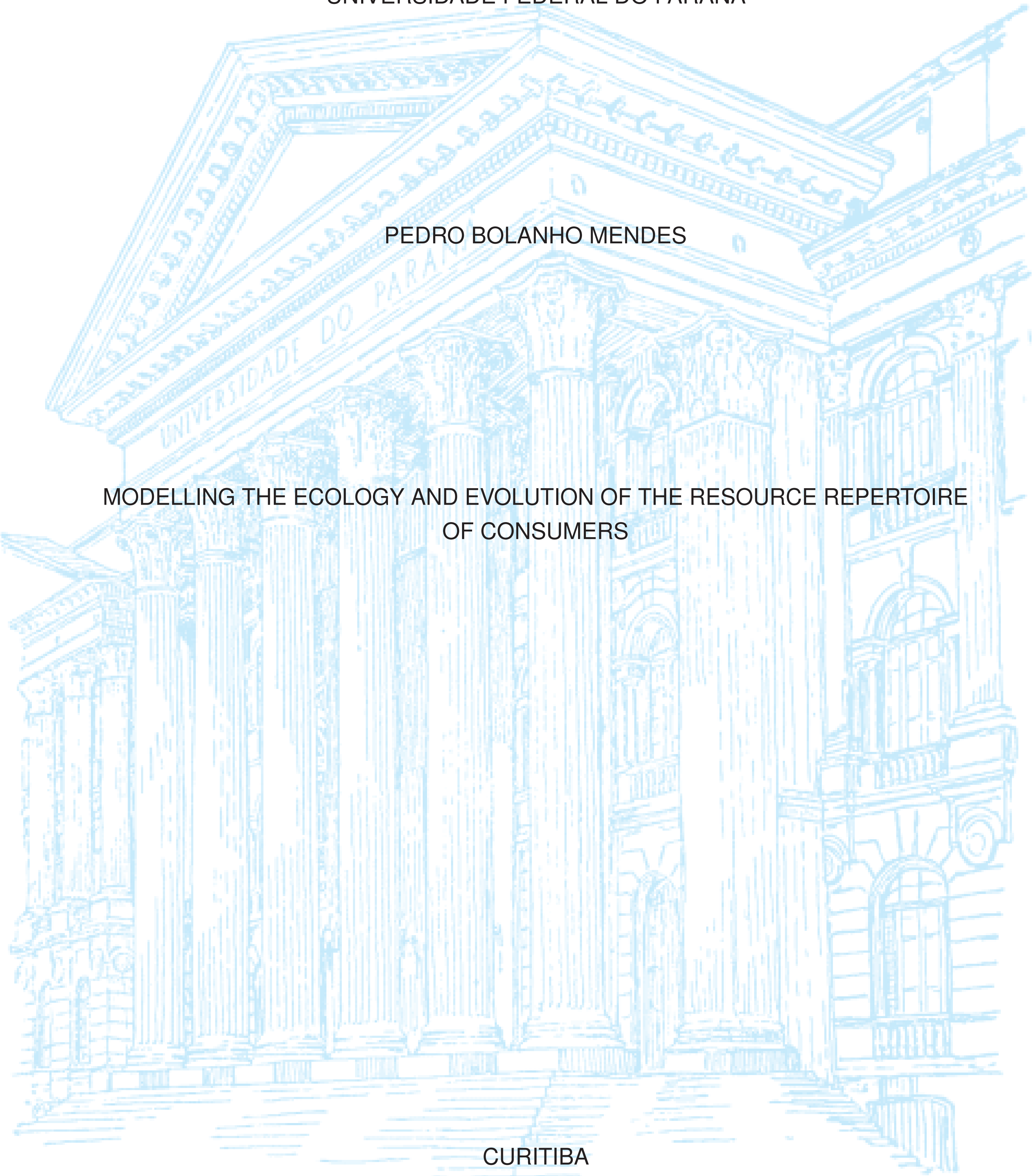

UNIVERSIDADE FEDERAL DO PARANÁ

PEDRO BOLANHO MENDES

MODELLING THE ECOLOGY AND EVOLUTION OF THE RESOURCE REPERTOIRE OF CONSUMERS

CURITIBA

2023

PEDRO BOLANHO MENDES

MODELLING THE ECOLOGY AND EVOLUTION OF THE RESOURCE REPERTOIRE OF CONSUMERS

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Orientador: Prof. Dr. Walter Antonio Pereira Boeger
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“The enigmatic oneness of the living organism, has as its corollary the fact that bodily traits are not merely physical, nor mental traits merely psychic. The continuity of nature knows nothing of those antithetical distinctions which the human intellect is forced to set up as aids to understanding.”

(Carl Jung)

RESUMO

A evolução do repertório de recursos dos consumidores desempenha um papel relevante em nossa compreensão sobre as populações naturais. Eventos como o surgimento de doenças infecciosas, a diversificação de patógenos e o resgate de populações ameaçadas estão relacionados aos mecanismos subjacentes às interações consumidor-recurso. A maioria dos estudos tem focado sua atenção em como as flutuações ambientais podem mudar o contexto ecológico dessas interações e, como consequência, promover mudanças no repertório de recursos. Por outro lado, fatores como a dinâmica interna das populações, *trade-offs* de história de vida e flexibilidade comportamental receberam relativamente menos atenção. Aqui, pretendemos abordar esses fatores para entender como eles se relacionam com a evolução de consumidores. Para tanto, exploramos um conjunto de modelos matemáticos que descrevem as interações hospedeiro-patógeno e predador-presa. Esta tese é composta por três capítulos. No primeiro, investigamos como a dinâmica interna de populações polimórficas de patógenos pode explicar pulsos de surtos de doenças em hospedeiros alternativos. No segundo capítulo, exploramos como as taxas de contato heterogêneas entre os hospedeiros se relacionam com a diversificação de patógenos com *trade-offs* de história de vida. No terceiro capítulo, avaliamos se a flexibilidade comportamental pode estabilizar a evolução da polifagia (generalismo). Nossos principais resultados são: 1) a dinâmica interna dos patógenos pode promover oscilações na composição das cepas. Essas oscilações mudam o repertório de hospedeiros de uma maneira que pode gerar pulsos de transbordamento de patógenos. 2) Por si só, taxas de contato heterogêneas são suficientes para diversificar o ambiente seletivo experimentado por patógenos. Essa diversificação ocorre mesmo quando o microambiente interno de diferentes hospedeiros é idêntico. 3) A dinâmica comportamental produz uma superfície de aptidão dinâmica que aprisiona a população de consumidores dentro de uma região de generalismo. Nossos resultados indicam que, dentro dessas regiões, o consumo generalista pode evoluir mesmo quando os especialistas têm maior aptidão. Discutimos a implicação de nossos resultados para a teoria atual e como eles podem melhorar nossa compreensão sobre ecologia e evolução.

Palavras-chaves: Ecologia teórica, teoria de jogos, dinâmica do replicador, redes, parasitas.

ABSTRACT

The evolution in the resource repertoire of consumers plays a relevant role in our understanding about natural populations. Events such as the emergence of infectious diseases, pathogen diversification and the rescue of endangered populations are related the mechanisms underlying the consumer-resource interactions. Most studies have focused their attention on how environmental fluctuations can shift the ecological context of these interactions, and, as a consequence, promote changes in the resource repertoire. Conversely, factors such as the internal population dynamics, life-history trade-offs and behavioral flexibility have received relatively less attention. Here, we aim to address these factors to understand how they relate to the evolution of pathogens and predators. To this end, we explore a set of mathematical models describing host-pathogen and predator-prey interactions. This dissertation is composed by three chapters. In the first, we investigated how the internal dynamics of polymorphic populations of pathogens may explain pulses of disease outbreaks in alternative hosts. In the second chapter, we explored how heterogeneous contact rates among hosts relate to the diversification of pathogens with life-history trade-offs. In the third chapter, we aimed to evaluate whether behavioral flexibility can stabilize the evolution of polyphagy (generalism). Our main results are: 1) the internal dynamics of pathogens can promote oscillations in the strain composition. These oscillations shifts the host range of pathogens in a manner that can generate pulses of pathogen spillover. 2) Alone, heterogeneous contact rates are sufficient to diversify the selective milieu experienced by pathogens. This diversification occurs even when the internal micro-environment of different hosts are identical. 3) Behavioral dynamics produces a dynamical fitness surface that traps consumer population within a region of generalism. Our results indicate that, within these regions, generalist consumption may evolve even when specialists have a greater fitness. We discuss the implication of our results to the current theory, and how they may enhance our understanding about ecology and evolution.

Key-words: Theoretical ecology, game theory, replicator dynamics, networks, parasites.

SUMÁRIO

1 INTRODUÇÃO GERAL

O principal objetivo da ciência é possibilitar a compreensão dos fenômenos naturais (McCain 2016). Para fazer isso, os cientistas são treinados para seguir um processo sistemático que começa com o reconhecimento de um determinado fenômeno e termina com algum tipo de conclusão sobre ele. De um modo geral, esse processo envolve dois tipos de atividades (Pickett; Kolasa; Jones, 2007). A primeira é a observação, pela qual as informações sobre o mundo externo podem ser reconhecidas pelo cientista. O segundo tipo de atividade é o raciocínio, que pode ser pensado como o processamento da informação que foi adquirida pela observação. Durante todo o processo de compreensão, essas atividades estão necessariamente entrelaçadas.

Enquanto as observações são responsáveis por adquirir informações de fenômenos naturais, o raciocínio é responsável por contextualizar essas informações para dar sentido a elas. Tal contextualização pode ser vista como a organização de uma construção conceitual (Pickett; Kolasa; Jones, 2007; Vellend, 2013; Jost, 2017). Isso não pode ser feito apenas pela lógica por pelo menos duas razões. A primeira é o fato de que, como membros da mesma espécie, nosso pensamento é influenciado por nossos órgãos sensoriais, assim como nossa percepção (Kokko, 2017). Isso faz com que a lógica evolua de acordo com nossas experiências, que são ampliadas pelos adventos da tecnologia e pelas discussões filosóficas e metafísicas (Menzel, 2011; Gershenson, 2018). A segunda razão é que os mecanismos causais dos fenômenos naturais são distribuídos de forma heterogênea no tempo e no espaço. Como consequência, as informações coletadas em um evento de observação muitas vezes são insuficientes para permitir que a lógica, sozinha, complete a atividade de raciocínio. Por esta razão, o exercício do raciocínio requer lógica associada ao conhecimento. É por isso que desenvolvemos teorias

Em ciência, uma teoria é um conjunto de proposições organizadas de uma maneira específica para que possam fornecer uma explicação para um determinado fenômeno (Craver, 2008). Nas palavras de Pickett, Kolasa & Jones (2007),

> "*Uma teoria é um sistema de construções conceituais que organiza e explica os fenômenos observáveis em um determinado domínio de interesse.*"
> -(Pickett; Kolasa; Jones, 2007).

Por *construções conceituais*, os autores se referem à natureza abstrata, sistematizada e contextual das teorias científicas. Isso porque os simbolismos usados dentro de uma teoria só têm sentido se interpretados ao longo da narrativa a partir da qual essa teoria foi construída (Otto; Rosales, 2020). Por exemplo, a teoria do forrageamento ótimo fornece uma construção conceitual que permite entender como os animais gastam seu tempo em busca de comida em função da distribuição de recursos (Pyke; Pulliam; Charnov, 1977). O significado da palavra "ótimo"nesta teoria refere-se à ideia de que consumidores otimizam seu forrageio de acordo com o custo-benefício de diferentes recursos (Okasha, 2011; Marquet *et al.*, 2014). Sem a perspectiva de que há uma quantidade a ser maximizada, a construção conceitual da teoria do forrageamento ótimo não tem significado preciso.

Uma maneira de formalizar teorias é traduzir suas preposições e conexões para uma linguagem matemática. Isso é comum em ciências exatas, como física e química, nas quais as representações matemáticas têm um poder preditivo forte e preciso (Griesemer, 2013). Isso significa que os modelos matemáticos têm uma conexão clara com a realidade e podem ser usados pragmaticamente. A biologia, no entanto, é uma ciência inexata. Assim, suas teorias e formulações matemáticas estão mais preocupadas com a compreensão do que com a realização de previsões precisas. Isso não quer dizer que não haja teorias ou formalizações matemáticas. Na verdade, a biologia tem um repertório elegante de teorias matematizadas (Pearson, 1903; Page; Nowak, 2002; Takeuchi; Iwasa; Sato, 2007; Otto; Day, 2007; Queller, 2017). Provavelmente, os campos matemáticos mais completos em biologia são a demografia e a evolução.

Demografia é o campo que estuda a estrutura, tamanho e dinâmica das populações (Xie, 2000). Apesar de ser muitas vezes considerada um ramo das ciências sociais (Sear, 2015), a demografia costuma ser abordada de forma interdisciplinar. Por exemplo, a famosa obra do economista Thomas Robert Malthus, ’Um ensaio sobre o princípio da população’, é o estudo dos padrões demográficos na perspectiva da economia e da filosofia. Em seu trabalho, Malthus argumenta que o crescimento geométrico da população humana junto com o crescimento aritmético da disponibilidade de recursos levaria a um colapso econômico (Malthus, 1798). Parte da fundamentação matemática da demografia inspirou o campo da ecologia teórica (May; Mclean, 2007; Case, 2000), que leva em conta qualquer fator que afete a persistência populacional. Estes vão desde fatores ecológicos como agressão/cooperação (White; Lewis; Murray, 1996; Teixeira Alves; Hilker, 2017; Berestycki; Zilio, 2019), fenologia (Bewick *et al.*, 2016) e histórias de vida (Roos, 2018); bem como elementos organísmicos como genótipos (Schreiber; Patel; Terhorst, 2016), fisiologia (Durinx; Metz; Meszéna, 2008) e cognição (Anderson, 1995; Olsson; Brown, 2010).

A teoria da evolução pode ser vista como uma teoria unificadora em biologia (Smocovitis, 1992). Essa propriedade unificadora pode ser justificada em duas perspectivas. A primeira é a ideia de que a diversidade de forma, função, comportamento e padrões de distribuição só pode ser compreendida à luz da evolução. Este é o argumento principal do artigo seminal de Theodozius Dobzhansky ’*Nada em biologia faz sentido exceto à luz da evolução*’ (Dobzhansky, 1973). Nessa perspectiva, a evolução unifica a biologia porque tem uma relação causal com os padrões observáveis da natureza. A segunda perspectiva na qual a evolução unifica a biologia é a observação de que os processos evolutivos são onipresentes nos sistemas biológicos. Independentemente da escala do sistema, os elementos da dinâmica evolutiva são intrínsecos à biologia (Smith, 2018; Kordium, 2021). Isso significa que qualquer sistema biológico evolui se o observarmos por um período de tempo suficiente. Isto é válido para sistemas em diferentes escalas biológicas. Nesta segunda perspectiva, a propriedade unificadora da evolução é fenomenológica.

A interação entre demografia e evolução é reconhecida desde os primórdios do pensamento evolutivo. Em ’*A Origem das Espécies*’, Charles Darwin e Alfred Wallace afirmam claramente que as ideias de Malthus justificam a ocorrência do que eles chamam de “*a luta pela existência*”:

> "*It is the doctrine of Malthus applied with manifold force to the whole animal and vegetable kingdoms; for in this case there can be no artificial increase of food, and no prudential restraint from marriage. Although some species may be now increasing, more or less rapidly, in numbers, all cannot do so, for the world would not hold them."* - (Darwin; Wallace, 1859) pp 64-65.

Darwin & Wallace (1859) sugerem que, no caso da população de animais e plantas, o única solução sugerida por Malthus (produção artificial de alimentos e reprodução controlada) não se aplicaria. Consequentemente, isso levaria a uma inevitável escassez de recursos, o que fundamenta os mecanismos que geram a luta pela existência:

> "*Hence, as more individuals are produced than can possibly survive, there must in every case be a struggle for existence, either one individual with another of the same species, or with the individuals of distinct species, or with the physical conditions of life*.- (Darwin; Wallace, 1859): page 64.

Dada essa conexão entre demografia e evolução, muitos esforços foram direcionados para entender as conexões lógicas entre esses campos. Estudos puramente teóricos também são uma contribuição necessária para o desenvolvimento de nossa compreensão. Estudar problemas que envolvem a matemática da interação entre entidades com significado biológico também é uma forma de ’fazer biologia’. A razão para isso é simplesmente porque a teoria pura também promove a compreensão. Além disso, tratamentos analíticos de modelos matemáticos que descrevem a dinâmica evolutiva podem nos ajudar a desenvolver modelos mais precisos. Isso não é um exercício matemático puro, mas sim um estudo teórico puro. A diferença é que o segundo requer conhecimento sobre o corpo teórico da biologia, enquanto o primeiro não.

Nesta tese, o nosso principal objetivo foi investigar alguns dos mecanismos teóricos subjacentes à evolução na gama de recursos. Especificamente, exploramos como interações intra-específicas, padrões de história de vida e dinâmicas comportamentais influenciam as trajetórias evolutivas em populações naturais de consumidores. Abordamos nosso objetivo por meio de modelos matemáticos que descrevem as interações entre os consumidores e seus recursos. Usamos ferramentas analíticas e simulações numéricas para analisar a dinâmica ecológica e evolutiva de nossos modelos. Todos os modelos analisados nesta tese são baseados em equações diferenciais e fortemente fundamentados na teoria das populações, ecologia teórica, epidemiologia e biologia evolutiva.

Esta tese é composta por três capítulos principais. No primeiro capítulo, intitulado *Game dynamics as a driver for pathogen spillover pulses*, focamos no papel da dinâmica interna dos patógenos (por exemplo, interações intra-específicas) para as oscilações da gama de hospedeiros do patógeno. Basicamente, demonstramos como as interações de cepa dentro de uma população de hospedeiro reservatório podem conduzir pulsos de transbordamento de patógenos. Tais eventos de transbordamento são representados por surtos de doenças na população de um hospedeiro acidental. Modelamos a interação entre cepas como uma rede de superinfecção e realizamos um conjunto de análises teóricas de jogos para o modelo. Este capítulo já foi publicado na revista *Ecological Modelling*.

No segundo capítulo, *Emerging patterns in heterogeneous contact networks*, estudamos o papel de redes de contato heterogêneas para a diversificação de patógenos com trade-offs de história de vida. Para tanto, desenvolvemos e analisamos um modelo de patógeno multi-hospedeiro no qual as populações hospedeiras são acopladas por uma rede de contato. Em seguida, analisamos a dinâmica adaptativa de patógenos residentes em diferentes populações hospedeiras.

No capítulo três, *The adaptive dynamics of a behaviorally flexible generalist*, avaliamos como a dinâmica comportamental pode moldar a superfície de aptidão de consumidores generalistas. Para abordar isso, analisamos uma versão modificada do clássico modelo predador-presa Lotka-Volterra, no qual os predadores (consumidores) podem ajustar seu esforço de forrageamento sobre as presas disponíveis (recursos). Esse ajuste depende da rentabilidade e abundância dos recursos no ecossistema. Em seguida, avaliamos a dinâmica adaptativa dos consumidores para entender quando a seleção dá vantagem aos generalistas.

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2 CAPÍTULO I: GAME DYNAMICS AS A DRIVER FOR PATHOGEN SPILLOVER PULSES

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Game dynamics as a driver for pathogen spillover pulses

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Multi-host pathogens

ABSTRACT

Understanding the underlying mechanisms for pathogen spillover is a main quest in epidemiology. Given the complexity of multi-host systems, several factors may play a relative role in spillover dynamics. A relatively less studied factor is the internal dynamics of pathogens that result from inter-strain competition. The competitive interactions among strains can cause deterministic changes in the population dynamics of pathogens. Such changes may affect the potential host-range of pathogens, influencing the probability of pathogen spillover. Here, our main goal is to show how an evolutionary game played by coexisting strains may induce pulses of pathogen spillover. To this end, we build a multi-host-multi-strain mean field model in which strains residing in a reservoir host have different capacities to explore an incidental host. We modelled inter-strain competition as a superinfection network and apply a game-theoretical analysis to it. The results indicate that game outcome depends on how reservoir hosts deal with the superinfection process (if superinfection is lethal, results in competitive exclusion or in coinfection). Our analysis suggests that game outcome can produce cycles in the strain composition that are cryptic in relation to the epidemiological dynamics of reservoirs (cycles in strain frequency composition cause small or no effect in the reservoir population). This suggests that relying solely on reservoir distribution and prevalence may subestimate the chance of pathogen spillover. Moreover, pulses of spillover can rely on the standing variation of pathogens, with no need for the emergence of novel mutations that increases pathogen host-range.

1. Introduction

In multihost systems, pathogens circulate from one species to another depending on the transmission route offered by each host (Woolhouse et al., 2001). As a consequence, disease outbreak in one species can be initiated by pathogens propagules that spill over from one host species to another. As in other studies (Daszak et al., 2000; Power and Mitchell, 2004; Borremans et al., 2019; Cross et al., 2019), we use the term "pathogen spillover" as a reference for this process. Given the complexity involved in multihost systems, there are many ecological factors driving events of spillover (Plowright et al., 2017). If the host–pathogen interaction produced by the spillover process is not stable, natural fluctuations in these ecological drivers may give rise to pulses of pathogen spillover (Plowright et al., 2016).

