the ability of a species to successfully copulate with a mate. It is the reason that makes peacocks maintain their magnificent tails, fruit flies to perform their courtship dances and certain birds to build elaborate nests. Thus, sexual selection gives rise to sexual dimorphism which is defined as the set of differences that are observed between two sexes of a species. This set does not include variations in sex organs. Sexual selection is also often powerful enough to produce features that are disadvantageous to the individual's survival. For example, extravagant and colorful tail feathers or fins useful for attracting interested members of the opposite sex are likely to attract predators as well.

## 3.3 Sex-specific differences in life-history traits

### 3.3.1 Ornamentation and sex roles

Intrasexual selection is performed between individuals of the same sex to attract and obtain mates from the other sex through fights, nuptial gifts, nests, sexual signals, ornament display and various types of 'attractiveness'. We refer to all of these as 'ornaments'. Sex roles are of two types, conventional and reversed. The former refers to males competing for females and the latter refers to females competing for male mates. Sex-role reversal has nothing to do with male pregnancy e.g. seahorse males undergo pregnancy, but still, seahorses are a conventional sex-role species as the males compete for the females (Vincent et al., 1992).

### 3.3.2 Parental investment

The collection of all the investments by an individual for all the offspring produced in that reproductive season is defined as parental investment (Trivers, 1972; Kokko and Jennions, 2008). It can be described in three parts or stages: initial parental investment or investment in the production of gametes (sperms or eggs), internal parental investment or pregnancy, and lastly, external parental investment or parental care which entails all the parental care provided after the birth of a juvenile. In nature, the amount of parental investment varies within members of the two sexes of a species, and also varies between species. For example, within a species, say, sticklebacks or seahorses, the males provide more parental investment as compared to females. Between species where males perform most of the parental investment, male sticklebacks provide all the parental care required for offspring, and seahorse males undergo pregnancy (Smith and Wootton, 1999; Wilson et al., 2003).

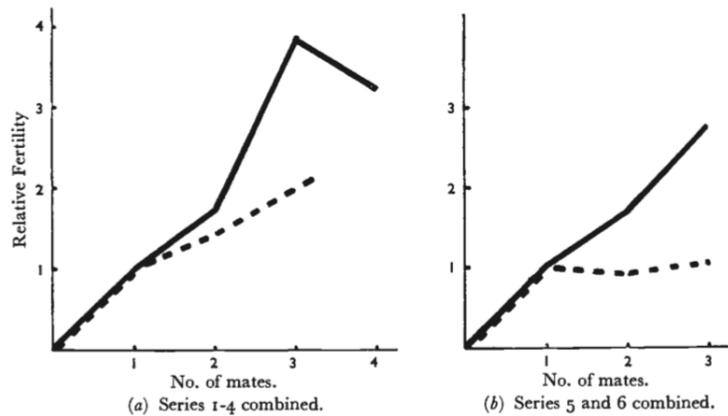


Figure 3.1: **The relation between number of males and fertility relative to that with one mate.** The following result is from the paper by Bateman where he performed a series of experiments with *Drosophila melanogaster* (Bateman, 1948). The solid lines correspond to males and the broken lines refer to the females. This plot shows that the relative fertility of males increases with the number of mates. Females do not gain as many mates as the male do in one reproductive season since females also go through parental investment.

### 3.3.3 Sex difference in immune response

Immunity is a crucial life-history parameter. In most sexually reproducing animals, females are more immunocompetent than males, but in species with male pregnancy (pipefishes, seahorses), males have a more efficient immune system. This sex-specific difference in immunocompetence is known as sexual immune dimorphism.

There are many hypotheses as to why there is sexual immune dimorphism. Initially, it was thought that only ornamentation (which is proportional to testosterone and other male hormones levels) was the proximate reason for sexual immune dimorphism since hormones such as testosterone are known to be immunosuppressors (immunocompetence handicap hypothesis) (ICHH)(Hillgarth and Wingfield, 1997). This hormone (restricted to vertebrate males) promotes secondary sexual characteristics. The higher its testosterone level, the more chances a male succeeds in the mating competition. Thus, this hormone strongly affects mating behaviors and sex traits. Despite having testosterone, seahorse males show higher immunocompetence than females. Invertebrates do not have testosterone and yet *Drosophila* males are less immunocompetent as compared to females

(Rolff, 2002; McKean and Nunney, 2008). In fact, lately, it has been in question if testosterone or sex-specific hormones are the only major immunosuppressants (Franco et al., 1990; Wedekind and Folstad, 1994; Jansson and Holmdahl, 1998; Roberts et al., 2004).

Sexual immune dimorphism could also be a result of parental investment. High levels of parental investment make the sex that undertakes it (usually females) more 'fragile' and prone to illness, as resources are rather allocated into parental investment than into immune defense. Pregnant females (or males in case of seahorses and pipefishes) are the ones that are the offspring are most dependent on and therefore, the sex that undergoes pregnancy needs to be 'healthy' and should also survive through the pregnancy period for being able to produce offspring.

According to Bateman's principle (Bateman, 1948), males increase their lifetime reproductive success by an increased number of matings (see Figure 3.1). Females (or males in case of seahorses and pipefishes) are the limiting resources for the males to compete for, as they spend most of the reproductive and breeding season in being pregnant, parturition and in some cases also undertake external parental care. A female thus needs to live longer than a male to have as much fertility as a male and higher immunocompetence enhances the probability to survive parasite and pathogens infections and thus increases longevity (Roth et al., 2011; Lin et al., 2016). The males have to mate with as many females as they can in each mating season, such that their genes can be passed on. Therefore they evolved certain traits or secondary sexual signals (different from females) that are attractive. For instance, male stags spend a lot of energy in developing larger antlers to increase their probability of winning in a fight with other males as this would give them increased chances of mates. Similarly, females may maximize their reproductive success or increase their chance of passing on their genes as much as possible by living longer. A high longevity requires a strong immune response (May, 2007; Caruso et al., 2013). The Bateman curves for females and males shown in Figure 3.1 was a result of experiments done with the fruit fly *Drosophila melanogaster*. In sex-role reversed species such as pipefishes where females

compete for mates while males perform mate choice, the Bateman curves would also be reversed. The features of Bateman's principles appear in diverse forms in different mating systems (Arnold and Duvall, 1994).

### **3.4 Emergence of sex-specific patterns from the model: bias in adult sex ratio**

Given the multiple examples of sexual dimorphism in various life-history traits across the animal kingdom, parental investment and ornamentation in many species are suggested to correlate with the efficiency of immune responses and longevity. (Trivers, 1972, 2002; Hedrick and Temeles, 1989; Austad, 2006; May, 2007; Roved et al., 2017). Our results showed that the combined dynamics of these life-history interactions with sex-specific differences produce a bias in adult sex ratio (ASR) as seen in nature (Pipoly et al., 2015) even if the offspring produced at every generation have an equal ratio. In our study, we refer to ASR as the ratio between the frequency of adults of Sex 1 and Sex 2 i.e. frequency of Sex 1 : frequency of Sex2. As shown in many empirical studies ASR has an impact on sex differences and roles (Liker et al., 2013, 2015; Székely et al., 2014). Here, we show that the reverse is also possible (Kokko and Jennions, 2008).

Our results reveal that sex differences in parental investment, ornamentation, and immunity give rise to skewed adult sex ratio (see 3.2) even when we set the sex ratio at birth to be equal for every generation.

Moreover, in the context of our model, when the equilibrium value of frequency of adults in one sex is higher when compared to the other, it would also mean that one sex tends to live longer than the other, and there are numerous examples in nature that validate the sex difference in longevity (Smith, 1959; Rolff, 2002; Austad, 2006; Litzgus, 2006; Clutton-Brock and Isvaran, 2007).

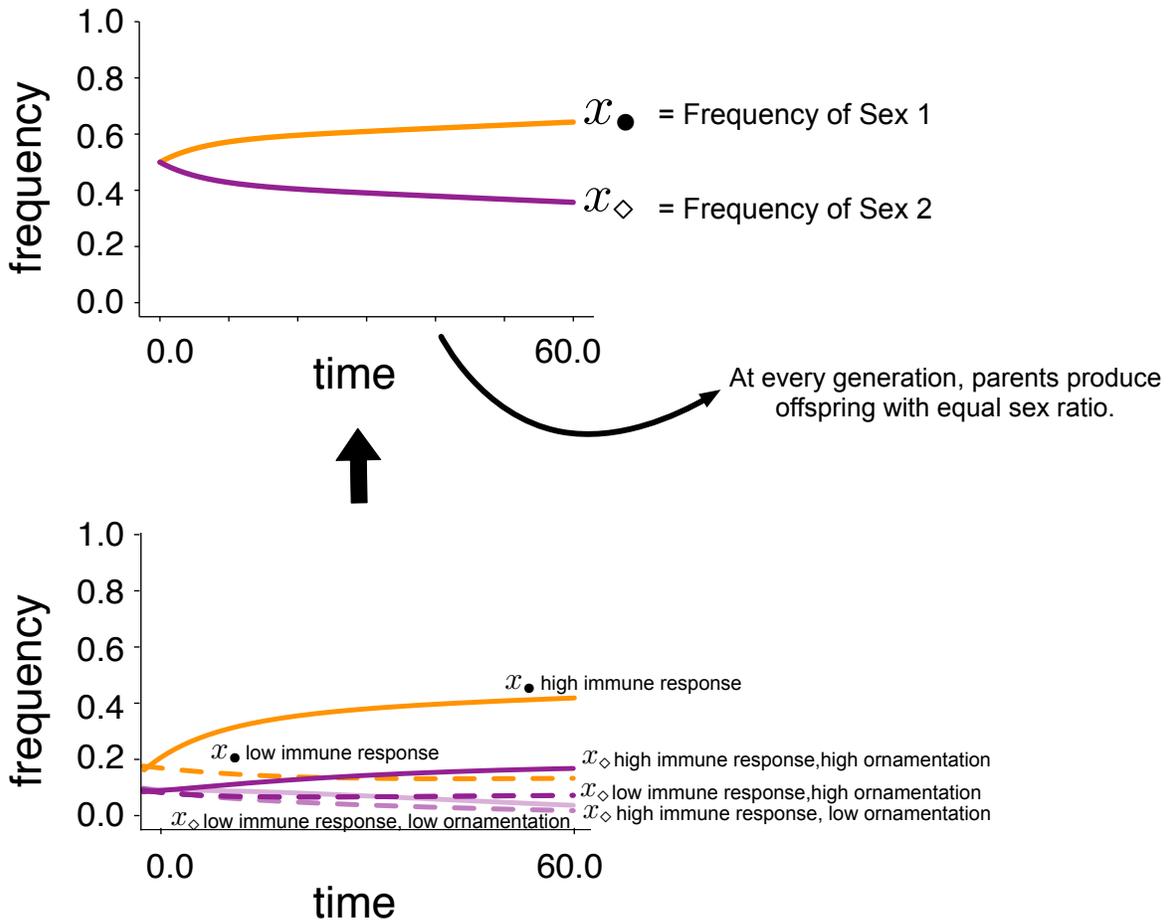


Figure 3.2: **Sex-differences in immunocompetence, parental investment and ornamentation skew the sex-ratio among the adults.** Sex 1 and Sex 2 exhibit sex-specific immune responses that determine their survivability which affects fitness. Here, both sexes have individuals with high and low levels of immune response, but Sex 1 has higher immunocompetence on average. Sex 1 undergoes parental investment and both sexes benefit from this interactions as they obtain offspring. This benefit (offspring produced) also contributes to fitness. Sex 2 performs mating competitions to gain more good mates and this also affects its fitness. The sex-specific traits evolve over a generation (time) by selection and therefore, get passed on to subsequent generations. Thus, even when the sex ratio is kept equal among offspring at every generation, their inherited sex-specific characteristics change their frequency in the population. After a certain time point, the frequencies of adult Sex 1 and Sex 2 individuals attain equilibrium as shown in the plots. Orange lines are for Sex 1 and purple for Sex 2. The solid lines are for the heterozygous immunity genotype and dashed lines for the immunity homozygotes. The lighter purple lines are for Sex 2 individuals with low ornamentation.

Our model provides theoretical support for patterns that emerges out of combining life-history traits with sex-specific differences. The model provides a generalized idea about the explanation for sex-specific lifetime reproductive success and skewed adult sex ratio by disentangling the different life-history strategies and sex itself. In the following section which contains our manuscript, the model and its outcomes are described and analyzed in detail. The supplementary material for this manuscript is provided in Appendix B of this thesis.

# Consequences of combining life-history traits with sex-specific differences

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Males and females evolved distinct life-history strategies, reflected in diverse life-history traits, summarized as sexual dimorphism. Life-history traits are highly interlinked. The sex that allocates more resources towards offspring is expected to increase its life span, and this might require an efficient immune system. However, the other sex might allocate its resources towards ornamentation, and this might have immunosuppressive effects. Activity of immune response may not be specific to the sex that produces the eggs but could correlate with the amount of parental investment given. Informed by experimental data, we designed a theoretical framework that combines multiple life-history traits. We disentangled sex-biased life-history strategies from a particular sex to include species with reversed sex-roles, and male parental investment. We computed the lifetime reproductive success from the fitness components arising from diverse sex-biased life-history traits, and observed a strong bias in adult sex ratio depending on sex-specific resource allocation towards life-history traits. Overall, our work provides a generalized method to combine various life-history traits with sex-specific differences to calculate the lifetime reproductive success. This was used to explain certain empirical observations as a consequence of sexual dimorphism in life-history traits.

**Keywords:** Life-history traits, theoretical biology, evolutionary game theory, population dynamics, lifetime reproductive success, adult sex ratio

## Introduction

Fitness is a complicated entity and describes the reproductive success of an individual reflecting the ability of individuals to produce offspring and survive. This arises from trade-offs between various

33 life-history traits. Theoretical models assessing the interaction of multiple life-history traits are  
34 thus crucial to understand organisms' overall life-history and how they impact fitness. Theoretical  
35 and experimental studies have shown how multiple life-history traits define an individual's lifetime  
36 reproductive success (Moore, 1990; Martin, 1992; Chapman and Partridge, 1996; Pusey et al., 1997;  
37 Fleming et al., 2000; Alonzo, 2002; Kalbe et al., 2009; Alonzo, 2010). However, typically, these  
38 traits have been studied in isolation.

39 In this study, we present a model that addresses the interaction of essential sex-specific life-history  
40 traits aiming to obtain the lifetime reproductive success of both sexes. This sheds light on how these  
41 traits are contributing to an individual's life-history. We further present the consequences of various  
42 sex-specific strategies affecting an evolving population.

43 Most life-history traits have sex-specific differences. Sex-specific life histories have evolved in  
44 the animal kingdom as a consequence of difference in gamete size known as anisogamy (Bell,  
45 1978); females contribute large costly eggs to reproduction and males small cheap sperm. The  
46 distinct resource allocation into the offspring asks for sex-specific life-history strategies (Trivers,  
47 1972; Hedrick and Temeles, 1989; Trivers, 2002; Austad, 2006; May, 2007; Roved et al., 2017). Here  
48 we focus on the sex-specific differences in three life history traits namely 1. Parental investment 2.  
49 Ornamentation and 3. Immunocompetence

50 In many species, parental investment is not restricted to sperm and egg production. Parental  
51 investment (PI) is any behavioural and physiological investment by a parent provided to the off-  
52 spring (Trivers, 1972, 2002). The sex that needs to allocate more resources towards the offspring  
53 strives for increased longevity since offspring survival also depends on the survival of the parent.  
54 Increased longevity requires the allocation of resources into parasite defence and, hence, immunity.  
55 Intense costly intrasexual competitions for obtaining mates are performed by allocating resources  
56 towards ornamentation (Hillgarth and Wingfield, 1997; Wong and Candolin, 2005; Andersson and  
57 Simmons, 2006). To this end, fewer resources may be available for the immune defence in the sex  
58 majorly investing in intrasexual interactions. This implies that both ornamentation and parental  
59 investment contribute to sexual immune dimorphism (Forbes, 2007; Nunn et al., 2008; Roth et al.,  
60 2011; Lin et al., 2016). Thus focusing only on one life-history trait in isolation will not shed light  
61 on the individual's true lifetime reproductive success.

62 We aimed for designing a framework in which multiple life-history traits and their interactions  
63 can be studied simultaneously. Particularly, we have constructed a holistic framework that captures  
64 sex-specific differences in parental investment, ornamentation and immune response and presents  
65 the outcomes of the overall life-history of a sex. We observed two important consequences of sex  
66 differences in life history interactions: 1) skewed adult sex ratios and 2) different ratios of homozygous  
67 and heterozygous individuals between the sexes with regard to immune alleles. We validated our  
68 findings using empirical data from a broad range of animal taxa and diverse life-history strategies to  
69 test the limits of our approach.

## 70 **Model**

71 We amalgamated approaches from standard population genetics and eco-evolutionary processes (Free-  
72 man and Herron, 2007; Otto and Day, 2007; Venkateswaran and Gokhale, 2019) (within and between  
73 populations) to investigate the interaction dynamics of multiple life-history traits (with sex-specific  
74 differences). We first developed a robust method (illustrated in Figure 1 to study the lifetime repro-  
75 ductive success (LRS) that arises from immune response, mating competition through ornaments  
76 and parental investment. Later, we used the LRS to investigate the consequences of combining the  
77 sex-specific interactions that are part of an individual's reproductive lifetime.

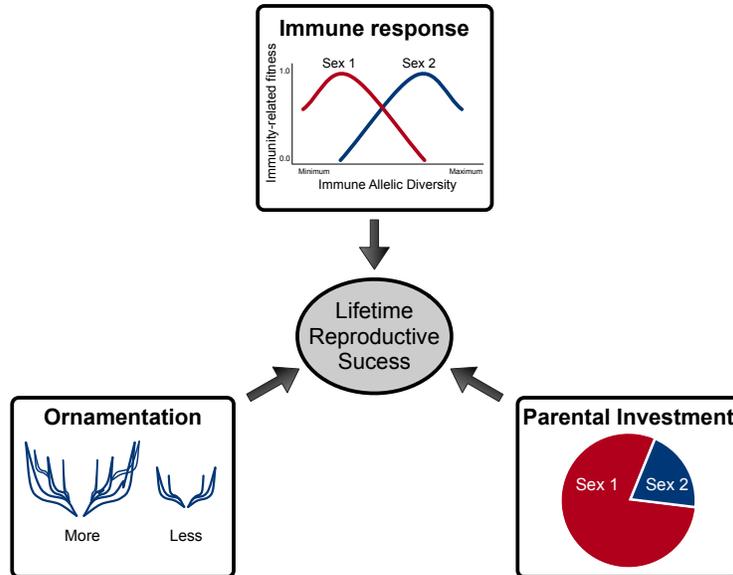


Figure 1: Model representation. Life-history traits affect the lifetime reproductive success. The fitness components from parental investment, immune system and ornamentation are offspring success, survival of the parent plus offspring and mating success, respectively. These contribute to an individual's lifetime reproductive success. We assumed that Sex 1 provides more parental investment (PI) than Sex 2. The sex-specific fitness from parental investment is modeled as frequency dependent since the number of copulations in one sex depends on the availability of the other sex. The individuals within a sex also have different levels of ornamentation, which they use to attract individuals of the other sex as potential mates. The model uses evolutionary game theory which gives frequency dependent fitnesses of two types of individuals: those with more and those with lesser levels of ornaments. The individuals also differ in their immune genotypes. Each immune genotype yields a certain immunity-related fitness value that depends on the type and number of different immune alleles. The strength of immune response differs between sexes (sexual immune dimorphism). We modeled the evolution of these immune genotypes using population dynamics. Finally, the fitness obtained from parental investment, ornamentation and immune response were used to measure the lifetime reproductive success of an individual.

78 Consider the two sexes in a population, Sex 1 denoted by a filled circle ●, and Sex 2 denoted by a  
 79 diamond ◇. We first consider one autosomal immunity locus  $A$  having two alleles  $A_1$  and  $A_2$ . The  
 80 three distinct zygotes genotypes would be  $A_1A_1$ ,  $A_1A_2$  and  $A_2A_2$ . For Sex 1, which throughout  
 81 this manuscript does major PI, the frequencies of the three genotypes are denoted by  $x_{\bullet 1}$ ,  $x_{\bullet 2}$ ,  $x_{\bullet 3}$ .  
 82 The fitnesses, of the same, are denoted by  $W_{\bullet 1}$ ,  $W_{\bullet 2}$  and  $W_{\bullet 3}$ . Similarly, we denote the frequencies  
 83 and fitnesses for Sex 2.

84 We used standard Mendelian segregation to model the evolution of the different types of individuals  
 85 in the population. The genotype dynamics following this segregation patterns are denoted in the  
 86 ESM. As with normal Mendelian segregation we assumed equal sex ratio; half of the offspring are  
 87 Sex 1 and the other half, Sex 2.

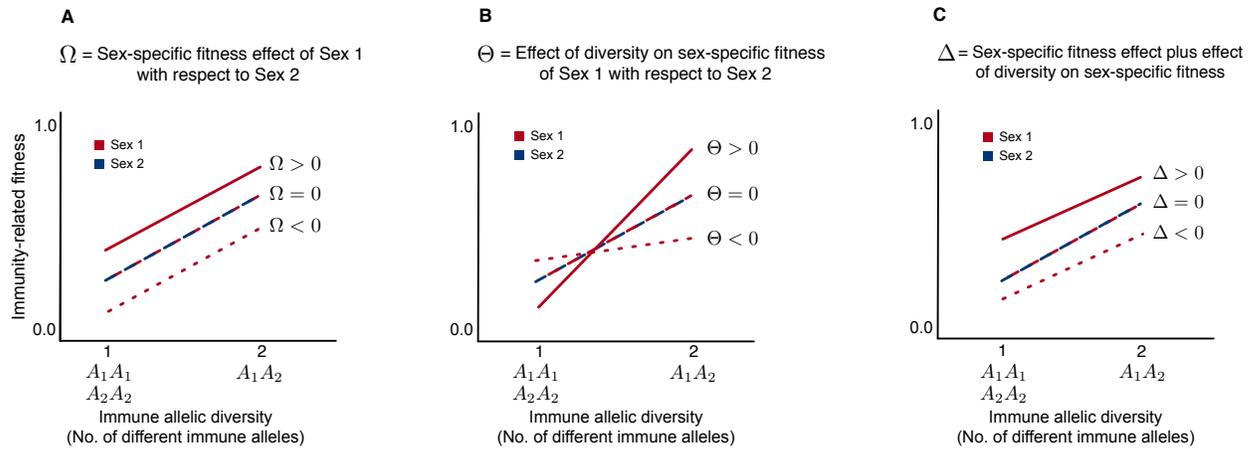


Figure 2: Schematic representation of different scenarios of sex-specific differences in host immunity-related fitness versus immune allelic diversity. We considered three distinct immune genotypes  $A_1A_1$ ,  $A_1A_2$ , and  $A_2A_2$  that result from mating between individuals having one immune gene locus  $A$  with two alleles  $A_1$  and  $A_2$  (Mendelian segregation, see ESM). Fitness positively correlates with the number of different alleles or allelic diversity (Apanius et al., 1997; Eizaguirre et al., 2009). So genotypes  $A_1A_1$  and  $A_2A_2$  (homozygotes) will have the same fitness value as they both have only one type of allele. But  $A_1A_2$  (heterozygote) which has two different types of alleles will have a higher fitness. This is known as heterozygous advantage and occurs within both sexes. However, between the sexes, there can be sex-specific differences (Roved et al., 2017). This is shown in panels (A), (B) and (C). In (A),  $\Omega > 0$  would imply that Sex 1 will have a higher value of immune response as compared to Sex 2 for any given allelic diversity. When  $\Omega < 0$ , Sex 1 has a lower values of immune response for any given allelic diversity as compared to Sex 2. Another situation is also possible: Sex 1 can have higher immune response for a homozygous locus, and lower immune response for a heterozygous locus when compared to Sex 2. This shown in (B), where  $\Theta$  is the difference between the angles of the two lines. In (C),  $\Delta$  differs from  $\Omega$  by considering lines that are not parallel to each other i.e. case C is a combination of cases A and B. When both sexes have the same immune response patterns,  $\Omega = \Theta = \Delta = 0$ .

## 88 Fitness

89 The lifetime reproductive success i.e. the overall fitness of an individual, is related to its immuno-  
 90 competence (the ability of an individual to produce a normal immune response following exposure  
 91 to a pathogen), and offspring success (Stoehr and Kokko, 2006; Kelly and Alonzo, 2010). Thus,  
 92 in our model the sex-specific fitness components resulting from immune response, ornamentation  
 93 and parental investment give the lifetime reproductive success of individuals of a sex as shown in  
 94 Figure 1. Below we introduce the fitness functions independently starting with immunity.