Host species that are able to stably sustain pathogen populations are called reservoir species (Haydon et al., 2002; Ashford, 2003). Given the stability of their interactions, reservoirs play a key role in promoting spillover events in multi-host communities (Wasik et al., 2019; Letko et al., 2020). In addition, many reservoir species seem to sustain high diversity of pathogen strains with no apparent change in the severity of the disease (Baum et al., 2012; Van Brussel and Holmes, 2022). A possible reason for this is that different strains may trigger the same immunological response in some hosts (Mandl et al., 2015). Another explanation is that reservoirs may be capable of producing a variety of antibody responses that confer host tolerance to a wide range of pathogens (Roy and Kirchner, 2000; Baum et al., 2012). Either way, the fact that different strains have similar epidemiology in reservoirs does not mean that their capacity to explore alternative hosts are equal (Morris et al., 2019). Instead, several studies suggest that increasing strain diversity in reservoirs may also increase the chance of spillover (Dennehy et al., 2010; Olival et al., 2017; Brooks et al., 2019; Letko et al., 2020; Remien and Nuismer, 2020; Feronato et al., 2021). This is because the region that a host occupies in the fitness landscape of a pathogen may vary depending on the pathogen strain (Smith-Tsurkan et al., 2010).

Besides being host-dependent, the fitness landscape of pathogens may also vary with the composition of their own population (Newton et al., 1998). This is because the within interactions among different strains may have alternative outcomes depending on the frequency composition (Kirkup and Riley, 2004; Andrade-Domínguez

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et al., 2014). Superinfection is one type of inter-strain competition in which a secondary strain infects an already infected host (Nowak and May, 1994). After superinfection, the outcome of the inter-strain competition may be either host death (Hamid et al., 2002), competitive exclusion from the within-host environment (Duncan et al., 2018), or strain coexistence (Leverich et al., 2008). Except in the case that is lethal to the host, superinfection can maintain pathogen diversity in the host population (Nowak and May, 1994; Mosquera and Adler, 1998). If strains have different capacities to explore novel hosts, the strain diversity maintained by the process of superinfection may increase the probability of pathogen spillover. If that is the case, spillover would be facilitated by the internal dynamics of pathogens, rather than by fluctuations on the external factors. However, we are unaware of studies about the role of superinfection to the dynamics of pathogen spillover.

Evolutionary game theory is one way to study the internal dynamics of populations. This is because it is centred in scenarios of intraspecific interactions among different phenotypes in a given population (Nowak and Sigmund, 2004). Depending on how interactions occur within the game, evolutionary dynamics yields to alternative population states (Kerr et al., 2002; Kirkup and Riley, 2004). Moreover, game dynamics may produce deterministic population cycles which maintains population polymorphism in a relatively predictable manner (Claussen and Traulsen, 2008; Laird, 2014; Szolnoki et al., 2014). In this sense, game dynamics may be a mechanism that bridges superinfection dynamics and events of pathogen spillover.

Here, our main goal is to show how game dynamics in inter-strain interactions can drive pulses of pathogen spillover. To this end, we build and analyse a set of mean field equations representing the epidemiological dynamics of a multi-host-multi-strain system in which different strains have different capacities to explore other hosts. Our analysis indicates that, if different strains have no differential effects to the reservoir host, the game dynamics may become cryptic in relation to the epidemiology of reservoirs (large fluctuations in the frequency composition of strains but small or absent fluctuations in the infected class of reservoir; see Mougi (2012) for an example of cryptic cycles in eco-evolutionary dynamics of predator–prey systems). The outcome of this game is mainly defined by whether inter-strain competition results in competitive exclusion, coinfection or host death. Then, pulses of pathogen spillover emerge whenever the game dynamics produces oscillations in the frequency composition of strains.

2. Models and analysis

Here, we present and analyse a multi-species version of the classical susceptible–infected–susceptible (SIS) model (Brauer et al., 2008). The model is composed by a set of ordinary differential equations (ODE's) describing the temporal change in the density of susceptible and infected classes during disease epidemics (the term 'density' is used as a reference for the fact that increasing the number of individuals also increases population crowding (Begon et al., 2007)). We consider two host species, namely a reservoir host and an incidental host (Ashford, 2003). The reservoir host sustains a polymorphic pathogen population composed by a diversity of strains. The frequency of each strain in the reservoir population depends on the inter-strain competition, which we describe as a superinfection evolutionary game. We assume that the incidental host cannot stably sustain the epidemics and thus outbreaks are always started by a spillover process. Only a particular strain is capable of infecting the incidental host. Consequently, the chance of spillover depends on the encounter between individuals in the susceptible class of the incidental host and reservoirs infected with that particular strain. The rate at which this encounter occurs is directly proportional to the frequency of that strain in the reservoir. Then, because the superinfection game affects the frequency composition in the pathogen population, patterns of spillover depends on the game outcome.

This section is organized as follows. First, we describe the epidemiological model in terms of their specific variables and parameters. Next, we show how the superinfection can be described by a competition network and how game dynamics can be derived from this network. Then, we use analytical treatments to derive the game outcomes and point out the conditions required for the emergence of cyclical outbreaks. We summarize our results with numerical simulations and their interpretations. The full R script used in our numerical analysis can be found in the Github repository (https://github.com/pbolanhom/gdsp).

2.1. Epidemiological dynamics

Consider a host community composed by a reservoir host and an incidental host. Hereafter, we use subscripts r and i to denote the variables and parameters of reservoir and incidental hosts, respectively. Let their population density be $N_r(t) = S_r(t) + I_r(t)$ and $N_i(t) = S_i(t) + I_i(t)$, where $S(t)$ and $I(t)$ are the densities of susceptible and infected classes in their populations. We assume that the reservoir host is initially infected with n pathogen strains $p = 1, \ldots, n$. Let these strains be contained in the set $\mathbb{P} = \{p\}_1^n$. Then, let $I_{r,p}$ be the population density of reservoirs that are infected with strain p. Thus, the overall density of the infected class of reservoir is the sum of the densities of classes infected by each strain, $I_r = \sum_p^n I_{r,p}$. The incidental host is only susceptible to infection by strain $p = 1$. For both hosts, Greek letters α, β and γ denote the epidemiological parameters for virulence, transmission rate and recovery rate, respectively. Assuming that hosts' populations grow logistically and transmission is direct (transmission depends on the direct contact between individuals), the system can be described by the following set of ODE's:

$$\frac{dS_r}{dt} = b_r N_r + \sum_{p=1}^{n} \gamma_{r,p} I_{r,p} - S_r \left[d_r + l_r N_r + \sum_{p=1}^{n} \beta_{r,p} I_{r,p} \right], \tag{1a}$$

$$\frac{dI_{r,p}}{dt} = I_{r,p} \left[\beta_{r,p} S_r - \Gamma_{r,p} + \sum_{q \in \mathbb{P}} I_{r,q} \left(\theta_{pq} - g\theta_{qp} \right) \right], \quad \forall p \in \mathbb{P}, \tag{1b}$$

$$\frac{dS_i}{dt} = b_i N_i + \gamma_i I_i - S_i \left[d_i + l_i N_i + \beta_i I_i + \beta_{ri} I_{r,1} \right], \tag{1c}$$

$$\frac{dI_i}{dt} = I_i \left[S_i \beta_i - \Gamma_i \right] + S_i \beta_{ri} I_{r,1}. \tag{1d}$$

Where symbol $\forall p \in \mathbb{P}$ reads 'for all p within set $\mathbb{P}$'. Parameters b and d stands for hosts' intrinsic born and death rates, respectively. l is the density-dependent mortality of hosts. This parameter reflects the intraspecific competition in hosts populations. Note that both infected and susceptible individuals contribute to the born rate (the term $bN = bS + bI$), which implies that infection does not impairs host reproduction. Also, note that newborn hosts are allocated to the susceptible class (the term bN only appears in Eqs. (1a) and (1c)), indicating that there is no vertical transmission of pathogens. $\Gamma = d + lN + \alpha + \gamma$ is the removal rate of the infected classes. The parameter β_{ri} is the rate of cross-transmission between the hosts. Note, however, that cross-transmission only occurs from reservoirs to incidental hosts and is thus unidirectional. Also, note that β_{ri} multiplies $I_{r,1}$ in Eqs. (1c) and (1d), which reflects the fact that I_i is only susceptible to strain $p = 1$. Fig. 1 depicts the motif describing the dynamics of model (1).

The term $\sum_{q \in \mathbb{P}} I_{r,q} \left(\theta_{pq} - g\theta_{qp} \right)$ in Eq. (1b) describes the superinfection process in the reservoir population (where index q represents another strain within the set $\mathbb{P}$ that is interacting with strain $p \in \mathbb{P}$). The upper limit in this summation term is omitted for simplicity (the subscript $q \in \mathbb{P}$ already indicates that the summation is over all the n elements of $\mathbb{P}$). Parameter θ_{pq} denotes the rate at which strain p superinfects another strain q, while θ_{qp} denotes the opposite direction. Parameter g represents a phenomenological class-transfer coefficient. This parameter describes how superinfected individuals move across the infected classes $I_{r,p}$. The range of this parameter is $0 < g < 2$ and different modes of transference emerge depending on its value interval. There are three possibilities. If $g = 1$, the superinfectant

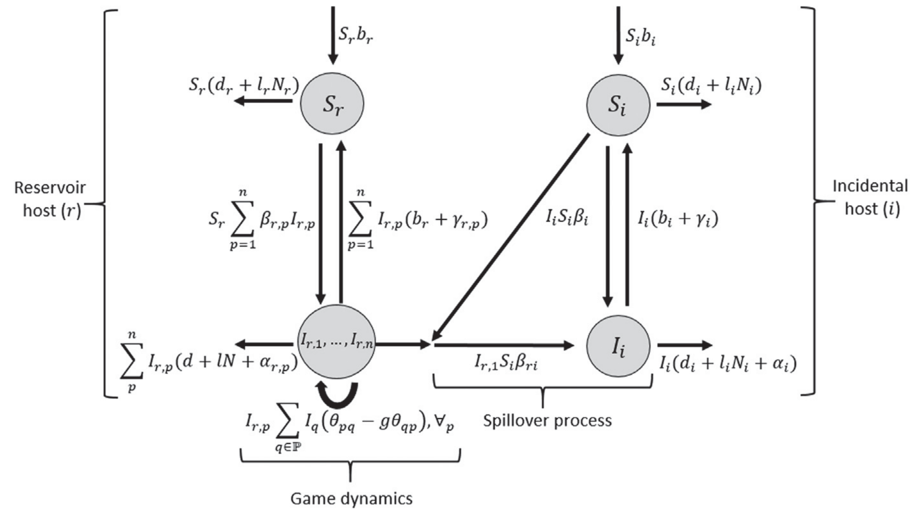

Fig. 1. The general motif describing the epidemiological model (1) (reservoir host populations in the left, and incidental host in the right) and n pathogen strains.

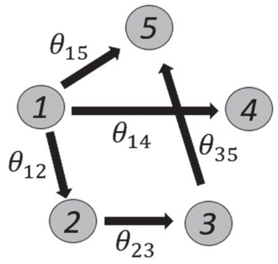

Fig. 2. An example of the superinfection network. Different nodes represent different strain and the arrows among the nodes denote the direction of the superinfection dynamics (e.g. strain 1 superinfects strain 2). The superinfection rate θ_{pq} is the weight of these links.

strain causes the competitive exclusion of the previous strain with no cost for the host. In this case, class transfer occurs immediately after superinfection and is the classical form of superinfection models (Nowak and May, 1994). In the case that $1 < g < 2$, the quantity of individuals that is removed from the superinfected class is larger than the quantity incremented in the superinfectant class. Thus, for this parameter interval, superinfection imposes a mortality cost for hosts. A comprehensive study on this type of process can be found in Mosquera and Adler (1998). The last case occurs when $0 < g < 1$. A g value within this interval means that less individuals leave one class than are incremented in the next. This means that some individuals remain coinfected with the two strains after superinfection.

2.1.1. The superinfection network

We use a network-theoretical approach to describe the superinfection dynamics. Consider a network in which nodes represent different strains and weighted directed links stands for the direction of superinfection. For example, if there is an arrow departing from node 1 towards node 2 it means that strain $p = 1$ superinfects strain $p = 2$. The weight of these links is denoted by the rate of superinfection θ_{pq}. Fig. 2 depicts an example of a superinfection network.

Mathematically, the link structure of any network can be described by its adjacency matrix, A. The entries of A are the weights of links between connected nodes (Newman, 2004). Here, the adjacency matrix of the superinfection network is

$$A = \begin{bmatrix} \theta_{11} & \cdots & \theta_{1n} \\ \vdots & \ddots & \vdots \\ \theta_{n1} & \cdots & \theta_{nn} \end{bmatrix}. \tag{2a}$$

Note that the entries of matrix A are in the form θ_{pq}. To get the values of θ_{qp} we simply evaluate the entries of the transposed matrix A^T. We define the following function for the value of θ_{pq}:

$$\theta_{pq} = \begin{cases} \beta_{r,p}\epsilon, & \text{if a link exists from } p \text{ to } q \\ 0, & \text{otherwise.} \end{cases} \tag{2b}$$

Where parameter ϵ is the rate to which superinfection occurs in relation to primary infections. This parameter have the same interpretation as parameter s in the seminal paper of Nowak and May (1994). Thus, if we build the superinfection network as if more virulent strains superinfect less virulent strains, and assume that $l_r = \gamma_{r,p} = 0$ (no density dependence and no recovery rate), and that $b_r N_r = B_r$ (constant immigration rate of uninfected individuals) Eqs. (1a) and (1b) goes back to exactly the same system developed and analysed in Nowak and May (1994).

2.2. Game dynamics

In this sub-section, we apply an evolutionary game analysis to the superinfection network. As depicted in the motif of Fig. 1, the game dynamics in model (1) is part of the dynamics of the infected reservoir class (Eq. (1b)). Thus, we start by showing how to evaluate any game dynamics in the superinfection network. Then we use this approach to analyse a rock–paper–scissors game and derive its outcome and relationships with pulses of pathogen spillover.

Evolutionary game theory describes the change in the frequency of players (or strategies) in a given population (Ray-mukherjee and Mukherjee, 2016; Cressman and Tao, 2014). The players of the superinfection game are the n strains $p = 1, \dots, n$ that coexist in the reservoir host. Let $f_{r,p}(t) = I_{r,p}(t)/I_r(t)$ be the frequency of each strain in the reservoir (where $I_r(t) = \sum_p^n I_{r,p}(t)$). Then, after taking the time derivative of $f_{r,p}(t)$ (see Appendix A), we get the following replicator equation describing the temporal dynamics of frequencies:

$$\frac{df_{r,p}}{dt} = f_{r,p}\left[\frac{dI_{r,p}}{I_{r,p}dt} - \frac{dI_r}{I_r dt}\right].$$

Where the first and second terms within the brackets of the above equation are the per capita growth rate of strain p and the per capita growth rate of the overall reservoir infected class. Under the assumption of well-mixed populations, which is an implicit assumption in mean field models (Morozov and Poggiale, 2012), these per capita growth rates are equivalent to the fitness of strains and of the overall population fitness (McPeek, 2017), respectively.

We assume that strains have no differential epidemiological parameters to the reservoir host. Thus, let the epidemiological parameters of

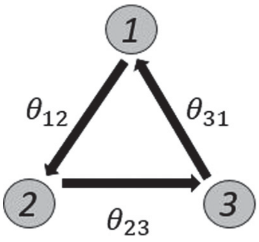

Fig. 3. The superinfection network with intransitive competition among $n = 3$ pathogen strains. Different nodes represent different strains and the arrows among the nodes denote the direction of the superinfection dynamics (e.g. strain 1 superinfects strain 2). θ_{pq} stands for the value of a given link. This graph has exactly the same configuration of the network describing the RPS game.

each strain in the reservoir be, for all $p \in \mathbb{P}$, $\beta_{r,p} = \beta_r$, $\alpha_{r,p} = \alpha_r$ and $\gamma_{r,p} = \gamma_r$. This yields to the following replicator equation:

$$\frac{df_{r,p}}{dt} = f_{r,p} I_r \left[\sum_{q\in\mathbb{P}} f_{r,q}\left(\theta_{pq} - g\theta_{qp}\right) - \sum_{p=1}^{n} f_{r,p} \sum_{q\in\mathbb{P}} f_{r,q}\left(\theta_{pq} - g\theta_{qp}\right) \right]. \tag{3}$$

The entire derivation of Eq. (3) can be found in Appendix A.

In evolutionary game theory, each player receives a payoff for playing the game with a specific opponent. Biologically, this payoff composes the reproductive success of those players (Nowak, 2006). If a strain p plays the superinfection game with another strain q, p receives a reproductive value that is a balance between how many infected individuals are incorporated into the class p and how many individuals leave the class p. Thus, the replicator Eq. (3) indicates that $\theta_{pq} - g\theta_{qp}$ is the payoff coefficient of any strain p playing the game with a given strain q. Remember that the values of θ_{pq} are the entries of superinfection network adjacency matrix A, while the values of θ_{qp} are the entries of A^T. Therefore, any game that can be described by the replicator Eq. (3) have the following payoff matrix:

$$\begin{aligned} P &= A - gA^T \\ &= \begin{bmatrix} \left(\theta_{11} - g\theta_{11}\right) & \cdots & \left(\theta_{1n} - g\theta_{n1}\right) \\ \vdots & \ddots & \vdots \\ \left(\theta_{n1} - g\theta_{1n}\right) & \cdots & \left(\theta_{nn} - g\theta_{nn}\right) \end{bmatrix}. \end{aligned} \tag{4}$$

Where P in Eq. (4) is a nxn matrix with the payoff coefficients $\theta_{pq} - g\theta_{qp}$ in its entries. The entries of each row represents the payoff that a player (in the row) get for playing the game with a given opponent. Eq. (4) also shows the connection between the superinfection network (which have the adjacency matrix A) and game dynamics. This connection allows us to analyse a variety evolutionary games among strains by drawing their network representation. To show this, we analyse the rock–paper–scissors (RPS) game (Zhou, 2016), a famous game that has relevant biological applications (Kerr et al., 2002; Kirkup and Riley, 2004; Szolnoki et al., 2014; Mark Rowland et al., 2017).

2.2.1. The RPS game

The RPS game describes a closed loop of intransitive competition (Gallien et al., 2017) in which rock beats paper, paper beats scissor, and scissor beats rock. To incorporate the RPS game in our model, we simply assume an analogous network structure (intransitive loop among $n = 3$ strains) to the superinfection network. This configuration is depicted in Fig. 3.

Then, we take the adjacency matrix of the network in Fig. 3 and use Eq. (4) to calculate the payoff matrix of the game. This yields to:

$$P = \begin{bmatrix} 0 & \theta_{12} & -g\theta_{31} \\ -g\theta_{12} & 0 & \theta_{23} \\ \theta_{31} & -g\theta_{23} & 0 \end{bmatrix}. \tag{5}$$

The row entries of matrix (5) denote the costs and benefits of a given strain (in each row), playing the game against their opponents (in each column). Positive values denote that the player/strain receives a benefit for playing against that opponent, while negative values denote a cost.

Generally speaking, there are two types of contests in this game, a tie or a win–lose contest (Zhou, 2016). The tie contest occurs when the two players pertain to the same strain. Because the payoff matrix (5) is hollow (the diagonal entries are zero), a tie outcome is always zero. The win–lose outcome of each player is the sum of all other entries of each row (except for the diagonal element). Here, this sum is always $\beta_r\epsilon(1 - g)$. Selection may give advantage to different population compositions depending on which contest provides a higher payoff. Whether a higher outcome comes from a tie or a win–lose contest depends on the signal of $(1 - g)$. We discuss the implications of this to our results further in the text.