95 **Immune response.** A host's immunological diversity helps eliminate a large number of pathogens  
 96 and disease causing agents. However, in some cases, having too high diversity may reduce efficient  
 97 immune response e.g. auto-immune diseases triggered by high Major Histocompatibility Complex  
 98 (MHC) diversity. Thus, having an optimal number of alleles (intermediate diversity) has been shown  
 99 to be ideal in many systems (Nowak et al., 1992; Milinski, 2006; Woelfing et al., 2009). The host's  
 100 immunological diversity can be coarsely split up into three parts: low diversity ( $LD$ , low efficiency of  
 101 the immune system), intermediate or optimal diversity ( $ID$ , optimal immune efficiency), and high

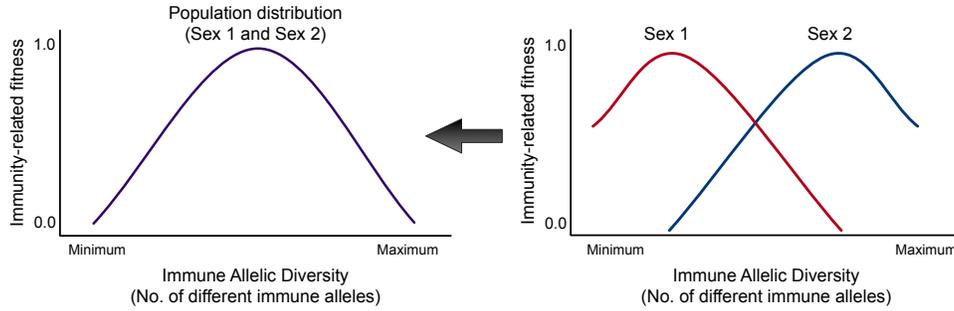


Figure 3: Schematic representation of host immunity-related fitness versus immune allelic diversity. For two immune gene loci  $A$  and  $B$  each having two alleles  $A_1, A_2$  and  $B_1, B_2$ , there would be ten distinct zygote genotypes. The population will comprise of individuals with these genotypes. Their immune responses would depend on these genotypes. The probability of immune response might reduce if the individual has too many immunity allele diversity. In the case of MHC, the auto-immune effect of having high MHC allele diversity reduces the probability of immune response (Nowak et al., 1992; Milinski, 2006; Woelfing et al., 2009). Thus there is an optimal allele diversity, which gives the parabolic shape to the curve. Recent studies have shown that males and females can have different optimal diversities ((Roved et al., 2017, 2018) and Winternitz et al., unpublished). Plotted here are hypothetical sex-specific optima of immune allelic diversity (Roved et al., 2017). The realized population distribution is what is typically looked at, but in our study we consider sex-specific optima of immune allelic diversity. Some immune genes may follow completely different sex-specific patterns from the one shown here (Roved et al., 2017; De Lisle, 2019), and this model can be used for most kinds of immune genes.

102 diversity ( $HD$ , might reduce the efficiency of the immune system). Recent experimental studies by  
 103 Roved et al. (2017, 2018) and Jamie Winternitz and Tobias Lenz (personal communication) show  
 104 that the optimal diversity could differ between the sexes. Based on these ideas, we have different  
 105 cases that are shown in the Figure 2 for one immune locus  $A$  with two alleles  $A_1$  and  $A_2$  that gives  
 106 three distinct parent and offspring genotypes  $A_1A_1$ ,  $A_1A_2$ , and  $A_2A_2$  denoted by  $j = \{1, 2, 3\}$ . We  
 107 denote their immune responses by  $W_{\bullet j}^I$  and  $W_{\circ j}^I$  for genotypes  $j = \{1, 2, 3\}$  in the two sexes. In our  
 108 model, we refer to immune allelic diversity as the number of different immune alleles in the immune  
 109 loci. A non-linear immune allelic diversity profile shown in Figure 3 where the negative effect of  $HD$   
 110 is also addressed is considered later.

111 These approaches can be generalised to any genetic system controlling the immune response or  
 112 a completely different causal mechanism devoid of the genetic correlation. For example, the ef-  
 113 fect of nutrients and its effect of the immune system can be captured by a non-genetic model as  
 114 well (Chandra, 1983). Thus, while we focus on the genetic mechanism in the current model, we  
 115 stress that our framework is independent of the exact mechanism of how the immune response curves  
 116 develop. Condition of an individual is directly proportional to immune response (resources allocated  
 117 to self-maintenance, immune defense) which in turn determines the survivability (Stoehr and Kokko,  
 118 2006).

119 **Parental investment.** Both sexes pay the costs for initial PI, i.e. egg and sperm production  
 120 (Hayward and Gillooly, 2011). Pregnancy and parental care vary massively among species (Trivers,  
 121 1972; Wade and Shuster, 2002; Trivers, 2002; Kokko and Jennions, 2003; Alonzo, 2010) (Figure 1).  
 122 We assume that Sex 1 provides major PI (e.g. male sticklebacks, male pipefish, most female

123 mammals). The fitness from PI will depend on the relative abundance of the other sex and are given  
 124 by,  $W_{\bullet}^P = (b^P - c_{\bullet}^P) \cdot \frac{x_{\diamond}}{x_{\bullet} + x_{\diamond}}$  and  $W_{\diamond}^P = (b^P - c_{\diamond}^P) \cdot \frac{x_{\bullet}}{x_{\bullet} + x_{\diamond}}$ . Here,  $b^P$  is the benefit (offspring produced)  
 125 from PI while  $c_{\bullet}^P$  and  $c_{\diamond}^P$  are the costs for PI by Sex 1 and Sex 2, respectively. The frequency of Sex  
 126 1 equals  $x_{\bullet} = x_{\bullet 1} + x_{\bullet 2} + x_{\bullet 3}$  and the frequency of individuals in Sex 2 equals  $x_{\diamond} = x_{\diamond 1} + x_{\diamond 2} + x_{\diamond 3}$ .  
 127 Since we have assumed that Sex 1 provides maximum parental investment,  $c_{\diamond}^P < c_{\bullet}^P < b^P$ .

128 **Ornamentation** Mating competitions occur among individuals of the same sex to attract and obtain  
 129 mates from the other sex. This is performed through fights, nuptial gifts, nests, sexual signals,  
 130 ornament display and various types of 'attractiveness'. We refer to all of these as 'ornaments'.  
 131 The investment into the display of ornaments will in most cases rise the chances of acquiring  
 132 mates (Carranza et al., 1990; Petrie et al., 1991; Berglund et al., 1997; Wong and Candolin, 2005).  
 133 However, ornamentation is often a costly signal (Zahavi, 1977; Andersson and Simmons, 2006;  
 134 Milinski, 2006; Kurtz, 2007)). Individual assessment of immune responses helps defining the costs.

135 When Sex 2 participates in mating competition as shown in Figure 1, two types of Sex 2 individuals  
 136 were considered in this interaction: one type displays more ornaments (*MO*) and the other type  
 137 displays less ornaments (*LO*). Therefore Sex 2 consists of six types of individuals -  $x_{\diamond j, MO}$  and  
 138  $x_{\diamond j, LO}$  where the genotype  $j = \{1, 2, 3\}$ . The frequency-dependent fitness that emerge from these  
 139 interactions are written as  $W_{\diamond MO}^O$  and  $W_{\diamond LO}^O$  (see ESM for details).

## 140 Overall dynamics

141 The lifetime reproductive success is a multiplicative effect of the fitness arising from immune re-  
 142 sponse, ornamentation and parental investment (Stoehr and Kokko, 2006; Kelly and Alonzo, 2010)  
 143 as shown in the ESM. Using the LRS values in the Mendelian population dynamics, we can obtain the  
 144 combined interaction dynamics of each type of individuals in the population (details and calculations  
 145 in the ESM). The population is divided into nine types of individuals - the three genotypes ( $j$ ) of  
 146 Sex 1,  $x_{\bullet j}$ , and the three genotypes of Sex 2 further split according to ornamentation into  $x_{\diamond j, MO}$   
 147 and  $x_{\diamond j, LO}$ . We refer to them as simply  $x_i$  with  $i$  as the type of individual. The classical selection  
 148 equation from population genetics (Crow and Kimura, 1970) gives the evolution of the frequency  
 149  $x_i$  having average fitness  $W_i$  (Crow and Kimura, 1970; Schuster and Sigmund, 1983; Hofbauer and  
 150 Sigmund, 1998; Gokhale et al., 2014). The equation can be written as,

$$\dot{x}_i = x_i (W_i - \bar{W}) \quad (1)$$

151 where  $\bar{W}$  is the average population fitness.

## 152 Results

### 153 Linear immune allelic diversity profile: single locus

154 The diversity levels in the immune alleles can result in differing immune response (e.g. MHC  
 155 homozygotes and heterozygotes, are known to have different immune responses (Apanius et al.,  
 156 1997; Eizaguirre et al., 2009)). For one immune locus with two alleles, higher allele diversity boosts  
 157 the immune response as shown Figure 2. The negative effect of very high diversity is not considered  
 158 here. Besides the null model of no sexual conflict within the allele diversity, we also include different  
 159 cases of sexual conflict (Roved et al., 2018) (Figure 2).

160 When we assume that both the sexes are not involved in mating competition i.e. ornamentation  
 161 competition game is neutral; we can vary the cost of PI and the immune response curves (shown in  
 162 Figure 2). The resulting equilibrium frequencies are shown in Figure 4. When the cost of PI is zero

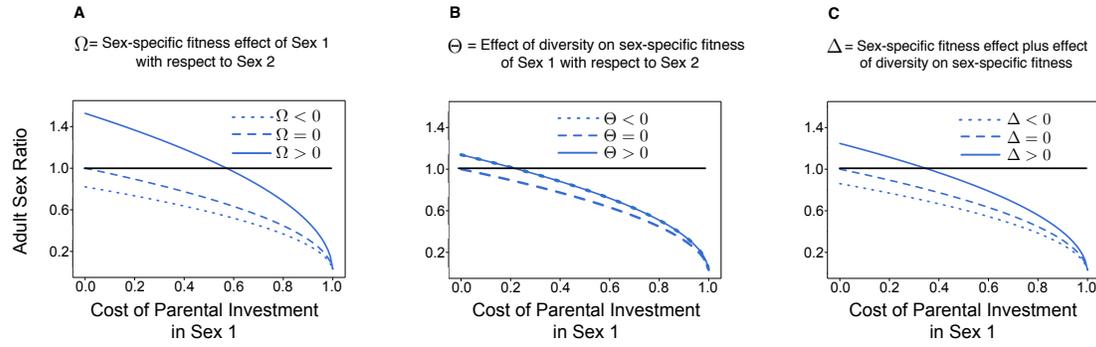


Figure 4: Adult sex ratio (Sex 1: Sex 2) for varying parental investment (PI) and various cases of sexual conflict within immune allelic diversity as shown in Figure 2. The ornamentation game is neutral, i.e. no selection acting on it (details in the ESM). As maintained throughout this study, Sex 1 does maximum PI. Sex 2 does negligible PI. Therefore, its cost is set to zero i.e.  $c_{\diamond}^P = 0$ . The black line highlights the even adult sex ratio i.e. 1:1. In (A), (B) and (C): When the cost of PI = 0 and there is no sex difference in immune response ( $\Omega = \Delta = \Theta = 0$ ), the obtained adult sex ratio is 1:1. In (A) and (C): when PI increases, the frequency of Sex 1 drops as PI is costly. When  $\Omega > 0$  and  $\Delta > 0$ , this sex difference in immune response compensates for the cost of PI. The fall in frequency of Sex 1 is lower than when  $\Omega = 0$  and  $\Delta = 0$  and Sex 1 has higher frequency than Sex 2 for most values of PI cost. However, when  $\Omega < 0$  and  $\Delta < 0$ , Sex 1's frequency decreases with an increase in PI. In (B): Frequency of Sex 1 is lower than Sex 2 for most values of PI cost for most  $\Theta$  values. Moreover,  $\Theta < 0$  and  $\Theta > 0$  give the same results. The above results highlight the fact that sexual conflict within immune allelic diversity can increase (when  $\Omega > 0$  and  $\Delta > 0$ ) or reduce (when  $\Omega < 0$ ,  $\delta < 0$ , almost all  $\Theta$ ) the adult sex ratio.

163 and there is no sex-biased difference in immune response, we observe that the sex ratio is 1 : 1. Here,  
 164 we focus on the adult sex ratio (ASR) (Kokko and Jennions, 2008). The classical definition of ASR  
 165 is number of males:total number of males and females, but in our Sex 1 could be male or female.  
 166 In this manuscript the term ASR is defined as the ratio between Sex 1 and Sex 2. Since in every  
 167 generation, offspring are produced in equal sex ratios (see ESM), what we obtain is the sex ratio of  
 168 the offspring after they become adults, perform mating interactions and parental investments. The  
 169 frequency of Sex 1 decreases with increasing PI. However, Sex 1 increases in frequency under certain  
 170 cases of sexual conflict over the immune allelic diversity (see  $\Delta > 0$ ,  $\Omega > 0$ , or  $\Theta \neq 0$  in Figure 2).  
 171 The results after including mating competitions are plotted in the figures in the ESM.

172 Under selection, the obtained genotypes deviated from the Hardy-Weinberg equilibrium (see Fig-  
 173 ures ??, ?? and ?? of the ESM). One sex has a higher number of heterozygotes when compared  
 174 to the other sex. In this setup, the heterozygous immune genotype ( $A_1A_2$ ) has a higher immune  
 175 response than the homozygous genotypes  $A_1A_1$  and  $A_2A_2$  (Figure 3). Thus, an increase in heterozy-  
 176 gotes within one sex compared to the other would also mean that this sex has a higher mean activity  
 177 of the immune system. There are scenarios, such as a recent study with wild songbird populations,  
 178 where the number of heterozygotes and homozygotes even under selection turned out to be equal  
 179 between the sexes (Roved, 2019). However, this could just be the result of a particular immune  
 180 response profile, parental investment and ornamentation costs in that species. Different profiles of  
 181 sexual conflict within the immune allelic diversity would determine different ratios of homozygotes

182 and heterozygotes. More empirical studies with various model organisms would shed light on how  
 183 species show diverse ways of sexual conflict within the immune allelic diversity.

184 **Nonlinear diversity profile**

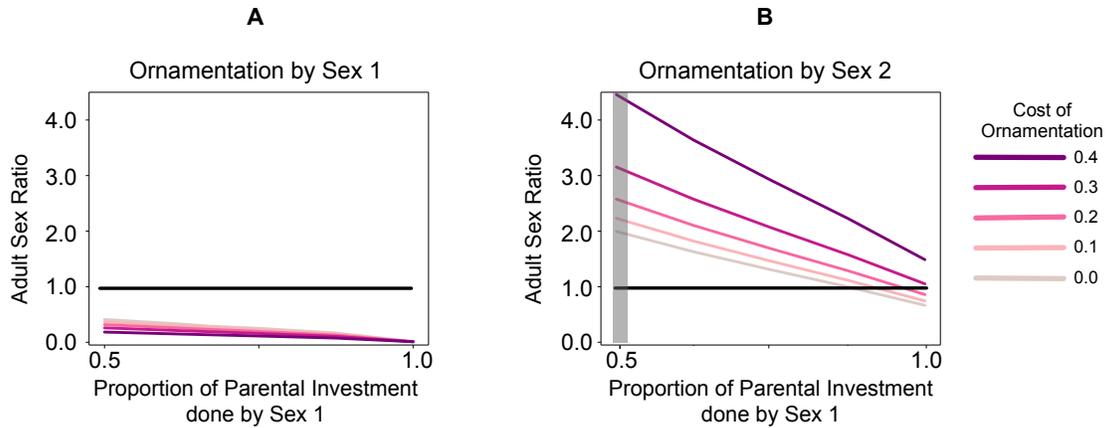


Figure 5: Qualitative difference in the adult sex ratio for diverse polygamous species with varying parental investment (PI) and ornamentation costs. As defined throughout the manuscript, Sex 1 is the major PI provider. For these calculations, we used the sexual conflict Case 4 shown in Figure 3. (A) Species such as sticklebacks where one sex performs both ornamentation and most PI. We observe that frequency of Sex 1 descends as its PI cost increases and this further decreases with a rise in its ornamentation cost. (B) The panel highlighted in gray shows bi-parental investment scenarios. In species where Sex 1 does most PI and Sex 2 performs elaborate mating competitions, the frequency of Sex 1 reduces with increasing PI. However, this value grows with ascending ornamentation cost in Sex 2. Note that for certain ornamentation and PI values, the adult sex ratios are equal. As shown by previous studies on multiple interactions between traits (Venkateswaran and Gokhale, 2019), even in the case where the cost of ornamentation is equal to zero in the mating competition game, the mere presence of that game will deviate the frequency of Sex 2 from a scenario where there is no ornamentation game.

185 In a multi-loci scenario, one can include non-linear density profiles (Nowak et al., 1992; Woelfing  
 186 et al., 2009) as shown in Figure 3. Across species, different sex-specific immune response profiles can  
 187 be found, depending on the sex-specific selection and phenotypic divergence (Uekert et al., 2006;  
 188 Love et al., 2008; Oertelt-Prigione, 2012). We hypothesize two such scenarios,

- 189 • the optimal diversity of immune alleles for both sexes is the same but the immune responses  
 190 at this optimal diversity could be different (for instance, females are more prone to acquir-  
 191 ing autoimmune diseases; sex hormones such as estrogen, testosterone also affect immune  
 192 response (Hillgarth and Wingfield, 1997; Törnwall et al., 1999; Whitacre, 2001) or,
- 193 • the two sexes have different optimal diversity of immune alleles and the immune response at  
 194 this optimal diversity is the same for both sexes. For instance, as shown in Roved et al. (2017,  
 195 2018), males and females have a different optimal diversity, where males need a higher number  
 196 of allele diversity to mount maximum immune response. We considered such a scenario for  
 197 this study (see Figure 3).

198 As done for the one locus scenario, we assume that only the number of different alleles i.e. allele  
199 diversity produces unequal fitness.

## 200 **Adult sex ratio in various species**

201 Our results showed that a sexual conflict within immune allelic diversity and varying parental invest-  
202 ment may result in adult sex ratio bias. The effect of ornamentation also plays an important role in  
203 skewing adult sex ratios as shown Figure 5. Diverse reproducing species have distinct ornamentation  
204 and parental investment costs. Figure 5 shows the values of adult sex ratios that our model predicts  
205 for a wide range of species.

## 206 **Discussion**

207 Various intersexual and intrasexual interactions during the reproductive lifespan of an individual  
208 determine its lifetime reproductive success (Stoehr and Kokko, 2006; Kalbe et al., 2009; Kelly  
209 and Alonzo, 2010). We have presented a model framework where several individual life-history  
210 interactions can be studied simultaneously. As shown in many empirical studies, the ASR has  
211 an impact on sex-specific differences and roles (Liker et al., 2013; Székely et al., 2014; Liker et al.,  
212 2015; Henshaw et al., 2019). Our results showed that the interaction of sex-specific life-history traits  
213 result in a biased adult sex ratio (ASR) (Pipoly et al., 2015). We showed that the vice versa is also  
214 possible (Kokko and Jennions, 2008) i.e., our results showed that ASR is a consequence of sex-specific  
215 differences. Our model incorporates the fact that fitness is a complex entity (Doebeli et al., 2017).  
216 The overall lifetime reproductive success is a combination of fitness values arising from the individual  
217 life-history strategies (here, parental investment, ornamentation and immunocompetence). This  
218 model showed that the variation in individuals' or the sex-specific lifetime reproductive success (based  
219 on their cost of parental investment, ornamentation and immune response levels) has population  
220 level consequences i.e. a skew in adult sex ratio (see Figures ?? and ?? in the ESM). Here, the  
221 females and males of one generation mate and produce equal numbers of daughters and sons in the  
222 next generation. Therefore, at birth, sex ratio of every generation was 1:1. The life-history traits are  
223 passed on from parents to offspring. Thus, even though every generation starts with equal sex ratio,  
224 their sex-specific traits change the adult sex ratio in every generation until it reaches an equilibrium  
225 state.

226 If a sex does both ornamentation and maximum parental investment, i.e. pays high costs of  
227 ornamentation and PI (eg. stickleback males), the ASR will be biased towards the sex that bears  
228 negligible costs for ornamentation and PI (e.g. female sticklebacks) (Hagen and Gilbertson, 1973)).  
229 Thus, the high costs for contributing to both PI and ornamentation cannot be compensated (Daly,  
230 1978) (Figure 5.A).

231 In birds and free-spawning fish both sexes exhibit similar levels of parental investment (equally  
232 little parental investment by both sexes in case of free-spawning fish) (Perrone Jr and Zaret, 1979;  
233 Gross and Sargent, 1985; Cockburn, 2006). Our model shows that these species could show equal  
234 ASR for certain parental investment and ornamentation levels (see Figure 5.B). However, in species  
235 where males have a higher ornamentation level, the ASR will be biased. For instance, free-spawning  
236 species such as the Atlantic salmon where males have elaborate ornaments, show a high adult sex  
237 ratio (7:1 ratio of males to females) (Mobley et al., 2019). Therefore, the high sex ratio values  
238 shown in the gray shaded region of Figure 5.B matches natural observations.

239 When one sex does maximum parental investment while the other displays ornaments, ASR is  
240 biased towards the sex that does more parental investment, as the other sex has to pay the costs  
241 of ornament display (Figure 5.B). Consider the pipefish species *N. ophidion* where males glue the

242 eggs on the belly and thus perform partial parental investment (Berglund et al., 1986). In contrast  
243 to pipefish species with placenta-like structure and an active transfer of nutrients and oxygen to the  
244 embryo (e.g. *S. typhle* (Berglund et al., 1986; Smith and Wootton, 1999)), *N. ophidion* only provide  
245 partial parental investment. We thus expect a decrease in frequency of *S. typhle* males compared  
246 to *N. ophidion* males (Berglund and Rosenqvist, 2003). However, with increasing ornamentation  
247 in females the frequency of males increases. Ornaments are costly as they make the bearer more  
248 vulnerable to predation. According to Bateman's principle (Bateman, 1948), the reproductive success  
249 of the sex that performs mating competition depends on the number of mating events. The sex  
250 limited by parental investment will have to live longer for more reproductive events to achieve  
251 the same reproductive success as the males (Roth et al., 2011). Thus sex differences in parental  
252 investment, ornamentation and immunity (Trivers, 1972; Hedrick and Temeles, 1989; Trivers, 2002;  
253 Roved et al., 2017) may also give rise to sexual differences in longevity, an important life-history  
254 trait (Austad, 2006; May, 2007).

255 Our model can be used to determine the lifetime reproductive success using fitness arising from  
256 sex-specific differences in life-history traits of a particular sex in a population e.g. parental invest-  
257 ment, ornamentation and immunocompetence. Studying the combined dynamics of life-history traits  
258 highlights population level consequences such as skewed adult sex ratio (Trivers, 2002; Kokko and  
259 Jennions, 2008) emerging due to sex-specific differences in life-history traits. With the aid of more  
260 empirical work directed towards investigating sexual conflict within the immune allelic diversity and  
261 other life-history strategies, we can obtain deeper understanding of the overall life-history of a sex or  
262 species. Disruptive selection leads to sexual dimorphism and in models that use tools like adaptive  
263 dynamics, traits that go through evolutionary branching may end up as two sex-specific traits i.e.  
264 sexual dimorphism. Recent studies addressed how coevolution of traits and resource competition  
265 drive the evolution of sexual dimorphism (Bolnick and Doebeli, 2003; Stoehr and Kokko, 2006;  
266 Vasconcelos and Rueffler, in press). Work by Vasconcelos and Rueffler (in press) demonstrated that  
267 even weak trade-offs between life-history traits can result in evolutionary branching that leads to  
268 evolution of two co-existing types. In this study, we investigated the eco-evolutionary consequences  
269 of interplay between two or more sex-specific life-history traits. Along with empirical evidence that  
270 matches our qualitative predictions, suggesting a skewed adult sex-ratio.

271 The functions in our model that describe fitness from parental investment and ornamentation  
272 consider polygamous species. While many sexually reproducing animals are polygamous, species  
273 like seahorses are monogamous throughout their lifetime (Vincent and Sadler, 1995). The trade-  
274 offs between ornamentation, parental investment and immunocompetence in monogamous species  
275 would be different. For instance, they may not have to bear costs of attracting mates after one  
276 brooding season. Our model can be modified to study the effect of integrating monogamous mating  
277 patterns. Also, with regard to immune genes such as the ones of the MHC, genetically dissimilar  
278 individuals mate more often as the evolutionary incentive is to produce optimal MHC diversity  
279 offspring (Milinski, 2006; Woelfing et al., 2009; Kalbe et al., 2009; Eizaguirre et al., 2009). To  
280 this end, mating is not random. Aspects of a model by Kirkpatrick (Kirkpatrick, 1982) for two  
281 autosomal loci with female mating preference for a trait that occurs in males is a potential extension  
282 of our model. Finally, novel studies directed at sexual conflict within the MHC and other immune  
283 genes as done by Roved et al. (2018) shall be very beneficial in providing further knowledge of how  
284 sex-specific immune defences manifest in different systems with distinct sex-specific ornamentation  
285 and parental investment patterns.

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# Chapter 4

## Emergence of species or sex specific differences in life-history traits

“Natural selection is ecology in action.”

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— Winfried Lampert

### 4.1 The relationship between parental investment and sexual immune dimorphism

The last two chapters (Chapter 2 and Chapter 3), highlighted the importance and consequences of probing combined interactions between individuals or traits. Chapter 3 investigated the outcomes of the combined dynamics of various life-history traits (ornamentation, parental investment, and immunocompetence) that have sex-specific differences. In the model described in the preceding chapter (Chapter 3), we used sex-specific differences in life-history traits as given parameters. But how does sexual dimorphism emerge in the first place? There might be different mechanisms for the presence of sexual dimorphism for each of the above-mentioned traits. Sex-specific differences

in one trait might influence sexual dimorphism in another due to the competition for reserves (between the traits) that result in sex-specific trade-offs. I used a state-dependent dynamic optimization method, where parental investment and immunocompetence are two coupled traits of an individual. I studied the trade-off between individuals' reserves allocated to parental effort and to immunity in such a way that they maximize an individual's fitness.

The evolution of sex-specific differences in the amount of parental investment (Trivers, 1972; Lonstein and De Vries, 2000; Alonzo and Klug, 2012; Liker et al., 2015) and ornamentation (Darwin, 1871; Jones et al., 2000; De Lisle, 2019) have been studied both experimentally and theoretically. This chapter presents a model that studied the emergence of sexual dimorphism in the ability to mount immune responses (immunocompetence) i.e. sexual immune dimorphism. There have been studies, both experimental and theoretical to investigate the causes of sexual immune dimorphism (Stoehr and Kokko, 2006; Forbes, 2007; Nunn et al., 2008; Restif and Amos, 2010). But the theoretical models were focused on the trade-off between ornamentation and immunocompetence. An important trait was missing in them which is parental investment. Parental investment has three parts: gamete production, pregnancy, and external parental care (Trivers, 1972). Experimental results show that parental investment affects immunocompetence. For instance, it has been shown that pregnancy, mouthbrooding, etc give rise to a better immunocompetence in the sex that performs them (Roth et al., 2011; Lin et al., 2016; Peck et al., 2016; Keller et al., 2017).