2.2.2. Game outcome and its relationship with spillover pulses

The frequency composition $\vec{f}_r = (f_{r,1}, f_{r,2}, f_{r,3})$ of pathogen strains reaches its equilibrium at $\vec{f}_r = \hat{\vec{f}}_r$ (whenever the superscript ^ appears in this paper it indicates that the quantity below it is evaluated at the interior equilibrium of model (1)). It is known that the RPS game has a unique interior equilibrium that is given by the homogeneous composition $\hat{\vec{f}}_r = (1/3, 1/3, 1/3)$ (Nowak, 2006; Cressman and Tao, 2014; Zhou, 2016). Here, $\hat{\vec{f}}_r$ is always stable, which means that if the initial frequency composition is exactly $\hat{\vec{f}}_r$, the system remains at its interior equilibrium. If, however, the initial composition is heterogeneous $(f_{r,p} \neq f_{r,q} | f_{r,p} > 0, \forall p)$, system dynamics depends on the asymptotic stability of $\hat{\vec{f}}_r$. Following Nowak (2006), the asymptotic stability (and thus the game outcome) can be evaluated by analysing the signal of the determinant $\det(P)$ of the payoff matrix (5):

$$\det(P) = \theta_{12}\theta_{23}\theta_{31}\left(1 - g^3\right) \tag{6a}$$

$$= \left(\epsilon\beta_r\right)^3 \left(1 - g^3\right). \tag{6b}$$

Note that, because parameters ϵ and β_r must be positive numbers, the signal of $\det(P)$ depends only on the value of parameter g. If $0 < g < 1$, $\det(P)$ is positive, indicating that the interior equilibrium $\hat{\vec{f}}_r$ is asymptotically stable. This means that the trajectories of the replicator system (3) oscillate around the interior equilibrium until they converge to a stable state at $\hat{\vec{f}}_r$ (bottom row of Fig. 4a and Fig. 4b). In the case that $g = 1$, $\det(P) = 0$ and thus $\hat{\vec{f}}_r$ is asymptotically unstable. In this case, the replicator dynamics will give rise to stable limit cycles (middle row of Fig. 4a and Fig. 4b). Under the $g = 1$ scenario, the superinfection term vanishes from the dynamics of the overall infected class I_r (the term $(\theta_{pq} - g\theta_{qp})$ vanishes; see Eq. (A.5) in Appendix A). As a consequence, the strain dynamics becomes decoupled from the overall infected class of reservoirs. Hence, while the game dynamics produces large oscillations in the strain composition, there is no signature of these oscillations on the dynamics of I_r. We call these 'cryptic cycles' as a reference to the cryptic cycles in the eco-evolutionary dynamics of predator–prey interactions in which cycles in predator population produce almost no change in the population dynamics of preys (Mougi, 2012; Kinnison et al., 2015).

If, however, g falls within the interval $1 < g < 2$, the determinant $\det(P)$ is negative. As in the previous case, a negative determinants also indicates that the interior equilibrium is asymptotically unstable. However, the oscillations that emerge from system dynamics are non-periodic and unstable. The trajectories starting at any non-zero heterogeneous composition oscillate around $\hat{\vec{f}}_r$ in periods that continuously increases (top row of Fig. 4a and Fig. 4b). This type of dynamical behaviour is called heteroclinic cycles and is a known result in superinfection dynamics (Nowak and May, 1994). Theoretically, as noted in May and Leonard (2003), heteroclinic cycles never lead to the total extinction of either one or two players of the RPS game. Even for the case in which $t \rightarrow \infty$, the frequency of the non-dominant players will never reach zero without an arbitrary decision or a rounding

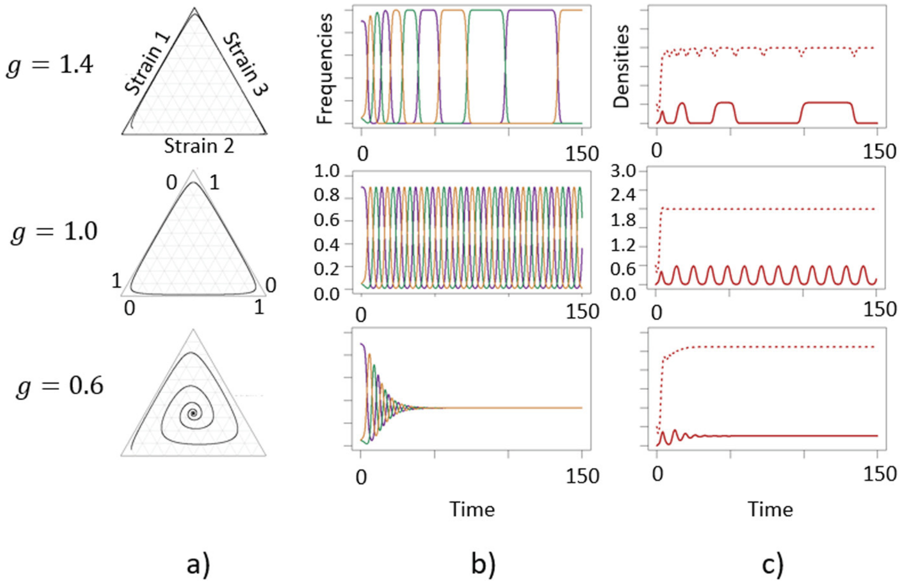

Fig. 4. The role of g in the game outcome. Parameter g (depicted at the left side of the panel) represents the manner at which infected hosts move across the infected classes of reservoirs, $I_{r,p}$ due to superinfection. Different rows in the panel shows the simulations for specific values of g. If $g > 1$ (top row), a fraction of superinfected individuals dies before changing their class. In this case, game outcome is depicted as heteroclinic cycles with increasing periods; If $g = 1$ (middle row), superinfection does not cause mortality and class change occurs immediately after superinfection. This game outcome produces limit cycles that stably oscillates around an unstable interior equilibrium; If $g < 1$ (bottom row), some individuals remain coinfected with two strains for a given fraction of time and thus remain in both classes simultaneously. In this case, the game outcome is a stable state. (a) The simplex projection produced by the game dynamics; (b) the dynamics of frequencies of different strains in the reservoir host described by the replicator Eq. (3) (the different of colours represent different strains); and (c) the dynamics of infected (red lines) classes of both reservoir (dashed lines) and incidental (solid lines) hosts. Initial conditions are $S_r = 0.5$, $I_r = 0.5$, $S_i = 1$, $I_i = 0$, $f_{r,1} = 0.9$, $f_{r,2} = 0.05$, $f_{r,3} = 0.05$. Common parameter values are: $b_r = 1.5$, $d_r = d_i = 0.2$, $l_r = l_i = 0.25$, $b_i = 2$, $\beta_{ri} = 0.1$, $\epsilon = 0.8$, $\alpha_{r,1} = \alpha_{r,2} = \alpha_{r,3} = 0.5$, $\beta_{r,1} = \beta_{r,2} = \beta_{r,3} = 1$, $\gamma_{r,1} = \gamma_{r,2} = \gamma_{r,3} = 0.5$, $\alpha_i = 1$, $\beta_i = 0.1$, $\gamma_i = 0.05$.

error (Nowak, 2006; Vandermeer, 2011). However, because these frequencies represent biological populations, it is plausible to expect lower limits of frequencies in which that population is driven to extinction (e.g. Allee effects, environmental fluctuations). For this reason, the biological interpretation of the heteroclinic cycles in Fig. 4 is that they eventually lead to monomorphic pathogen populations.

At the interior equilibrium, the density of the infected class of the incidental host is:

$$\hat{I}_i(\hat{S}_i) = \frac{\left[\hat{S}_i(\beta_i - l_i) - (d_i + \alpha_i + \gamma_i)\right] + \sqrt{[d_i + \alpha_i + \gamma_i - \hat{S}_i(\beta_i - l_i)]^2 + 4l_i\hat{S}_i\beta_{ri}I_r f_{r,1}}}{2l_i} \tag{7}$$

In the Appendix B, we show the calculation of Eq. (7) and that it is an increasing function of $f_{r,1}$. The incidental host may or may not stably sustain the epidemics in absence of the reservoir. This depends on the value of the reproductive ratio of the pathogen in the incidental host, $R_{0,i}$:

$$R_{0,i} = \frac{\beta_i \hat{S}_i}{\Gamma_i}. \tag{8}$$

The derivation of $R_{0,i}$ follows Lion and Metz (2018) and can be found in Appendix C. $R_{0,i}$ measures how many secondary infections is produced by each infected individual. The condition for stable maintenance of epidemics requires that $R_{0,i} > 1$. Here, we set our parameter values so that $R_{0,i} < 1$. Therefore, stable epidemics on the incidental host is necessarily maintained by spillover from reservoirs. This means that, if the equilibrium composition $\hat{\vec{f}}_r$ is not reached, the density of the infected class of the incidental host I_i follows the oscillatory trajectory of the replicator dynamics. This can be seen in Fig. 4c. Note how the density of infected incidental hosts (solid red lines in column c) abruptly increases following the increases in $f_{r,1}$ (purple lines in column b). This sudden increases are analogous to pulses of spillover since they produce outbreaks that are started (and maintained) by the reservoir population (Power and Mitchell, 2004). Because oscillations in the frequency composition are caused by the shape of the payoff matrix, game dynamics has an indirect causal effect in these pulses of pathogen spillover.

3. Discussion

Given the complexity of epidemiological systems containing multiple host species, several factors can be evoked in order to study causal pathways involved in spillover events. Here, we have shown that pulses of pathogen spillover may be a consequence of oscillations in the frequency composition of pathogen strains. These oscillations are deterministic and happens as the outcome of an evolutionary RPS game played by different strains that coexist in a reservoir population. Our analysis suggests that pulses of spillover does not necessarily depends on seasonal fluctuations of environmental conditions, nor on the emergence of new mutations that increases potential host-range of pathogens. Instead, they can be caused by the internal game dynamics of a pathogen population.

The outcome of the evolutionary RPS game was driven by parameter g. In terms of game theory, g defines whether the pathogen population mostly benefits from a tie or from a win–lose outcome (see Zhou (2016)). In the RPS game analysed here, the tie outcome is always 0, whereas the win–lose outcome is approximately $1 - g$. Our results suggests that selection gives advantages to alternative population compositions depending on the balance between these outcomes. If $g > 1$, then $1-g < 0$ and thus the tie outcome is greater. This means that the overall fitness of pathogen population is higher if the game is played between individuals of the same strain. In accordance, our simulations have shown that, in this case, system dynamics generates heteroclinic cycles which eventually produces monomorphic populations (Nowak, 2006). If, however, $g < 1$, $1 - g > 0$, indicating that a win–lose outcome is greater. This is coherent with our results in which trajectories converge towards a polymorphic composition with homogeneous frequency distribution. This homogeneous distribution minimizes the probability that the game will be played by two individuals of the same strain. Finally, if $g = 1$, $1 - g = 0$, both outcomes are equal. In this case, heterogeneous polymorphism is maintained by stable limit cycles. A possible interpretation is that the limit cycles alternates the dominance of each outcome.

In terms of epidemiology, g describes how inter-strain competition affects the superinfected hosts. This means that g is likely to vary with host traits since that tolerance to superinfection may be related to host physiology (Mosquera and Adler, 1998; Cronin et al., 2010; Portugal et al., 2011; Mandl et al., 2015; Irving et al., 2021). Therefore, host traits can modulate pathogen diversity even when different strains have no differential effect to the host. This has some interesting implications to the evolution of host–pathogen interactions. First, if hosts tolerate superinfection, pathogen diversification may occur even in pathogens considered highly fit to their hosts. The reason is that, if diversification occurs in pathogen traits that has no differential effect to the host, their lineage can be maintained by superinfection. Second, these lineages may have different capacities to explore novel host species through spillover processes (Mandl et al., 2015). Then, even if novel hosts are unable to sustain the interaction, parameter g of the novel host may give opportunity to the emergence of new variants in that host (Wasik et al., 2019). In this sense, spillover can be seen as a process by which pathogens explore their repertoire of potential host species. This idea suggests that the emergence of infectious diseases in novel host–pathogen interactions may actually be the rule rather than the exception in the evolutionary dynamics of pathogenic species.

We find that superinfection affects reservoir demography for the parameter interval $1 < g < 2$. This is not surprising since that, for this interval, inter-strain competition is lethal for some hosts. Consequently, host death causes small declines in the infected class even though strains have no differential virulence to the reservoir ($\alpha_{r,p}$). This happens because whenever a rare strain rises in frequency, it does it by superinfecting host individuals, causing their death. Then, because there is no vertical transmission in our model, newborn individuals are rapidly added to the susceptible class, maintaining the stability of the overall population density N_r. Whenever the rising strain is strain $p = 1$, these demographic effects on the reservoir matches the spillover pulses. Studies have shown that replacing infected by susceptible individuals may increase pathogen circulation (Peel et al., 2014; Field et al., 2015). As noted in Plowright et al. (2016), increased circulation may cause pathogen shedding to other populations, augmenting the chance of spillover. This implicates a causal relationship between cohort replenishment and pathogen spillover. In our model, however, that is not the case since replenishment and spillover are correlated process with no direct causality between them. In fact, both are driven by oscillations in the strain composition associated with the partial lethality of the inter-strain competition. That is not to say that such a causality does not exist. Rather, it is to point out that the internal dynamics of pathogens may naturally change its potential host-range even when the current host–pathogen system is considered stable.

Other studies have focused on the role of internal dynamics to the emergence of cyclic outbreaks. Although not the only relevant factor, cross-immunity seems to be a key element for the emergence of these cycles (Andreasen et al., 1997; Kamo and Sasaki, 2002; Zhang and Cao, 2014; Zhang, 2016). Cross-immunity occurs when infection by one strain confers total or partial host protection against other strains (Gill and Murphy, 1977). Often in superinfection models, cross-immunity is inversely proportional to parameter ϵ (Nowak and May, 1994). If $\epsilon = 0$, it means that cross-immunity is total and thus hosts infected with a given strain becomes immune to infection by another strain. As the value of ϵ increases, cross-immunity decreases and thus infected hosts are susceptible to secondary infections by other strains. In the scenario studied here, however, cyclic outbreaks occurs to the incidental host, which is susceptible to the infection by a single strain and thus cross-immunity does not play a role in this cycles. Instead, the cycles are caused by oscillations in the frequency of reservoirs infected by different strains.

The realism of the model analysed here can be enhanced in several ways. For example, our model only encompasses one reservoir species and one incidental host. In nature, however, host–pathogen interactions may involve several other species (Woolhouse et al., 2001; Legget et al., 2013). In a more complex environment, spillover may be followed by spillback events (Auld et al., 2017). One possible problem of this limitation is that, since our incidental host is only susceptible to strain $p = 1$, spillback events may cause an additional insertion of strain 1 back to its original host. Depending on which point of the dynamics this occurs, the other strains may be excluded from the reservoir host. In such a case, Strain $p = 1$ may become dominant in the reservoir and pulses would stop. Furthermore, this may occur with other strains and other host species. Thus, it would be interesting to evaluate our results in a more complex host community. This can be done by evaluating the reservoir system (Eqs. (1a) and (1b)) coupled with a more complex host community. Another possibility is to evaluate the result of other game dynamics. For example, the Hawk–Dove game (Sigmund and Nowak, 1999) can be used to study the evolution of mutualistic interactions among strains. This can be done by assuming a superinfection network which produces the payoff matrix that describes the Hawk–Dove game.

As we have shown, the internal dynamics of pathogens within reservoir populations may change the risk of spillover with no apparent signatures in the epidemiological dynamics of reservoirs. This means that changes in strain composition may occur cryptically in relation to the size of the infected class. This suggests that evaluating only the distribution of reservoirs and their seroprevalence may subestimate the probability of pathogen spillover. Furthermore, such changes in frequency composition can occur rapidly since they rely on the standing variation of pathogen population. In this paper, we have assumed that spillover does not results in host exploitation by the pathogen since that the incidental host cannot self-sustain the host–pathogen interaction. However, in terms of evolutionary trajectories, the spillover process can represent a window of opportunity for the pathogen to evolve in an alternative fitness landscape This can lead to a further diversification process (Braga et al., 2018). To conclude, we emphasize that changes in pathogen frequency distribution due to its internal game dynamics may be a key mechanism underlying the emergence of new host–pathogen interactions.

CRediT authorship contribution statement

Pedro B. Mendes: Conceptualization, Data curation, Formal analysis, Funding acquisition, Investigation, Methodology, Project administration, Resources, Software, Validation, Visualization, Writing – original draft, Writing – review & editing. **Walter A. Boeger:** Conceptualization, Funding acquisition, Investigation, Project administration, Resources, Supervision, Writing – original draft, Writing – review & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

No data was used for the research described in the article.

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Appendix A. Deriving the replicator equation

The frequency $f_{r,p}(t)$ of strain p in the reservoir population is

$$f_{r,p}(t) = \frac{I_{r,p}(t)}{I_r(t)}. \tag{A.1}$$

Where $I_r(t) = \sum_{p=1}^{n} I_{r,p}(t)$ is the overall density of the infected class in the reservoir population. To evaluate the temporal dynamics of $f_{r,p}(t)$ we apply the quotient rule from calculus, which yields to the following general form of the replicator equation:

$$\begin{aligned}\frac{d f_{r,p}}{dt} &= \frac{I_r \frac{dI_{r,p}}{dt} - I_{r,p}\frac{dI_r}{dt}}{I_r^2} \\ &= \frac{1}{I_r^2}\left[I_r \frac{dI_{r,p}}{dt} - I_{r,p}\frac{dI_r}{dt}\right] \\ &= \frac{I_{r,p}}{I_r}\left[\frac{1}{I_{r,p}}\frac{dI_{r,p}}{dt} - \frac{1}{I_r}\frac{dI_r}{dt}\right] \\ &= f_{r,p}\left[\frac{dI_{r,p}}{I_{r,p}dt} - \frac{dI_r}{I_r dt}\right].\end{aligned} \tag{A.2}$$

The first term between the brackets in the right-hand side of Eq. (A.2) can be obtained by dividing both sides of Eq. (1b) by $1/I_{r,p}$ and replacing the remaining $I_{r,p}$ by $I_r f_{r,p}$. Thus,

$$\begin{aligned}\frac{dI_{r,p}}{I_{r,p}dt} &= \beta_{r,p}S_r - \Gamma_{r,p} + \sum_{q\in\mathbb{P}} I_{r,q}\left(\theta_{pq} - g\theta_{qp}\right) \\ &= \beta_{r,p}S_r - \Gamma_{r,p} + I_r \sum_{q\in\mathbb{P}} f_{r,q}\left(\theta_{pq} - g\theta_{qp}\right).\end{aligned} \tag{A.3}$$

Since that $I_r = \sum_{p=1}^{n} I_{r,p}$, the second term between the brackets in the right-hand side of (A.2) can be obtained by adding the n equations $dI_{r,p}/I_{r,p}dt$:

$$\begin{aligned}\frac{dI_r}{I_r dt} &= \frac{1}{I_r}\sum_{p=1}^{n}\frac{dI_{r,p}}{dt} \\ &= \frac{1}{I_r}\sum_{p=1}^{n} I_{r,p}\left[\beta_{r,p}S_r - \Gamma_{r,p} + \sum_{q\in\mathbb{P}} I_{r,q}\left(\theta_{pq} - g\theta_{qp}\right)\right] \\ &= \frac{1}{I_r}\left[\sum_{p=1}^{n} I_{r,p}\beta_{r,p}S_r - \sum_{p=1}^{n} I_{r,p}\Gamma_{r,p} + \sum_{p=1}^{n} I_{r,p}\sum_{q\in\mathbb{P}} I_{r,q}\left(\theta_{pq} - g\theta_{qp}\right)\right] \\ &= \frac{1}{I_r}\left[I_r\sum_{p=1}^{n} f_{r,p}\beta_{r,p}S_r - I_r\sum_{p=1}^{n} f_{r,p}\Gamma_{r,p}\right. \\ &\left.+ I_r^2\sum_{p=1}^{n} f_{r,p}\sum_{q\in\mathbb{P}} f_{r,q}\left(\theta_{pq} - g\theta_{qp}\right)\right] \\ &= \frac{I_r}{I_r}\left[\sum_{p=1}^{n} f_{r,p}\beta_{r,p}S_r - \sum_{p=1}^{n} f_{r,p}\Gamma_{r,p} + I_r\sum_{p=1}^{n} f_{r,p}\sum_{q\in\mathbb{P}} f_{r,q}\left(\theta_{pq} - g\theta_{qp}\right)\right] \\ &= \sum_{p=1}^{n} f_{r,p}\beta_{r,p}S_r - \sum_{p=1}^{n} f_{r,p}\Gamma_{r,p} + I_r\sum_{p=1}^{n} f_{r,p}\sum_{q\in\mathbb{P}} f_{r,q}\left(\theta_{pq} - g\theta_{qp}\right)\end{aligned} \tag{A.4}$$

Under our assumption that strains do not differ in their epidemiological parameters in the reservoir population we have that $\alpha_{r,p} = \alpha_r$, $\beta_{r,p} = \beta_r$ and $\gamma_{r,p} = \gamma_r$, $\forall p \in \mathbb{P}$. This assumption leads to the following simplification of Eq. (A.4):

$$\begin{aligned}\frac{dI_r}{I_r dt} &= \sum_{p=1}^{n} f_{r,p}\beta_{r,p}S_r - \sum_{p=1}^{n} f_{r,p}\Gamma_{r,p} + I_r\sum_{p=1}^{n} f_{r,p}\sum_{q\in\mathbb{P}} f_{r,q}\left(\theta_{pq} - g\theta_{qp}\right) \\ &= \beta_{r,p}S_r\sum_{p=1}^{n} f_{r,p} - \Gamma_{r,p}\sum_{p=1}^{n} f_{r,p} + I_r\sum_{p=1}^{n} f_{r,p}\sum_{q\in\mathbb{P}} f_{r,q}\left(\theta_{pq} - g\theta_{qp}\right) \\ &= \beta_{r,p}S_r - \Gamma_{r,p} + I_r\sum_{p=1}^{n} f_{r,p}\sum_{q\in\mathbb{P}} f_{r,q}\left(\theta_{pq} - g\theta_{qp}\right)\end{aligned} \tag{A.5}$$

Then, replacing (A.5) and (A.3) into (A.2) and simplifying it yields to

$$\begin{aligned}\frac{df_{r,p}}{dt} &= f_{r,p}\left[\frac{dI_{r,p}}{I_{r,p}dt} - \frac{dI_r}{I_r dt}\right] \\ &= f_{r,p}\left[\beta_{r,p}S_r - \Gamma_{r,p} + I_r\sum_{q\in\mathbb{P}} f_{r,q}\left(\theta_{pq} - g\theta_{qp}\right) - \beta_{r,p}S_r + \Gamma_{r,p}\right. \\ &\left.- I_r\sum_{p=1}^{n} f_{r,p}\sum_{q\in\mathbb{P}} f_{r,q}\left(\theta_{pq} - g\theta_{qp}\right)\right] \\ &= f_{r,p}\left[I_r\sum_{q\in\mathbb{P}} f_{r,q}\left(\theta_{pq} - g\theta_{qp}\right) - I_r\sum_{p=1}^{n} f_{r,p}\sum_{q\in\mathbb{P}} f_{r,q}\left(\theta_{pq} - g\theta_{qp}\right)\right] \\ &= f_{r,p}I_r\left[\sum_{q\in\mathbb{P}} f_{r,q}\left(\theta_{pq} - g\theta_{qp}\right) - \sum_{p=1}^{n} f_{r,p}\sum_{q\in\mathbb{P}} f_{r,q}\left(\theta_{pq} - g\theta_{qp}\right)\right].\end{aligned} \tag{A.6}$$

Which is exactly the replicator equation depicted in Eq. (3).

Appendix B. The equilibrium density of the infected class of the incidental host population

Assuming that reservoir variables are constant, we can write the following model for the incidental host

$$\frac{dS_i}{dt} = b_i N_i + \gamma_i I_i - S_i\left[d_i + l_i N_i + \beta_i I_i + \beta_{ri} I_{r,1}\right] \tag{B.1}$$

$$\frac{dI_i}{dt} = I_i\left[S_i\beta_i - \left(d_i + l_i N_i + \alpha_i + \gamma_i\right)\right] + S_i\beta_{ri}I_{r,1}. \tag{B.2}$$

At the equilibrium, the per capita growth rate of I_i must be zero. Thus, setting $dI_i/I_i dt = 0$ and rearranging the terms leads to the following second degree polynomial:

$$\begin{aligned}\frac{dI_i}{I_i dt} &= S_i\beta_i - \left(d_i + l_i N_i + \alpha_i + \gamma_i\right) + \frac{S_i}{I_i}\beta_{ri}I_{r,1} = 0 \\ &= S_i\beta_i - \left(d_i + l_i S_i + l_i I_i + \alpha_i + \gamma_i\right) + \frac{S_i}{I_i}\beta_{ri}I_{r,1} = 0 \\ &= S_i\beta_i - \left(d_i + l_i S_i + l_i I_i + \alpha_i + \gamma_i\right) + \frac{S_i}{I_i}\beta_{ri}I_r f_{r,1} = 0 \\ &= I_i^2 l_i + I_i\left[d_i + \alpha_i + \gamma_i - S_i\left(\beta_i - l_i\right)\right] + \left(-S_i\beta_{ri}I_r f_{r,1}\right) = 0.\end{aligned} \tag{B.3}$$

Then, using the quadratic formula to solve (B.3) for I_i yields to

$$I_i = \frac{\left[S_i(\beta_i - l_i) - (d_i + \alpha_i + \gamma_i)\right] \pm \sqrt{[d_i + \alpha_i + \gamma_i - S_i(\beta_i - l_i)]^2 + 4 l_i S_i \beta_{ri} I_r f_{r,1}}}{2 l_i}. \tag{B.4}$$

Note that Eq. (B.4) has two versions. One in which the square root term is negative, and one in which it is positive. Our numerical simulations indicates that the interior equilibrium $\hat{I}_r$ is the positive version:

$$\hat{I}_i(\hat{S}_i, f_{r,1}) = \frac{\left[\hat{S}_i(\beta_i - l_i) - (d_i + \alpha_i + \gamma_i)\right] + \sqrt{[d_i + \alpha_i + \gamma_i - \hat{S}_i(\beta_i - l_i)]^2 + 4 l_i \hat{S}_i \beta_{ri} I_r f_{r,1}}}{2 l_i} \tag{B.5}$$

where the superscript ˆ denotes equilibrium values. Because the density of $\hat{S}_i$ is also evaluated at its equilibrium, it is also a function of the strain frequency $\hat{S}_i(f_{r,1})$. For this reason, it is not clear from Eq. (B.5) how $\hat{I}_i$ varies as a function of $f_{r,1}$. Fig. B.1 shows a numerical analysis to understand how $\hat{I}_i$ varies with $f_{r,1}$ for a range of transmission rates.

Note that, for all the evaluated values of β_i, $\hat{I}_i$ is an increasing function of $f_{r,1}$.

Appendix C. Deriving the reproductive ratio in Eq. (8)

The reproductive ratio $R_{0,i}$ can be derived from the per capita growth rate $\frac{dI_i}{I_i dt}$ (Lion and Metz, 2018). The reproductive ratio of pathogens is a quantity used to describe how many individuals is

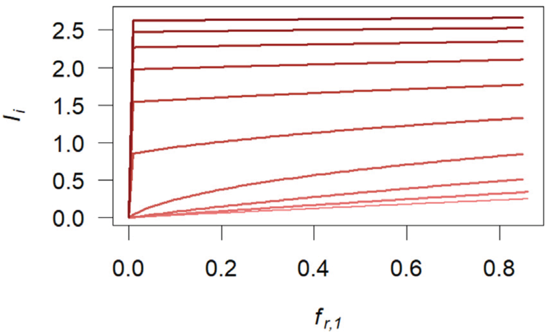

Fig. B.1. The relationship between $\hat{I}_i(\hat{S}_i)$ (in the y-axis) and $f_{r,1}$ (in the x-axis) for different β_i values (the different red lines). β_i values range from 0.1 (the line with the lightest shade of red) to 1 (the line with the darkest shade of red). Initial values are $S_i = 1$ and $I_i = 0$. Common parameter values are: $I_r = 1$, $l_i = 0.25$, $b_i = 2$, $d_i = 0.2$, $\beta_{ri} = 0.1$, $\alpha_i = 1$, $\gamma_i = 0.05$.

infected by each already infected individual within a given population. Eq. (1d) incorporates the infections caused by the reservoir host. In this sense, in order to do the derivation, we first need to set the cross-infection term to zero, $\beta_{ri} I_{r,1} = 0$. This leads to

$$\begin{aligned}\frac{dI_i}{I_i dt} &= S_i\beta_i - \left(d_i + l_i N_i + \alpha_i + \gamma_i\right). \\ &= S_i\beta_i - \Gamma_i \end{aligned} \tag{C.1}$$

Now, the above quantity describes the per capita growth rate of the incidental host in absence of the reservoir population. This quantity is the difference between the rate of which individuals that are inserted into the infected class $S_i\beta_i$, and the rate at which they are removed Γ_i. Then, following Lion and Metz (2018), the reproductive ratio can be written as the ratio between these terms:

$$R_{0,i} = \frac{S_i\beta_i}{\Gamma_i}. \tag{C.2}$$

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3 CAPÍTULO II: EMERGING PATTERNS IN HETEROGENEOUS CONTACT NETWORKS

Emerging patterns in heterogeneous contact networks

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Abstract: Several studies have shown that heterogeneous contact rates among groups of individuals strongly affect the epidemiological and evolutionary dynamics of pathogens. We propose a theoretical mechanism which we call the heterogeneous contact rates (HCR) hypothesis. This hypothesis states that heterogeneous contact rates, alone, are enough to diversify pathogen evolution. We develop a theoretical framework to study the epidemiological and evolutionary dynamics of pathogens that are inserted in communities composed of host populations. Our results show that the heterogeneity in the network describing the contact rates among host populations interplay with the density of susceptible classes in order to diversify the fitness surfaces of pathogens. This suggests that the subsequent evolutionary trajectory of pathogens occurring in multi-population systems can generate a diversity of pathogenic strains, even if hosts only vary in terms of their contact rates. Furthermore, we found a source-sink dynamics in which some host populations cannot self-sustain epidemics, but stability is reached by propagule pressure coming from other populations. Whether a population of host is a source or a sink depends on its position within the contact network. We discuss the implications of our analysis for understanding pathogen emergence.

Introduction

The rate at which disease epidemics spreads among host populations (or sub-populations) affects both the ecology and evolution of pathogens. For directed transmitted pathogens, transmission rates can be modeled as the product of three factors: the infectiousness of infected individuals; the susceptibility of susceptible individuals; and the contact rate between infected and susceptible individuals (McCallum et al. 2001). Host infectiousness and susceptibility relate to the capacities built by the evolutionary history of hosts and pathogens (Cronin et al. 2010, Park et al. 2018, Braga et al. 2018, Farrell & Davies 2019, Brooks et al. 2019). Contact rates, on the other hand, represent the ecological opportunity of pathogens to access hosts that are available within a given environment (Slingenbergh et al. 2004). This is because contact rates emerge from ecological processes that structure groups of individuals according to factors such as their social and/or spatial structure (Craft et al. 2011, Wang et al. 2013), diet (Simpson et al. 2012, Malmberg et al. 2021), behaviour (Borg et al. 2017) and microhabitat preference (Brooks et al. 2006); although contact may also vary with the organismic nature of the host-pathogen interaction (Franz et al. 2018).

Especially for pathogens with obligate parasitism, transmission dynamics is also a relevant property of their life-histories. Generally speaking, there are three main phases in the life-history of pathogens, namely, in the words of Smith (1904): "[1]*the entry into the host*, [2] *the temporary multiplication therein*, and lastly, [3] *the escape to another host*". While phase [1] and [3] are part of the transmission dynamics, phase [2] relates to the interactions in the within-host environment (Antia et al. 1996, Refardt 2011). Often, life-history traits are pleiotropic and may affect both pathogen transmission and its within-host performance (Templeton 1980, Ordon & Trewler 2000). For example, within-host replication rate may increase transmission but also increase host mortality (e.g. the virulence-transmission trade-off; Crossan et al. 2007*a*, Acevedo et al. 2019). Thus, depending on the relative contribution of transmission and within-host performance on fitness, pathogens may evolve towards alternative trajectories.

The theory of epidemiology predicts that the long-term persistence of pathogens depends on the number of secondary infections produced by infected individuals (André & Day 2005, Heffernan et al. 2005, Day et al. 2020). This means that transmission must occur at a greater rate than host death (Heesterbeek' & Dietz 1996, Diekmann et al. 1990). Since contact rates constrain transmission, they may alter the actual value of host survival for pathogen persistence. For example, if contacts are rare, opportunity to infect is scarce and thus the death of the current host may be the deadline of a pathogen lineage (although long-living propagules can maintain the risk of infection after host's death; Bonhoeffer et al. 1996). If, however, contacts are frequent, pathogen persistence can be secured before host succumbs and hence becomes less constrained by host survival (Combes 1997). This dependence of persistence on host survival influences several aspects of epidemics (e.g. prevalence, final epidemic size) and, as a consequence, influences the selective milieu experienced by pathogens (Lion & Metz 2018). If pathogen evolution is constrained by life-history trade-offs (André & Day 2005, Crossan et al. 2007*a*), selection may give advantage to alternative life-history strategies of pathogens depending on the current context. This suggests that, alone, heterogeneity in contact rates among hosts is enough to diversify evolutionary trajectories of pathogens. This is what we call the heterogeneous contact rate hypothesis (hereafter HCR hypothesis; see Box 1).

Here, our main goal is to investigate the theoretical foundations of the HCR hypothesis. Specifically, we aim to investigate the role of heterogeneous contact rates in the selective milieu experienced by pathogens. To this end, we build and analyze an epidemiological model in which host populations are coupled by a contact network. Then, we analyze the adaptive dynamics of a pathogen's quantitative trait to understand how the structure of the network shapes its evolution. Our analysis reveals that heterogeneous contacts have the potential to diversify pathogen evolution even when hosts are identical. Moreover, a source-sink epidemiological dynamics emerges from the network structure. We discuss the implications of our findings for the emergence of infectious diseases and how the HCR may help to understand the epidemiological consequences of habitat fragmentation.

Box 1: The *HCR* hypothesis

The mechanism of the *HCR* hypothesis is as follows. Ecological pleiotropy in life-history traits affects both pathogen transmission dynamics and its within-host performance. This generates life-history trade-offs that constrain pathogen evolution. Depending on the environmental context, these trade-offs will have different solutions. Then, selection gives advantage to phenotypes that are closer to the trade-off solution. Meanwhile, heterogeneous contact rates produce variability in the cost of host death. In other words, the value of host survival varies depending on the environmental context in which that host is embedded. As a consequence, the environmental context of pathogens also variegates and so does the solution of their life-history trade-offs. The result is then an increase in the diversity of strains with selective advantage.

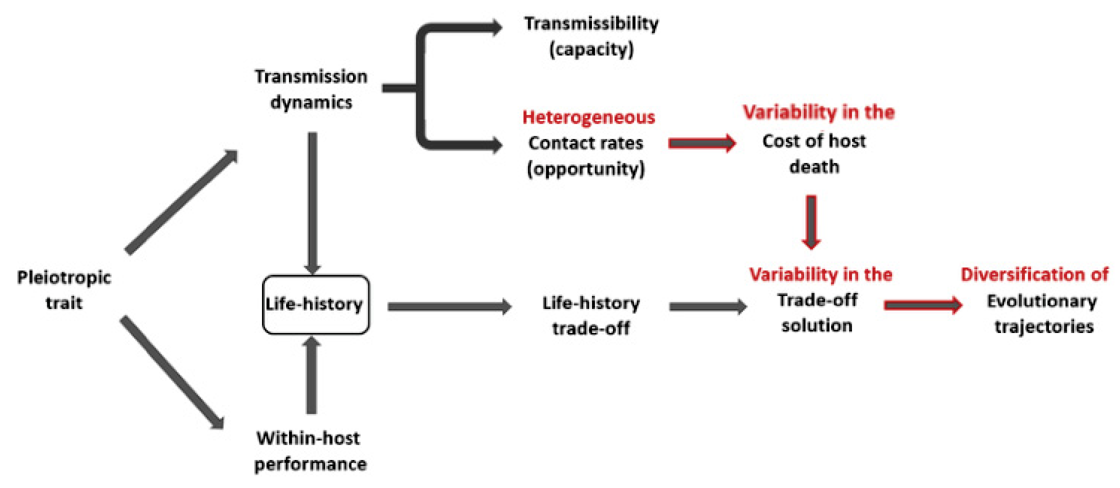


Figure B1.1: Illustration of the *HCR* hypothesis. Arrows with red contour mark the causal path of contact heterogeneity.

Models and analysis

Our analysis is based on a multi-host version of the susceptible-infected-susceptible (SIS) model (Brauer et al. 2008). The model is composed by a set of ordinary differential equations (ODE'S) describing the temporal dynamics in the densities of susceptible and infected classes of each host population. We assume that the dynamics of these populations are coupled by a contact network in which nodes represent host populations and links are the contact rates among them. In our system, the life-history trade-off of pathogens is represented by a correlation between transmission and virulence. This trade-off is produced by a pleiotropic trait that relates to pathogen's replication rate (Acevedo et al. 2019). To investigate the role of contact heterogeneity, we analyze how the network structure affects the evolution of the replication of pathogens. Except for their contact rates, all hosts have the same parameter values. In this sense, any effect on the stability of the system is due to the structure of the contact network.

This section is organized as follows. First we describe the SIS model with its variables and parameters. Then, we describe the life-history trade-off and the properties of the contact network. Next, we show how a source-sink transmission dynamics emerges from heterogeneous structure of the contact network. Finally, we perform an invasion analysis to evaluate the adaptive dynamics of pathogens and show how graph heterogeneity can lead to pathogen diversification. The full R script used in our numerical analysis is available at github.com/pbolanhom/hcr.

The model

Consider a set $\mathbb{H} = \{i\}_1^n$ containing n host populations with identity $i = 1, ..., n$. The overall population density of each host is $N_i = S_i + I_i$, where S_i and I_i are the density of the susceptible and infected classes in the ith host population. Let letters b and d be theirs intrinsic birth and death rates, respectively. We also assume a density-dependent mortality that decelerates population growth at a rate l. Infected individuals face an additional mortality which is quantified by pathogen's virulence $\alpha(z)$ (Thomas & Elkinton 2004). z is a pathogen's quantitative trait that defines its intrinsic within-host per capita growth rate (Alizon & van Baalen 2005). Hosts that survive infection recovers from disease at a rate γ. In the model, the transmission rate from a given host i to j (direction $i \longrightarrow j$) is the product $\beta_{ij} = \tau(z)\mathcal{C}_{ij}$. Where $\mathcal{C}_{ij}$ is the ijth contact rate (the rate at which an infected i effectively encounters a susceptible j). $\tau(z)$ is pathogen's transmissibility, calculated as the product between the ith infectivity and jth susceptibiility (O'Regan et al. 2015). We show the full derivation of the transmissibility function $\tau(z)$ in the Appendix I. Given these assumptions, the epidemiological dynamics of our system can be described by the following set of ODE's:

$$\frac{dS_i}{dt} = bN_i + \gamma I_i - S_i \left[d + lN_i + \tau(z) \sum_{j \in \mathbb{H}} \mathcal{C}_{ji} I_j \right], \tag{1a}$$

$$\frac{dI_i}{dt} = S_i \tau(z) \sum_{j \in \mathbb{H}} \mathcal{C}_{ji} I_j - I_i \left[d + lN_i + \alpha(z) + \gamma \right], \quad \forall i \in \mathbb{H} \tag{1b}$$

Notation $\forall i \in \mathbb{H}$ reads "for all hosts i within set $\mathbb{H}$". The term bN_i in Eq.1a means that newborns are always incorporated at the susceptible class. In this sense, model 1 assumes that there is no vertical transmission of pathogens. Furthermore, there are no other interaction between hosts except for their contact rate. Thus, in absence of pathogens, all host populations grow to their respective carrying capacities $K_i = (b-d)/l$.

The virulence-transmission trade-off

To incorporate the virulence-transmission trade-off, we follow Alizon et al. (2009) and assume that both $\alpha(z)$ and $\tau(z)$ are increasing functions of z:

$$\alpha(z) = \alpha_{(1)} z + \alpha_{(2)} z^2, \tag{2a}$$

$$\tau(z) = \tau_{(0)} + \tau_{(1)} z. \tag{2b}$$

Where $\alpha_{(1)}$ and $\alpha_{(2)}$ are scaling parameters that weigh the influence of trait z in the virulence. $\tau_{(0)}$ and $\tau_{(1)}$ are the intercept and the scaling parameter of transmissibility, respectively. These functions state that, while the transmissibility increases linearly with trait z, the virulence increases quadratically. In other words, increasing z increases both the rate at which pathogens are transmitted, and the rate at which pathogens causes the death of their hosts. In the appendix I, we detail the derivation Eq.2 from the within-host dynamics.

The contact network

Here, a contact network (CN) is a weighted directed graph in which nodes represent the populations of hosts and links are the contact rates between them. The fact that the graph is directed means that node degree (the number of links connected to it) is composed by the links arriving at the node (in-degree) and those departing from it (its out-degree), deg(i) =indeg(i)+outdeg(i). Here, we only analyze networks in which indeg(i) = outdeg(i) = deg(i)/2. These graphs are known as *balanced directed graphs* (Euler & Bridges 2011). Information about the structure of any graph can be found in its adjacency matrix (Newman 2010). The adjacency matrix of the contact network has the form:

$$\mathsf{A}_{\mathcal{C}} = \begin{bmatrix} \mathcal{C}_{11} & \cdots & \mathcal{C}_{1n} \\ \vdots & \ddots & \vdots \\ \mathcal{C}_{n1} & \cdots & \mathcal{C}_{nn} \end{bmatrix}. \tag{3}$$

Figure 1 depicts an example of the contact network.

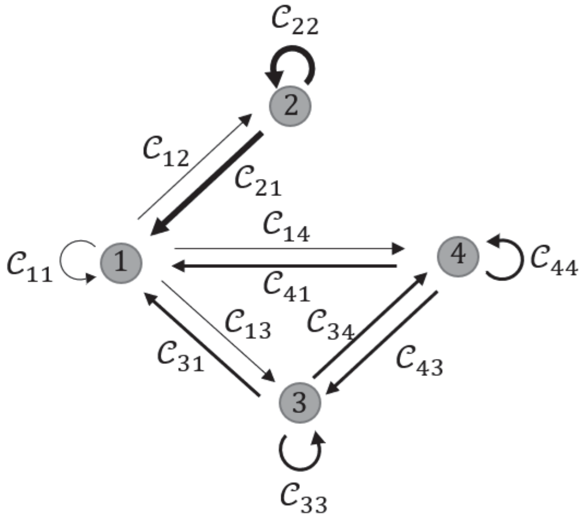


Figure 1: An illustration of a contact network describing the contact dynamics between $n = 4$ nodes. Each node represents a host population ($i = 1, 2, 3, 4$). Arrows are the directed weighted links indicating the direction $i \longrightarrow j$ of pathogen transmission. The $\mathcal{C}_{ij}$'s values are the weights of those links and stands for the contact rates of model 1. Note that increasing node neighborhood dilutes node contact rates (depicted as the thickness of the departing arrows). In the example, $\mathcal{C}_{11} = \mathcal{C}_{12} = \mathcal{C}_{13} = \mathcal{C}_{14} = 1/4$, $\mathcal{C}_{21} = \mathcal{C}_{22} = 1/2$, $\mathcal{C}_{31} = \mathcal{C}_{33} = \mathcal{C}_{34} = \mathcal{C}_{41} = \mathcal{C}_{43} = \mathcal{C}_{44} = 1/3$.