Using mathematical models where parental investment and immunocompetence can be considered as different traits of an individual of a particular sex, I have developed a model to show how these traits affect each other.

The model introduced in this chapter studies the ecology of sexually reproducing animals during their reproductive lifetime. We look at sets of individuals and track them throughout their reproductive lifetime to investigate how they allocate resources for parental investment and

immunocompetence, respectively. Here, we do not track their offspring or the evolution of the parents' traits. This is a theoretical ecology, life-history and resource allocation model whose outcomes are similar to empirical works that track at a population throughout its lifetime minus the offspring. In these cases, one studies selection acting on the variation of traits. Evolution by natural selection happens when these variations are inherited to the next generations. Thus, future extensions of this model should be towards the direction of studying evolution by natural selection i.e. how do the fittest individuals in the first generation pass on their traits to the offspring? If they do, how do the traits in that species evolve? For now, I focused on how individuals in a population make decisions based on the ecological factors affecting them.

# The effect of parental investment on immunocompetence and sexual immune dimorphism

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Sexes of a species show different characteristics beyond the differences in their sexual organs; this is known as sexual dimorphism and applies to immunocompetence as well. Immunocompetence is the ability of an individual to mount an immune response when exposed to pathogens. Females are shown to have increased longevity that comes with higher immunocompetence as compared to males and this may also lead to an increased probability of autoimmune disease in females. However, for some species such as pipefishes and seahorses belonging to the Syngnathid family, studies show that the males have a higher immunocompetence. Experimental evidences suggest that this could be due to the fact that these males undergo pregnancy i.e. the males have brood pouches where the eggs are fertilized; the fathers provide oxygen and nutrition to their offspring until they give birth to the juveniles. Therefore, an increase in immunocompetence may also be related to the amount of parental investment. In this study, using state dependent life-history theory, we show that for most species systems it is optimal to invest more in immunocompetence when the time spent in parental investment is longer. Our findings also show that an increase in parental investment brings about an earlier immunosenescence i.e. the gradual deterioration of the immune system that occurs with aging. We observe that an increase in investment towards immunocompetence is more pronounced in short-lived species with long brooding periods whereas species with a longer lifespan allocate more reserves towards offspring production. Our model also accounts

for intraspecies scenarios: if a sex spends a longer fraction of its reproductive season in pregnancy or brooding (as compared to the other sex), then we find that this sex would invest more towards immunocompetence.

## Introduction

Crucial life-history parameters such as body size, reproductive investment, immunocompetence (the ability of an individual to mount an immune response when exposed to pathogens) have sex-specific differences. The evolution of sex-specific differences in the amount of parental investment (Liker et al., 2015; Lonstein and De Vries, 2000; Alonzo and Klug, 2012; Trivers, 1972) and mating competition through ornamentations (Jones et al., 2000; Darwin, 1871; De Lisle, 2019) have been studied experimentally and theoretically. Sexes of a species also show sexual dimorphism in immunocompetence i.e. sexual immune dimorphism. There have been studies, both experimental and theoretical to study the occurrence of sexual immune dimorphism (Stoehr and Kokko, 2006; Restif and Amos, 2010; Nunn et al., 2008; Forbes, 2007). Nunn et al. (2008) presented a means to study Bateman's principle for immunity independently of the immunocompetence handicaps hypothesis (ICHH) due to lack of testosterone in insects. They found a female bias in immunocompetence and showed that ICHH lacks a generic empirical support. They highlighted the fact that immunocompetence can be explained using fitness-related differences between the sexes. A review by Forbes (2007) illuminated a model by Stoehr and Kokko (2006) that tested two common hypotheses that longevity is more important to a female's fitness than for a male's fitness and the benefit from investing in immunocompetence is an increase in longevity. The model showed that the male immunocompetence is lower than that of females when the above hypotheses are satisfied. But Stoehr and Kokko (2006) further showed that sex differences in parasitic impact might cause males to invest more into immunocompetence. They modeled the impact of parasites on condition and that this might differ between the sexes. Another study by Medley (2002) also demonstrated an increased investment in immunocompetence when the parasitic impact increases. An adaptive dynamic model which considered both host sexual reproduction host-pathogen population dynamics was developed by Restif and Amos (2010) although it does not involve sexual selection. Their results revealed the importance of ecological feedback loops in the eco-evolution of immune system.

The previous theoretical models mentioned above that investigated evolution of immune systems were mostly in the lines of studying trade-offs between ornamentation and immunocompetence. These studies missed an important factor - parental investment, which is the focus of our study.

Parental investment has three parts: gamete production, pregnancy, and parental care. Gamete production is common to both sexes and the difference in gamete size (anisogamy) is where the definition of the sexes comes about: large gamete (egg) producers are referred to as females, and smaller gamete (sperm) producers are called males. The time taken for parental investment varies across taxa. In some taxa, parental investment involves preparing a nest, in others, it also involve taking care of the eggs until they hatch. In some species, the maximum time taken for parental investment is in brooding the eggs. Female mammals and males of the Syngathidae family undergo pregnancy. During gestation or brooding period, the offspring is particularly dependent on the sex that performs pregnancy or brooding.

Experimental results across diverse taxa show that increase in parental investment can be linked to higher investments in immunocompetence in that sex (Roth et al., 2011; Keller et al., 2017; Lin et al., 2016; Peck et al., 2016). This might be because the sex that performs mating competitions can maximize their reproductive fitness earlier in life if they are able to successfully compete for mating opportunities. In intrasexual competition, benefit from investing more in ornaments, weaponry,

and size might come at an expense of immunocompetence. The sex that is involved in mating competitions through ornamentation usually has a higher variance (as compared to other sex) in reproductive success (Bateman, 1948). It may hence be beneficial for this sex to invest more in the current reproductive season even if it may affect their survival to the next reproductive season. On the other hand, the sex that is limited by pregnancy and/or parental care can maximize fitness by living longer through greater investment in immune defenses (Nunn et al., 2008). Thus, an increase in immunocompetence must be related to the amount of parental investment in addition to ornamentation and other factors. For instance, in mammals, we know that females tend to have increased longevity that comes with higher immunocompetence and this could be because female mammals go through pregnancy i.e. periods of gestation (Forbes, 2007; Rolff, 2002; May, 2007). However, in pipefishes, the males undergo pregnancy. In most pipefish species, the males have a pouch on its stomach. The male receives eggs from females in this brood pouch and the eggs are fertilized here. In this pouch, the fathers provide his embryos with nutrients (Wilson et al., 2003). Experimental studies show that the males of these species have a higher immunocompetence (Roth et al., 2011). In seahorses, males perform mating competitions as well as pregnancy (Vincent et al., 1992). The mode of pregnancy in seahorses is similar to that of pipefishes. The males have a completely enclosed saclike fleshy pouch where eggs are incubated. Here, virgin males showed higher levels immunocompetence (Lin et al., 2016).

In this study, we focus on the effect of parental investment on immunocompetence. Using state dependent life history theory (Houston and McNamara, 1999; Mangel and Clark, 1988; Clark and Mangel, 2000), where parental investment and immunocompetence are considered to be two different traits of an individual, we studied the trade-offs between resources allocated to these traits in such a way that they maximize individuals' fitness. We also observe that immunosenescence, which is the dip in immune responses that happens later in life, is also affected by parental investment as observed in experimental data (Das et al., 2008; Yan et al., 2010).

## Methods

Let us consider an individual that has  $R(t)$  amount of reserves at a time  $t$ . These reserves are either allocated to improve immunocompetence or directed towards offspring production. There are natural observations and empirical studies that highlight such resource allocation across species of the animal kingdom (Peck et al., 2016; Mangel and Heimpel, 1998; Lin et al., 2016; Loiseau et al., 2008; Contreras-Garduño et al., 2006; Wedell and Karlsson, 2003; Verhulst et al., 2005). Reproductive success mainly results from trade-offs in allocations of resources between an individual's survival and its offspring success as shown in Figure 1.

We shall refer to the reserves allocated towards one's own immunity and the parental effort as  $r_i$  and  $r_o$ , respectively. Here, the number of offspring produced by a parent is a decelerating function of the amount of resources allocated to care i.e.  $\Phi = r_o^\alpha$ , with  $0 < \alpha < 1$  (see Figure 2a). Investment towards the immune system will increase the parent's survival. Thus, we assume that the probability of survival in the current reproductive period is  $S_{current} = e^{\frac{-Mf}{1+\gamma r_i}}$ , where  $f$  is the fraction of time spent in gamete production plus pregnancy or brooding (depending on the taxa) as discussed in the Introduction,  $M$  is the background mortality and  $\gamma$  determines the effect that investment in immunocompetence has on this background mortality i.e. it is a scaling factor of the effect of investment in immunocompetence ( $r_i$ ) on survival. The parent that the offspring are dependent on has to survive throughout the current reproductive period for the offspring to survive. Thus, the current reproductive success would be a multiplicative function written as  $\Phi \cdot S_{current}$ . The probability that an individual survives to the next reproductive period is given by  $S_{future} = e^{\frac{-M}{1+\gamma r_i}}$ .

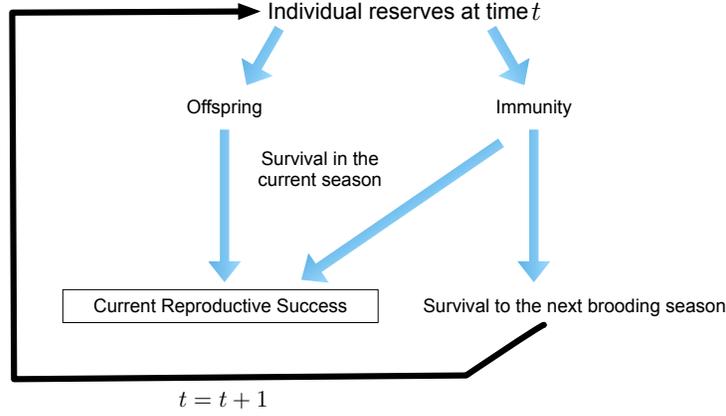


Figure 1: **Allocation of an individual's reserves** towards offspring and parents' survivability together determine the reproductive success in the current reproductive season. Survivability to the next season depends on the amount of reserves for immunity allocated towards future survival.

In this discrete time model, the reserves available for immunocompetence or reproduction change throughout the lifetime of an individual. The function describing the gain in reserves  $g(t)$  at time  $t$ , is shown in Figure 2b. The function for  $g(t)$  is a decelerating one which saturates since the total amount of reserves that an individual gains cannot increase indefinitely.

We use a state dependent dynamic model (Mangel and Clark, 1988) to investigate the optimal allocation towards immunocompetence and parental effort. We do this for individuals that differ in the length of time spent in pregnancy or brooding  $f$ , across environments that vary in background mortality  $M$  and the effect that immunocompetence has on survival  $\gamma$ . As a metric of fitness, we use accumulated lifetime reproductive success (Stearns, 1992; Roff, 1993). To do so, we let  $W(r, t)$  denote the maximum (taken over investment) expected (taken over stochastic events) accumulated lifetime reproductive success from time  $t$  onwards, given that  $R(t) = r$ . We will call  $W(r, t)$  the fitness function. At time  $t + 1$ , its reserves will change to,

$$R(t + 1) = r - r_o^*(r, t) - r_i^*(r, t) + g(t), \quad (1)$$

where  $r_o^*(r, t)$  and  $r_i^*(r, t)$  are the optimal values for  $(r_o, r_i)$  for  $r$  reserves at time  $t$  and  $g(t)$  is the gain in reserves after each reproductive period. The value  $r$  is bound by  $R_{max}$  which is the maximum value of the total reserves. An individual is no longer alive when  $r \leq 0$ .

For all combinations of the reserve state  $R(t)$  and time  $t$ , we find the allocation to immunocompetence  $r_i$  and parental effort  $r_o$  that maximize  $W(r, t)$ . Assuming that no reproductive success can be accumulated at or beyond time  $T$  i.e  $T$  is the time of reproductive senescence, we have the end condition  $W(r, T) = 0$ . For previous times,

$$W(r, t) = \max_{r_i, r_o} \left[ \Phi \cdot S_{current} + S_{future} \cdot W(r', t + 1) \right]. \quad (2)$$

That is, expected cumulative fitness  $W(r, t)$  for  $R(t) = r$  reserves used at time  $t$  is equal to its current reproductive success plus the future reproductive success (see Figure 1). In table 1, we summarize the variables and parameters in the model. Solution of Equation 2 gives the optimal values of investment,  $r_o^*(r, t)$  and  $r_i^*(r, t)$  for every possible value of  $r$  and at each time. As seen in Equation 2, one must go backwards from end time  $T$  to a starting time to get the optimal values

for  $(r_o, r_i)$  from  $r$  that the individual would allocate for all the times.

Table 1: A list and summary of parameters used in the model.

Parameter	Definition
$R(t) = r$	Reserves at time $t$
$R_{max} = 1.0$	Maximum value of total reserves
$r_o$	Reserves allocated towards (energy required for) parental effort i.e. for producing eggs and offspring
$r_i$	Immune reserves allocated towards the parent's survival in a reproductive season
$f$	Fraction of one time period used in pregnancy or brooding
$\Phi = r_o^\alpha$	Offspring produced
$M$	Background mortality
$\gamma$	Scaling factor for the effect of investment in immunocompetence ( $r_i$ ) on survival
$g(t)$	Gain in reserves after each reproductive period
$S_{current} = e^{\frac{-Mf}{1+\gamma r_i}}$	Probability that the parent survives in the current reproductive season
$\Phi \cdot S$	Current fitness i.e. offspring success (the number of offspring surviving)
$S_{future} = e^{\frac{-M}{1+\gamma r_i}}$	Probability of survival to next reproductive season
$W(r, t)$	Fitness function
$\sigma$	Variance in $g(t)$ in a fluctuating (stochastic environment).
$g_h = g(t) + \sigma$ and $g_l = g(t) - \sigma$	Highest and lowest values that $g(t)$ can take

### Including stochasticity in the amount of resources gained between each reproductive season

In the previous section we assumed that the reserves increased deterministically between seasons. In Equation 2, we have assumed that survival is a stochastic event, but that the increment in reserves is deterministic. We now consider the situation in which the gain in reserves fluctuates depending on the time and environment. Here,  $g_h(t) = g(t) + \sigma$  and  $g_l(t) = g(t) - \sigma$  are the two upper and lower values of resource that can be gained by an individual where  $0 < \sigma < R_{max}$ . Let  $x$  be the probability of receiving  $g_h(t)$ . For this study, we assumed equal probability of obtaining  $g_h(t)$  and  $g_l(t)$ . Thus  $g_h(t)$  and  $g_l(t)$  average to  $g(t)$  and this can happen for different values of  $\sigma$ . i.e resource gain can fluctuate according to  $\sigma$  around  $g(t)$ . To capture this, one would use a stochastic version of the model where Equation 2 would be re-written as,

$$W(r, t) = \max_{r_i, r_o} \left[ \Phi \cdot S_{current} + \frac{1}{2} [S_{future} \cdot W(r_h, t + 1)] + \frac{1}{2} [S_{future} \cdot W(r_l, t + 1)] \right] \quad (3)$$

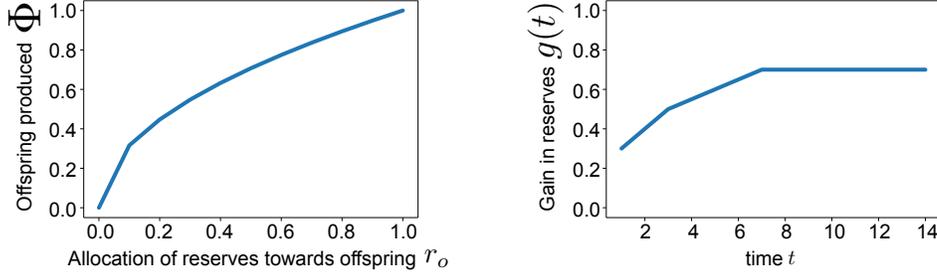


Figure 2: **Functions describing offspring produced  $\Phi = r_o^\alpha$  and gain in reserves after each reproductive period  $g(t)$ .** (a) For the results presented in this study, offspring success  $\Phi$  is an increasing function with respect to reserves allocated towards offspring success ( $r_o$ ). Here,  $\alpha = 0.5$ . (b) Gain in reserves after each reproductive period  $g(t)$  increases with time  $t$  (here,  $g(t) = \frac{0.3}{2+4e^{-0.5t}}$ ). In the beginning of an individual's lifetime, an individual is smaller and would spend more on growth. But over time it stops growing and therefore, more resources can be spend on reproduction. The function  $g(t)$  saturates since the total amount of reserves that an individual gains cannot increase indefinitely.

where  $r = R(t)$  and  $r_h = r - r_o^*(r, t) - r_i^*(r, t) + g_h(t)$ ,  $r_l = r - r_o^*(r, t) - r_i^*(r, t) + g_l(t)$ .

### Forward Monte Carlo simulation algorithm

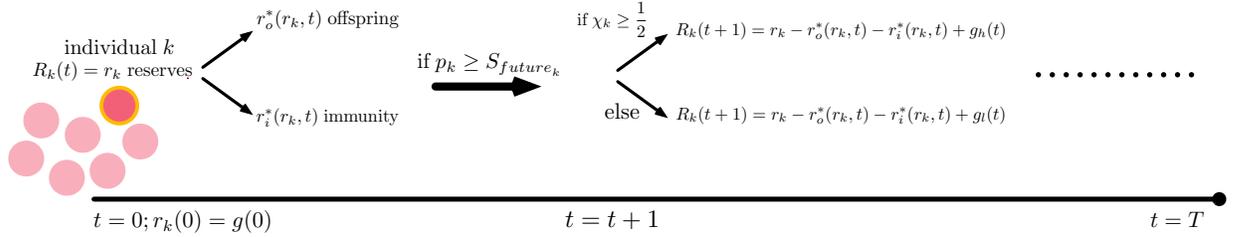


Figure 3: **Forward stochastic routine** Algorithm of the forward Monte Carlo simulation used for tracking individuals' dynamics. Here,  $g(t)$  equals gain in reserves at each reproductive period, and  $g_h(t)$  and  $g_l(t)$  are the two upper and lower values of resource that can be gained by an individual  $k$  at time  $t$ . As described previously,  $x$  be the probability of receiving  $g_h(t)$  and  $S_{future_k} = e^{\frac{-M}{1+\gamma r_i^*(r_k, t)}}$  is the probability of survival to next reproductive season. The values  $p_k \in [0, 1]$  and  $\chi_k \in [0, 1]$  are two numbers drawn at random for each individual at every time which determine their survival to the next brooding season and gain in reserves to the upper value  $g_h(t)$ , respectively.

The backward (in time) method provides us with a 'handbook' of all possible optimal values of  $(r_o, r_i)$  at any given  $r$  for time  $t$ . However, this method shows all possible outcomes. At any given  $r$  for time  $t$ . We simulate the dynamics of an individual  $k$  starting with an initial amount of reserves  $R_k(0) = g(0)$  (Forward routine). With an equal probability, the individual will encounter the high or low reserve gain values  $g_h$  and  $g_l$ , respectively. This way, at time  $t + 1$ , an individual's reserves

will be,

$$R_k(t+1) = r_k - r_o^*(r_k, t) - r_i^*(r_k, t) + \begin{cases} g_h(t) \\ \text{or} \\ g_l(t) \end{cases} \quad (4)$$

where  $r_o^*(r_k, t)$  and  $r_i^*(r_k, t)$  are the optimal values ( $r_o, r_i$ ) for  $r_k$  reserves at time  $t$  for individual  $k$ . The individuals die when their reserves drop to a critical level. Here, when  $r_k \leq 0$ , the individual dies as it no longer can invest in its own immunity. The survival function  $S_{future_k}$  gives the probability of an individual  $k$ 's survival to the next reproductive season. A flowchart illustrating this algorithm is shown in Figure 3.

## Results

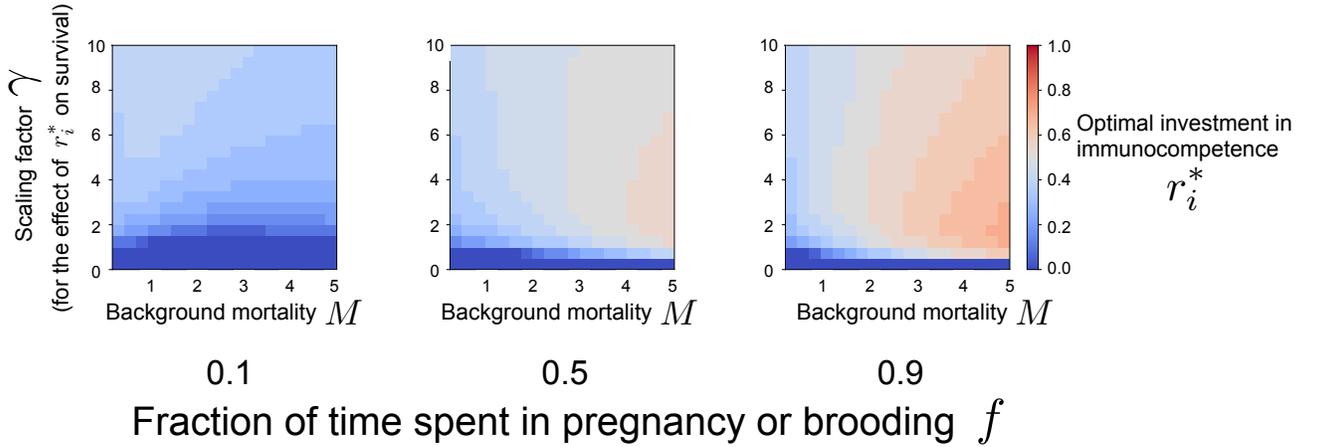


Figure 4: **Investment in immunocompetence as a function of background mortality ( $M$ ), scaling factor for the effect of investment in immunocompetence on survival ( $\gamma$ ) for different lengths of the brooding or pregnancy period ( $f$ ).** In general, more resources are allocated towards immunocompetence as parental investment increases. Ascending background mortality further makes the individual allocate more towards immunocompetence. When background mortality is high, having a low value of  $\gamma$  allows individuals to invest more reserves into immunocompetence. These results are for  $R(1) = R_{max}$ , where  $R_{max} = 1.0$  is the maximum value of total reserves as mentioned in Table 1.

The optimal invest in immunocompetence  $r_i$  for a range of background mortality  $M$  and scaling factor for the effect of investment in immunocompetence ( $r_i$ ) on survival  $\gamma$  values using the backward (in time) deterministic Equation 2 is shown in Figure 4. This result manifests the effect that the fraction of time spent in parental investment has on immunocompetence. For a given value of  $M$  and  $\gamma$ , with increasing fraction of time spent in internal parental investment or brooding ( $f$ ), the optimal investment of reserves towards immunocompetence also increases. We can also observe that an increasing amount of background mortality, further increases an individual to boost its immune responses by investing more in immunocompetence and generally more so for smaller values of  $\gamma$ .

A general pattern of the effect of fraction of time spent in pregnancy or brooding on immunocompetence is shown in Figure 5(a). The individuals of this population were tracked from the beginning i.e. time  $t = 0$  until the time of reproductive senescence  $T$ . From the results shown in this figure,

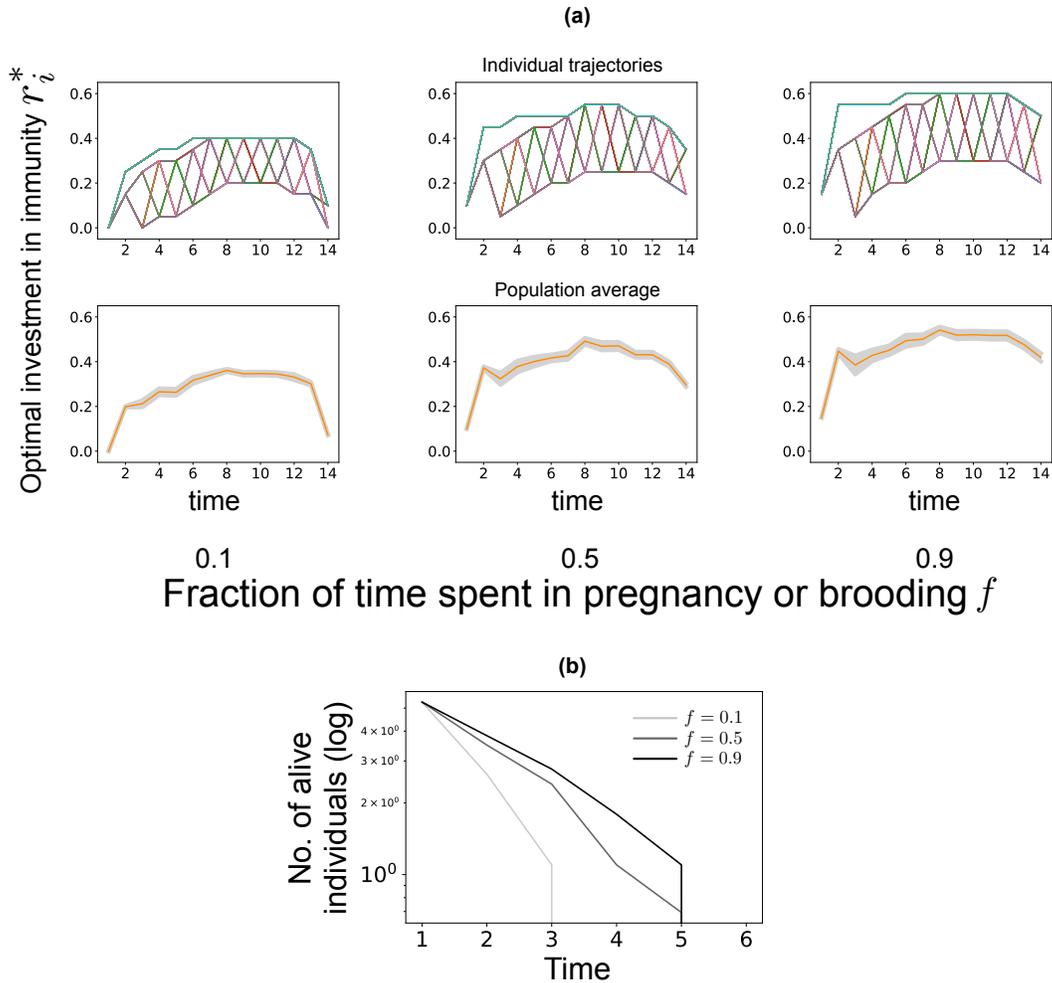


Figure 5: **Effect of time spent in pregnancy or brooding on immunocompetence** The values for background mortality and mortality scaling factor are  $M = 2.5$  and  $\gamma = 5$ . Population size is 200 and  $\sigma = 0.3$ . (a) The individual trajectories are plotted for the 200 individuals in the population. These trajectories rise and fall since at each brooding season, an individual can receive a high ( $g_h$ ) or low ( $g_l$ ) value of gain in reserves. Immunocompetence initially increases with time  $t$ , and drops later in life which is referred to as immunosenescence. There is a slight rise in the peak with increasing fraction of time spent in parental investment,  $f$ . The population average is plotted by the orange lines in the second row. The gray shaded area shows the variance between these 200 individuals. (b) Population density decreases with time. This decrease is slower for a higher  $f$ , and this highlights the positive effect parental investment in life-span. The slope of each line is the realized mortality experienced by the population and this is a property that has emerged out of this model.

we infer that investment in immunocompetence increases with time  $t$  for all values of pregnancy or brooding periods  $f$ . Investment towards immunocompetence initially increases, but drops with time. Therefore, these results also show the occurrence of immunosenescence, which is the phenomenon

of immunocompetence dropping later in life (Metcalf et al., 2019). We could also compare the investment of a parent having a long pregnancy or brooding period (high  $f$ ) to another parent having a negligible investment ( $f = 0.1$ ). This would correspond to different sexes of a species where one parent undergoes pregnancy or brooding. The plots in Figure 5(a) also show that this increase of investment in immunocompetence is higher for the parent having larger  $f$ . Figure 5(b) shows how the population density decreases with time. However, this decrease is slower for a higher  $f$ , and this highlights how pregnancy affects mortality i.e. when an individual undergoes pregnancy, it becomes beneficial to invest more resources in immunocompetence leading to higher survival.