The weight of an edge departing from node i towards node j is given by the contact rate $\mathcal{C}_{ij}$, which is defined as:

$$\mathcal{C}_{ij} = \begin{cases} 1/k_i, & \text{if an edge exists from } i \text{ to } j \\ 0, & \text{otherwise} \end{cases}. \tag{4}$$

Letter $k_i = \text{outdeg}(i)$ in Eq.4 is the out-degree of node i (the amount of edges departing from i). Note that function 4 implies that $\sum_{j \in \mathbb{H}} \mathcal{C}_{ij} = 1$ for all nodes. Thus, function 4 assumes that the weight of an edge $i \longrightarrow j$ decreases as k_i increases. In other words, we assume that the rate at which an infected i effectively encounters j decreases as i effectively encounters other hosts. In a sense, this incorporates a dilution effect in transmission dynamics: increasing the diversity of edges dilutes the contact rate with the other nodes in the neighborhood. The neighborhood of each host is the sub-graph that is directly connect to it (Green et al. 2018). We use $\mathbb{N}_i$ as a reference for the ith neighborhood.

To measure graph heterogeneity, we use the index proposed in Estrada (2010). The index is based on the irregularity of each node (the squared difference between the inverse of the square root of the degree of two adjacent nodes). The irregularity measures how much two adjacent nodes differ in terms of their degree (Estrada 2010). The index has the form:

$$\rho(CN) = \sum_{i}^{n} \sum_{j}^{n} \left[\frac{1}{\sqrt{k_i}} - \frac{1}{\sqrt{k_j}} \right]^2 \tag{5}$$

Instead of using node degree, we use node out-degree k_i. However, since the graph is balanced, k_i is just $\deg(i)/2$. The heterogeneity index $\rho(CN)$ is 0 for homogeneous graphs and increases with node irregularity.

Pathogen fitness

In a single population epidemiological model, pathogen fitness can be evaluated by analyzing the per capita rate of increase in the infected class (Day & Gandon 2006). This yields to a quantity that increases with the density of susceptible class (see appendix II). In a multihost model such as eq.1, the ith per capita quantity $dI_i/I_i dt$ only varies with the density of the ith susceptible class, S_i:

$$\frac{dI_i}{I_i dt} = \frac{S_i}{I_i} \tau(z) \sum_{j \in \mathbb{H}} \mathcal{C}_{ji} I_j - \left[d + l N_i + \alpha(z) + \gamma \right].$$

This cannot be regarded as pathogen fitness because we know that the susceptible class of any other population in the neighborhood of node i, $\mathbb{N}_i$, must affect the fitness of pathogens within host i (Crossan et al. 2007*b*). To incorporate this, we derive pathogen fitness as follows.

Consider an overall infected class with density $I = \sum_{i \in \mathbb{H}} I_i$. The temporal dynamics of I is then given by

$$\frac{dI}{dt} = \sum_{i \in \mathbb{H}} \frac{dI_i}{dt} \tag{6a}$$

$$= \tau(z) \sum_{i \in \mathbb{H}} S_i \sum_{j \in \mathbb{H}} \mathcal{C}_{ji} I_j - \sum_{i \in \mathbb{H}} I_i \left[d + lN_i + \alpha(z) + \gamma\right] \tag{6b}$$

$$= \tau(z) \sum_{i \in \mathbb{H}} I_i \sum_{j \in \mathbb{H}} \mathcal{C}_{ij} S_j - \sum_{i \in \mathbb{H}} I_i \left[d + lN_i + \alpha(z) + \gamma\right] \tag{6c}$$

$$= \sum_{i \in \mathbb{H}} I_i \left[\tau(z) \sum_{j \in \mathbb{H}} \mathcal{C}_{ij} S_j - (d + lN_i + \alpha(z) + \gamma)\right] \tag{6d}$$

Note that, in equation 6b the contact rate $\mathcal{C}_{ji}$ describes the contact rate in the direction $j \longrightarrow i$. Then, after rearranging the terms in 6c, it appears in the form $\mathcal{C}_{ij}$, which describes contacts in the direction $i \longrightarrow j$. We can also write the above equation in terms of its per capita rate:

$$\frac{dI}{Idt} = \sum_{i \in \mathbb{H}} q_i \left[\tau(z) \sum_{j \in \mathbb{H}} \mathcal{C}_{ij} S_j - (d + lN_i + \alpha(z) + \gamma)\right] \tag{6e}$$

Where $q_i = I_i/I$ is the relative abundance host i to the overall infected class I. The term between the brackets in equation 6e measures the contribution of the ith infected class to the per capita rate of increase in the overall infected class I. Here, we argue that this term can represent pathogen fitness because it quantifies the reproductive success of pathogens in a given host i accounting for the fact that pathogen's offspring may develop in another host j. Thus, replacing $\mathcal{C}_{ij}$ by $1/k_i$ (according to equation 4) we get the fitness F_i of pathogens occupying host i:

$$F_i(z, \mathbf{S}, N_i) = \tau(z) \frac{1}{k_i} \sum_{j \in \mathbb{N}_i} S_j - \left[d + lN_i + \alpha(z) + \gamma\right], \quad \forall i \in \mathbb{H}. \tag{7}$$

Where $\mathbf{S}$ in the left-hand side of equation 7 is a vector with the densities $\mathbf{S} = (S_1, ..., S_n)^T$. Note that, replacing $\mathcal{C}_{ij}$ by $1/k_i$ requires writing the summation term over the neighborhood $\mathbb{N}_i$, instead of over the overall pool of hosts $\mathbb{H}$ as in eq.6e.

The emergence of a source-sink epidemiological network

At the **appendix II** we show that, for small graphs, the interior equilibrium of model 1 is approximately the equilibrium for homogeneous graphs:

$$\hat{I}_i(\hat{S}_i, z) \approx \frac{\hat{S}_i(z)[\tau(z)\sum_{j\in\mathbb{H}} \mathcal{C}_{ji} - l] - [d + \alpha(z) + \gamma]}{l}, \tag{8a}$$

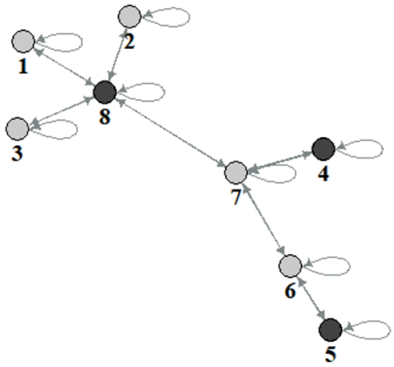
$$\hat{S}_i(z) \approx \frac{b + d + \alpha(z)[1 + l] + 2\gamma}{2\tau(z)\sum_{j\in\mathbb{H}} \mathcal{C}_{ji}} - \frac{\sqrt{\Delta_i}}{2\left[\tau(z)\sum_{j\in\mathbb{H}} \mathcal{C}_{ji}\right]^2} \tag{8b}$$

With $\Delta_i = \tau(z)\sum_{j\in\mathbb{H}} \mathcal{C}_{ji}\left[\tau(z)\sum_{j\in\mathbb{H}} \mathcal{C}_{ji}[r - \alpha(z)]^2 + 2\alpha(z)l[b + d + \alpha(z) + 2\gamma]\right] + \alpha(z)^2 l^2$. Superscript $\hat{}$ stands for equilibrium densities. For homogeneous graphs, all nodes have the same $\sum_j^n \mathcal{C}_{ji} = 1$. Then, the interior equilibrium becomes the same as if only loops were allowed ($\mathcal{C}_{ii} = 1$), which is exactly the interior equilibrium of single-population models (appendix II). Thus, if contacts are homogeneous, the interior equilibrium of system 1 can be fully described by the equilibrium densities of a single-population model.

In heterogeneous graphs, nodes vary in degree. This causes the sums $\sum_j^n \mathcal{C}_{ji}$ in 8 to vary depending on node i, variegating in the interior equilibrium of each host. However, the interior equilibrium of small heterogeneous graphs, eq.8 yields a value close to the numerical simulations. As the graph increases in number of nodes, the effect of network structure also increases. At this point, eq.8 no longer describes its interior equilibrium. We go back to this further in the text.

As a consequence of contact heterogeneity, the fitness of pathogens $F_i(z, \hat{\mathbf{S}}, \hat{N}_i)$ also varies. Still, the equality $\sum_i q_i F_i(z, \hat{\mathbf{S}}, \hat{N}_i) = 0$ must be satisfied at a stable state. Since that q_i is a proportion and thus is constrained within the interval $0 \leq q_i \leq 1$, only a summation of positives and negative values of the $F_i(z, \hat{\mathbf{S}}, \hat{N}_i)$ terms can result in zero. In other words, under heterogeneous contact networks (in which node neighborhoods are variable), the interior equilibrium of system 1 produces fitness values that must vary from negative to positive values, depending on the node.

Nodes in which $F_i(z, \hat{\mathbf{S}}, \hat{N}_i) \geq 0$ represent host populations that self-sustain the infection within the network context. In analogy to the theory of metapopulations, these nodes act as "sources" of pathogen transmission because they can export propagules to the remaining of the network. Nodes in which $F_i(z, \hat{\mathbf{S}}, \hat{N}_i) < 0$, pathogen's population cannot be sustained and thus are called ”sink” nodes. In these sink nodes, stable epidemics are possible but are necessarily maintained by propagules that are imported from the source nodes. Figure 2 shows the emergence of the source-sink dynamics. Note that, although the approximation 8 differs from the numerical simulations, both produced the same source-sink pattern.



i	k_i	$\sum_{j=1}^{n} \mathcal{C}_{ji}$	$\hat{S}_i$(sim/calc)	$\hat{q}_i$	$F_i(z, \hat{\mathbf{S}}, \hat{N}_i)$
1	2	0.700	4.979/4.867	0.123	−0.249
2	2	0.700	4.979/4.867	0.123	−0.249
3	2	0.700	4.979/4.867	0.123	−0.249
4	2	0.750	4.441/4.479	0.126	0.089
5	2	0.833	3.926/3.949	0.129	0.141
6	3	1.083	2.930/2.898	0.131	−0.450
7	4	1.283	2.444/2.381	0.129	−0.273
8	5	1.950	1.369/1.478	0.116	0.900

a) b)

Figure 2: The emergence of a source-sink epidemiological dynamics. a) The contact network with $n = 8$ nodes. Dark and light colored nodes are the source and sink host populations, respectively. Numbers in each node stands for their identity i. b) Table with the information referent to the aside network. i is host identity, k_i is the ith out-degree, $\sum_j^n \mathcal{C}_{ji}$ is the weighted sum of the ith in-degree, S_i is the susceptible class density (simulated/calculated with approximation 8); $q_i = I_i/I$ is the ith relative abundance within the overall infected class; and $F_i(z, \hat{\mathbf{S}}, \hat{N}_i)$ is pathogen fitness. Common parameters are: $b = 2$, $d = 0.2$, $l = 0.15$, $\gamma = 0.2$, $z = 2.533773$, $\alpha_{(1)} = 0.248$, $\alpha_{(2)} = 0.2$, $\tau_{(0)} = 0.004$ and $\tau_{(1)} = 0.4$. Initial conditions: $S_i = 0.5$, $I_i = 0.5$.

A general interpretation of the source-sink pattern

For the parameter values that we have used, all nodes can sustain infection when disconnected from the contact network. This indicates that the source-sink pattern is an emergent property of the graph structure. As we have shown, source nodes have a positive fitness, while sink nodes have negative fitness. However, the fitness function also incorporate information about other properties of the system besides the network structure. In appendix III we show that the actual effect of the graph structure can be written as:

$$\mathcal{M}_i = \frac{1}{k_i} \sum_{j \in \mathbb{N}_i} \frac{1}{\sum_{g \in \mathbb{N}_j} \mathcal{C}_{gj}}. \tag{9a}$$

Where g in $\sum_{g \in \mathbb{N}_j}$ stands for the nodes within the jth neighborhood. We call this quantity $\mathcal{M}_i$ to emphasize that is relates to the meta-population (or source-sink) state of node i. A node will be a source (positive fitness) whenever the following criteria is true:

$$\mathcal{M}_i \geq 1. \tag{9b}$$

If the above inequality is reversed, pathogen fitness is negative and thus represents a sink. Equation eq.9a points out how the source-sink dynamics is an emergent property of the graph structure. This is because is clarifies that the value of $\mathcal{M}_i$ varies not only with its own out-degree, but also with the out degree of the of the nodes g within the neighborhood of nodes j within the neighborhood i ($\mathcal{C}_{gj} = 1/k_g$). Moreover, $\mathcal{M}_i$ only incorporate information about the network, being independent of system parameters. Still, it can predict which nodes behave as a source of pathogens, and which behaves as a sink. This emphasizes the emergent property of the source-sink pattern.

Evolutionary dynamics

Here, we apply the theoretical framework of the Adaptive Dynamics toolbox (Dieckmann & Law 1996, Dieckmann 2002). Let $z_{m,i}$ be the trait value of a rare mutant strain that emerges in host i. If we assume that this strain emerges after system 1 reaches its interior equilibrium, its increase in frequency depends on its ability to invade the environment promoted by the resident strain with trait z (the stable state of 1 given the trait value z of the resident pathogen). This can be evaluated by analyzing the ith invasion fitness $\mathcal{F}_i(z, z_{m,i})$ of the rare variant. Based on the resident fitness 7 and following Brannstrom et al. (2013), the invasion fitness of our model is:

$$\mathcal{F}_i(z, z_{m,i}) = \tau(z_{m,i}) \sum_{j \in \mathbb{H}} \mathcal{C}_{ij} \hat{S}_j(z) - \left[d + l\hat{N}_i(z) + \alpha(z_{m,i}) + \gamma \right]. \tag{10}$$

Note that, in 10, both transmissibility and virulence are functions of the trait value of the mutant strain, $\tau(z_{m,i})$ and $\alpha(z_{m,i})$. These functions have the same format as in 2 but are evaluated for $z = z_{m,i}$. Then, the shape, strength and direction of the selective pressures acting over the emergence of the rare strain can be captured by its selection gradient, which describes the curvature of the fitness surface for a given trait value (Lehtonen 2017, Lion 2017). The selection gradient is calculated as the partial derivative of the invasion fitness with respect to the mutant trait $z_{m,i}$:

$$\frac{\partial \mathcal{F}_i(z, z_{m,i})}{\partial z_{m,i}} = \tau_{(1)} \sum_{j \in \mathbb{H}} \mathcal{C}_{ij} \hat{S}_j(z) - 2\alpha_{(2)} z_{m,i} - \alpha_{(1)}. \tag{11a}$$

There is a singular point $\hat{z}_i$ for which the natural selection vanishes:

$$\left. \frac{\partial \mathcal{F}_i(z, z_{m,i})}{\partial z_{m,i}} \right|_{z_{m,i} = \hat{z}_i} = 0. \tag{11b}$$

and solving 11b for the $\hat{z}_i$ reveals that the singular point is given by the quantity

$$\hat{z}_i = \frac{\tau_{(1)} \sum_{j \in \mathbb{H}} \mathcal{C}_{ij} \hat{S}_j(z) - \alpha_{(1)}}{2\alpha_{(2)}}. \tag{12}$$

Then, because the second derivative,

$$\left. \frac{\partial^2 \mathcal{F}_i(z, z_{m,i})}{\partial z_{m,i}^2} \right|_{z_{m,i} = z = \hat{z}_i} = -2\alpha_{(2)}, \tag{13}$$

is always negative, we know that the singular point 12 is an evolutionary stable strategy (ESS).

Following Doebeli & Ispolatov (2010), the singular point is also a convergent stable strategy (attracts trajectories towards the ESS) if:

$$\left. \frac{\partial^2 \mathcal{F}_i(z, z_{m,i})}{\partial z \partial z_{m,i}} \right|_{z_{m,i} = z = \hat{z}_i} + \left. \frac{\partial^2 \mathcal{F}_i(z, z_{m,i})}{\partial z_{m,i}^2} \right|_{z_{m,i} = z = \hat{z}_i} < 0. \tag{14a}$$

Which, in our model, yields

$$\tau_{(1)} \sum_{j \in \mathbb{H}} \mathcal{C}_{ij} \frac{\partial \hat{S}_i(z)}{\partial z} - 2\alpha_{(2)} < 0. \tag{14b}$$

The differential invasibility of rare variants

The singular point in equation 12 can be rewritten as

$$\hat{z}_i = \frac{\tau_{(1)} \frac{1}{k_i} \sum_{j \in \mathbb{N}_i} \hat{S}_j(z, \mathbb{N}_j) - \alpha_{(1)}}{2\alpha_{(2)}}. \tag{15}$$

This format shows that, $\hat{z}_i$ is indirectly affected by the contact network. This effect is indirect because if all S_j values in eq.15 are forced to be identical, there is no diversification in despite of graph heterogeneity. This happens because, in case, the summation term becomes $\frac{1}{k_i} \sum_{j \in \mathbb{N}_i} \hat{S}_j(z, \mathbb{N}_j) = \frac{1}{k_i} k_i S_i = S_i$. If, on the other hand, the graph is homogeneous but susceptible classes differ, diversification occurs because the sum $\frac{1}{k_i} \sum_{j \in \mathbb{N}_i} \hat{S}_j(z, \mathbb{N}_j)$ varies with node position. In this sense, the evolutionary effect of heterogeneity depends on the feedback between the contact network and the actual availability of hosts.

In system 1, however, heterogeneous graphs necessarily produce variability in the susceptible classes $\hat{S}_i(z)$ and thus the sum $\frac{1}{k_i} \sum_j^n \mathcal{C}_{ij} \hat{S}_j(z)$ varies with node position. In this sense, if there are no other forces holding the densities at a specific value, heterogeneous contact rates are enough to diversify the ESS's. This produces a "differential invasibility" of rare strains throughout the network: the invasion ability of a given strain depends on node position.

Figure 3 shows the differential invasibility promoted by the heterogeneous contact rates. The panel shows Barabasi-Albeirt network models with different powers of preferential attachment (Barabási & Albert 1999). Decreasing the power of preferential attachment increases the variance in degree distribution (appendix IV) and increases graph heterogeneity (Estrada 2010). Graphs with the same number of links but different topologies produce different ESS compositions. This is evident when we compare panel a) and b) in figure 3. Both networks have the same number of links. But, while link distribution is homogeneous in a), it is heterogeneous in b). As shown, this is enough to diversify the fitness surfaces of pathogens. The curves and dots in figure 3 were produced with the interior equilibrium produced by the numerical simulations (actually solving the temporal dynamics of system 1 and picking up the densities at the stable state). Nonetheless, the interior equilibrium in 8 is a good approximation for small graphs and produces similar patterns.

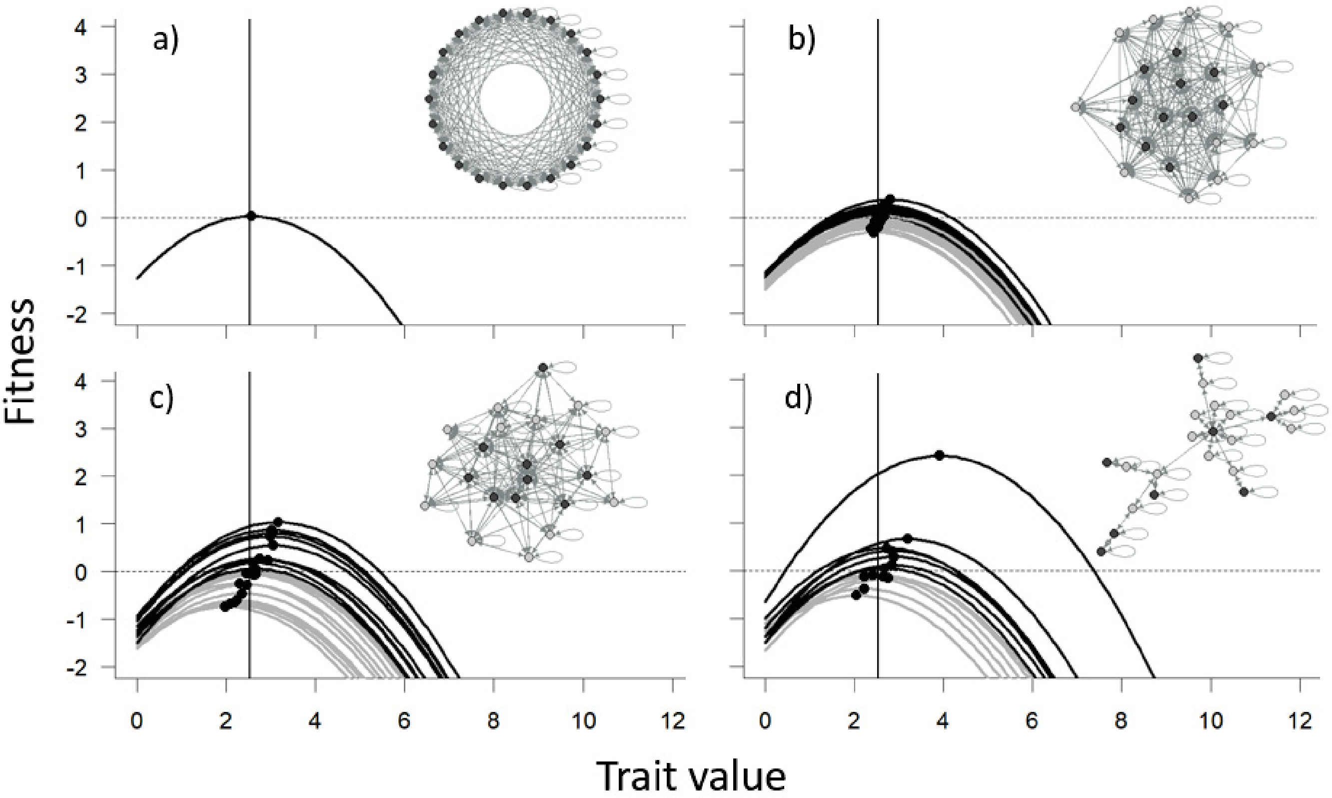


Figure 3: The invasion fitness of pathogens in different hosts as a function of the contact network structure. Curves are the parametric curve $[z_{m,i}, \mathcal{F}_i]$ of each node and the black dots over them are the ESS of each node (the $z_{m,i} = \hat{z}_i$ given by 12). Dark and light colored nodes and curves are the source and sink host populations, respectively. Horizontal dashed lines indicates the region where $\mathcal{F}_i = 0$. Vertical solid lines denotes the ESS for the homogeneous network scenario and the resident trait value (panel a). All networks have $n = 22$ nodes, but they differ in their number of links and heterogeneity (calculated with index 5): a) $E = 374$, $\rho(CN) = 0$; b) $E = 374$, $\rho(CN) = 0.476$; c) $E = 244$, $\rho(CN) = 2.387$; d) $E = 64$, $\rho(CN) = 11.930$. Common parameters are: $b = 2$, $d = 0.2$, $l = 0.15$, $\gamma = 0.2$, $z = 2.533773$, $a_{(1)} = 0.248$, $a_{(2)} = 0.2$, $m_{(0)} = 0.004$ and $m_{(1)} = 0.4$. Initial conditions: $S_i = 0.5$, $I_i = 0.5$.

Discussion

In the model, the stability of system 1 is insensible to node degree when the network is homogeneous. In such a case, the densities of infected and susceptible classes are described by the interior equilibrium of a single-host system. This is due to the conditional function 4, which causes the out-degree of a given node to dilute the weight of the links departing from that node. This function reflects our assumption that the secondary infections of pathogens varies with host availability but not with the amount of routes for infection. Using any other function is likely to introduce modeling artifacts in the fitness of pathogens. If transmission is not constrained by $\sum_{j=1}^{n} \mathcal{C}_{ij} = 1$

(as imposed by function 4), the number of secondary infections would increase with node out-degree. Although this has evident biological explanation (Franz et al. 2018), it generates a problem of order-sensitivity in the transmission dynamics: the effect of link diversity is not solely driven by changes in link distribution, but also by changes in the actual number of links (Moisset de Espanés et al. 2021). As a consequence, the actual effect of the graph structure would be inflated by node out-degree. This means that, two homogeneous graphs with different number of links would produce different attractors, confounding the actual effect of graph structure. Under function 4 any variation in the equilibrium densities is necessarily caused by the heterogeneous structure of the contact network.

The main outcome of contact heterogeneity is the variability in host availability. Here, host availability is the amount of susceptible individuals available for a pathogen residing in a given host population (the quantity $\sum_{j \in \mathbb{N}_i} S_j$ in the fitness function 7). Then, as we have shown, a source-sink epidemiological dynamics emerges from the variability in host availability. In this emerging pattern, epidemics in sink populations are stable, but necessarily maintained by the sources. This, however, does not indicates that isolating sink nodes will deplete pathogen prevalence. In fact, all hosts can maintain the epidemics when they are isolated from the network and thus the source-sink pattern is necessarily an emergent property of the graph structure. This differs from the case studied by Okano et al. (2020), for example, in which prevalence is depleted in sink populations when they are isolated from the network. One way to interpret this dependence of sinks in our model is that their local outbreaks are driven by what is known as ”stuttering chains” of host individuals (Blumberg & Lloyd-Smith 2013). Following Dalziel et al. (2014), stuttering chains may be produced by either variation in contact rates, or by the poor compatibility between pathogens and some hosts. Carefully, Dalziel et al. (2014) also noted that both processes may occur simultaneously. Indeed, our model indicates that, not only they do occur simultaneously, but also have a relationship of causality. There is, variation in contact rates may actually diversify the compatibility of the host-pathogen interaction.

The source-sink pattern that emerges at the interior equilibrium is a property of the fitness measure that we have used (see appendix III). Usually, pathogen fitness is either measured as the reproductive ratio R_0 (Gilligan & Van Den Bosch 2008) or by the per capita rate of increase in the infected class dI_i/I_idt (Day & Gandon 2007). While the former accounts for reproductive success per generation, the latter is continuous in time (Day & Gandon 2006). Likewise the latter, our approach is continuous in time, but it does not measures the rate of increase in the infected class. Instead, function $F_i(z, \mathbf{S}, N_i)$ measures the contribution of pathogens in a given node i to the rate of increase in the overall infected class ($\frac{dI}{dt} = \sum_i^n \frac{dI_i}{dt}$). The main difference is that at the non-trivial equilibrium, quantity dI_i/I_idt is necessarily null, but $F_i(z, \mathbf{S}, N_i)$ is not. As we have shown throughout our analysis, this property of $F_i(z, \mathbf{S}, N_i)$ is the main driver of the source-sink pattern. Furthermore, fitness function 7 does not affect the epidemiological dynamics, but rather emerges from it. This is because the actual dynamics of the system is given by the SIS model, which depends on per capita rates. This allows the derivation of $F_i(z, \mathbf{S}, N_i)$ for other epidemiological and/or ecological scenarios involving multi-host pathogens. Unsurprisingly, different systems may generate alternative source-sink patterns. Yet, the pattern may emerge whenever the transmission dynamics variegate the size/density of the susceptible classes.

Our results indicates that the fitness of pathogens residing in sink nodes may be negative even for optimal trait values. This means that, if optimal strains emerge in a sink it could not persist since these nodes cannot self-sustain epidemics. As a consequence, evolution by natural selection in sink hosts depends on strains that emerge elsewhere. After reaching the sink, strains with greater fitness may vanish in a slower rate than their residents. Then, as long as strains are continuously imported, we expect an increase the prevalence of adapted strains in despite of their negative fitness. Likewise, meta-population dynamics predicts that sink patches evolve more due to gene flow then by the local emergence of variants (Kawecki 2004).

In this study, pathogen diversification is due to the interplay between host availability and the virulence-transmission trade-off. Whilst the trade-off inserts convexity to the fitness function, host availability determines the trait value that maximizes it. Then, as heterogeneous contact rates variegate host availability, the fitness maxima in each host diversifies. In terms of the Adaptive Dynamics framework that we have used (Diekmann 2004), this means that the ability of a given rare variant to invade the resident pathogen depends on the host position within the contact network. This differential ability to invade different nodes is what we call the ”differential invasibility” of rare strains. A direct consequence of differential invasibility is that it increases the diversity of emerging variants. Apparently, this contrasts with the results of Leventhal et al. (2015) in which contact heterogeneity actually decreases the invasibility of rare variants. However, Leventhal et al. (2015) evaluated strain ability to invade whole clusters of nodes in the contact network. In such a case, invading strains are those compatible with the average requirements of that cluster, consequently decreasing the space in which invasion is feasible. Here, on the other hand, we are interested in the invasibility per node and independently of its persistence throughout the network. Whether or not the variant persists after a successful invasion depends on how it interacts with the resident strain and on its ability to resist the environmental change caused by its emergence (e.g. change in the density of infected and susceptible classes). Notwithstanding, a sudden increase in the frequency of a given variant may be sufficient to promote disease outbreaks in both humans and wildlife (Brooks et al. 2019).

The virulence-transmission trade-off is related to the pace-of-life of pathogens. This means that, depending on the trade-off solution, pathogens may evolve towards slower or faster life-histories (Franco et al. 1996, Oli 2004, Salguero-Gómez et al. 2016). The link between pathogen life-history and the virulence-transmission trade-off is the within-host replication rate (Alizon & van Baalen 2005). If the benefit of high transmission rates are greater then the costs of higher virulence, selection favors pathogens with higher rates of replication and thus fast life-histories. If, however, the costs of virulence are higher then the benefits of transmission, advantageous phenotypes are those with lower replication rates and thus slower life-histories. As a consequence, the evolution of replication rate is sensible to host availability. Even in the absence of qualitative differences in the selective milieus (e.g. behavioural differences between hosts occupying different nodes), the heterogeneous distribution of contact rates promotes differential opportunity to access the overall pool of susceptible hosts. A similar result was found by Crossan et al. (2007*b*), in which pathogen life-history rapidly evolved along a fast-slow continuum depending on the availability of hosts. Likewise, Poulin (2003) found that increasing host mortality can accelerate the development in a trematode parasite. Indeed, the evidence shows that virulence and transmission rates are not always correlated (Acevedo et al. 2019, Hector & Booksmythe 2019), and when they are, within-host replication rate may not always be a pleiotropic trait (De Roode & Altizer 2010). Nonetheless, we argue that heterogeneous contact rates can diversify pathogens selective milieu whenever selection over the evolving trait is sensible to host availability.

The *HCR* hypothesis states that heterogeneous contact rates can diversify the evolutionary trajectories of pathogens with life-history trade-off's. As we have shown, the underlying mechanism for diversification is composed by two consecutive phases: (1) heterogeneous contacts variegates host availability; and (2) life-history trade-off's produce differential invasibility. Phase (2) can take place after any other ecological process have shifted host availability (Ebert & Mangin 1997, Johnson & Thieltges 2010, Hamback et al. 2014, O'Regan et al. 2015). Nevertheless, the temporal scale of disease outbreak is often faster then that of most factors driving the population dynamics of hosts (May & Anderson 1979, Penczykowski et al. 2016, De Meester et al. 2019). Hence, effects of contacts may appear earlier in host availability than most ecological processes do. This give some insights about the effect of habitat fragmentation on pathogen evolution. For instance, the role of habitat fragmentation on contact rates (and thus on host availability) may be immediate, but its influence on host population dynamics can take longer (Gibbs 2001, Ewers & Didham 2006, Fletcher et al. 2018). Then, differential invasibility may appear almost immediately after fragmentation. A similar mechanism is proposed by Zohdy et al. (2019) as the "coevolution effect hypothesis". This hypothesis states that the increased isolation promoted by habitat fragmentation produces "coevolutionary engines" (sets of hosts and pathogens) with diverging evolutionary trajectories. Although there is no coevolution in our model, the diversification of pathogen's ESS corroborates with the idea that these "coevolutionary engines" would take diverging trajectories after fragmentation. Conversely, however, the *HCR* hypothesis has more to do with how patches remain connected to each other after fragmentation than to how they become isolated as in the coevolution hypothesis. That is not say that the two hypotheses are antithetical, but rather complementary.

Our model incorporates heterogeneity in the contact rates between host populations, but assumes that the contacts between individuals are homogeneous. Such an homogeneity is a simplifying assumption that facilitates the analytic tractability of epidemiological models, but it often fail to predict disease dynamics in their full dynamics (Bansal et al. 2007). Hence, a possible expansion to our model is to use the network approximations in Newman (2002) to incorporate individual heterogeneity. This would be interesting to test whether the within-node dynamics of sink populations are formed by stuttering chains. Another interesting expansion is to assume a meta-population dynamics among host populations (e.g. allowing the migration of hosts from one node to another). This is likely to increase the realism of the model since that same species populations that are close enough to share pathogens may also have other demographic interactions (Craft et al. 2011, Craft & Caillaud 2011).

Although deterministic, the dynamics of our model occurs as an emergent property of the network structure. By definition, emergent properties cannot be reduced to the individual behavior of system's elements (Regenmortel 2004, Fisher & Pruitt 2020). In the model analyzed here, host populations have identical parameter values and thus produces the exactly same fitness surface when isolated. When heterogeneously connected, on the other hand, the interior equilibrium idiosyncratically diversifies according to the graph structure. This happened because, although deterministic, the systems attractor depends on the net-effect of pathogen circulation throughout populations. Thus, although the observed pathogen diversification is localized, it is not the outcome of local pressures. This suggests that natural selection driving the local adaptation of pathogens are not necessarily limited by the selective pressures operating at the local scale (Buckee et al. 2004). Probably, this happens because the actual state of a node varies with its neighborhood. Since that contact networks are naturally heterogeneous (Leventhal et al. 2015, Craft & Caillaud 2011), the differential invasibility may be a common mechanism driving the emergence of pathogenic variants.

Acknowledgments

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Appendix I: Within-host dynamics

Here, we follow the method and model proposed in Alizon & van Baalen (2005) in order to derive functions virulence and transmission from the within-host dynamics. Let P_i and L_i be the ith within-host population density of pathogens and lymphocytes, respectively. P_i increases with its intrinsic growth rate z and decreases with a lymphocyte killing rate θ. ν and δ stands for lymphocyte's baseline production and death rate, respectively. The presence of pathogens increases the production of lymphocytes at a rate ρ. Hence, the within-host system can be written as:

$$\frac{dP_i}{dt} = P_i\left[z - \mu L_i\right], \tag{AI.1a}$$

$$\frac{dL_i}{dt} = \nu + yP_i - \delta L_i. \tag{AI.1b}$$

The interior equilibrium of the above system AI.1 is

$$\hat{P}(z) = \frac{1}{y}\left(z\frac{\delta}{\mu} - \nu\right), \tag{AI.2a}$$

$$\hat{L}(z) = \frac{z}{\mu}. \tag{AI.2b}$$

Where superscript ˆ stands for equilibrium densities. Note that, since we assume that all hosts have the same within-host parameter values, and node position does not affect within-host dynamics, all hosts $i = 1, ..., n$ have exactly the same interior equilibrium described in AI.2.

Following Alizon & van Baalen (2005), the virulence and transmission rate are functions of the interior equilibrium AI.2. Virulence, as the the disease-induced mortality of hosts, increases with the replication rate of pathogens $uz\hat{P}(z)$,where u is the rate at which pathogen replication rate increases death rate. Also, there is a side-effect w of lymphocyte load which may also increases pathogen virulence. Thus, virulence function becomes:

$$\begin{aligned}\alpha(z) &= uz\hat{P}(z) + w\hat{L}(z)\\ &= \frac{u\delta}{y\mu}z^2 + \frac{yw - \mu uv}{y\mu}z\\ &= \alpha_{(1)}z + \alpha_{(2)}z^2. \end{aligned} \tag{AI.3}$$

Where $\alpha_{(1)}$ and $\alpha_{(2)}$ reads

$$\alpha_{(1)} = \frac{yw - \mu uv}{y\mu} \tag{AI.4a}$$

$$\alpha_{(2)} = \frac{u\delta}{y\mu}. \tag{AI.4b}$$

For the transmission rate, we understand that what Alizon & van Baalen (2005) called "transmission", is more likely to reflect the infectiousness of i. This is because their derivation does not account for the susceptibility of susceptible individuals. In their paper, this is not a problem since they have focused on a single-host population with monomorphic parameter values. Nonetheless, theory predicts that transmission rate β_{ij} is the product of the ith infectiousness ι_i, the jth susceptibility ζ_j and the contact rate c_{ij} (O'Regan et al. 2015), $\beta_{ij} = \iota_i \zeta_j c_{ij}$. However, because we are interested in the effects of the structure of the contact network, rather then in the variation of host parameters, we have assumed that all hosts that the same infectiousness $\iota_i = \iota$ and the same susceptibility $\zeta_i = \zeta = 1$. This allows us to set pathogen transmissibility as $\tau(z) = \iota(z)\zeta$ and thus the transmission rate as $\beta_{ij} = \tau(z)c_{ij}$. Then, to derive the function $\tau(z)$ from the within-host dynamics we use the same idea that Alizon & van Baalen (2005) have used for transmission rate. Thus assuming that transmissibility increases with the pathogen load at a rate a:

$$\begin{aligned}\tau(z) &= a\hat{P}(z)\\ &= \frac{a\delta}{y\mu}z - \frac{av}{y}\\ &= \tau_{(0)} + \tau_{(1)}z \end{aligned} \tag{AI.5}$$

Where

$$\tau_{(0)} = -\frac{a\nu}{y}, \tag{AI.6a}$$

$$\tau_{(1)} = \frac{a\delta}{y\mu}. \tag{AI.6b}$$

Equations AI.3 and AI.5 are exactly equations in 2 in the text.

In our simulations, the parameter values of the within host dynamics that produces the desired values of α_1, α_2, τ_0 and τ_1 are: $\delta = 1$, $\nu = 0.01$, $\mu = 1$, $y = 5$, $u = 1$, $w = 0.25$ and $a = 2$.

Appendix II: Interior equilibrium of the homogeneous case

By definition, nodes have identical degree in homogeneous graphs. Thus, $k_i = k_j \; \forall i, j \in \mathbb{N}$. Given our function for the contact rate $\mathcal{C}_{ij}$ in 4, homogeneous graphs also imply in identical contact rates $\mathcal{C}_{ji} = 1/k_i \; \forall i, j \in \mathbb{N}$. Then, we also know that, since all parameters are identical, all hosts should have the same population density $\hat{N}_i$ and prevalence $\hat{p}_i$ (and thus the same $\hat{I}_i = \hat{N}_i\hat{p}_i$) at their interior equilibrium. This means that the size of the infected class connected to a given node i is k_i times its own density $\hat{I}_i$. Thus, for homogeneous graphs, the transmission term in 1 can be simplified to

$$\begin{aligned}
S_i\tau(z)\sum_j^n \mathcal{C}_{ji}I_j &= S_i\tau(z)\mathcal{C}_{ij}\sum_j^n I_j && \text{(AII.1)}\\
&= S_i\tau(z)\mathcal{C}_{ij}k_iI_i && \text{(AII.2)}\\
&= S_i\tau(z)\frac{1}{k_i}k_iI_i && \text{(AII.3)}\\
&= S_i\tau(z)I_i. && \text{(AII.4)}
\end{aligned}$$

Hence, replacing the transmission term AII.4 in 1 yields the following model:

$$\frac{dS_i}{dt} = bN_i + \gamma I_i - S_i\left[d + lN_i + \tau(z)I_i\right] \tag{AII.5a}$$

$$\frac{dI_i}{dt} = S_i\tau(z)I_i - I_i\left[d + lN_i + \alpha(z) + \gamma\right], \quad \forall i \in \mathbb{N} \tag{AII.5b}$$

the above model describes the single-host epidemiological dynamics. The interior equilibrium of the susceptible class $\hat{S}_i$ can be found by setting the per capita quantity $\frac{dI_i}{I_i dt} = 0$ ans solving for S_i, which leads to

$$\hat{S}_i(\hat{N}_i) = \frac{d + l\hat{N}_i + \alpha(z) + \gamma}{\tau(z)} \tag{AII.6a}$$

To find for the interior equilibrium of I_i, we also use the per capita quantity $\frac{dI_i}{I_i dt} = 0$. However, we write it evidencing that $N_i = S_i + I_i$:

$$\frac{dI_i}{I_i dt} = S_i\tau(z) - \left[d + l(S_i + I_i) + \alpha(z) + \gamma\right] = 0 \tag{AII.6b}$$

Then, solving for $\hat{I}_i$ yields

$$\hat{I}_i(\hat{S}_i) = \frac{\hat{S}(\tau(z) - l) - (d + \alpha(z) + \gamma)}{l}. \tag{AII.6c}$$

Now, we need to solve for the overall population density $\hat{N}_i$. To do it, we can rewrite our system in terms of overall population density and prevalence:

$$\frac{dN_i}{dt} = N_i\left[r - lN_i - \alpha(z)p_i\right] \tag{AII.7a}$$

$$\begin{aligned}\frac{dp_i}{dt} &= p_i\left[\frac{dI_i}{I_i dt} - \frac{dN_i}{N_i dt}\right] \\ &= p_i\left[(1 - p_i)(\tau(z)N_i - \alpha(z)) - b - \gamma\right], \quad \forall i \in \mathbb{N}\end{aligned} \tag{AII.7b}$$

the prevalence equation is a replicator equation that results from differentiating $pi(t) = Ii(t)/Ni(t)$ in respect to time t. We then set $\frac{dN_i}{N_i dt} = 0$ and $\frac{dp_i}{p_i dt} = 0$ and solve for N_i

and p_i, respectively. This results in the following interior equilibrium:

$$\hat{N}_i(\hat{p}_i) = \frac{r - \alpha(z)\hat{p}_i}{l} \tag{AII.8a}$$

$$\hat{p}_i = \frac{[\tau(z)r + \alpha(z)(\tau(z) - l)] \pm \sqrt{\Delta_i}}{2\alpha(z)\tau(z)} \tag{AII.8b}$$

With $\Delta_i = \tau(z)\left[\tau(z)[r - \alpha(z)]^2 + 2\alpha(z)l[b + d + \alpha(z) + 2\gamma]\right] + \alpha(z)^2 l^2$. Then, replacing AII.8 in the susceptible and infected equilibrium densities leads to the following interior equilibrium for the homogeneous situations:

$$\hat{I}_i(\hat{S}_i, z) = \frac{\hat{S}_i(z)[\tau(z) - l] - [d + \alpha(z) + \gamma]}{l}, \tag{AII.9a}$$

$$\hat{S}_i(z) = \frac{b + d + \alpha(z)[1 + l] + 2\gamma}{2\tau(z)} - \frac{\sqrt{\Delta_i}}{2\left[\tau(z)\right]^2}. \tag{AII.9b}$$

Now, the product $\tau(z)\sum_{j\in\mathbb{H}}\mathcal{C}_{ji}$ is the ith rate at which an individual in class S_i is allocated to I_i following an encounter with a infected individual in its infected neighborhood. Our simulations shown that using $\tau(z)\sum_{j\in\mathbb{H}}\mathcal{C}_{ji}$ in AII.9 leads to an approximation for heterogeneous graphs (quantities 8). These approximations assumes that the density of the neighborhoods have no differential effect and thus only depends on graph structure. The approximations can predict equilibrium accurately under small networks. Increasing the size of the graph, however, increases the net-effect of the system, causing their densities to change idiosyncratically with graph structure.