### Optimal allocation of reserves in long-lived and short-lived species

The resource allocation towards immunocompetence in relation to pregnancy or brooding period, for both long and short-lived species is shown in Figures 6 and 7. Here, long-lived and short-lived species have low  $M$  and high  $M$ , respectively. The effect of  $\sigma$  (variation in high and low resource gain between seasons) in the optimal allocation of resources in short-lived species do not differ much between the different  $\sigma$  value (see Appendix). Our results show that short-lived species spend more reserves for producing offspring rather than their own immunocompetence. This further decreases for a low  $\gamma$  (see Figure 6). However, for high  $f$  values, we see an increase in investment of resources towards immunocompetence i.e. investment towards immunocompetence is stronger in short-lived species with long brooding periods. This is more pronounced under high  $\gamma$  conditions. We see that when the effect of invest towards immunocompetence on survival is low, it drives the individuals to allocate more towards their offspring since they most likely would not live for more than one reproductive season. On the other hand, for long-lived species shown in Fig. 7, individuals live longer as compared to short-lived individuals shown in Fig. 6. For such species, the background mortality is low which allows them to focus their reserves to offspring production since they would anyway survive to next reproductive seasons even without much invest to the immune system. As observed in short-lived species, here too we see that when  $\gamma$  is low, the individuals allocate more towards their offspring to ensure as much reproductive success as possible while the individual is alive. For both low and higher  $\gamma$  values, the investment towards immunocompetence increases in long-lived species for longer brooding or pregnancy periods (higher  $f$  values). For both short-lived and long-lived species, the results also show how species with high  $\gamma$  value allocate more towards their immunocompetence and this effect is stronger for short-lived species and low  $\gamma$ . We also observe that the rise in immunocompetence with time  $t$  increases for higher fraction of time spent in pregnancy or brooding ( $f$ ) values. Our study illuminates the effect that pregnancy and brooding periods have on optimal investment in mounting a higher immune response.

## Discussion

Empirical evidence suggests that sexual dimorphism in immunocompetence could be due to sex-specific differences in life-history traits and relative differences in reproductive investments (Vincent and Gwynne, 2014; Stoehr and Kokko, 2006; Restif and Amos, 2010; Nunn et al., 2008; Forbes, 2007) such as sex differences in parental investment (Roth et al., 2011; Lin et al., 2016; Keller et al., 2017). Higher immunocompetence is not attached to a particular sex. Commonly females have higher immunocompetence. For instance, evidences from nature such as those done among humans, show that women live longer than men (Austad, 2006; May, 2007) due to sex-specific differences in immunocompetence. However, in some species males are known to invest more towards immunocompetence. For instance, in pipefish species, *Syngnathus typhle* and *Syngnathus rostellatus* males have a placenta-like structure and perform internal parental investment i.e. pregnancy. However,

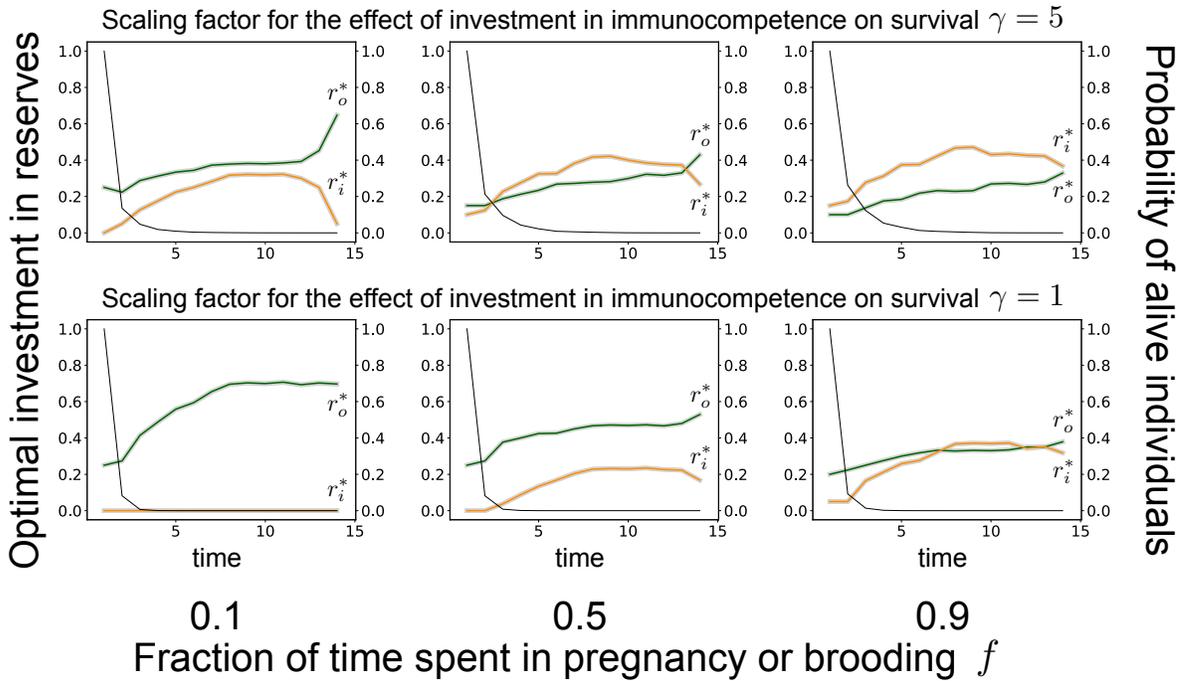


Figure 6: **Optimal allocation of resources in short-lived species.** Here, the value for background mortality is  $M = 2.5$ . Here, high and low scaling factor for the effect of investment in immunocompetence ( $r_i$ ) on survival are represented by  $\gamma = 1$  and  $\gamma = 5$ , respectively. The population size is 200. The results shown here for optimal values of  $r_i$  and  $r_o$  are average values of the population of 200 individuals for  $\sigma = 0.8$ . The shaded area in gray is the variance between the individuals. The black curve shows the frequency of alive individuals in the population i.e. it highlights the population density. Short-lived species spend more reserves for offspring production instead of their own immunocompetence. For high  $f$  values, we see an increase in investment of resources towards immunocompetence; especially under high  $\gamma$  conditions. When  $\gamma$  is low, individuals allocate more resources towards offspring since they most likely would not live for more than one reproductive season.

*Nerophis ophidion* males only glue the eggs on their belly, thus providing partial parental investment. (Keller, Hildebrand and Roth, in review) showed that in species with a higher parental investment (*S. typhle* and *S. rostellatus*) males have stronger immune response than females whereas in *Nerophis ophidion*, sexual immune dimorphism was absent. In the seahorse *Hippocampus erectus*, which is a conventional sex-role species with males undergoing pregnancy, sexually mature virgin seahorse males showed a stronger immune response than females. Results from Roth et al. (2011) and Lin et al. (2016) showed that male pipefishes and seahorses which are the sexes that undergo pregnancy have an increase immune responses. Keller et al. (2017) present similar findings from their experiments with the female mouthbrooding cichlid *Astatotilapia burtoni*. They showed that maternal and offspring immune defense is affected by parental investment.

Our results reveal that the increase in the amount of parental investment increases the investment in immunocompetence for most species systems. This is a trade-off in resources which favor higher investment in immunocompetence ( $r_i$ ) at the cost of lower investment in offspring ( $r_o$ ). We show

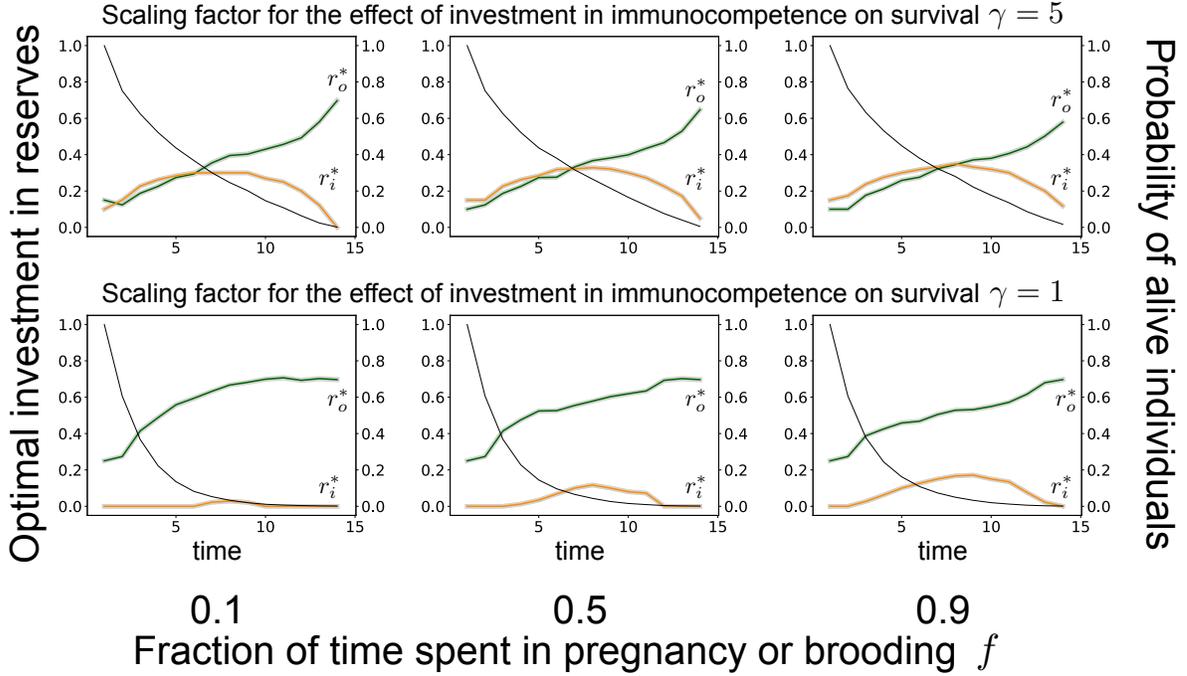


Figure 7: **Optimal allocation of resources in long-lived species.** The value for background mortality species is  $M = 0.5$ . High and low scaling factor for the effect of investment in immunocompetence ( $r_i$ ) on survival are represented by  $\gamma = 1$  and  $\gamma = 5$ , respectively. The population size is 200. The results shown here are the optimal values of  $r_i$  and  $r_o$  are average values of the population of 200 individuals for  $\sigma = 0.8$ . The shaded gray area is the variance between the individuals. The black curve shows the frequency of alive individuals in the population i.e. it represents the population density. For long-lived individuals the background mortality is low which allows them to focus their reserves to offspring production since they would anyway survive to next reproductive seasons even without much invest to the immune system. When  $\gamma$  is low, the individuals allocate more towards their offspring to ensure as much reproductive success as possible while the individual is alive. Overall, we see that the investment towards immunocompetence increases in long-lived species for longer brooding or pregnancy periods.

that in both short-lived and long-lived species, investment towards immunocompetence increases with increasing brooding or pregnancy periods. Our model also accounts for intraspecies scenarios where a higher value of  $f$  corresponds to the sex having long brooding or pregnancy periods, and a negligible value of  $f$  refers to the other sex that does negligible investment. So if a sex spends a long fraction of its reproductive season in internal parental investment or brooding as compared to the sex that does not spend as much time in parental investment, then our results show that this sex would invest more towards immunocompetence. This suggests that the one sex would have a higher immunocompetence as compared to the other i.e. sexual immune dimorphism can arise out of sex-specific differences in parental investment, as supported by empirical studies (Roth et al., 2011; Lin et al., 2016; Keller et al., 2017). Immunosenescence is commonly observed phenomena that occurs after a certain lifetime. Studies suggest that sex-specific immunosenescence can occur due to sex differences in traits such as immunocompetence, parental investment (Metcalf et al., 2019).

We show that an increase in the time spent in internal parental investment could bring about an earlier immunosenescence. More experiments across a wide range of taxa, focused in the direction of measuring immunocompetence between the sexes at various stages of their lifetime and in relation to parental investment, would shed more light on the understanding of sexual immune dimorphism across the animal kingdom.

The works by [Stoehr and Kokko \(2006\)](#) and [Medley \(2002\)](#) showed that sex differences in parasitic impact might cause either sex to invest more into immunocompetence. Therefore, a potential enhancement to our model related to immunology, should involve host-pathogen dynamics. The parameter  $\gamma$  that describes an individual's intrinsic scaling factor for the effect of investment in immunocompetence ( $r_i$ ) on survival could also be used to describe different kinds of species. In figures 6 and 7, low and high values of  $\gamma$  could denote species with high and low intrinsic ability to 'fight' pathogens. The first row shows species with high  $\gamma$  i.e. they are better adapted to the environment by investing more towards immunocompetence and therefore, the number of alive individuals is also higher here (especially for long-lived species) as compared to species with a lower  $\gamma$  value. Varying  $\gamma$  along with another parameter that describes either the pathogen impact in a certain environment, or parameters that describe low and high infectious environments could tackle finer details and provide more thorough results.

The immunocompetence handicap hypothesis (ICHH) cannot be completely dismissed ([Zahavi, 1977](#); [Houle and Kondrashov, 2002](#); [Roberts et al., 2004](#); [Stoehr and Kokko, 2006](#); [Kurtz, 2007](#)). Attracting potential mates through ornamentation which gives rise to increased mating success is also crucial for the lifetime reproductive success. However, these ornaments might be very costly and thus, there could be a trade-off between maintaining elaborate ornamentation and immunocompetence. In sticklebacks, for instance, females have a better immunocompetence than the males who have to attract mates using ornamentation and also perform almost entire parental care. However, seahorses males that also have to undergo both pregnancy and attracting mates are shown to have a higher immunocompetence, unlike stickleback males. This might be due to their highly monogamous mating system where a males obtains a lifetime mate during its first reproductive season itself and therefore, can invest more towards its immunocompetence for its own survival and the survival to future reproductive seasons. As mentioned earlier, in most pipefishes, the males can undergo pregnancy (whereas the females attracting mates through ornamentation) and the males exhibit a higher immunocompetence. In summary, with regard to mating roles and parental investment we can list three different types of systems (i) conventional sex role species where females provide significant PI (peacocks, primates), (ii) conventional sex role species where males provide significant PI (sticklebacks, seahorses) and (iii) sex-role reversed species where males provide significant PI (pipefishes, seadragons, phalarope birds) where conventional role species are ones where the males perform male-male (intrasexual) mating competitions whereas in sex-role reversed species, the females perform female-female competitions. Sexual immune dimorphism is biased towards males, females or neither depending on the system. Therefore, a third trait, ornamentation ([Candolin, 2000](#)) could also be included in future extensions of this model.

As mentioned earlier, parental investment consists of three parts (initial gamete production, internal pregnancy, and external parental care) and this varies between species, and within the sexes of a species ([Trivers, 1972](#)). In this model, our assumption that the time of parental investment directly relates to the amount of parental investment is a legitimate approximation since parental takes up both time and energy ([Trivers, 1972](#); [Sargent and Gross, 1985](#)). However, we only studied species systems where pregnancy or brooding is necessary for offspring success. A clear distinction between the three components of parental investment may help to look into the details of parental investment systems. Different taxa perform diverse forms of parental investment. Thus, the different parts of parental investments, and ornamentation can be studied separately as well. It might be practical to

study their effect on the immune response by varying one trait and keeping the others as constant values.

Informed by our theoretical predictions, further empirical studies that investigate sexual immune dimorphism in relation to parental investment for long and short-lived species; species with diverse reproductive strategies (semelparity or iteroparity); species in niches with varying pathogen impact or virulence, will shed more light on the general understanding of how diverse sex-specific life-history traits affect each other.

## Appendix

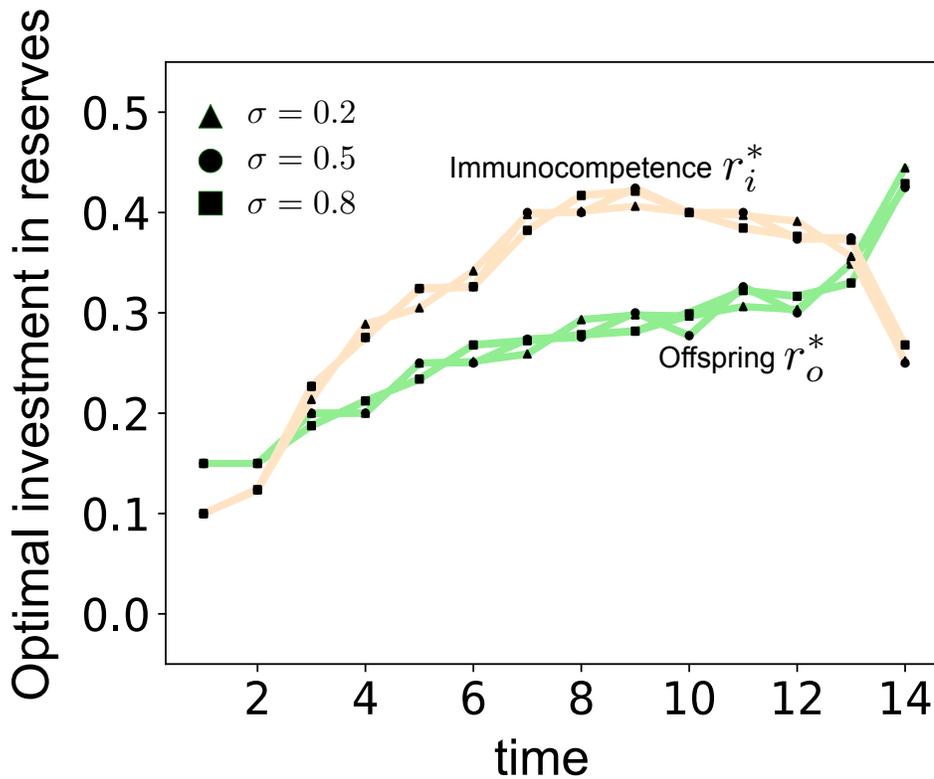


Figure 8: **Effect of  $\sigma$  in the optimal allocation of resources in short-lived species.** Here, the value for background mortality is  $M = 2.5$ . The values  $\gamma = 5$  and  $f = 0.5$ . The population size is 200. The results shown here for optimal values of  $r_i$  and  $r_o$  are average values of the population of 200 individuals for  $\sigma = 0.2$  (triangle marker),  $\sigma = 0.5$  (circle marker) and  $\sigma = 0.8$  (square marker). The shaded area is the variation between the individuals. Here the probability for obtaining  $g_h(t)$  i.e.  $x = 0.5$  is fixed. Thus, the values for  $g_h(t)$  and  $g_l(t)$  average to  $g(t)$  and this can happen for different values of  $\sigma$ . i.e resource gain can fluctuate according to  $\sigma$  around  $g(t)$ . We see that the results do not differ much between the different  $\sigma$  value i.e there is no effect of  $\sigma$ . Using a normally distributed  $g(t)$  with  $\sigma$  variance might show better pronounced differences.

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# Chapter 5

## Conclusions

“Mathematics without natural history is sterile, but natural history without mathematics is muddled.”

---

— John Maynard Smith

The universe, our planet, and its inhabitants can be described by mathematical equations. This allows us to understand causes for empirical observations, find patterns in nature, factors that shape evolution. Theoretical results from mathematical models have also given rise to new empirical questions. Galileo quoted that ‘The laws of Nature are written in the language of mathematics’, and eco-evolutionary dynamics are no exception.

The commencement of the modern synthesis by mathematicians and statisticians such as Wright, Fisher, Haldane, which marked the birth of population genetics, also gave rise to the application of mathematical analyses to study natural selection (Haldane, 1927, 1937; Wright, 1930; Fisher, 1949). Using mathematical tools to study eco-evolutionary dynamics has been in use in a plethora of fields such as population genetics (Crow and Kimura, 1970), microbial communities (Sala et al., 2016; Wu and Ross, 2016; Rakoff-Nahoum et al., 2016), cancer dynamics (Komarova et al., 2003; Michor et al., 2004; Zhang et al., 2017), antibiotic resistance (Conlin et al., 2014), and

agriculture (Clay and Kover, 1996; Thrall et al., 2011). A well-used and established mathematical tool, evolutionary game theory, is used to investigate situations where fitness is dependent on the frequencies of the types of individuals in the population. Game theory, a term coined by John von Neumann, has its origins in economics in which it was used to study optimal strategies in competitions between adversaries (von Neumann and Morgenstern, 1944). This was extended to evolutionary game theory by John Maynard Smith and George R. Price (Maynard Smith and Price, 1973). In fact, game theory is a branch of optimization theory useful for understanding many aspects of animal behavior (Mangel and Clark, 1988).

In this thesis, I used population dynamics, evolutionary game theory, and state-dependent dynamic optimization modeling in order to study the outcome of multiple interlinked interactions between individuals or between traits within individuals.

## **Brief summary of the results from this thesis work**

Species are connected within and between each other through interactions, and probably no interspecies or intraspecies interaction happens in isolation from other interactions. Various examples ranging across diverse taxa show the importance of investigating combined evolutionary dynamics as seen in the introduction. The previous three chapters of this thesis investigated the dynamics of combined interactions and the conclusions from these studies are as follows:

- **Combined multiple interaction dynamics differs from looking at each interaction in isolation** with regard to the evolution of frequencies of types of individuals in the population, and the equilibrium values which these frequencies converge over time.

I constructed a mathematical framework using evolutionary game theory and stochastic Moran process, that combines many multiplayer games (interactions) with a diverse set of strategies in each game. Through this framework, one can combine diverse multiplayer interactions and study their resulting dynamics. In infinite populations, I found that the

composition of a population in which individuals are involved in combined games showed different dynamical trajectories and different equilibrium points as compared to a population in which individuals only take part in single games considered in isolation. For finite populations, I derived an expression for calculating the fixation probability of a strategy, which is an important stochastic property and here too, I found that combining even a neutral game with another game could change the dynamics.

- **An important outcome that emerged out of studying a combination of multiple life-history traits with sex-specific differences is a bias in the adult sex ratio.**

For this study, in addition to evolutionary game theory, I also used population genetics, frequency-dependent fitness models and life-history theory to measure the lifetime reproductive success of an individual. Fitness components from three crucial life-history traits with sex-specific differences i.e parental investment, mating competition, and immune response were used to determine the overall fitness (here, lifetime reproductive success) of the different types of individuals in both the sexes. This allowed me to probe the consequences of the combined dynamics of life-history traits with sex-specific differences. The outcome of my model showed a population-level skew in the adult sex ratio, and my results matched various empirical evidence.

- **Sexual dimorphism can occur in one trait as an effect of sex-specific differences in another trait that it interacts with.**

Using state-dependent dynamic programming, the occurrence of sexual immune dimorphism in relation to the fraction of time spent in parental investment was investigated.

In the model that I constructed, parental investment and immunocompetence were considered to be two different traits of an individual. I modeled and studied the trade-off between allocating the energy reserves (of individuals in a population) to parental effort and to immunity in such a way that they maximize the individual's fitness. Through this

study, I showed that the differences in the proportion of time spent in parental investment and overall resources allocated to parental investment have an effect on the amount of resources allocated to immune response when exposed to an environment with fluctuating amount of resources. My results showed that for most species systems, it is optimal to invest more towards immunocompetence when the time spent in pregnancy or brooding is longer; and within a species, the sex that can undergo pregnancy or brooding invests more in immunocompetence.

A general conclusion from all the chapters is that studying coupled interactions between individuals or between the traits within an individual is necessary to obtain more realistic and meticulous results. This is because our results show that combined interactions dynamics differs from studying an isolated interaction. The dynamics differ in terms of trajectories of frequencies of individuals of every type and the equilibrium values for the frequencies, both. Coupling even a neutral interaction to another interaction affects their dynamics. I successfully applied the multiple interactions approach to interactions between life-history traits as well. By doing so, I was able to investigate how one trait affects another and also how the combined dynamics of the traits can result in population-level consequences.

## **A couple of future outlooks**

Differences in life-history trade-offs due to different fitness expectations can result in the evolution of diverse animal personalities. The multiple interactions approach can be used in this recently well-discussed and relatively young field i.e. the evolution of personalities through interactions between multiple personality traits (Wolf et al., 2007; Carter et al., 2013). Many personality traits determine the personality of an individual, and different personalities govern the behavior of the individual. If we look at each personality as a game where different traits are the strategies, then the multiple games dynamics framework that uses 'simple' equations can be used even in

these complex studies. Using this, one can study the coexistence of behavior types and behavior correlations more clearly.

Another future outlook would be to extend the multiple-game dynamics to spatial structure (Shibasaki and Shimada, 2018). The multiple-game dynamics framework can be extended to evolutionary graphs (Szabó and Fáth, 2007) to study interacting agents that are connected through diverse social networks.

Interspecies and intraspecies interactions shape evolution and each interaction is coupled with other interactions. One can use mathematical tools to study various questions related to natural history, eco-evolutionary interactions being one of them. Therefore, my mathematical models that investigate the combined dynamics of multiple interactions would allow one to capture more realistic eco-evolutionary scenarios and provide more insight. This is another feather in the cap for the pursuit of understanding evolution.



# **Appendix A**

**Electronic Supplementary Material of the  
article in Chapter 1**

# Electronic Supplementary Material: Evolutionary dynamics of complex multiple games

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## 1 Infinite population

### 1.1 Single Game Dynamics (SGD)

#### A two player replicator approach

Consider a  $2 \times 2$  (two player two strategy) payoff matrix (A.1) : There are two players and each of them can adopt two strategies. The two types of strategies they could employ are 1 and 2 and their respective frequencies are  $x_1$  and  $x_2$ .

$$\begin{matrix} & \begin{matrix} 1 & 2 \end{matrix} \\ \begin{matrix} 1 \\ 2 \end{matrix} & \begin{pmatrix} a_{1,(1,0)} & a_{1,(0,1)} \\ a_{2,(1,0)} & a_{2,(0,1)} \end{pmatrix} \end{matrix} \quad (\text{A.1})$$

In matrix A.1, we write the elements in the form  $a_{i,\alpha}$ , where  $i$  is the strategy of the focal player. Using multiindex notation,  $\alpha$ , is a vector written as  $\alpha = (\alpha_1, \alpha_2)$ , together representing the group composition. The average payoffs of the two strategies are given by  $f_1 = a_{1,(1,0)}x_1 + a_{1,(0,1)}x_2$  and  $f_2 = a_{2,(1,0)}x_1 + a_{2,(0,1)}x_2$ . The replicator equation Eq. (A.2) [1, 2] describes the change in frequency  $x_i$  of strategy  $i$  over time.

$$\dot{x}_i = x_i[(f_i - \phi)] \quad (\text{A.2})$$

where  $f_i$  is the fitness of strategy  $i$  and  $\phi$  is the average fitness. For an infinitely large population size we have  $x_1 = x$ ,  $x_2 = 1 - x$  Thus the replicator equation for the change in the

17 frequency of strategy 1 is,

$$\begin{aligned}\dot{x} &= x(1-x)(f_1 - f_2) \\ &= x(1-x)[(a_{1,(1,0)} - a_{1,(0,1)} - a_{2,(1,0)} + a_{2,(0,1)})x + a_{2,(1,0)} - a_{2,(0,1)}].\end{aligned}\tag{A.3}$$

18 Apart from the trivial fixed points ( $x = 0$  and  $x = 1$ ), there is an internal equilibrium given  
19 by,

$$\mathbf{x}^* = \frac{a_{2,(0,1)} - a_{2,(1,0)}}{a_{1,(1,0)} - a_{1,(0,1)} - a_{2,(1,0)} + a_{2,(0,1)}}.\tag{A.4}$$

## 20 Multiplayer games

21 We now extend the dynamics to multiplayer games [3]. The payoff matrix (A.5), represents a  
22 three player ( $d = 3$ ) two strategy ( $n = 2$ ) game; a  $2 \times 2 \times 2$  game.

$$\begin{array}{ccc} & 11 & 12 & 22 \\ \begin{array}{c} 1 \\ 2 \end{array} & \begin{pmatrix} a_{1,(2,0)} & a_{1,(1,1)} & a_{1,(0,2)} \\ a_{2,(2,0)} & a_{2,(1,1)} & a_{2,(0,2)} \end{pmatrix} & & \end{array}\tag{A.5}$$

23 The rows correspond to the focal player. Focal player interacting with two other players, both  
24 with strategy 1 will receive a payoff  $a_{1,(2,0)}$ . While interacting with a one strategy 1 player and  
25 a strategy 2 player, he will get  $a_{1,(1,1)}$ . When interacting with two other strategy 2 individuals,  
26 the payoff is equal to  $a_{1,(0,2)}$ . Assuming that the order of players does not matter, the average  
27 payoffs (or in this case, the fitnesses) will be,

$$\begin{aligned}f_1 &= x^2 a_{1,(2,0)} + 2x(1-x)a_{1,(1,1)} + (1-x)^2 a_{1,(0,2)} \\ f_2 &= x^2 a_{2,(2,0)} + 2x(1-x)a_{2,(1,1)} + (1-x)^2 a_{2,(0,2)}.\end{aligned}\tag{A.6}$$

28 The replicator equation in this case is given by,

$$\begin{aligned}\dot{x} &= x(1-x)((a_{1,(0,2)} - 2a_{1,(1,1)} + a_{1,(2,0)} - a_{2,(0,2)} + 2a_{2,(1,1)} - a_{2,(2,0)})x^2 \\ &\quad + (-a_{1,(0,2)} + a_{1,(1,1)} + a_{2,(0,2)} - a_{2,(1,1)})2x + a_{1,(0,2)} - a_{2,(0,2)}).\end{aligned}\tag{A.7}$$

29 The quadratic  $x^2$  term in Eq. (A.7) can give rise to a maximum of two interior fixed points. In  
30 general, for a  $d$ -player two strategy game, the replicator equation can result in  $d - 1$  interior  
31 fixed points (maximum). For an  $n$  strategy  $d$ -player game, the maximum number of internal  
32 equilibria is  $(d - 1)^{(n-1)}$  as shown in [4].

## 33 1.2 Multi Game Dynamics (MGD)

### 34 Linear combination of two $2 \times 2$ games

35 To start looking into the dynamics of combinations of games i.e. Multi Game Dynamics  
36 (MGD) in contrast with the Single Game Dynamics (SGD), consider the example: two games

37 with two strategies in each. Let the payoff matrix of Game 1 and Game 2 be,

$$A^1 = \begin{matrix} & A_1^1 & A_2^1 \\ A_1^1 & \begin{pmatrix} a_{1,(1,0)}^1 & a_{1,(0,1)}^1 \\ a_{2,(1,0)}^1 & a_{2,(0,1)}^1 \end{pmatrix} & \end{matrix} \quad A^2 = \begin{matrix} & A_1^2 & A_2^2 \\ A_1^2 & \begin{pmatrix} a_{1,(1,0)}^2 & a_{1,(0,1)}^2 \\ a_{2,(1,0)}^2 & a_{2,(0,1)}^2 \end{pmatrix} & \end{matrix}$$

38 The individuals can be partitioned into four classes. Individuals playing strategy 1 in game  
 39  $A^1$  and game  $A^2$ , strategy 1 in  $A^1$  and 2 in  $A^2$ , strategy 2 in  $A^1$  and 1 in  $A^2$ , and strategy 2 in  
 40  $A^1$  and  $A^2$ . So, there are four types of strategies,  $A_1^1 A_1^2$ ,  $A_1^1 A_2^2$ ,  $A_2^1 A_1^2$  and  $A_2^1 A_2^2$ . We refer to  
 41 them as ‘‘categorical types’’. Their respective frequencies are written as  $x_{11}$ ,  $x_{12}$ ,  $x_{21}$  and  $x_{22}$ .  
 42 We shall now use a new notation,  $p_{ji}$  or playing strategy  $i_j$  in game  $j$ , which is just a variable  
 43 transformation that can be written as (here,  $i_j \in \{1, 2\}$  and  $j \in \{1, 2\}$ ),

$$\begin{aligned} p_{11} &= x_{11} + x_{12} \\ p_{12} &= x_{21} + x_{22} \\ p_{21} &= x_{11} + x_{21} \\ p_{22} &= x_{12} + x_{22}. \end{aligned} \tag{A.8}$$

44 The fitnesses for playing strategy  $i_j$  in game  $j$  can be written out as,

$$\begin{aligned} f_{11} &= x_{11} a_{1,(1,0)}^1 + x_{12} a_{1,(1,0)}^1 + x_{21} a_{1,(0,1)}^1 + x_{22} a_{1,(0,1)}^1 \\ f_{12} &= x_{11} a_{2,(1,0)}^1 + x_{12} a_{2,(1,0)}^1 + x_{21} a_{2,(0,1)}^1 + x_{22} a_{2,(0,1)}^1 \\ f_{21} &= x_{11} a_{1,(1,0)}^2 + x_{12} a_{1,(0,1)}^2 + x_{21} a_{1,(1,0)}^2 + x_{22} a_{1,(0,1)}^2 \\ f_{22} &= x_{11} a_{2,(1,0)}^2 + x_{12} a_{2,(0,1)}^2 + x_{21} a_{2,(1,0)}^2 + x_{22} a_{2,(0,1)}^2. \end{aligned} \tag{A.9}$$

45 A crucial assumption here is that the effective average payoff is a linear composite of the  
 46 constituent games. The replicator dynamics will be given by the following set of coupled  
 47 different differential equations:

$$\begin{aligned} \dot{x}_{11} &= x_{11}[(f_{11} + f_{21}) - \phi] \\ \dot{x}_{12} &= x_{12}[(f_{11} + f_{22}) - \phi] \\ \dot{x}_{21} &= x_{21}[(f_{12} + f_{21}) - \phi] \\ \dot{x}_{22} &= x_{22}[(f_{12} + f_{22}) - \phi]. \end{aligned} \tag{A.10}$$

48 The average fitness  $\phi$  is given by,

$$\begin{aligned} \phi &= x_{11}(f_{11} + f_{21}) + x_{12}(f_{11} + f_{22}) + x_{21}(f_{12} + f_{21}) + x_{22}(f_{12} + f_{22}) \\ &= f_{11}(x_{11} + x_{12}) + f_{12}(x_{21} + x_{22}) + f_{21}(x_{11} + x_{21}) + f_{22}(x_{12} + x_{22}) \\ &= f_{11} p_{11} + f_{12} p_{12} + f_{21} p_{21} + f_{22} p_{22}. \end{aligned} \tag{A.11}$$

49 The single games' dynamics and their multi game dynamics will be the same or in other  
50 words, an MGD can be separated back into all its SGDs if  $p_{ji} = x_{ij} \forall i, j$  in a game  $j$ , for all  
51  $N$  games. At times, even if this equality holds, the trajectories in the MGD space might be  
52 different from the SGD space. Both these cases are shown in the examples in the main article.  
53 A previous study with two player games with two strategies [5], showed that the SGDs can  
54 be separated from their MGD. The dynamics lie on the generalized invariant manifold. [1, 6]  
55 in the  $S_4$  simplex which is given by  $W_K = \{x \in S_4 \mid x_{11}x_{22} = Kx_{12}x_{21}\}$  for  $K > 0$ . When  
56  $K = 1$ , we have  $W = \{x \in S_4 \mid x_{11}x_{22} = x_{12}x_{21}\}$  which is the *Wright manifold*. The Wright  
57 manifold  $W_K$  [6, 1] is a population dynamic concept. The states belonging to the Wright  
58 manifold are for the population in linkage equilibrium i.e. the games (or loci/traits, in biology)  
59 are inherited completely independently in each generation. Thus, on this manifold, MGD can  
60 be separated back into the SGDs of the constituent games. The attractor for a combination of  
61 two 2-player games having two strategies each is a line  $E$ , an evolutionarily stable set [5]. The  
62 point where the line  $E$  intersects the Wright manifold indicates a rest point. All the trajectories  
63 in the simplex depicting the MGD fall onto an attractor given by a line (ES set) on  $W_K$ . The  
64 dynamics on  $W_K$  and the trajectories on each  $W_K$  were analyzed in the same study [5] using  
65 methods used in dynamical systems to show they are qualitatively the same as on the Wright  
66 manifold.

67 However, for multiple games having more than two strategies in at least one game, the  
68 MGD cannot be separated even into a linear combination of the constituent SGDs unless they  
69 are on  $W$  [7]. Increase in the number of games and the number of strategies increases the  
70 dimension of MGD simplex. This high dimensional space of MGD, which would be equal  
71 to  $\sum_{i=1}^N (m_j - 1)$  (where  $N$  is the number of games and  $m_j$  is the number of strategies in a  
72 game  $j$ ), is densely packed with manifolds. All the manifolds are non-intersecting while  $W$   
73 is the invariant. Even for a simplified example of 2 games each with  $m_1$  and  $m_2$  number  
74 of strategies the generalised invariant manifold is given by  $W_K = \{x \in \Delta^{m_1 \times m_2} \mid x_{i,k}x_{j,l} =$   
75  $K_{ik,jl} x_{i,l}x_{j,k} \forall 1 \leq i, j \leq m_1, 1 \leq k, l \leq m_2\}$  where  $K = \{K_{ik,jl}\}$  is a set of positive  
76 constants for which  $W_K$  is a non-empty set. When  $K_{ik,jl} = 1$ , we have the Wright manifold  
77 on which the MGD can be separated back into its SGDs. While combining two 2-player  
78 games with three strategies [7], the evolutionarily stable set  $E$  would be in a four-dimensional  
79 hyperplane [6]. So while combining many games, even if one individual game has more than  
80 two strategies, the ES set may no longer be a line. It would be a hyperplane in the  $W_K$   
81 hyperspace. Thus, it is important to know on which manifold the initial conditions are, for  
82 only if they start from the Wright manifold  $W$ , will the dynamics be a perfect match to the  
83 SGDs [7].

84 If the initial condition is not on  $W$ , if the strategies between the different games are allowed

85 to recombine then the dynamics converges to  $W$ . While the relationship between strategies  
 86 under recombination is genetically plausible, for phenotypic strategies, social learning or hor-  
 87 izontal adoption of traits could have a similar effect [8, 9].

## 88 2 Finite population

### 89 2.1 Single game dynamics

90 In a population of size  $Z$  consisting of strategy 1 and strategy 2 players, the probability that  
 91 one of the strategies, say 1, fixates, is given by the fixation probability  $\rho_1$ . An individual  
 92 is chosen proportional to its fitness to reproduce an identical offspring. Another individual  
 93 is chosen randomly and discarded from the group. Therefore, the group size is kept at a  
 94 constant value  $Z$ . Fitness of a strategy  $s$  can be a linear function of its average payoff  $\pi_s$  i.e  
 95  $f_s = 1 - w + w\pi_s$ . In a population that has  $i$  strategy 1 players, the fitnesses can be used to  
 96 calculate the transition probabilities  $T_i^+$  and  $T_i^-$  for the number of type 1 players to increase  
 97 and decrease by one, respectively.

$$\begin{aligned} T_i^+ &= \frac{if_1}{if_1 + (Z-i)f_2} \frac{Z-i}{Z} \\ T_i^- &= \frac{(Z-i)f_2}{if_1 + (Z-i)f_2} \frac{i}{Z}. \end{aligned} \quad (\text{A.12})$$

98 With probability  $1 - T_i^+ - T_i^-$  the system does not change. Using the transition probabilities,  
 99 the fixation probability can be calculated [2, 10] to be,

$$\rho_1 = \frac{1}{1 + \sum_{m=1}^{Z-1} \prod_{i=1}^m \frac{T_i^-}{T_i^+}}. \quad (\text{A.13})$$

100 Since  $\frac{T_i^-}{T_i^+} = \frac{f_2}{f_1} = \frac{1-w+w\pi_2}{1-w+w\pi_1} \approx 1 - w(\pi_1 - \pi_2)$  for selection intensity  $w \ll 1$  i.e. weak selection.

101 Therefore,

$$\rho_1 \approx \frac{1}{1 + \sum_{m=1}^{Z-1} \prod_{i=1}^m (1 - w(\pi_1 - \pi_2))}. \quad (\text{A.14})$$

102 For a  $d$ -player game, the payoffs are obtained using a hypergeometric distribution given by,

$$H(k, d; i, Z) = \frac{\binom{i-1}{k} \binom{Z-i}{d-1-k}}{\binom{Z-1}{d-1}}. \quad (\text{A.15})$$

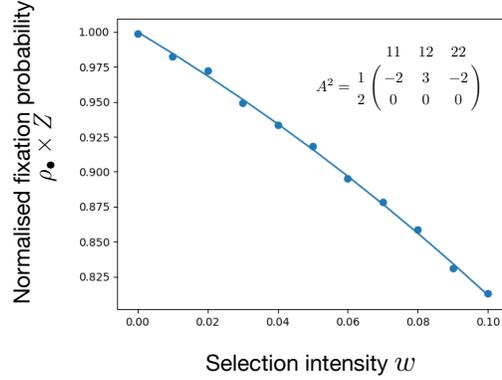


Figure A.1: **Fixation probability for a single individual playing strategy 1 varying with selection intensity for a three player game having two strategies.** For the game shown in this figure, the payoff of strategy 2 is greater than strategy 1 ( $\pi_2 > \pi_1$ ), the fixation probability decreases, according to equation (A.17). The results from analytics and simulations (averaged over  $10^6$  realizations) are plotted as solid lines and solid circles, respectively.

103 Thus,

$$\begin{aligned} \pi_1 &= \sum_{k=0}^{d-1} \frac{\binom{i-1}{k} \binom{Z-i}{d-1-k}}{\binom{Z-1}{d-1}} a_{1,\alpha} \\ \pi_2 &= \sum_{k=0}^{d-1} \frac{\binom{i}{k} \binom{Z-i-1}{d-1-k}}{\binom{Z-1}{d-1}} a_{2,\alpha}. \end{aligned} \quad (\text{A.16})$$

104 Maintaining weak selection, then from [4] we have,

$$\rho_1 \approx \frac{1}{Z} + \frac{w}{Z^2} \sum_{m=1}^{Z-1} \sum_{i=1}^m (\pi_1 - \pi_2). \quad (\text{A.17})$$

105 Figure A.1 contains the fixation probabilities of strategy 1 with respect to varying selection  
106 intensities for a three player game with two strategies.

## 107 2.2 Multiple game dynamics

108 We begin with the same example that was used to explain the combination of two  $d$ -player  
109 games where both games have two strategies; and use the same notations for a finite population  
110 of size  $Z$ . The population consists of individuals of four types :  $A_1^1 A_1^2$ ,  $A_1^1 A_2^2$ ,  $A_2^1 A_1^2$  and  
111  $A_2^1 A_2^2$ . The combined dynamics results in an  $S_4$  simplex as shown in Fig. A.2. We perform  
112 pairwise comparisons for all the edges of the simplex. On a particular edge, only the two

$$A^1 = \frac{1}{2} \begin{pmatrix} 1 & 2 \\ -1 & 1 \\ 0 & 0 \end{pmatrix} \quad A^2 = \frac{1}{2} \begin{pmatrix} 11 & 12 & 22 \\ -2 & 3 & -2 \\ 0 & 0 & 0 \end{pmatrix}$$

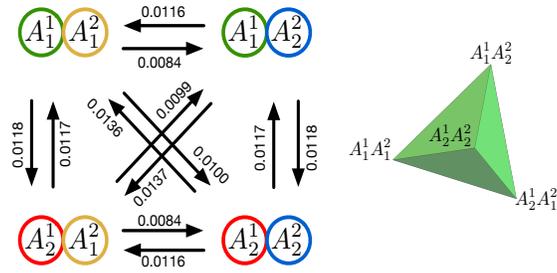


Figure A.2: **Fixation probabilities over pure strategies.** Figure shows the fixation probabilities and the direction of selection between the vertices in a tetrahedron (which contains the MGD of the two games  $A^1$  and  $A^2$  shown in the matrices). Here selection intensity  $w = 0.01$  and population size  $Z = 100$ . It has been assumed that both the games have the same selection intensity and hence the average payoffs have been added first and then the mapping (linear or exponential mapping from payoffs to fitness) has been performed i.e. Method II (Method I would produce a different figure). For the edges where one of the games does not change (e.g.  $A_1^1, A_1^2 \rightleftharpoons A_1^1, A_2^2$ ), only one of the game (here game 2) matters and hence the fixation probabilities are the same as if *only* one game.

113 vertex strategies are present. Let us start with the edge containing  $x_{11}$  and  $x_{12}$  vertices. If  
 114 there are  $\gamma_{11}$  individuals playing strategy  $A_1^1 A_1^2$ , then there are  $\gamma_{12} = Z - \gamma_{11}$  individuals  
 115 of type  $A_1^1 A_2^2$ . The number of  $A_2^1 A_1^2$  and  $A_2^1 A_2^2$  individuals i.e.  $\gamma_{21}$  and  $\gamma_{22}$  is zero. In the  
 116 individual games, the number of players adopting strategy  $i_j$  in a game  $j$  is given by  $p_{ji_j}$ .  
 117 Since we are looking at the edge with  $A_1^1 A_1^2$  and  $A_1^1 A_2^2$  individuals, we have

$$\begin{aligned}
 p_{11} &= \gamma_{11} + \gamma_{12} = Z \\
 p_{12} &= \gamma_{21} + \gamma_{22} = 0 \\
 p_{21} &= \gamma_{11} + \gamma_{21} = \gamma_{11} \\
 p_{22} &= \gamma_{12} + \gamma_{22} = Z - \gamma_{11}.
 \end{aligned} \tag{A.18}$$

118 In contrast to the binomial distribution which is used for infinite populations where the draws  
 119 can be considered independent, the hypergeometric distribution was used for sampling with-  
 120 out replacement in the case of finite populations [4, 11]. For infinite population, we used the  
 121 multinomial distribution to calculate the average payoffs for a combination of  $N$  multiplayer  
 122 games in an infinite population size. Therefore, for finite populations, we shall use the multi-  
 123 variate hypergeometric distribution. For a population of size  $Z$  containing  $\gamma_{11}$  type  $A_1^1 A_1^2$  and  
 124  $Z - \gamma_{11}$  type  $A_1^1 A_2^2$  individuals, the average payoffs  $\pi_{ji_j}$  for playing strategy  $i_j$  in game  $j$  (in  
 125 our example,  $i_j \in \{1, 2\}$  and  $j \in \{1, 2\}$ ) are

$$\begin{aligned}
 \pi_{11} &= \sum_{|k|=d_1-1} \frac{\binom{p_{11}-1}{k_1} \binom{p_{12}}{k_2}}{\binom{Z-1}{d_1-1}} a_{1,k}^1 \\
 \pi_{12} &= \sum_{|k|=d_1-1} \frac{\binom{p_{11}}{k_1} \binom{p_{12}-1}{k_2}}{\binom{Z-1}{d_1-1}} a_{2,k}^1 \\
 \pi_{21} &= \sum_{|k|=d_2-1} \frac{\binom{p_{21}-1}{k_1} \binom{p_{22}}{k_2}}{\binom{Z-1}{d_2-1}} a_{1,k}^2 \\
 \pi_{22} &= \sum_{|k|=d_2-1} \frac{\binom{p_{21}}{k_1} \binom{p_{22}-1}{k_2}}{\binom{Z-1}{d_2-1}} a_{2,k}^2.
 \end{aligned} \tag{A.19}$$

126 In general, for  $N$  multi-strategy  $d$ -player games,

$$\pi_{ji_j} = \sum_{|k|=d_j-1} \frac{\binom{p_{ji_j}-1}{k_{i_j}} \prod_{n=1, n \neq i_j}^{m_j} \binom{p_{jn}}{k_n}}{\binom{Z-1}{d_j-1}} a_{i_j,k}^j. \tag{A.20}$$

127 We can calculate the fitnesses using linear or exponential mapping. If  $w_j$  is the intensity

128 of selection in game  $j$ , then

$$f^{ji_j} = \begin{cases} 1 - w_j + w_j \pi_{ji_j} & \text{for linear mapping} \\ e^{w_j \pi_{ji_j}} & \text{for exponential mapping.} \end{cases} \quad (\text{A.21})$$

129 Thus, in the combined dynamics, the fitness (assuming it to be additive) of type  $A_{i_1}^1 A_{i_2}^2 \dots A_{i_N}^N$   
130 is

$$F_{i_1 i_2 \dots i_N} = \sum_{j=1}^N f^{ji_j}. \quad (\text{A.22})$$

131 If we are looking at an edge with types  $A_{i_1}^1 A_{i_2}^2 \dots A_{i_N}^N$  and  $A_{h_1}^1 A_{h_2}^2 \dots A_{h_N}^N$ , the transition prob-  
132 ability  $T_\gamma^+$  for type  $A_{i_1}^1 A_{i_2}^2 \dots A_{i_N}^N$  to increase from  $\gamma$  to  $\gamma + 1$  (and type  $A_{h_1}^1 A_{h_2}^2 \dots A_{h_N}^N$  to be  
133 randomly selected for death) is

$$T_\gamma^+ = \frac{\gamma F_{i_1 i_2 \dots i_N}}{\gamma F_{i_1 i_2 \dots i_N} + (Z - \gamma) F_{h_1 h_2 \dots h_N}} \frac{Z - \gamma}{Z}. \quad (\text{A.23})$$

134 Likewise,  $T_\gamma^-$  will be

$$T_\gamma^- = \frac{(Z - \gamma) F_{h_1 h_2 \dots h_N}}{\gamma F_{i_1 i_2 \dots i_N} + (Z - \gamma) F_{h_1 h_2 \dots h_N}} \frac{\gamma}{Z}. \quad (\text{A.24})$$

135 So, for a  $A_{i_1}^1 A_{i_2}^2 \dots A_{i_N}^N$  and  $A_{h_1}^1 A_{h_2}^2 \dots A_{h_N}^N$  edge, the fixation probability  $\rho_{A_{i_1}^1 A_{i_2}^2 \dots A_{i_N}^N}$  of type  
136  $A_{i_1}^1 A_{i_2}^2 \dots A_{i_N}^N$  is

$$\rho_{A_{i_1}^1 A_{i_2}^2 \dots A_{i_N}^N, A_{h_1}^1 A_{h_2}^2 \dots A_{h_N}^N} = \frac{1}{1 + \sum_{m=1}^{Z-1} \prod_{\gamma=1}^m \frac{T_\gamma^-}{T_\gamma^+}}. \quad (\text{A.25})$$

## 137 Method I

138 As  $\frac{T_\gamma^-}{T_\gamma^+} = \frac{F_{h_1 h_2 h_3 \dots h_N}}{F_{i_1 i_2 i_3 \dots i_N}}$ , Eq. (A.25) can be written as,

$$\begin{aligned} \rho_{A_{i_1}^1 A_{i_2}^2 \dots A_{i_N}^N, A_{h_1}^1 A_{h_2}^2 \dots A_{h_N}^N} &= \frac{1}{1 + \sum_{m=1}^{Z-1} \prod_{\gamma=1}^m \frac{F_{h_1 h_2 h_3 \dots h_N}}{F_{i_1 i_2 i_3 \dots i_N}}} \\ &= \frac{1}{1 + \sum_{m=1}^{Z-1} \prod_{\gamma=1}^m \frac{\sum_{j=1}^N f^{jh_j}}{\sum_{j=1}^N f^{ji_j}}} \\ &= \frac{1}{1 + \sum_{m=1}^{Z-1} \prod_{\gamma=1}^m \left( \frac{N + \sum_{j=1}^N -w_j + w_j \pi_{jh_j}}{N + \sum_{j=1}^N -w_j + w_j \pi_{ji_j}} \right)}. \end{aligned} \quad (\text{A.26})$$

139 where the fitness is obtained using a linear mapping. In order to further simplify the model,  
 140 we consider that all games have the same selection intensity. In this case,

$$\begin{aligned} \rho_{A_{i_1}^1 A_{i_2}^2 \dots A_{i_N}^N, A_{h_1}^1 A_{h_2}^2 \dots A_{h_N}^N} &= \frac{1}{1 + \sum_{m=1}^{Z-1} \prod_{\gamma=1}^m \left( \frac{N - Nw + w(\sum_{j=1}^N \pi_{jh_j})}{N - Nw + w(\sum_{j=1}^N \pi_{ji_j})} \right)} \\ &= \frac{1}{1 + \sum_{m=1}^{Z-1} \prod_{\gamma=1}^m \left( \frac{1 - w + \frac{w}{N}(\sum_{j=1}^N \pi_{jh_j})}{1 - w + \frac{w}{N}(\sum_{j=1}^N \pi_{ji_j})} \right)}. \end{aligned} \quad (\text{A.27})$$

141 It is worth mentioning here that the assumption of having equal intensities for all games is  
 142 strong. Many times, the selection on one game may be more intense than others. These have  
 143 to be taken into account as it strengthens the precision of the model and Eq. (A.26) must be  
 144 used in these scenarios. However for the sake of our analysis, we shall assume  $w_j = w$  for all  
 145  $j \in [0, N]$ .