Appendix III: Deriving the general condition for the source-sink state of nodes

At the interior equilibrium, the fitness of pathogens occupying the ith node can be written as

$$F_i(z, \hat{S}, \hat{N}_i) = \tau(z)\frac{1}{k_i}\sum_{j\in\mathbb{N}_i}\hat{S}_j - \left[d + l\hat{N}_i + \alpha(z) + \gamma\right] \tag{AIII.1}$$

Now, if we take the interior equilibrium of the single population dynamics in eq.AII.6a and replace at the fitness function, we get that the following quantity

$$\tau(z)\frac{1}{k_i}\sum_{j\in\mathbb{N}_i}\frac{d+l\hat{N}_j+\alpha(z)+\gamma}{\tau(z)\sum_{g\in\mathbb{N}_j}\mathcal{C}_{gj}}-\left[d+l\hat{N}_i+\alpha(z)+\gamma\right]. \tag{AIII.2}$$

Where $g\in\mathbb{N}_j$ stands for the nodes g that pertain to the neighborhood of node j. Assuming that all hosts have the same parameter values implies that, when decoupled from the contact network, they reach the exactly same interior equilibrium. This allows us to perform the following algebra:

$$\begin{aligned}
&\tau(z)\frac{1}{k_i}\sum_{j\in\mathbb{N}_i}\frac{d+l\hat{N}_j+\alpha(z)+\gamma}{\tau(z)\sum_{g\in\mathbb{N}_j}\mathcal{C}_{gj}}-\left[d+l\hat{N}_i+\alpha(z)+\gamma\right]=\\
=&\tau(z)\frac{1}{k_i}\frac{d+l\hat{N}_j+\alpha(z)+\gamma}{\tau(z)}\sum_{j\in\mathbb{N}_i}\frac{1}{\sum_{g\in\mathbb{N}_j}\mathcal{C}_{gj}}-\left[d+l\hat{N}_i+\alpha(z)+\gamma\right]=\\
=&\frac{1}{k_i}(d+l\hat{N}_i+\alpha(z)+\gamma)\sum_{j\in\mathbb{N}_i}\frac{1}{\sum_{g\in\mathbb{N}_j}\mathcal{C}_{gj}}-\left[d+l\hat{N}_i+\alpha(z)+\gamma\right]=\\
=&(d+l\hat{N}_i+\alpha(z)+\gamma)\left[\frac{1}{k_i}\sum_{j\in\mathbb{N}_i}\frac{1}{\sum_{g\in\mathbb{N}_j}\mathcal{C}_{gj}}-1\right]=\\
=&(d+l\hat{N}_i+\alpha(z)+\gamma)\left[\mathcal{M}_i-1\right].
\end{aligned} \tag{AIII.3}$$

Where $\mathcal{M}_i$ is an approximation of the effect of the network structure to the meta-population (or source-sink) state of the fitness function. Its value, is:

$$\mathcal{M}_i=\frac{1}{k_i}\sum_{j\in\mathbb{N}_i}\frac{1}{\sum_{g\in\mathbb{N}_j}\mathcal{C}_{gj}}. \tag{AIII.4}$$

Appendix IV: Relationship between the power of preferential attachment and degree distribution

Here, we show that the power of preferential attachment decreases the variance in node degree (figure). This happens because increasing. the power of preferential attachment in a Barabasi-Albeirt model increases the chance of a new node make a connection with more connected nodes. Then, increasing this power ultimately produces a star graph in which 1 central node connects with all other nodes.

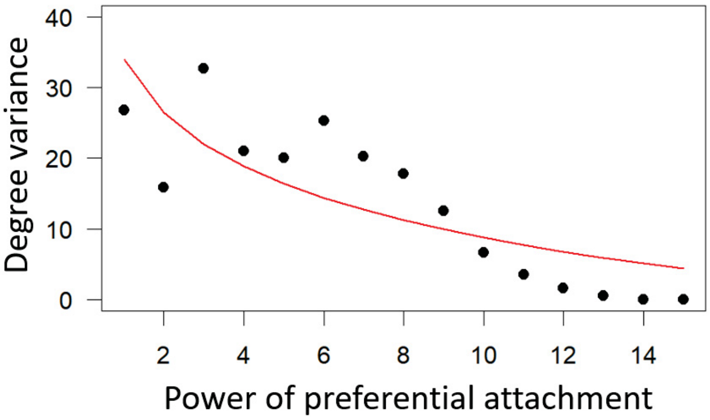

Figure 4: The relationship between the variance in node degree and the power of preferential attachment in Barabasi-Albeirt graphs. Dots are the value of node degree in different networks. The red line is a tendency line.

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4 CAPÍTULO III: THE ADAPTIVE DYNAMICS OF A BEHAVIORALLY FLEXIBLE GENERALIST

The adaptive dynamics of a behaviorally flexible generalist

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Abstract: Foraging behavior is a specific type of organism plasticity that affects both the ecology and evolution of consumer populations. Some evidences indicate that increasing the diet range of consumers also increases their repertoire of foraging behavior. This suggests that the ability to behaviorally optimize foraging activity may be specially relevant for polyphagous consumers (generalists). Yet, the mechanistic connection between behavioral flexibility and the evolution of generalist consumption is still obscure. In this paper, our main goal is to investigate the relationship between flexible foraging behavior and the evolution of generalism. To do so, we analyze a modified version of the classical Lotka-Volterra consumer-resource system. The model is composed by a consumer that must distribute its foraging effort over its two potential resources depending on their availability and profitability. We then analyze how foraging behavior affects the evolutionary dynamics of consumer digestibility. We found that generalism can be both evolutionary stable and convergence stable, indicating that selection may give advantage to polyphagous consumption even when specialism confers a greater fitness. Also, our analysis indicate that generalism can represent branching points in the fitness surface of consumers. These points of fitness minima act as attractors even when consumers are optimal specialists. We discuss the implications of our results for the role of behavior in evolution and how it can provide insights on the classical idea that evolution always leads to specialization.

Introduction

Understanding the role of plasticity in the evolutionary trajectory of populations is at the frontiers of ecology and evolution (Miner et al. 2005, Sutherland et al. 2013). Plasticity is the manifestation of different phenotypes depending on the interaction between organism development and the environmental context in which development occurs (Schlichting 1986). Plastic traits can either be neutral, reduce or increase the rate of adaptation (Wyles et al. 1983), and also either deviate or canalize the trajectories of populations towards ecological and evolutionary attractors (Miner et al. 2005). This inserts uncertainty to the biological system, hampering our ability to understand and predict evolutionary dynamics. Behavioral dynamics is a class of plasticity that is known to influence the evolution of ecological interactions (Lima & Dill 1990, Dall et al. 2004, Chang et al. 2017). Differently from other plastic traits, however, behavioral change is more reversible than developmental plasticity (Sih et al. 2004). Behavioral change is rapid and is only manifested given specific environmental cues. The consequence of behavioral reversibility is that they insert a greater uncertainty in the actual repertoire of activity of a given organism then plastic traits which are based on level of genetic expression (Duckworth 2009).

The interest in the role of individual behaviour to the evolutionary trajectory of populations have a long history in evolutionary ecology (Bogert 1949, Dill 1983, Arnold 1988, Wcislo 1989, Lima & Dill 1990, Dall et al. 2004, Chang et al. 2017). Since evolution emerges from the interplay between the nature of the organism and the nature of the conditions (Darwin & Wallace 1859), behavior is what mediates the intensity of such interplay (Duckworth 2009, Chang et al. 2017). For example, the use of a specific resource may increase the fitness of a population by promoting a set of selective pressures that gives advantage to a certain phenotype. However, adaptation may be hindered if individuals are not motivated to use that resource (even when the resource is available; Parent et al. 2014). As a consequence, the fitness landscape experienced by organisms is a function of how individuals behaves and perform in their environment. In theoretical studies, the interest on behaviors associated with foraging activities (e.g. foraging effort, optimal give-up time, predator avoidance) produced a repertoire of mathematical expressions as an attempt to describe behavioral flexibility (Abrams 1982, McNair 1982, Olsson & Holmgren 2000, Kondoh 2003, McKane 2004, Beckerman et al. 2006). Generally speaking, these models trace behavioral distributions as a function of the payoff received for behaving in a certain manner. Together, they form the great area optimal foraging behavior (Pyke et al. 1977). This theory predicts that consumers will optimize their foraging effort over a given resource in order to maximize their overall energy intake (Krivan & Sikder 1999). This theory is considered one of the efficient theories in ecology given its simplicity and predictive power (Marquet et al. 2014).

Foraging behavior seems to be specially relevant to species that are resource generalists. This is because generalists often face a cost of being inferior competitors when compared to specialists (Futuyma & Moreno 1988, Dall & Cuthill 1997). Then, adjusting their behavior may dampen the costs of generalism (Bogert 1949). Moreover, generalist species are usually able to display a greater repertoire of behavior (Ducatez et al. 2015, Rosati 2017, Daniels et al. 2019), indicating that such a flexibility may play a relevant role in the evolution of generalism. For example, Komatsu et al. (2009) note that, while specialist crickets were able to avoid host recognition, generalists could not. However, generalists displayed alternative behaviors that circumvent the problem of being detected. This raises questions such as: is foraging behavior a cause or a consequence of evolution? How does behavioral dynamics affects the evolution of consumers? Some authors already explored these questions (Rueffler et al. 2007, Overington et al. 2008, Duckworth 2009). Yet, the mechanistic foundations connecting behavior flexibility and the evolution of generalism consumption are still unclear.

The nature of generalist strategies is a core theme in both theoretical ecology and evolutionary theory (Jaenike & Selander 1978, Lapchin 2002, Dennis et al. 2011, Loxdale et al. 2016, Clarke 2018). The word ”generalism” does not describes the absolute properties of organisms activities (e.g. niche width, fitness spaces). Rather, it describes the range of capacities that are manifested within a given ecological dimension (e.g. habitat, diet). Such a description must be relative with a plausible reference such as the relative range of resource that a species of a given taxonomic group is able to use (Leggett et al. 2013). Moreover, a consumer may be specialist in terms of resource species but generalist in terms of resource phenology. For example, some bruchid species are highly specialized in species of Fabaceae but may consume fruits in different phenological phases, while others may be specialized in a given phase (Johnson

& Romero 2004). Here, we use the word generalism as a proxy for polyphagy.

There are two main narratives to elucidate the evolution of generalism. In the first, generalism is thought to be a transient state that invariably evolves towards a specialist strategy (Loxdale et al. 2011). This is based on the classical idea that ”the jack-of-all-trades is the master of none” (MacArthur 1972), which implies that generalists often face a fitness cost when compared to specialists. Hence, adaptation would drive species towards resource specialization. The second narrative is that the evolution of generalism requires temporal fluctuations in resource quality that cancel-out the selective pressures driving the process of specialization (Lapchin 2002, Frank 2011). As a consequence, generalism would evolve not as an adaptation, but as a by-product of multiple selective pressures acting over a given trait. Theoretically, instantaneous measures of fitness (e.g. arithmetic fitness) will favor specialist strategies whenever generalism is not a global maxima (e.g. the generalist is not a master-of-all; Hellgren et al. 2009). In this later case, the evolution towards generalism only occurs under long-term measures of fitness such as the geometric mean fitness (Lande et al. 2009, Starrfelt & Kokko 2012). This points out to a theoretical gap that hinders our understanding on how generalists can evolve in relatively stable environments. Here, we argue that behavioral decisions can serve as a mechanism to fill this gap.

In this study, our main goal is to explore the role of behavioral dynamics in the evolution of generalist strategies. To do so, we study the ecological, behavioral, and evolutionary stability of a mathematical model describing the interaction between a consumer and its two resources. We use both analytical treatments and numerical simulations to evaluate the general conditions driving the evolution of generalism. We found that behavior can stabilize the evolution of generalist strategies in stable environments even when the jack-of-all-trades is the master-of-none. Basically, our results indicate that behavioral flexibility produces a dynamical fitness surface that traps the consumer population in a generalist region. Moreover, the analytic tractability of the model allows for insights on the role of generalist consumers to the stability of communities. We discuss the insights provided by our analysis to the influence of behavior in evolutionary trajectories of consumer populations.

Models and analysis

Our analysis is based on a two-resource version of the classical Lotka-Volterra predator-prey system (Berryman 1992). This version describes the interaction between the population of a consumer and its two resources. We incorporate two types of trade-off constraining the consumer-resource interaction. The first is a behavioral trade-off in which consumers can adjust their foraging effort over each resource in order to maximize fitness. This adjustment occurs by accommodating the proportion of time they spend searching for each specific resource according to its availability and profitability. We assume that resources co-occur at the same environment. This means that, while consumers spend no time changing patches to forage for the other resource, they only find the resource they are searching for. Biologically, this can be interpreted as the formation of a search image, so that while foraging for a given resource consumers are unable to find the other (Uchida et al. 2007). The second trade-off is represented by a physiological constraint. We assume that the digestibility of each resource depends on a consumer quantitative trait. Then, the performance of consumers in digesting a given resource depends on its trait value. Since different resources require different trait values, it is impossible to have an optimal digestion for both resources. In the model, whether the consumer is a generalist or a specialist depends on how the system solves these two trade-offs. For example, the solution of the behavioral trade-off may promote a generalist behavior, but subsequent trait evolution may promote specialism. Alternately, as we will show, the behavioral trade-off may promote a specialist behavior, but further adaptation produces generalism. Then, we use the adaptive dynamics framework (Brannstrom et al. 2013) to evaluate how consumer foraging behavior affects the evolution of digestibility and how this relates with the evolution of generalists.

This section is organized as follows. First, we present the population and behavioral dynamics of the model together with the interpretation of its variables and parameters. Then, we evaluate the stability of model's interior equilibrium. Next, we show how to find (when there is one) the generalist strategy in our model, here themed the generalist singularity. Finally, we perform an invasion analysis to understand the possibles evolutionary outcomes for a consumer population that resides in the generalist singularity. The full R script used in the numerical simulations of this paper can be found in the github repository (http://github.com/pbolanhom/abeg)

The model

Population dynamics: Consider a community composed by a consumer and $n = 2$ resources $i = 1, 2$, each of which with population density C, R_1 and R_2, respectively. Let the foraging effort of the consumer over resource 1 be denoted by p. This foraging effort is measured as the proportion of time spent foraging for 1 in relation to the total foraging time (Uchida et al. 2007). This produces a linear constraint in which the sum of the time spent foraging for each resource must equal 1. Thus, $(1 - p)$ is the foraging effort over resource 2 ($p + (1 - p) = 1$). Moreover, we assume that the conversion efficiency of consumers differs depending on the resource it consumes. Thus, let e_i be its conversion efficiency for each resource i. Assuming that consumer's functional response is linear (also known as Holling type I functional response; Case 2000) with attack rate a, the system can be described by the following set of ODE's:

$$\frac{dC}{dt} = C\left[e_1 paR_1 + e_2(1-p)aR_2 - d\right], \tag{1a}$$

$$\frac{dR_1}{dt} = R_1\left[r_1 - lR_1 - Cap\right], \tag{1b}$$

$$\frac{dR_2}{dt} = R_2\left[r_2 - lR_2 - Ca(1-p)\right]. \tag{1c}$$

Where d in 1a is the intrinsic death rate of consumers. The quantities $e_1 paR_1$ and $e_2(1-p)aR_2$ are known as the numerical response of consumers (the amount of consumers produced per prey consumed; Gotelli 2008). Note that these numerical responses are linear in relation to the density of each resource. Letters r_i and l in eq.1b and eq.1c are the intrinsic growth rate and density-dependent mortality of each resource, respectively. This means that, in absence of consumers, resources grow logistically until densities stabilizes at their carrying capacities $K_i = r_i/l$.

Behavioral dynamics: We assume that consumers can behaviorally adjust their foraging efforts over each of its resources depending on its profitability. We use the replicator-based adaptive behavior framework (Uchida et al. 2007, Valdovinos et al. 2010) to describe the temporal dynamics of p. The framework describes the dynamical change in a given foraging effort p_i as

$$\frac{dp_i}{dt} = \tau p_i \left[\frac{\partial}{\partial p_i} \left(\frac{dC}{Cdt} \right) - \sum_{i=1}^{n} p_i \frac{\partial}{\partial p_i} \left(\frac{dC}{Cdt} \right) \right].$$

Which, in our case, leads to the following ODE for the temporal change in p:

$$\frac{dp}{dt} = \tau p(1-p)a \left[e_1 R_1 - e_2 R_2 \right]. \tag{1d}$$

Where the Greek letter τ in the above equation represents the time scale of behavioral change in relation to the population dynamics. Because behavioral change happens much faster then population dynamics, τ must be always greater than 1. Since that p is a proportion, it is constrained between the interval $0 \leq p \leq 1$.

Consumer digestibility: In order to evaluate the evolutionary dynamics of consumers we need to assume an evolving trait. Thus, we assume that consumers have a quantitative trait z that represents the digestibility of resources. To incorporate a trade-off between the optimum digestibility for each resource, let x_1 and x_2 be the optimum value of z for the consumption of resource $i = 1$ and $i = 2$, respectively. Then, to incorporate these assumptions, we assume that the conversion efficiencies (the e_i quantities in eq.1a) are Gaussian functions of z with optimum values at their respective x_i values:

$$e_1 = \exp\left[-\frac{(x_1 - z)^2}{2\sigma_1^2} \right] \text{ and } e_2 = \exp\left[-\frac{(x_2 - z)^2}{2\sigma_2^2} \right], \tag{2}$$

Where the variances σ_i^2 are inversely proportional to the strength of the selective pressures from each resource as the z value get far from their optimum values. In other words, increasing the variances decreases the strength of selection.

The ecological stability of generalism

System 1 may reach its equilibrium at either a generalist ($0 < p < 1$) or a specialist ($p = 0$ or $p = 1$) scenario. In the generalist scenario, the foraging effort p finds its equilibrium at

$$\hat{p} = \frac{e_2(e_1 a r_1 - ld)}{e_1 e_2 a(r_1 + r_2) - ld(e_1 + e_2)}. \tag{3a}$$

Where superscript ˆ denotes equilibrium values. The above quantity leads to the following equilibrium densities for consumers, resource 1 and resource 2, respectively:

$$\hat{C} = \frac{r_1 + r_2}{a}\left[1 - \frac{ld(e_1 + e_2)}{ae_1 e_2(r_1 + r_2)}\right], \tag{3b}$$

$$\hat{R}_1 = \frac{d}{ae_1}, \tag{3c}$$

$$\hat{R}_2 = \frac{d}{ae_2}. \tag{3d}$$

We show the full derivation of eq.3 in appendix I. It is well known that the instability of the interior equilibrium of classical predator-prey models requires a saturating functional response (e.g. Holling type II functional response; Holling 1959, Abrams & Matsuda 1997, Case 2000). Since that we have assumed a linear functional response we conclude that, if feasible, the interior equilibrium of the generalist scenario is always stable.

The feasibility of eq.3 requires the the densities of resources to stabilize bellow their carrying capacity $K_i = r_i/l$. This condition is also valid for classical Lotka-Volterra predator-prey model (Abrams 2000) and can be written as the inequality

$$\hat{R}_i < K_i \text{ or } \frac{d}{ae_i} < \frac{r_i}{l}. \tag{4}$$

As long as inequality eq.4 is true, consumers behave as generalists. This holds even if their digestibility is at its optimal value for a specialist consumption ($z = x_i$). This suggests that, under the assumption of adaptive behavior, even specialist consumers can behave as ecological generalists as long as resource density stabilizes bellow their respective carrying capacities (Robinson & Wilson 1998).