146 For weak selection intensity,

$$\begin{aligned} \rho_{A_{i_1}^1 A_{i_2}^2 \dots A_{i_N}^N, A_{h_1}^1 A_{h_2}^2 \dots A_{h_N}^N} &\approx \frac{1}{1 + \sum_{m=1}^{Z-1} \prod_{\gamma=1}^m [1 - w\{1 - \frac{(\sum_{j=1}^N \pi_{jh_j})}{N}\}] \times [1 + w\{1 - \frac{(\sum_{j=1}^N \pi_{ji_j})}{N}\}]} \\ &\approx \frac{1}{1 + \sum_{m=1}^{Z-1} \prod_{\gamma=1}^m [1 - \frac{w}{N}(\sum_{j=1}^N (\pi_{ji_j} - \pi_{jh_j}))]}. \end{aligned} \quad (\text{A.28})$$

147 Eq. (A.28) can be written as,

$$\rho_{A_{i_1}^1 A_{i_2}^2 \dots A_{i_N}^N, A_{h_1}^1 A_{h_2}^2 \dots A_{h_N}^N} \approx \frac{1}{Z - \frac{w}{N} \sum_{m=1}^{Z-1} \sum_{\gamma=1}^m (\sum_{j=1}^N (\pi_{ji_j} - \pi_{jh_j}))}. \quad (\text{A.29})$$

148 Following Taylor expansion and since  $w \ll 1$ , we get

$$\rho_{A_{i_1}^1 A_{i_2}^2 \dots A_{i_N}^N, A_{h_1}^1 A_{h_2}^2 \dots A_{h_N}^N} \approx \underbrace{\frac{1}{Z}}_{\text{Under neutrality (w=0)}} + \frac{w}{NZ^2} \left[ \sum_{m=1}^{Z-1} \sum_{\gamma=1}^m \left( \sum_{j=1}^N (\pi_{ji_j} - \pi_{jh_j}) \right) \right]. \quad (\text{A.30})$$

149 For  $w = 0$  and  $N = 1$  i.e. neutrality condition while there is only one game, the above  
 150 equation is also equal to the classic neutral fixation probability  $\frac{1}{Z}$  for single games. For  $N = 1$   
 151 in Eq. (A.30), we can retrieve Eq. (A.17) for a single multiplayer game i.e.

$$\rho_{A_{i_1}^1, A_{h_1}^1} \approx \underbrace{\frac{1}{Z}}_{\text{Under neutrality}} + \frac{w}{Z^2} \sum_{m=1}^{Z-1} \sum_{\gamma=1}^m (\pi_{1i_1} - \pi_{1h_1}). \quad (\text{A.31})$$

152 For  $N = 2$  Eq. (A.28) becomes,

$$\rho_{A_{i_1}^1 A_{i_2}^2, A_{h_1}^1 A_{h_2}^2} \approx \frac{1}{1 + \sum_{m=1}^{Z-1} \prod_{\gamma=1}^m \left[ 1 - \frac{w}{2} [(\pi_{1i_1} + \pi_{2i_2}) - (\pi_{1h_1} + \pi_{2h_2})] \right]}. \quad (\text{A.32})$$

153 While looking at an edge for which, say, game 1 in both vertices has the same strategy and  
 154 thus, we need to only look at differences in one game i.e. only game 2 matters ( $\pi_{1i_1} = \pi_{1h_1}$ ),

$$\begin{aligned} \rho_{A_{i_1}^1 A_{i_2}^2, A_{h_1}^1 A_{h_2}^2} &\approx \frac{1}{1 + \sum_{m=1}^{Z-1} \prod_{\gamma=1}^m \left[ 1 - \frac{w}{2} [(\pi_{2i_2} - \pi_{2h_2})] \right]} \\ &= \frac{1}{Z} + \frac{w}{2Z^2} \sum_{m=1}^{Z-1} \sum_{i=1}^m (\pi_{2i_2} - \pi_{2h_2}) \end{aligned} \quad (\text{A.33})$$

155 We can make pairwise comparisons between all categorical types (all the edges of the  $S_4$   
 156 simplex in containing the MGD of the two games with two strategies). Using these compar-  
 157 ative fixation probabilities we can determine the flow of the dynamics over pure strategies as  
 158 shown Fig. A.2.

## 159 Method II

160 If all games have the same intensity, we could also add the payoffs first and then perform the  
 161 fitness mappings, then  $F_{i_1 i_2 i_3 \dots i_N} = 1 - w + w \left( \sum_{j=1}^N \pi_{j i_j} \right)$  and  $F_{h_1 h_2 h_3 \dots h_N} = 1 - w +$   
 162  $w \left( \sum_{j=1}^N \pi_{j h_j} \right)$ . Thus, the combined fitness (of a vertex) is not just a sum of the fitnesses  
 163 of strategies used in the inherent games (in that vertex). The combined fitness is obtained  
 164 by summing the average payoffs of playing the respective strategies in the games involved in  
 165 a particular vertex and using that to calculate the fitness of that vertex. Only the payoffs of  
 166 the games that have the same selection intensity can be added together and mapped to fitness  
 167 through this method. An example of a situation where the combined effect of the payoffs for  
 168 the strategies of the games on that vertex leads to the combined fitness, would be in models of  
 169 mating and sexual selection. Numerous interactions (parenting, mating, brooding) or games  
 170 during a mating season decides the reproductive success or fitness of an individual during that  
 171 period. This combination of games is not trivial as bringing all the smaller games into one  
 172 larger game but we cannot always deconstruct the multi-game back to all the inherent single  
 173 games. The fixation probability, Eq. (A.25), in this case will be,

$$\rho_{A_{i_1}^1 A_{i_2}^2 \dots A_{i_N}^N, A_{h_1}^1 A_{h_2}^2 \dots A_{h_N}^N} = \frac{1}{1 + \sum_{m=1}^{Z-1} \prod_{\gamma=1}^m \left( \frac{1-w+w(\sum_{j=1}^N \pi_{j h_j})}{1-w+w(\sum_{j=1}^N \pi_{j i_j})} \right)}. \quad (\text{A.34})$$

174 For weak selection intensities,

$$\begin{aligned} \rho_{A_{i_1}^1 A_{i_2}^2 \dots A_{i_N}^N, A_{h_1}^1 A_{h_2}^2 \dots A_{h_N}^N} &\approx \frac{1}{1 + \sum_{m=1}^{Z-1} \prod_{\gamma=1}^m \left(1 - w[1 - (\sum_{j=1}^N \pi_{jh_j})] + w[1 - (\sum_{j=1}^N \pi_{ji_j})]\right)} \\ &= \frac{1}{1 + \sum_{m=1}^{Z-1} \prod_{\gamma=1}^m \left(1 - w[(\sum_{j=1}^N \pi_{ji_j} - \sum_{j=1}^N \pi_{jh_j})]\right)}. \end{aligned} \quad (\text{A.35})$$

175 and this can be further written as,

$$\rho_{A_{i_1}^1 A_{i_2}^2 \dots A_{i_N}^N, A_{h_1}^1 A_{h_2}^2 \dots A_{h_N}^N} \approx \underbrace{\frac{1}{Z}}_{\text{Under neutrality (w=0)}} + \frac{w}{Z^2} \left[ \sum_{m=1}^{Z-1} \sum_{\gamma=1}^m \left( \sum_{j=1}^N (\pi_{ji_j} - \pi_{jh_j}) \right) \right]. \quad (\text{A.36})$$

176 If we consider two games, then Eq. (A.35) will be reduced to

$$\rho_{A_{i_1}^1 A_{i_2}^2, A_{h_1}^1 A_{h_2}^2} \approx \frac{1}{1 + \sum_{m=1}^{Z-1} \prod_{\gamma=1}^m (1 - w[(\pi_{1i_1} + \pi_{2i_2}) - (\pi_{1h_1} + \pi_{2h_2})])}. \quad (\text{A.37})$$

177 Here, if we look at an edge for which, say, game 1 in both vertices has the same strategy  
178 ( $\pi_{1i_1} = \pi_{1h_1}$ ), then looking at differences in game 2 is what matters. In this scenario,

$$\rho_{A_{i_1}^1 A_{i_2}^2, A_{h_1}^1 A_{h_2}^2} \approx \frac{1}{1 + \sum_{m=1}^{Z-1} \prod_{\gamma=1}^m (1 - w(\pi_{2i_2} - \pi_{2h_2}))}. \quad (\text{A.38})$$

179 This corresponds to equation Eq. (A.14) for a single game with two strategies  $i_1$  and  $h_1$ . This  
180 can also be written as ,

$$\rho_{A_{i_1}^1 A_{i_2}^2, A_{h_1}^1 A_{h_2}^2} \approx \frac{1}{Z} + \frac{w}{Z^2} \sum_{m=1}^{Z-1} \sum_{i=1}^m (\pi_{2i_2} - \pi_{2h_2}) \quad (\text{A.39})$$

181 and this is similar to Eq. (A.17) for single game dynamics. We can make pairwise comparisons  
182 between all categorical types (all the edges of the  $S_4$  simplex in containing the MGD of the two  
183 games with two strategies). Using these comparative fixation probabilities we can determine  
184 the flow of the dynamics over pure strategies as shown Fig. A.2.

## 185 **Difference between Method I and II**

186 The difference between Method I and II is given by,

$$\begin{aligned} & \left| \left( \frac{1}{Z} + \frac{w}{Z^2} \left[ \sum_{m=1}^{Z-1} \sum_{\gamma=1}^m \left( \sum_{j=1}^N (\pi_{ji_j} - \pi_{jh_j}) \right) \right] \right) - \left( \frac{1}{Z} + \frac{w}{NZ^2} \left[ \sum_{m=1}^{Z-1} \sum_{\gamma=1}^m \left( \sum_{j=1}^N (\pi_{ji_j} - \pi_{jh_j}) \right) \right] \right) \right| \\ &= \left| \frac{w}{Z^2} \left[ \sum_{m=1}^{Z-1} \sum_{\gamma=1}^m \left( \sum_{j=1}^N (\pi_{ji_j} - \pi_{jh_j}) \right) \right] \cdot \left[ 1 - \frac{1}{N} \right] \right|. \end{aligned} \quad (\text{A.40})$$

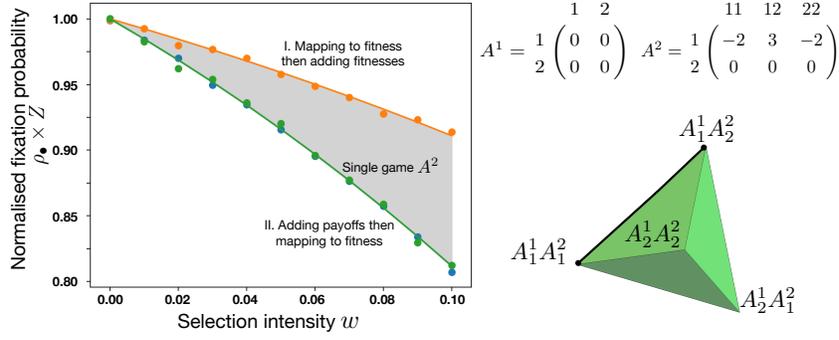


Figure A.3: **Fixation probability of a single individual playing  $A_1^1 A_1^2$  strategy on the edge  $A_1^1 A_1^2 \rightleftharpoons A_1^1 A_2^2$  varying with selection intensity for a combination of two games having two strategies each (special case  $A^1$ ).** For a population of  $Z = 10$  the fixation probabilities are normalised according to the neutral fixation probability,  $\frac{1}{Z} = 0.1$ . We look at the edge  $A_1^1 A_1^2 \rightleftharpoons A_1^1 A_2^2$  where  $A^1$  is the same for both vertices i.e. neutral in both the vertices, and  $A^2$  is what matters. The payoffs in Game  $A^1$  are zero. Since the payoff of playing strategy 2 in  $A^2$  is greater than playing strategy 1 ( $\pi_{22} > \pi_{21}$ ), the fixation probability decreases as shown in the earlier sections of the ESM. The line labeled ‘single game’ corresponds to single game dynamics of  $A^2$ . The plots from Method I (mapping payoffs to fitnesses and then adding the fitnesses) and Method II (adding the payoffs first, and then mapping to fitness) for a combination of the two games  $A^1$  and  $A^2$ . Since  $\pi_{11} (= \pi_{12}) = 0$ , results from Method II and the SGD of  $A^2$  are the same. However, Method I shows a different result. Here, MGD differs from the SGD. Adding another game to  $A^2$  modifies the dynamics. Thus, within the MGD, the two methods of mapping from payoffs to fitness i.e. Method I and Method II differ from each other (by Eq. A.41 shaded region). The difference is due to the different baseline payoffs that the different mappings produce. The results from analytics and stochastic simulations are plotted as solid lines and symbols, respectively. The simulations are averaged over  $10^6$  realisations. Thus while looking at a combination of various games, there can be different methods of mapping and one needs to choose a mapping method that reflects their model best as they can bring about different results.

187 As  $N$  increases, the difference between the two methods becomes independent of the number  
 188 of games. For  $N = 2$ , if we look at an edge where game 1 at both vertices has the same strategy  
 189 ( $\pi_{1i_1} = \pi_{1h_1}$ ) then game 2 is what matters. Here, the difference between Methods I and II is the  
 190 difference between the equations (A.39) and (A.33) which is equal to  $\frac{w}{Z^2} [\sum_{m=1}^{Z-1} \sum_{\gamma=1}^m (\pi_{2i_2} -$   
 191  $\pi_{2h_2})] \cdot \frac{1}{2}$ . In the main text Fig. 6 shows the fixation probability  $\rho_{A_1^1 A_1^2, A_1^1 A_2^2}$  (both Method  
 192 I and Method II) with respect to varying selection intensities in the  $A_1^1 A_1^2, A_1^1 A_2^2$  edge of the

193 tetrahedron simplex that contains the multiple game dynamics for a combination of two games  
 194 with two strategies each. While this is the general case where both the games matter, Fig. A.3  
 195 is a particular case where the payoff in game  $A^1$  is zero. Here, there is no difference between  
 196 Method II and SGD. However, in Method I, its results differ from SGD. Eq. A.40 becomes,

$$\begin{aligned}
 & \left| \left( \frac{1}{Z} + \frac{w}{Z^2} \left[ \sum_{m=1}^{Z-1} \sum_{\gamma=1}^m (\pi_{21} - \pi_{22}) \right] \right) - \left( \frac{1}{Z} + \frac{w}{2Z^2} \left[ \sum_{m=1}^{Z-1} \sum_{\gamma=1}^m (\pi_{21} - \pi_{22}) \right] \right) \right| \\
 & = \left| \left( \frac{w}{Z^2} \left[ \sum_{m=1}^{Z-1} \sum_{\gamma=1}^m (\pi_{21} - \pi_{22}) \right] \cdot \frac{1}{2} \right) \right|.
 \end{aligned} \tag{A.41}$$

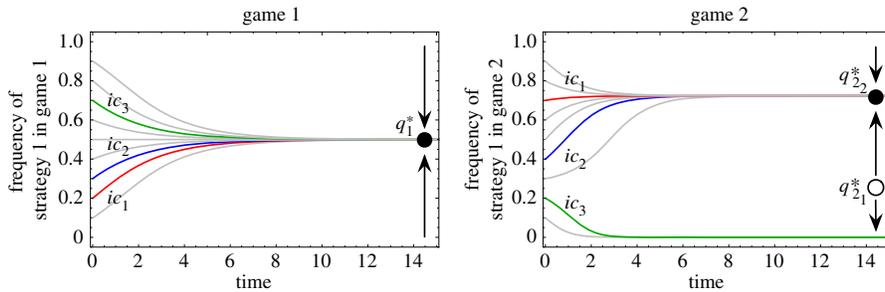
197 Thus the kind of mapping method that one chooses becomes important in multi game dynam-  
 198 ics as there are various ways of mapping payoffs to fitness especially when we remove the  
 199 assumption that the selection intensity are the same value  $w$  for all  $N$  games i.e. the value  $w_j$   
 200 would be different from one game  $j$  to another.

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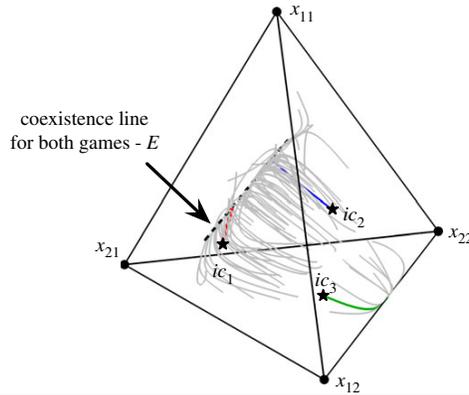
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single game dynamics

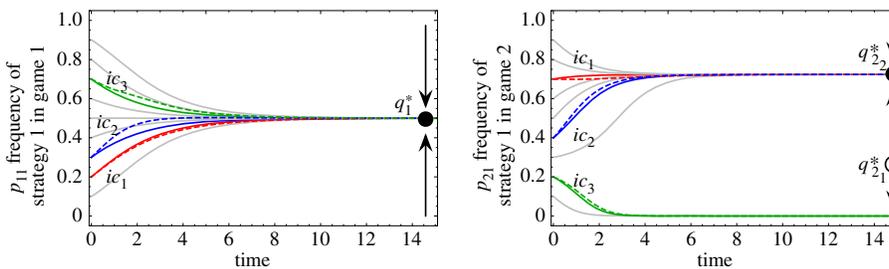


multi-game dynamics

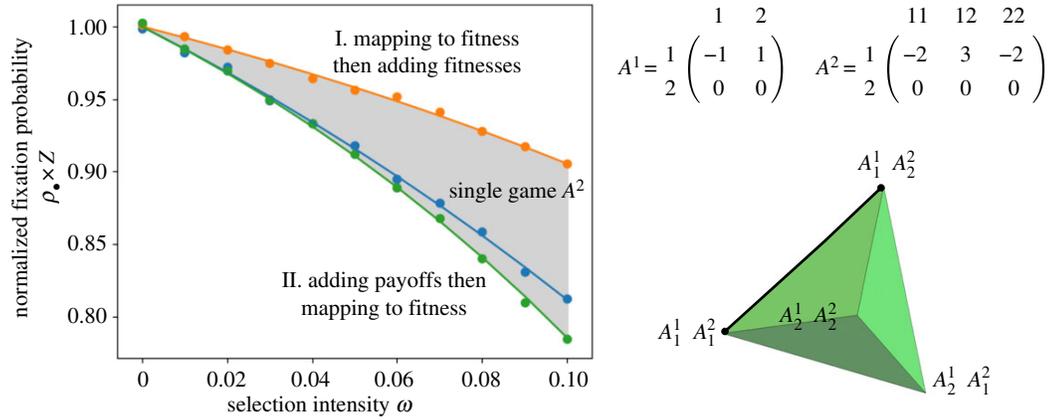


recovering single game dynamics from MGD

recovered dynamics (dashed) as compared to the SGD



**Figure A.4.** Two games with two strategies. The SGD of a 2-player and a 3-player game from equations (3.1) are shown in the top panel. Initial conditions of the highlighted trajectories correspond to the ones used in the MGD. The vertices of an  $S_4$  simplex (tetrahedron) denote these ‘categorical strategies’. The asterisks depict the initial conditions ( $ic_1$ ,  $ic_2$ , and  $ic_3$ ) chosen to correspond to the initial conditions from the SGD. Other random initial conditions are plotted in grey. Recovering the SGDs from the MGD, we see that  $p_{11}$  (playing strategy 1 in game 1, dashed lines) converges to  $q_1^* = 0.5$  which is the equilibrium solution for strategy 1 in game 1. If we start above the unstable equilibrium solution for game 2, i.e.  $q_{2_1}^* = 0.27$ , then  $p_{21}$  (playing strategy 1 in game 2, dashed lines) converges to  $q_{2_2}^* = 0.73$ , the stable equilibrium solution. For trajectories commencing below the unstable equilibrium, strategy 1 goes extinct. Comparing the recovered (dashed) dynamics to the SGD (solid), we see that while the equilibria of the recovered dynamics are the same as that of the SGD, the trajectories do not follow the same path. This is because the trajectories traverse a higher dimension which offers optional paths to the same equilibrium solutions. The initials conditions for ( $x_{11}$ ,  $x_{12}$ ,  $x_{21}$ ,  $x_{22}$ ) used in these plots are:  $ic_1 = (0.1, 0.1, 0.6, 0.2)$ ,  $ic_2 = (0.2, 0.1, 0.2, 0.5)$ , and  $ic_3 = (0.1, 0.6, 0.1, 0.2)$ . (Online version in colour.)



**Figure A.5.** Fixation probability of a single individual playing  $A_1^1 A_1^2$  strategy on the edge  $A_1^1 A_1^2 \rightleftharpoons A_1^1 A_2^2$ , i.e.  $\rho_{A_1^1 A_1^2, A_1^1 A_2^2}$  varying with selection intensity for a combination of two games having two strategies each. For a population of  $Z = 10$ , the fixation probabilities are normalized according to the neutral fixation probability,  $(1/Z) = 0.1$ . We look at the edge  $A_1^1 A_1^2 \rightleftharpoons A_1^1 A_2^2$  where  $A^1$  is the same for both vertices, i.e. neutral in both the vertices, and  $A^2$  is what matters. Since the payoff of playing strategy 2 in  $A^2$  is greater than playing strategy 1 ( $\pi_{22} > \pi_{21}$ ), the fixation probability decreases (see the electronic supplementary material for more details). The line labelled 'single game' corresponds to  $A^2$ . The plots from Method I (mapping payoffs to fitnesses and then adding the fitnesses to get the combined fitness) and Method II (adding the payoffs first, and then performing the payoff to fitness mapping) for a combination of the two games  $A^1$  and  $A^2$  show how the MGD is different from the SGD. Adding another game to  $A^2$  modifies the dynamics. Within the MGD, the two methods of mapping from payoffs to fitness, i.e. Methods I and II show different results. The shaded region (calculated in the electronic supplementary material) shows this difference between the two methods with increasing selection intensity. The results from analytics and stochastic simulations are plotted as solid lines and symbols, respectively. The simulations are averaged over  $10^6$  realizations. (Online version in colour.)

# **Appendix B**

## **Supplementary Material for the manuscript in Chapter 2**

# Electronic Supplementary Material: Consequences of combining life-history traits with sex-specific differences

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## S.1 One locus, two alleles

Let the fitnesses of the three genotypes be  $A_1A_1$ ,  $A_1A_2$  and  $A_2A_2$  are  $W_1$ ,  $W_2$  and  $W_3$ . The frequencies of the three genotypes are denoted by  $x_1$ ,  $x_2$  and  $x_3$ . Thus,

$$p(t+1) = x_1 + \frac{1}{2}x_2 = p(t)^2 \cdot \frac{W_1}{\bar{W}} + \frac{1}{2} \frac{2p(t)q(t) \cdot W_2}{\bar{W}}. \quad (\text{S.1})$$

Similarly,

$$q(t+1) = x_3 + \frac{1}{2}x_2 = q(t)^2 \cdot \frac{W_3}{\bar{W}} + \frac{1}{2} \frac{2p(t)q(t) \cdot W_2}{\bar{W}}. \quad (\text{S.2})$$

The  $W$ s could be survivability (viability) or fertility or both. Under neutrality they are all equal to unity (Otto and Day, 2007).

## S.2 Separate population into males and females

If the population is separated into the two sexes, Sex 1 which could be male (or female) denoted by a solid circle symbol  $\bullet$ , and Sex 2 which could be female (or male) denoted by a diamond symbol  $\diamond$ . We stick to calling the sexes as Sex 1 and Sex 2 instead of males and females (and we also do not use the standard  $\text{♀}$  and  $\text{♂}$  symbols as it might be misleading) because we want to show a generalized idea of the dependence of sexual immune dimorphism on the amount of parental investment (or mating competition and other factors) given and not to the sex itself.

For Sex 1, let frequency of  $A_1A_1 = x_{\bullet 1}$ , frequency of  $A_1A_2 = x_{\bullet 2}$  and frequency of  $A_2A_2 = x_{\bullet 3}$ . Similarly, for Sex 2, let frequency of  $A_1A_1 = x_{\diamond 1}$ , frequency of  $A_1A_2 = x_{\diamond 2}$  and frequency of  $A_2A_2 = x_{\diamond 3}$ .

In Sex 1, let the fitness of individuals with genotype  $A_1A_1 = W_{\bullet 1}$ , fitness of  $A_1A_2 = W_{\bullet 2}$  and fitness of  $A_2A_2 = W_{\bullet 3}$ . Similarly, for Sex 2, let the fitness of individuals with genotype  $A_1A_1 = W_{\diamond 1}$ , fitness of  $A_1A_2 = W_{\diamond 2}$  and fitness of  $A_2A_2 = W_{\diamond 3}$ . The sex that performs mating competitions (say, Sex 2) is further divided into individuals with Less or More Ornamentation (*LO* or *MO*). Through Mendelian population dynamics we obtain the of frequency of each genotype at subsequent generations (Gokhale et al., 2014),

$$\begin{aligned}\bar{W} x'_{\bullet 1} &= \frac{W_{\bullet 1}}{2} \left( x_{\bullet 1} x_{\diamond 1} + \frac{x_{\bullet 1} x_{\diamond 2}}{2} + \frac{x_{\bullet 2} x_{\diamond 1}}{2} + \frac{x_{\bullet 2} x_{\diamond 2}}{4} \right) \\ \bar{W} x'_{\bullet 2} &= \frac{W_{\bullet 2}}{2} \left( x_{\bullet 1} x_{\diamond 3} + x_{\bullet 3} x_{\diamond 1} + \frac{x_{\bullet 1} x_{\diamond 2}}{2} \right. \\ &\quad \left. + \frac{x_{\bullet 2} x_{\diamond 1}}{2} + \frac{x_{\bullet 3} x_{\diamond 2}}{2} + \frac{x_{\bullet 2} x_{\diamond 2}}{2} + \frac{x_{\bullet 2} x_{\diamond 3}}{2} \right) \\ \bar{W} x'_{\bullet 3} &= \frac{W_{\bullet 3}}{2} \left( x_{\bullet 3} x_{\diamond 3} + \frac{x_{\bullet 3} x_{\diamond 2}}{2} + \frac{x_{\bullet 2} x_{\diamond 3}}{2} + \frac{x_{\bullet 2} x_{\diamond 2}}{4} \right).\end{aligned}\tag{S.3}$$

and

$$\begin{aligned}\bar{W} x'_{\diamond 1, MO} &= \frac{W_{\diamond 1, MO}}{2} \left( x_{\diamond 1, MO} x_{\bullet 1} + \frac{x_{\diamond 1, MO} x_{\bullet 2}}{2} + \frac{x_{\diamond 2, MO} x_{\bullet 1}}{2} + \frac{x_{\diamond 2, MO} x_{\bullet 2}}{4} \right) \\ \bar{W} x'_{\diamond 2, MO} &= \frac{W_{\diamond 2, MO}}{2} \left( x_{\diamond 1, MO} x_{\bullet 3} + x_{\diamond 3, MO} x_{\bullet 1} + \frac{x_{\diamond 1, MO} x_{\bullet 2}}{2} \right. \\ &\quad \left. + \frac{x_{\diamond 2, MO} x_{\bullet 1}}{2} + \frac{x_{\diamond 3, MO} x_{\bullet 2}}{2} + \frac{x_{\diamond 2, MO} x_{\bullet 2}}{2} + \frac{x_{\diamond 2, MO} x_{\bullet 3}}{2} \right) \\ \bar{W} x'_{\diamond 3, MO} &= \frac{W_{\diamond 3, MO}}{2} \left( x_{\diamond 3, MO} x_{\bullet 3} + \frac{x_{\diamond 3, MO} x_{\bullet 2}}{2} + \frac{x_{\diamond 2, MO} x_{\bullet 3}}{2} + \frac{x_{\diamond 2, MO} x_{\bullet 2}}{4} \right) \\ \bar{W} x'_{\diamond 1, LO} &= \frac{W_{\diamond 1, LO}}{2} \left( x_{\diamond 1, LO} x_{\bullet 1} + \frac{x_{\diamond 1, LO} x_{\bullet 2}}{2} + \frac{x_{\diamond 2, LO} x_{\bullet 1}}{2} + \frac{x_{\diamond 2, LO} x_{\bullet 2}}{4} \right) \\ \bar{W} x'_{\diamond 2, LO} &= \frac{W_{\diamond 2, LO}}{2} \left( x_{\diamond 1, LO} x_{\bullet 3} + x_{\diamond 3, LO} x_{\bullet 1} + \frac{x_{\diamond 1, LO} x_{\bullet 2}}{2} \right. \\ \bar{W} x'_{\diamond 3, LO} &= \frac{W_{\diamond 3, LO}}{2} \left( x_{\diamond 3, LO} x_{\bullet 3} + \frac{x_{\diamond 3, LO} x_{\bullet 2}}{2} + \frac{x_{\diamond 2, LO} x_{\bullet 3}}{2} + \frac{x_{\diamond 2, LO} x_{\bullet 2}}{4} \right)\end{aligned}\tag{S.4}$$

where  $\bar{W}$  is the average fitness of all genotypes. Also,  $x'_{\bullet i}$  and  $x'_{\diamond i}$  is the change in

frequencies of the genotypes  $i$  (for the different sexes) with time. Also, here we assume equal sex ratio; half of the offspring are males and the other half, females.

Now, let  $W_{\diamond 1} = W_{\bullet 1} = W_1$ ,  $W_{\diamond 2} = W_{\bullet 2} = W_2$  and  $W_{\diamond 3} = W_{\bullet 3} = W_3$ . where  $W_{\diamond i} = W_{\diamond i, MO} + W_{\diamond i, LO}$ . Then,

$$\begin{aligned}
\bar{W}x'_1 &= \bar{W}x'_{\diamond 1} + \bar{W}x'_{\bullet 1} = W_1 \left[ (x_{\bullet 1} \cdot x_{\diamond 1}) + \frac{(x_{\bullet 1} \cdot x_{\diamond 2})}{2} + \frac{(x_{\bullet 2} \cdot x_{\diamond 1})}{2} + \frac{(x_{\bullet 2} \cdot x_{\diamond 2})}{4} \right] \\
\bar{W}x'_2 &= \bar{W}x'_{\diamond 2} + \bar{W}x'_{\bullet 2} = W_2 \left[ (x_{\bullet 1} \cdot x_{\diamond 3}) + (x_{\bullet 3} \cdot x_{\diamond 1}) + \frac{(x_{\bullet 1} \cdot x_{\diamond 2})(x_{\bullet 2} \cdot x_{\diamond 1})}{2} \right. \\
&\quad \left. + \frac{(x_{\bullet 3} \cdot x_{\diamond 2})}{2} + \frac{(x_{\bullet 2} \cdot x_{\diamond 2})}{2} + \frac{(x_{\bullet 2} \cdot x_{\diamond 3})}{2} \right] \\
\bar{W}x'_3 &= \bar{W}x'_{\diamond 3} + \bar{W}x'_{\bullet 3} = W_3 \left[ (x_{\bullet 3} \cdot x_{\diamond 3}) + \frac{(x_{\bullet 3} \cdot x_{\diamond 2})}{2} + \frac{(x_{\bullet 2} \cdot x_{\diamond 3})}{2} + \frac{(x_{\bullet 2} \cdot x_{\diamond 2})}{4} \right].
\end{aligned} \tag{S.5}$$

### S.3 Mating competition

Mating competition is performed through fights, sexual signals, nuptial gifts, ornament display and various types of attractiveness. We shall refer to all of these as 'ornaments'. Let's assume there are individuals of two types in this interaction: ones that display more ornaments (MO) and ones that display less (LO). Consider the mating competition interaction between individuals of Sex 2. For the three different genotypes  $i$  the population in Sex 2 will consist of six different kinds of individuals,  $x_{\diamond j, MO}$  and  $x_{\diamond j, LO}$ .

We model this interaction as an evolutionary game (Maynard Smith, 1986; Sigmund and Nowak, 1999). The payoff matrix is written as,

$$\begin{array}{cc}
& \begin{array}{cc} MO & LO \end{array} \\
\begin{array}{c} \text{More Ornament or } MO \\ \text{Less Ornament or } LO \end{array} & \begin{pmatrix} \frac{b^O}{2} - c_{\diamond}^O & b^O - c_{\diamond}^O \\ 0 & \frac{b^O}{2} \end{pmatrix}
\end{array} \tag{S.6}$$

where  $b^O$  is the benefit arising from mating competitions i.e. mating gain and  $c_{\diamond}^O$  is the cost that Sex 2 bears to maintain ornament(s). The frequency dependent fitnesses resulting from these interactions are given by,

$$\begin{aligned}
W_{\diamond MO}^O &= \left( \frac{b^O}{2} - c_{\diamond}^O \right) \frac{x_{\diamond, MO}}{x_{\diamond, MO} + x_{\diamond, LO}} + (b^O - c_{\diamond}^O) \frac{x_{\diamond, LO}}{x_{\diamond, MO} + x_{\diamond, LO}} \\
W_{\diamond LO}^O &= 0 \frac{x_{\diamond, MO}}{x_{\diamond, MO} + x_{\diamond, LO}} + \frac{b^O}{2} \frac{x_{\diamond, LO}}{x_{\diamond, MO} + x_{\diamond, LO}}
\end{aligned} \tag{S.7}$$

where  $x_{\diamond, MO} = \sum_{j=1}^3 x_{\diamond j, MO}$  and  $x_{\diamond, LO} = \sum_{j=1}^3 x_{\diamond j, LO}$  and  $c^O < \frac{b^O}{2}$ .

The payoff matrix (S.6) is an interaction between a pair of individuals i.e. two player game. We can extend this to  $d$ -players (Gokhale and Traulsen, 2014; Chen et al., 2017) and the payoffs are given by,

$$P_{MO} = \begin{cases} b^O - c_{\diamond}^O & k = 1 \\ \frac{b^O}{k} - c_{\diamond}^O & k > 1 \end{cases} \quad (\text{S.8})$$

$$P_{LO} = \begin{cases} \frac{b^P}{n} & k = 0 \\ 0 & k > 0 \end{cases} \quad (\text{S.9})$$

where  $k$  is the number of  $MO$  (More Ornament) players and  $n$  is the total number of players.  $k$  and  $n$  can vary between the sexes.

## S.4 Dynamics

### S.4.1 One locus

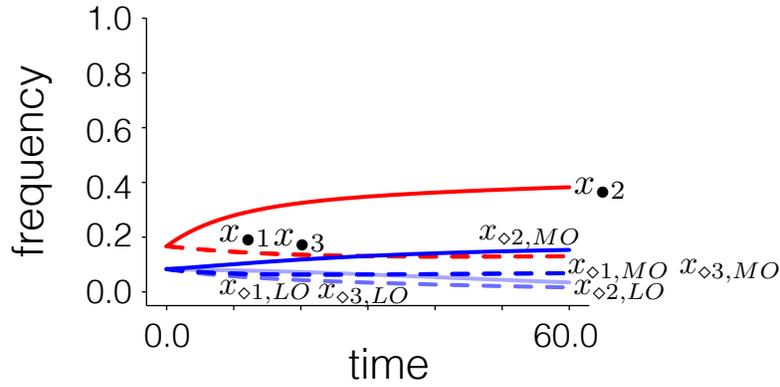


Figure S.1: **Evolution of frequency of all possible type of individuals in a population that exhibits sexual dimorphism in immunity and ornamentation, and sex difference in parental investment.** When sex 1 performs major parental investment and individuals of Sex 2 perform mating competitions, then the population will have nine types of individuals -  $x_{\bullet i}, x_{\diamond i, MO}$  and  $x_{\diamond i, LO}$  for the three immunity genotypes  $i$ . For the results shown in this figure,  $c_{\bullet}^P = 0.6$ ,  $c_{\diamond}^P = 0$  and  $c_{\diamond}^O = 0.4$ . Fitness from immune response comes from  $\Omega > 0$  of the linear immune allelic diversity vs immune response profile in Figure 2 in the main article. Red lines are for Sex 1 and blue for Sex 2. The solid lines are for the heterozygous genotype and dashed lines for the homozygotes. The lighter blue lines in Sex 2 are for individuals with low ornamentation.

If we consider that Sex 1, the sex undergoes major parental investment does not involve in mating competitions and individuals of Sex 2 perform mating competitions, then the population will have nine types of individuals -  $x_{\bullet i}, x_{\diamond i, MO}$  and  $x_{\diamond i, LO}$  for the three genotypes  $i$ . We shall refer to them as  $x_1, x_2, x_3, x_4, x_5, x_6, x_7, x_8$  and  $x_9$ .

The lifetime reproductive success of each type within a sex is a multiplicative combination of mating gains, fertility and survival probability (Stoehr and Kokko, 2006; Kelly and Alonzo, 2010). These are given by,

$$\begin{aligned}
W_{\bullet 1} &= \frac{W_{\bullet}^P}{1 - W_{\bullet 1}^I} & W_{\bullet 2} &= \frac{W_{\bullet}^P}{1 - W_{\bullet 2}^I} & W_{\bullet 3} &= \frac{W_{\bullet}^P}{1 - W_{\bullet 3}^I} \\
W_{\diamond 1,MO} &= \frac{W_{\diamond}^P \cdot W_{\diamond MO}^O}{1 - W_{\bullet 1}^I} & W_{\diamond 2,MO} &= \frac{W_{\diamond}^P \cdot W_{\diamond MO}^O}{1 - W_{\bullet 2}^I} & W_{\diamond 3,MO} &= \frac{W_{\diamond}^P \cdot W_{\diamond MO}^O}{1 - W_{\bullet 3}^I} \\
W_{\diamond 1,LO} &= \frac{W_{\diamond}^P \cdot W_{\diamond LO}^O}{1 - W_{\bullet 1}^I} & W_{\diamond 2,LO} &= \frac{W_{\diamond}^P \cdot W_{\diamond LO}^O}{1 - W_{\bullet 2}^I} & W_{\diamond 3,LO} &= \frac{W_{\diamond}^P \cdot W_{\diamond LO}^O}{1 - W_{\bullet 3}^I}.
\end{aligned} \tag{S.10}$$

Here,  $W_{\bullet i}^I$  and  $W_{\diamond i}^I$  are the fitness from immune responses (survivability) of type  $i$  for Sex 1 and Sex 2 as described in the main text. Similarly,  $W_{\bullet}^P$  and  $W_{\diamond}^P$  are the fitness that arise from parental investments performed by members of Sex 1 and Sex 2, respectively. The fitness from More and Less ornamentation ( $W_{MO}^O$  and  $W_{LO}^O$ ) are as defined in the previous section. Using equations (S.3) and (S.4), we can obtain the average fitnesses for each type of individuals in the population. For Sex 1 they are given by,

$$W_{\bullet j} = \frac{\bar{W} x'_{\bullet j}}{x_{\bullet j}} \tag{S.11}$$

where  $j = \{1, 2, 3\}$  and  $\bar{W}$  is the average fitness of all types. For Sex 2 they are given by,

$$\begin{aligned}
W_{\diamond j,MO} &= \frac{\bar{W} x'_{\diamond j,MO}}{x_{\diamond j,MO}} \\
W_{\diamond j,LO} &= \frac{\bar{W} x'_{\diamond j,LO}}{x_{\diamond j,LO}}
\end{aligned} \tag{S.12}$$

where again  $j = \{1, 2, 3\}$ . Here,  $MO$  and  $LO$  correspond to individuals with more and less ornamentation, respectively. From equations (S.11) and (S.11) we know that there are nine different types of individuals whose frequencies can be just described by  $x_i$  for  $i = \{1, 2, 3, \dots, 9\}$  and their respective average fitnesses are denoted by  $W_i$  (for  $i = \{1, 2, 3, \dots, 9\}$ ).

Using the above given equations we have,

$$x'_i - x_i = \left( \frac{x_i W_i}{\bar{W}} \right) - x_i. \tag{S.13}$$

The classical selection equation (Crow and Kimura, 1970; Hofbauer and Sigmund, 1998) that gives the evolution of each type (see Figure S.1) is then obtained by taking the time derivative of (S.13) given by,

$$\dot{x}_i = x_i (W_i - \bar{W}). \tag{S.14}$$

The frequencies of all types reach an equilibrium value at some time point. This is our value of interest that is used in the results throughout this ESM and the main article.

The frequencies of each sex is a summation of frequencies of all types of individuals in a sex. Figure S.2 shows how the frequency of the sexes changes with sex-specific differences in immunocompetence, parental investment, and ornamentation.

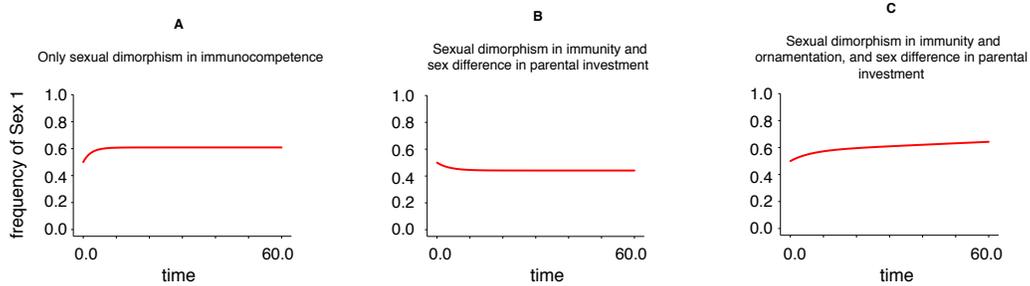


Figure S.2: **Evolution of frequency of Sex 1 in the population.** Since frequency of Sex 1 ( $x_{\bullet}$ ) and frequency of Sex 2 ( $x_{\diamond}$ ) equals unity,  $x_{\diamond}$  equals  $1 - x_{\bullet}$ . These frequencies are obtained by summing up all types of individuals within the sexes. (A) When Sex 1 has a higher values of immune response a compared to Sex 2 for all immune allelic diversity ( $\Omega$ ). (B) When condition A is met, but Sex 1 also performs parental investment, while Sex 2 does not. (C) When conditions A and B are met, and Sex 2 also exhibits ornamentation. The sex-specific traits evolve over generation (time) by selection and therefore, get passed on to subsequent generations (for example, case C is shown in Figure S.1). Therefore, even when the sex ratio is kept equal among offspring at every generation, their sex-specific characteristics change their frequency in the population.

## S.4.2 One locus: Results

### Heterozygosity vs Homozygosity

Under Hardy Weinberg or when all interactions are neutral, the number of heterozygous and homozygous individuals within a sex would be equal. However, under selection (through different probabilities of immune response for homozygotes and heterozygotes), varying cost of parental investment and ornamentation the number of heterozygotes and homozygotes would deviate from neutrality. An increase in heterozygotes within one sex compared to the other, would also mean that it has a higher immune response on average. When we allow for selection to act on all the three factors (parental investment, immunity genes and ornamentation), we can observe their combined effect on the increase in frequency of heterozygous individuals within a sex (results shown in Figures S.3, S.4 and S.5).

## S.5 Two loci having two alleles each

### S.5.1 Population dynamics with separation of population into males and females

For Sex 1, let the frequency of  $A_1B_1|A_1B_1 = f_{\bullet}(A_1B_1|A_1B_1) = x_{\bullet 1}$ ,  $f_{\bullet}(A_1B_1|A_1B_2) = x_{\bullet 2}$ ,  $f_{\bullet}(A_1B_2|A_1B_2) = x_{\bullet 3}$ ,  $f_{\bullet}(A_1B_1|A_2B_1) = x_{\bullet 4}$ ,  $f_{\bullet}(A_1B_2|A_2B_1) = x_{\bullet 5}$ ,  $f_{\bullet}(A_1B_2|A_2B_2) = x_{\bullet 6}$ ,  $f_{\bullet}(A_2B_1|A_2B_1) = x_{\bullet 7}$ ,  $f_{\bullet}(A_2B_1|A_2B_2) = x_{\bullet 8}$ ,  $f_{\bullet}(A_2B_2|A_2B_2) = x_{\bullet 9}$  and  $f_{\bullet}(A_1B_1|A_2B_2) = x_{\bullet 10}$ . Similarly, for Sex 2.

From Mendelian population dynamics (as done in the one locus case), the frequency of the homozygotes in Sex 1 will be:

$$\begin{aligned} \bar{W}x'_{\bullet 1} = & \frac{W_1}{2} \left( x_{\bullet 1} \cdot x_{\bullet 1} + \frac{x_{\bullet 1} \cdot x_{\bullet 2} + x_{\bullet 1} \cdot x_{\bullet 4} + x_{\bullet 1} \cdot x_{\bullet 10} + x_{\bullet 2} \cdot x_{\bullet 1} + x_{\bullet 4} \cdot x_{\bullet 1} + x_{\bullet 10} \cdot x_{\bullet 1}}{2} \right. \\ & \left. + \frac{x_{\bullet 2} \cdot x_{\bullet 2} + x_{\bullet 2} \cdot x_{\bullet 4} + x_{\bullet 2} \cdot x_{\bullet 10} + x_{\bullet 4} \cdot x_{\bullet 2} + x_{\bullet 4} \cdot x_{\bullet 4} + x_{\bullet 4} \cdot x_{\bullet 10} + x_{\bullet 10} \cdot x_{\bullet 2} + x_{\bullet 10} \cdot x_{\bullet 4} + x_{\bullet 10} \cdot x_{\bullet 10}}{4} \right). \end{aligned} \quad (\text{S.15})$$

$$\begin{aligned} \bar{W}x'_{\bullet 3} = & \frac{W_3}{2} \left( x_{\bullet 3} \cdot x_{\bullet 3} + \frac{x_{\bullet 2} \cdot x_{\bullet 3} + x_{\bullet 3} \cdot x_{\bullet 2} + x_{\bullet 3} \cdot x_{\bullet 5} + x_{\bullet 3} \cdot x_{\bullet 6} + x_{\bullet 5} \cdot x_{\bullet 3} + x_{\bullet 6} \cdot x_{\bullet 3}}{2} \right. \\ & \left. + \frac{x_{\bullet 2} \cdot x_{\bullet 2} + x_{\bullet 2} \cdot x_{\bullet 5} + x_{\bullet 2} \cdot x_{\bullet 6} + x_{\bullet 5} \cdot x_{\bullet 2} + x_{\bullet 5} \cdot x_{\bullet 5} + x_{\bullet 5} \cdot x_{\bullet 6} + x_{\bullet 6} \cdot x_{\bullet 2} + x_{\bullet 6} \cdot x_{\bullet 5} + x_{\bullet 6} \cdot x_{\bullet 6}}{4} \right). \end{aligned} \quad (\text{S.16})$$

$$\begin{aligned} \bar{W}x'_{\bullet 7} = & \frac{W_7}{2} \left( x_{\bullet 7} \cdot x_{\bullet 7} + \frac{x_{\bullet 4} \cdot x_{\bullet 7} + x_{\bullet 5} \cdot x_{\bullet 7} + x_{\bullet 7} \cdot x_{\bullet 5} + x_{\bullet 7} \cdot x_{\bullet 8} + x_{\bullet 8} \cdot x_{\bullet 7}}{2} \right. \\ & \left. + \frac{x_{\bullet 4} \cdot x_{\bullet 4} + x_{\bullet 4} \cdot x_{\bullet 5} + x_{\bullet 4} \cdot x_{\bullet 8} + x_{\bullet 5} \cdot x_{\bullet 4} + x_{\bullet 5} \cdot x_{\bullet 5} + x_{\bullet 5} \cdot x_{\bullet 8} + x_{\bullet 8} \cdot x_{\bullet 4} + x_{\bullet 8} \cdot x_{\bullet 5} + x_{\bullet 8} \cdot x_{\bullet 8}}{4} \right). \end{aligned} \quad (\text{S.17})$$

$$\begin{aligned} \bar{W}x'_{\bullet 9} = & \frac{W_9}{2} \left( x_{\bullet 9} \cdot x_{\bullet 9} + \frac{x_{\bullet 6} \cdot x_{\bullet 9} + x_{\bullet 8} \cdot x_{\bullet 9} + x_{\bullet 9} \cdot x_{\bullet 6} + x_{\bullet 9} \cdot x_{\bullet 8} + x_{\bullet 9} \cdot x_{\bullet 10} + x_{\bullet 10} \cdot x_{\bullet 9}}{2} \right. \\ & \left. + \frac{x_{\bullet 6} \cdot x_{\bullet 6} + x_{\bullet 6} \cdot x_{\bullet 8} + x_{\bullet 10} \cdot x_{\bullet 8} + x_{\bullet 8} \cdot x_{\bullet 6} + x_{\bullet 8} \cdot x_{\bullet 8} + x_{\bullet 8} \cdot x_{\bullet 10} + x_{\bullet 10} \cdot x_{\bullet 6} + x_{\bullet 10} \cdot x_{\bullet 9} + x_{\bullet 10} \cdot x_{\bullet 10}}{4} \right). \end{aligned} \quad (\text{S.18})$$

Frequency of the single heterozygotes will be:

$$\begin{aligned}
\overline{W}x'_{\bullet 2} = & \frac{W_2}{2} \left( x_{\bullet 1} \cdot x_{\circ 3} + x_{\bullet 3} \cdot x_{\circ 1} + \frac{x_{\bullet 1} \cdot x_{\circ 2} + x_{\bullet 1} \cdot x_{\circ 5} + x_{\bullet 1} \cdot x_{\circ 6} + x_{\bullet 2} \cdot x_{\circ 1} + x_{\bullet 2} \cdot x_{\circ 2} + x_{\bullet 2} \cdot x_{\circ 3} + x_{\bullet 3} \cdot x_{\circ 2}}{2} \right. \\
& + \frac{x_{\bullet 3} \cdot x_{\circ 4} + x_{\bullet 3} \cdot x_{\circ 10} + x_{\bullet 4} \cdot x_{\circ 3} + x_{\bullet 5} \cdot x_{\circ 1} + x_{\bullet 5} \cdot x_{\circ 2} + x_{\bullet 6} \cdot x_{\circ 1} + x_{\bullet 10} \cdot x_{\circ 2}}{2} \\
& + \frac{x_{\bullet 2} \cdot x_{\circ 4} + x_{\bullet 2} \cdot x_{\circ 5} + x_{\bullet 2} \cdot x_{\circ 6} + x_{\bullet 2} \cdot x_{\circ 10} + x_{\bullet 4} \cdot x_{\circ 2} + x_{\bullet 4} \cdot x_{\circ 5} + x_{\bullet 4} \cdot x_{\circ 6} + x_{\bullet 5} \cdot x_{\circ 4} + x_{\bullet 5} \cdot x_{\circ 10}}{4} \\
& \left. + \frac{x_{\bullet 6} \cdot x_{\circ 2} + x_{\bullet 6} \cdot x_{\circ 4} + x_{\bullet 6} \cdot x_{\circ 10} + x_{\bullet 10} \cdot x_{\circ 2} + x_{\bullet 10} \cdot x_{\circ 5} + x_{\bullet 10} \cdot x_{\circ 6}}{4} \right). \tag{S.19}
\end{aligned}$$

$$\begin{aligned}
\overline{W}x'_{\bullet 4} = & \frac{W_4}{2} \left( x_{\bullet 1} \cdot x_{\circ 7} + x_{\bullet 7} \cdot x_{\circ 1} + \frac{x_{\bullet 1} \cdot x_{\circ 4} + x_{\bullet 1} \cdot x_{\circ 5} + x_{\bullet 1} \cdot x_{\circ 8} + x_{\bullet 4} \cdot x_{\circ 1} + x_{\bullet 4} \cdot x_{\circ 4} + x_{\bullet 4} \cdot x_{\circ 7} + x_{\bullet 5} \cdot x_{\circ 1}}{2} \right. \\
& + \frac{x_{\bullet 7} \cdot x_{\circ 2} + x_{\bullet 7} \cdot x_{\circ 4} + x_{\bullet 7} \cdot x_{\circ 10} + x_{\bullet 8} \cdot x_{\circ 1} + x_{\bullet 10} \cdot x_{\circ 7} + x_{\bullet 10} \cdot x_{\circ 8}}{2} \\
& + \frac{x_{\bullet 2} \cdot x_{\circ 4} + x_{\bullet 2} \cdot x_{\circ 5} + x_{\bullet 2} \cdot x_{\circ 7} + x_{\bullet 2} \cdot x_{\circ 8} + x_{\bullet 4} \cdot x_{\circ 2} + x_{\bullet 4} \cdot x_{\circ 5} + x_{\bullet 4} \cdot x_{\circ 8} + x_{\bullet 4} \cdot x_{\circ 10} + x_{\bullet 5} \cdot x_{\circ 2} + x_{\bullet 5} \cdot x_{\circ 4}}{4} \\
& \left. + \frac{x_{\bullet 5} \cdot x_{\circ 10} + x_{\bullet 8} \cdot x_{\circ 2} + x_{\bullet 8} \cdot x_{\circ 4} + x_{\bullet 8} \cdot x_{\circ 10} + x_{\bullet 10} \cdot x_{\circ 4} + x_{\bullet 10} \cdot x_{\circ 5}}{4} \right). \tag{S.20}
\end{aligned}$$

$$\begin{aligned}
\overline{W}x'_{\bullet 6} = & \frac{W_6}{2} \left( x_{\bullet 9} \cdot x_{\circ 3} + x_{\bullet 3} \cdot x_{\circ 9} + \frac{x_{\bullet 2} \cdot x_{\circ 9} + x_{\bullet 3} \cdot x_{\circ 6} + x_{\bullet 3} \cdot x_{\circ 8} + x_{\bullet 3} \cdot x_{\circ 10} + x_{\bullet 5} \cdot x_{\circ 9} + x_{\bullet 6} \cdot x_{\circ 3} + x_{\bullet 6} \cdot x_{\circ 6}}{2} \right. \\
& + \frac{x_{\bullet 6} \cdot x_{\circ 9} + x_{\bullet 8} \cdot x_{\circ 3} + x_{\bullet 9} \cdot x_{\circ 2} + x_{\bullet 9} \cdot x_{\circ 5} + x_{\bullet 9} \cdot x_{\circ 6} + x_{\bullet 10} \cdot x_{\circ 3}}{2} \\
& + \frac{x_{\bullet 2} \cdot x_{\circ 6} + x_{\bullet 2} \cdot x_{\circ 8} + x_{\bullet 2} \cdot x_{\circ 10} + x_{\bullet 5} \cdot x_{\circ 6} + x_{\bullet 5} \cdot x_{\circ 8} + x_{\bullet 5} \cdot x_{\circ 10} + x_{\bullet 6} \cdot x_{\circ 2} + x_{\bullet 6} \cdot x_{\circ 5} + x_{\bullet 6} \cdot x_{\circ 8}}{4} \\
& \left. + \frac{x_{\bullet 6} \cdot x_{\circ 10} + x_{\bullet 8} \cdot x_{\circ 2} + x_{\bullet 8} \cdot x_{\circ 5} + x_{\bullet 8} \cdot x_{\circ 6} + x_{\bullet 10} \cdot x_{\circ 2} + x_{\bullet 10} \cdot x_{\circ 5} + x_{\bullet 10} \cdot x_{\circ 6}}{4} \right). \tag{S.21}
\end{aligned}$$

$$\begin{aligned}
\overline{W}x'_{\bullet 8} = & \frac{W_8}{2} \left( x_{\bullet 7} \cdot x_{\circ 9} + x_{\bullet 9} \cdot x_{\circ 7} + \frac{x_{\bullet 4} \cdot x_{\circ 9} + x_{\bullet 5} \cdot x_{\circ 9} + x_{\bullet 6} \cdot x_{\circ 7} + x_{\bullet 7} \cdot x_{\circ 6} + x_{\bullet 7} \cdot x_{\circ 8} + x_{\bullet 7} \cdot x_{\circ 10} + x_{\bullet 8} \cdot x_{\circ 7}}{2} \right. \\
& + \frac{x_{\bullet 8} \cdot x_{\circ 8} + x_{\bullet 8} \cdot x_{\circ 9} + x_{\bullet 8} \cdot x_{\circ 10} + x_{\bullet 9} \cdot x_{\circ 4} + x_{\bullet 9} \cdot x_{\circ 5} + x_{\bullet 9} \cdot x_{\circ 8} + x_{\bullet 10} \cdot x_{\circ 7}}{2} \\
& + \frac{x_{\bullet 4} \cdot x_{\circ 6} + x_{\bullet 4} \cdot x_{\circ 8} + x_{\bullet 4} \cdot x_{\circ 10} + x_{\bullet 5} \cdot x_{\circ 6} + x_{\bullet 5} \cdot x_{\circ 8} + x_{\bullet 5} \cdot x_{\circ 10} + x_{\bullet 6} \cdot x_{\circ 4} + x_{\bullet 6} \cdot x_{\circ 5} + x_{\bullet 6} \cdot x_{\circ 8}}{4} \\
& \left. + \frac{x_{\bullet 8} \cdot x_{\circ 4} + x_{\bullet 8} \cdot x_{\circ 5} + x_{\bullet 8} \cdot x_{\circ 6} + x_{\bullet 10} \cdot x_{\circ 4} + x_{\bullet 10} \cdot x_{\circ 5} + x_{\bullet 10} \cdot x_{\circ 8}}{4} \right). \tag{S.22}
\end{aligned}$$

Frequency of the double heterozygotes will be:

$$\begin{aligned}
\overline{W}x'_{\bullet 5} = & \frac{W_5}{2} \left( x_{\bullet 3} \cdot x_{\diamond 7} + x_{\bullet 7} \cdot x_{\diamond 3} + \frac{x_{\bullet 2} \cdot x_{\diamond 7} + x_{\bullet 3} \cdot x_{\diamond 4} + x_{\bullet 3} \cdot x_{\diamond 5} + x_{\bullet 3} \cdot x_{\diamond 8} + x_{\bullet 4} \cdot x_{\diamond 3} + x_{\bullet 5} \cdot x_{\diamond 3}}{2} \right. \\
& + \frac{x_{\bullet 5} \cdot x_{\diamond 5} + x_{\bullet 5} \cdot x_{\diamond 7} + x_{\bullet 6} \cdot x_{\diamond 5} + x_{\bullet 7} \cdot x_{\diamond 2} + x_{\bullet 7} \cdot x_{\diamond 5} + x_{\bullet 7} \cdot x_{\diamond 6} + x_{\bullet 8} \cdot x_{\diamond 3}}{2} \\
& + \frac{x_{\bullet 2} \cdot x_{\diamond 4} + x_{\bullet 2} \cdot x_{\diamond 5} + x_{\bullet 2} \cdot x_{\diamond 8} + x_{\bullet 4} \cdot x_{\diamond 2} + x_{\bullet 4} \cdot x_{\diamond 5} + x_{\bullet 4} \cdot x_{\diamond 6} + x_{\bullet 5} \cdot x_{\diamond 2} + x_{\bullet 5} \cdot x_{\diamond 4} + x_{\bullet 5} \cdot x_{\diamond 6}}{4} \\
& \left. + \frac{x_{\bullet 5} \cdot x_{\diamond 8} + x_{\bullet 6} \cdot x_{\diamond 4} + x_{\bullet 6} \cdot x_{\diamond 7} + x_{\bullet 6} \cdot x_{\diamond 8} + x_{\bullet 8} \cdot x_{\diamond 2} + x_{\bullet 8} \cdot x_{\diamond 5} + x_{\bullet 8} \cdot x_{\diamond 6}}{4} \right). \tag{S.23}
\end{aligned}$$

$$\begin{aligned}
\overline{W}x'_{\bullet 10} = & \frac{W_{10}}{2} \left( x_{\bullet 1} \cdot x_{\diamond 9} + x_{\bullet 9} \cdot x_{\diamond 1} + \frac{x_{\bullet 1} \cdot x_{\diamond 6} + x_{\bullet 1} \cdot x_{\diamond 8} + x_{\bullet 1} \cdot x_{\diamond 10} + x_{\bullet 2} \cdot x_{\diamond 8} + x_{\bullet 4} \cdot x_{\diamond 8} + x_{\bullet 6} \cdot x_{\diamond 1} + x_{\bullet 8} \cdot x_{\diamond 1}}{2} \right. \\
& + \frac{x_{\bullet 9} \cdot x_{\diamond 2} + x_{\bullet 9} \cdot x_{\diamond 4} + x_{\bullet 9} \cdot x_{\diamond 10} + x_{\bullet 10} \cdot x_{\diamond 1} + x_{\bullet 10} \cdot x_{\diamond 9} + x_{\bullet 10} \cdot x_{\diamond 10}}{2} \\
& + \frac{x_{\bullet 2} \cdot x_{\diamond 6} + x_{\bullet 2} \cdot x_{\diamond 9} + x_{\bullet 2} \cdot x_{\diamond 10} + x_{\bullet 4} \cdot x_{\diamond 6} + x_{\bullet 4} \cdot x_{\diamond 9} + x_{\bullet 4} \cdot x_{\diamond 10} + x_{\bullet 6} \cdot x_{\diamond 2} + x_{\bullet 6} \cdot x_{\diamond 4}}{4} \\
& \left. + \frac{x_{\bullet 6} \cdot x_{\diamond 10} + x_{\bullet 8} \cdot x_{\diamond 2} + x_{\bullet 8} \cdot x_{\diamond 4} + x_{\bullet 8} \cdot x_{\diamond 10} + x_{\bullet 10} \cdot x_{\diamond 2} + x_{\bullet 10} \cdot x_{\diamond 4} + x_{\bullet 10} \cdot x_{\diamond 6} + x_{\bullet 10} \cdot x_{\diamond 8}}{4} \right). \tag{S.24}
\end{aligned}$$

Here, the  $W_i$ s are the fitnesses of each genotype  $i$  with frequency  $x_i$  and  $\overline{W}$  is their mean fitness. Similarly, we can obtain the frequencies of the genotypes in Sex 2.

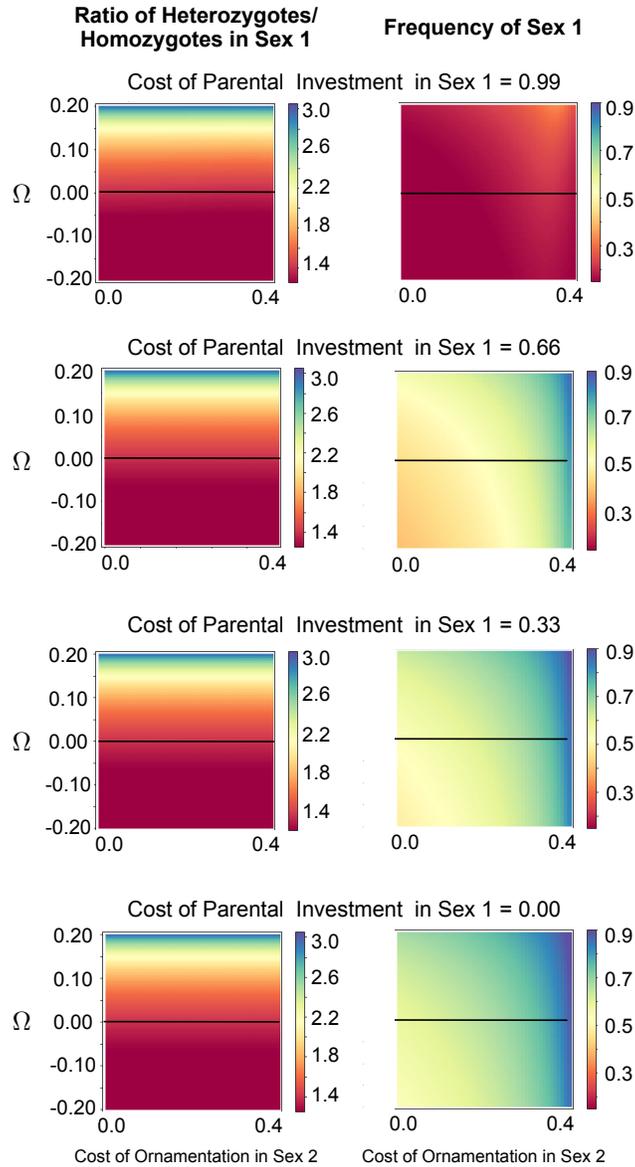


Figure S.3: Ratio of Heterozygotes: Homozygotes in Sex 1 and the frequency of Sex 2 for a full range of  $\Omega$ . The parameter  $\Omega$  is a measure of the sex difference in immune response through sexual conflict within the MHC as shown in Figure 2 in the main article. It represents the sex-specific fitness effect of Sex 1 relative to Sex 2. When  $\Omega = 0$ , there is so sex-specific difference in immune response. There is no effect of ornamentation and parental investment (PI) on the ratio of allele diversity. However,  $\Omega$  has an effect on this ratio. All factors: coat of PI, cost of ornamentation and  $\Omega$  have an effect on the frequency of the sexes. Thought the effect of  $\Omega$  is not profound, the cost of ornamentation in Sex 2 and cost of PI in Sex 1 reduce their frequency, respectively.

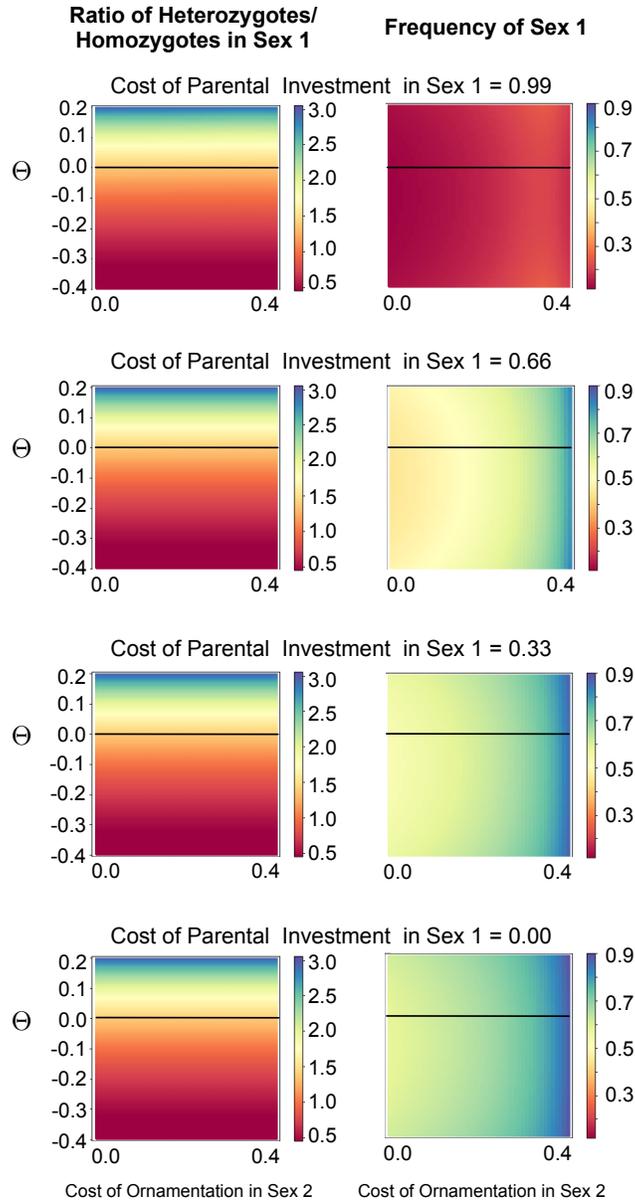


Figure S.4: Ratio of Heterozygotes: Homozygotes in Sex 1 and the frequency of Sex 2 for a full range of  $\Theta$ . The parameter  $\Theta$  is a measure of the sex difference in immune response through sexual conflict within the MHC as shown in Figure 2 in the main article. It represents the effect of allelic diversity on sex-specific fitness of Sex 1 relative to Sex 2. When  $\Theta = 0$ , there is so sex-specific difference in immune response. The parameter  $\Theta$  has an effect on the allele diversity ratio. But there is no effect of ornamentation and parental investment (PI) on this ratio. There is no effect of  $\Theta$  on the frequency of Sex 1. The cost of ornamentation in Sex 2 increases the frequency of Sex 1 while the cost of PI decreases its frequency.

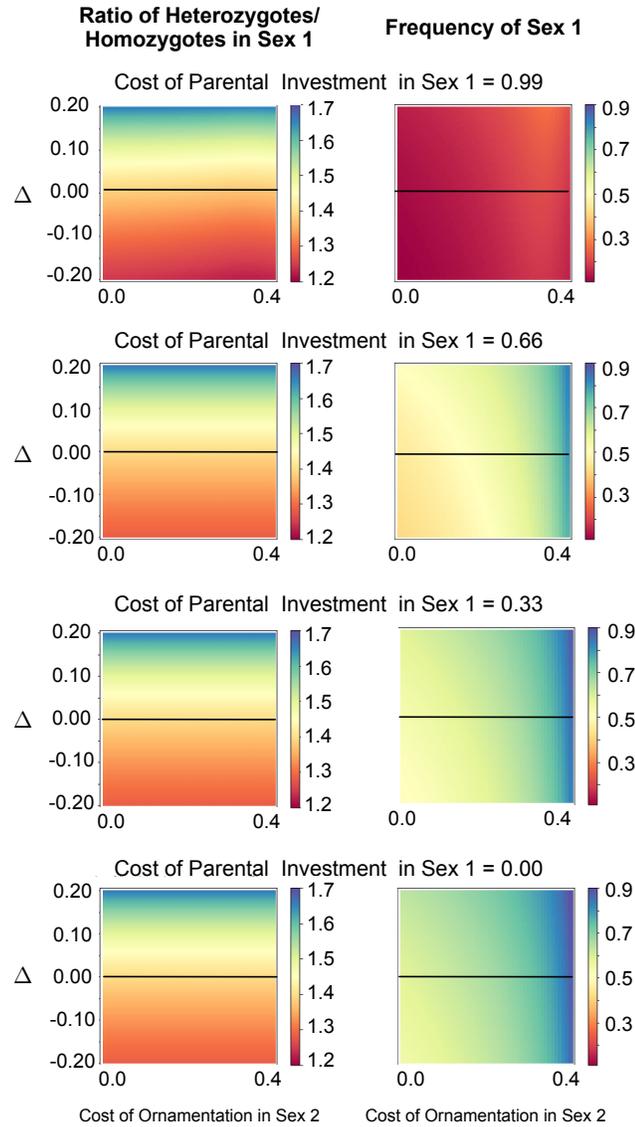


Figure S.5: Ratio of Heterozygotes: Homozygotes in Sex 1 and the frequency of Sex 2 for a full range of  $\Delta$ . The parameter  $\Delta$  is a measure of the sex difference in immune response through sexual conflict within the MHC as shown in Figure 2 in the main article. It represents the sex-specific fitness effect (that also includes the effect of diversity on sex-specific fitness) of Sex 1 relative to Sex 2. When  $\Delta = 0$ , there is no sex-specific difference in immune response. There is no effect of ornamentation and parental investment (PI) on the ratio of allele diversity. But  $\Delta$  has an effect on this ratio. As observed in the previous figure, here too, the cost of ornamentation in Sex 2 and cost of PI in Sex 1 reduce their frequency, respectively while the effect of  $\Delta$  is not as profound.

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# Publications and author contributions

## Publications

- Chapter 2: V.R Venkateswaran, C.S Gokhale, 2019. Evolutionary dynamics of complex multiple games. *Proceedings of the Royal Society B: Biological Sciences*.
- Chapter 3: V.R Venkateswaran, O Roth and C.S Gokhale. Consequences of combining life-history traits with sex-specific differences. *bioRxiv* doi: <https://doi.org/10.1101/2020.01.03.892810>.
- Chapter 4: V.R Venkateswaran, C.S Gokhale, M. Mangel and S. Eliassen. The effect of parental investment on immunocompetence and sexual immune dimorphism. *Manuscript under preparation*.

## Author contributions

- Chapter 2: CSG conceived the model. VRV and CSG developed the model. VRV performed the analyses.
- Chapter 3: OR and VRV devised the study. VRV and CSG conceived the model. VRV and CSG developed the model. VRV performed the analyses.
- Chapter 4: VRV devised the study. VRV, MM and SE conceived the model. VRV, MM,

CSG and SE developed the model. VRV performed the analyses.

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# Declaration

I hereby declare,

- i. that apart from my supervisor, Chaitanya S. Gokhale's guidance, the content and design of the thesis is my own work.
- ii. that the thesis has not been submitted partly or wholly as a part of a doctoral degree to any other examining body. Apart from the included published papers and the submitted papers no other part of the thesis has been published or submitted for publishing.
- iii. that the thesis has been prepared according to the rules of Good Scientific Practice of the German Research Foundation.
- iv. that I have not attempted and failed to obtain a doctoral degree prior to this thesis.



Vandana Revathi Venkateswaran

Plön, March 6, 2020



# Curriculum vitae

## PERSONAL DETAILS

First Name: Vandana

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## EDUCATIONAL DETAILS

YEAR	DEGREE	SCHOOL/UNIVERSITY
1997-2009	Secondary School Examination Certificate	DTEA Sr. Sec. School, New Delhi
2009-2011	Senior Secondary School Certificate	Sri Sankara Sr. Sec. School, Chennai
2011-2014	Bachelor of Science (Hons) Physics	University of Delhi
2014-2016	M.Sc in Nuclear Fusion, Engineering Physics	University of Gent (home institution)
2016-2019	Ph.D candidate (Kiel University)	MPI for Evolutionary Biology, Plön

## SELECTED WORKSHOPS AND CONFERENCES

- Was awarded a travel grant by IAST, Toulouse to attend the Toulouse Economics and Biology Workshop in June 2017 at the Institute for Advanced Study (IAST), Toulouse, France.
- Participant of the Santa Fe Institute's Complex Systems Summer School (10th June - 6th July at Santa Fe, NM, USA).
- Was selected to present a talk at the German Physics Society Conference's Evolutionary Game Theory session (April, 2019).
- Gave a talk on my work on sex differences in life-history interactions at ESEB (Congress of the European Society for Evolutionary Biology) 2019, Turku, Finland (August, 2019).

## WORKSHOPS ORGANIZED

- Organised the workshop "Talking Evolution" (at the Max Planck Institute for Evolutionary Biology, Germany from 26th September to the 28th September, 2018) that focused on discussing topics such as niche construction theory, extra-genetic inheritance, developmental bias, and phenotypic plasticity.
- Organised (and presented a talk) at the Aquavit Symposium 2017 at the Max Planck Institute for Evolutionary Biology, Plön.

## REVIEWING EXPERIENCE

*Proceedings of the Royal Society A: Mathematical, Physical and Engineering Sciences*