Any parameter that reverts inequality 4 for one of the two resources causes the consumer the behave as a specialist ($p = 0$ or $p = 1$). In such case, the interior equilibrium is the fixed point for the classical Lotka-Volterra system:

$$\hat{C}^s = \frac{r_i}{a}\left[1 - \frac{dl}{e_i a r_i}\right] \tag{5a}$$

$$\hat{R}_i^s = \frac{d}{a e_i} \tag{5b}$$

$$\hat{R}_j^s = \frac{r_j}{l} = K_j \tag{5c}$$

Where superscript s in eq.5 denotes for the specialist scenario. Subscript i and j denotes for the consumed and non-consumed resource, respectively. Note that the non-consumed resource j reaches its carrying capacity $K_j = r_j/l$ and has no effect to the population dynamics of consumers and the consumed resource i. This happens because we assume no direct interaction between the two resources. As a consequence, the non-consumed resource has effect in the community (see appendix I). This indicates that, in our model, consumers behaving as generalists can maintain the flow of information throughout the community.

Evolutionary dynamics

To evaluate the evolutionary dynamics of the consumer population we apply the classical Adaptive Dynamics (AD) framework (Geritz et al. 1997, Dieckmann & Doebeli 1999, Brannstrom et al. 2013). The AD is a set of analytical tools useful to study the long-term evolution of quantitative traits. Given its analytical property, this framework is adequate to study relatively simple ecological models such as model 1. The main assumptions of the AD is that mutations are rare and evolution is slow (Lion 2017). This means that, whenever a mutant emerges in the population, the resident population is at its ecological equilibrium. This means that there is no feedback between trait adaptation and populations dynamics. This makes no difference in our model since that, as we will show further in the analysis, a behavioral-evolutionary feedback loop emerges in our model. This feedback leads to exactly the same result as if an eco-evolutionary feedback were considered. Another common assumption in AD is that the resident population is monomorphic for a given trait value; although some studies already have expanded the analysis for polymorphic populations (Doebeli & Ispolatov 2010, Champagnat & Méléard 2011).

Following Doebeli & Ispolatov (2010) and Lion (2017), the AD assumes that the rate of change in the trait z of a monomorphic population can be described by the following canonical equation:

$$\frac{dz}{dt} = M(z)\left.\frac{\partial F}{\partial z'}\right|_{z'=z}. \tag{6}$$

Where z' is the mutant trait, and function $M(z)$ describes the rate of mutation. This functions is assumed to be a scalar in 1-dimensional traits such as in our case. The partial derivative term in the right-side of equation 6 is the gradient of selection. This term indicates the direction, strenght and shape of natural selection acting over the evolving population (Lehtonen 2017). Letter F within this term is the individual fitness. The $z' = z$ subscript indicates that the gradient of selection is evaluated at the point where the mutant trait is equal the resident trait. Evaluating at this point will inform how selection acts over residents.

In order to apply the AD toolbox, one must have information of about the z value for which the gradient of selection vanishes. This point is known as evolutionary singular points (Geritz et al. 1998). Since that we are interested in how selection behaves when residents are generalists, we first need to understand whether our model have an evolutionary singularity that corresponds to a generalist.

The evolutionary singularity of generalism

The first step to evaluate the evolutionary dynamics of consumer digestibility z is to find a proper measure of individual fitness. In mean field models such as system 1, populations are assumed to be well-mixed (Morozov & Poggiale 2012). In such case, theory predicts that fitness can be described by the per capita rate of increase of the evolving population (McPeek 2017). Here, this leads to the following quantity describing the individual fitness $F = dC/Cdt$ of consumers:

$$F(z, p, \mathbf{R}) = a\left[e_1(z)pR_1 + e_2(z)(1-p)R_2\right] - d. \tag{7}$$

Where $\mathbf{R} = (R_1, R_2)$ is a vector containing the densities of resources.

We can use the fitness function 7 to evaluate the properties of natural selection acting over individuals. These properties are described by the gradient of selection, which is calculated by taking the partial derivative of the population fitness in relation to the evolving trait (Lehtonen 2017). This leads to

$$\frac{\partial F(z,p,\mathbf{R})}{\partial z} = a\left[p\frac{(x_1-z)}{\sigma_1^2}e_1(z)R_1 + (1-p)\frac{(x_2-z)}{\sigma_2^2}e_2(z)R_2\right]. \tag{8a}$$

Note that conversion efficiency functions (equations 2) maintain their original form after taking the derivatives. As a consequence, the numerical responses (quantities e_1aR_1 and e_2aR_2) appears in the selection gradient eq.8a. Given the form of the behavioral dynamics that we have assumed (eq.1d), we already know that $e_1a\hat{R}_1 = e_2a\hat{R}_2$ when system 1 reaches the ecological equilibrium at the generalist scenario (equations 3). This causes the numerical responses to cancel-out when natural selection vanishes (the gradient of selection 8a is null) at the generalist scenario. The z value for which the gradient of selection vanishes is known the *evolutionary singular point* (Geritz et al. 1998). Hence, in such a case, there is a $z = z^*$ value to which the following quantity must be true:

$$\hat{p}\frac{(x_1-z^*)}{\sigma_1^2} + (1-\hat{p})\frac{(x_2-z^*)}{\sigma_2^2} = 0. \tag{8b}$$

Then, solving 8b for z^* we get

$$z^* = \frac{\hat{p}x_1\sigma_2^2 + (1-\hat{p})x_2\sigma_1^2}{\hat{p}\sigma_2^2 + (1-\hat{p})\sigma_1^2}. \tag{8c}$$

z^* can only be regarded as the evolutionary singularity if consumers with trait value $z = z^*$ behaves as $\hat{p}(z^*) = \hat{p}(z)$. However, since that we have assumed adaptive behavior, consumers with trait z^* may behave differently. Simply replacing $\hat{p}(z^*)$ (eq.3a) in eq.8b and solving for the z^* is not straightforward given the fact that $\hat{p}$ is an exponential function of z. If, however, we replace $\hat{p}$ in eq.8c and rearrange it, we get a function $\zeta(z)$:

$$\zeta(z) = \frac{e_1(z)e_2(z)\left[r_1x_1\sigma_2^2 + r_2x_2\sigma_1^2\right] - ld\left[e_1(z)x_2\sigma_1^2 + e_2(z)x_1\sigma_2^2\right]}{e_1(z)e_2(z)\left[r_1\sigma_2^2 + r_2\sigma_1^2\right] - ld\left[e_1(z)\sigma_1^2 + e_2(z)\sigma_2^2\right]}. \tag{9a}$$

Then, we iterate

$$z^o = \zeta(z^{o-1}), \tag{9b}$$

where the superscript ^{o} is the iteration number and thus z^o is the oth iteration of ζ. If the generalist singularity exist, a sufficient large ^{o} will lead to the following convergence:

$$z^o \longrightarrow z^\Phi. \tag{9c}$$

Where z^Φ is the fixed point of the iteration and is also the generalist singularity. We call $\zeta(z)$ the *behavioral update function*, which represents a behavioral-evolutionary feedback loop. Since that it does not depends on population densities, it indicates that an eco-evolutionary model would lead to the same result as iterating this function. This means that, in the model analyzed here, foraging behavior obscures eco-evolutionary feedback loops.

The existence of the fixed point z^Φ indicates whether adaptive behavior promotes the evolution of specialists or generalists when generalism is ecological stable ($0 < \hat{p} < 1$ and system 1 interior equilibrium is described by equations 3). Figure 1 shows both cases. In 1a) the fixed point z^Φ does not exists and thus the feedback between trait adaptation and behavioral dynamics drives the consumer population towards specialization. In such a situation, there is no generalist trait to which natural selection vanishes. In 1b), on the other hand, z^Φ exists and thus the feedback promotes the evolution of generalism. Panel 1b) bottom row suggests that adaptive evolution may drive the population towards the generalist singularity even when consumers are initially optimal specialists for resource $i = 1$ ($z = x_1 = -1$).

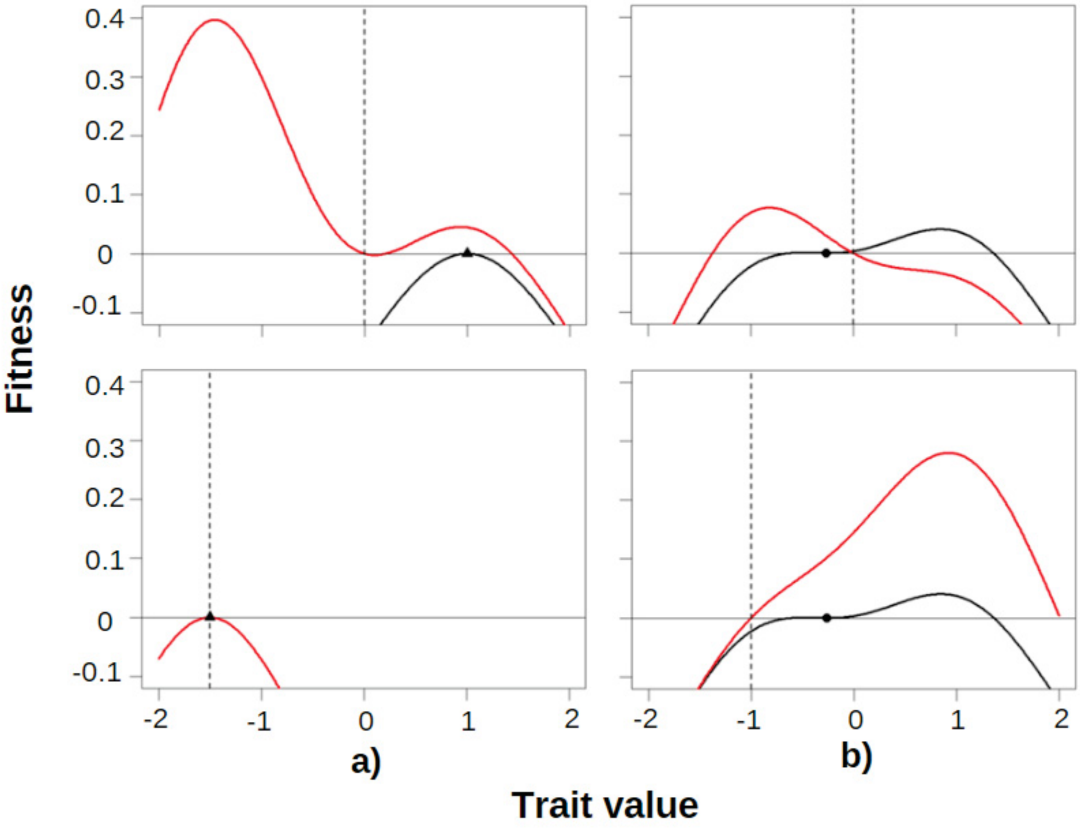

Figure 1: The role of adaptive behavior in the evolution of consumer digestibility. Red and black curves represent the fitness surface of consumers (the parametric curve $[z, F(z, \hat{\mathbf{R}})]$) before and after iterating the behavior update function $\zeta(z^o)$. The vertical dashed line stands for the z value that generated the red curve: a generalist trait ($z = 0$) in top row and for a specialist trait ($z = x_1$) in the bottom row. In a) $x_1 = -1.5$ and z^ϕ is not the fixed point of $\zeta(z^o)$ thus evolution leads to specialism (marked by the triagules). In b) $x_1 = 1$ and z^ϕ is the fixed point and thus adaptation promotes generalism (marked by the dots). Initial conditions: $C = 0.5$, $R_1 = 1$, $R_2 = 1$, $p = 0.5$. Common parameter values are: $x_2 = 1$, $a = 1$, $d = 0.4$, $r_1 = r_2 = 1$, $\sigma_1 = 0.8$, $\sigma_2 = 1$, $l = 0.25$, $\tau = 2$.

The existence of z^ϕ, however, does not indicates whether a resident population that is monomorphic for generalism is evolutionary stable. To understand this, z^ϕ must be uninvadable by rare phenotypes with trait value near z^ϕ. Next, we perform an invasion analysis to access this information.

Invasion analysis

The invasion analysis is part of the Adaptive Dynamics framework (Brannstrom et al. 2013). The goal of this analysis is to evaluate whether or not a resident population that is monomorphic for a given trait z can be invadable by a rare mutant with a trait value z' (we use primes to indicate the mutant trait). First part of the analysis is to derive the invasion fitness function, which is the fitness of the rare mutant trying to invade the resident population. Assuming that rare mutants emerge after the ecological and behavioral system reaches its interior equilibrium, we write the following expression for the invasion fitness:

$$\mathcal{F}(z,z') = e_1(z')\hat{p}(z)a\hat{R}_1(z) + e_2(z')[1-\hat{p}(z)]a\hat{R}_2(z) - d. \tag{10}$$

Note that, in the invasion fitness $\mathcal{F}(z,z')$, the foraging effort is is a function of the resident trait. This is because we focus in understanding if there is another trait value that can increase the population fitness for a given foraging effort. Assuming that $\hat{p}(z')$ would obscure the evolutionary stability of the resident behavior.

The natural selection experienced by rare mutants can be captured by the gradient of selection, which is the first derivative of the invasion fitness in relation to the mutant trait z':

$$\frac{\partial \mathcal{F}(z,z')}{\partial z'} = a\left[\hat{p}(z)\frac{(x_1-z')}{\sigma_1^2}e_1(z')\hat{R}_1(z) + (1-\hat{p}(z))\frac{(x_2-z')}{\sigma_2^2}e_2(z')\hat{R}_2(z)\right]. \tag{11a}$$

Which, evaluated at the generalist singularity ($z = z^{\phi}$) yields

$$\left.\frac{\partial \mathcal{F}(z,z')}{\partial z'}\right|_{z'=z=z^{\phi}} = \frac{d}{\sigma_2^2}\left[\frac{e_2(z^{\phi})(e_1(z^{\phi})ar_1 - ld)[(x_1-z^{\phi})\sigma_2^2 - (x_2-z^{\phi})\sigma_1^2]}{\sigma_1^2[e_1(z^{\phi})e_2(z^{\phi})a(r_1+r_2) - ld(e_1(z^{\phi})+e_2(z^{\phi}))]} + x_2 - z^{\phi}\right]. \tag{11b}$$

The conditions for the generalist trait z^{ϕ} to be an an evolutionary stable strategy (ESS) requires that the second derivative of the invasion fitness AII.1b with respect to the mutant trait and evaluated at the singular point to be negative:

$$\left.\frac{\partial^2 \mathcal{F}(z,z')}{\partial z'^2}\right|_{z'=z=z^{\phi}} < 0. \tag{12a}$$

Even if the generalist is evolutionary stable, it does not means that near trajectories converge towards it. There is, if selection gives advantage to phenotypes that are near z^{ϕ}. To understand if generalism is a convergent stable strategy (CSS), the following condition must be true:

$$\left.\frac{\partial^2 \mathcal{F}(z,z')}{\partial z \partial z'}\right|_{z'=z=z^{\phi}} + \left.\frac{\partial^2 \mathcal{F}(z,z')}{\partial z'^2}\right|_{z'=z=z^{\phi}} < 0. \tag{12b}$$

In the appendix II, we show the calculations necessary to evaluate the above conditions.

If both conditions in eq.12 are met, then z^{ϕ} is both and ESS and a CSS. This indicates that the population moves along its fitness surfaces towards the generalist strategy and, once there, remains there. If, on the other hand, z^{ϕ} is a CSS but not an ESS, the population escalates the surface towards z^{ϕ} but, once there, the population can be invaded by near mutants and then departs from generalism. This phenomena is known as evolutionary branching points (Geritz et al. 1997).

Figure 2 shows these possible evolutionary outcomes depending on the strenght of selection σ_1^2 and σ_2^2. The top row of figure 2 shows the pairwise invasibility plots (PIP's), which are the classical numerical simulations for AD (Brannstrom et al. 2013). PIP's shows the trait space for which residents can be invaded by mutants. Black regions demarks the area to which the resident is invadable. The bottom row of these figures shows the respective evolutionary outcomes from the perspective of fitness surfaces. If selection is weak (e.g. $\sigma = \sigma_1 = \sigma_2 = 1.1$), generalism is both convergent and evolutionary stable (figure 2a). Increasing the strength of selection (decreasing σ), generalism becomes evolutionary unstable but is still convergence stable, producing evolutionary branching points in figure 1b). If, however, selection is too strong (e.g. $\sigma = 0.91$), even the condition for the CSS 12b is reversed. In such such a case, generalism is an evolutionary repellor (figure 1c). This means that, although the behavior update function $\zeta(z^o)$ converges to the singularity, a resident population that is monomorphic for z^{ϕ} will not stay there if near mutants arrive. However, the fact that the singularity exists indicates that adaptive behavior causes repellors to act as branching points in our model. This is because the repellor point is formed after the population reaches that point, similar to the dynamics of branching points.

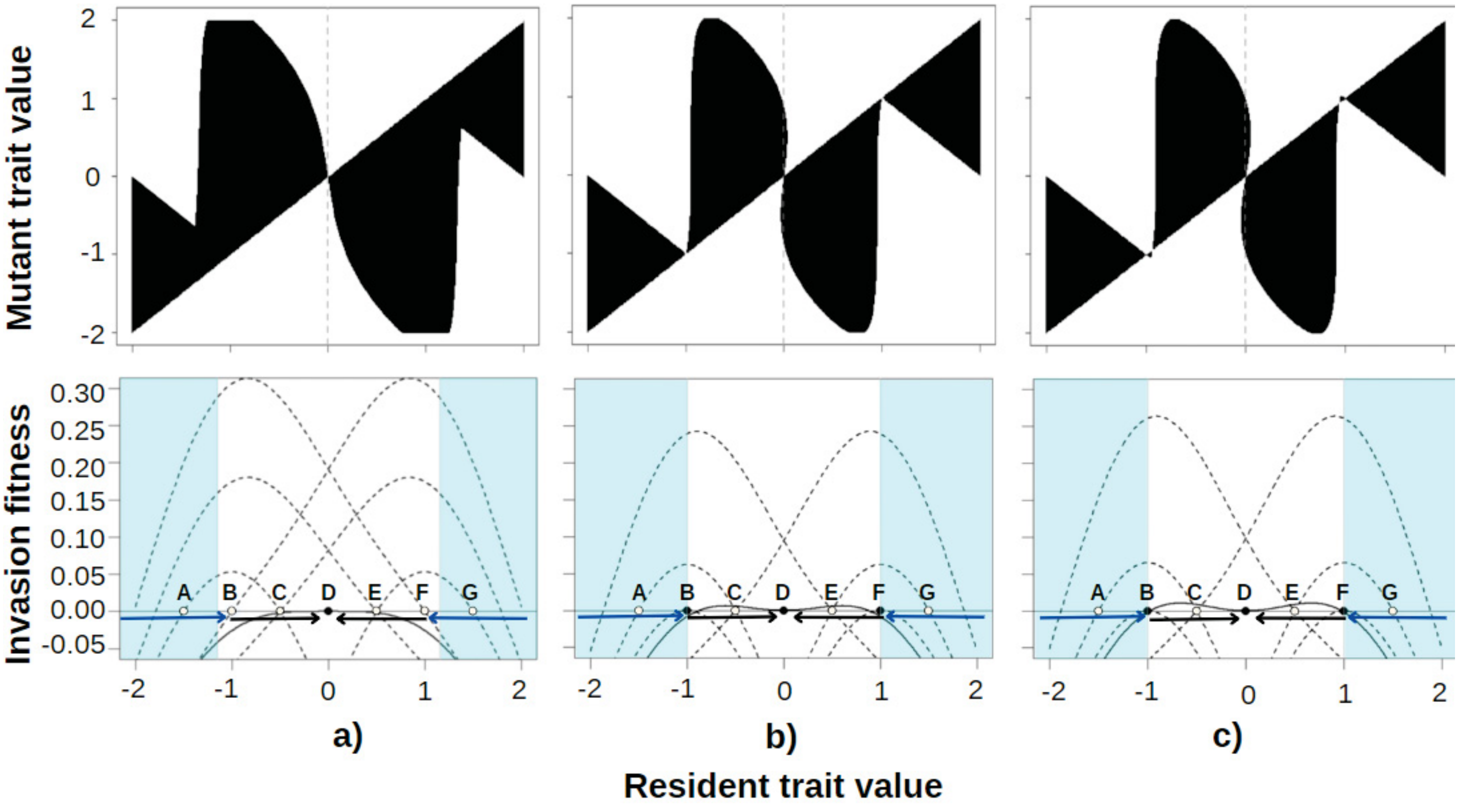

Figure 2: Evolutionary outcomes depending on the resident trait value. Top row how the pairwise invasibility plots (PIP's). The dark regions are the parameter space in which the resident population can be invaded by rare mutants. The vertical dashed lines in the PIP's shows the generalist singularity. The bottom row shows the fitness surfaces. Dashed curves are the fitness surfaces generated by residents that are not at the generalist singularity and solid curve is the surface promoted by the generalist singularity. Black dots are z values that are evolutionary stable, while white dots are not. Dots over the curves are the z values that generated each curve. Black and white dots stands for values that are evolutionary stable and unstable, respectively. The value of the points are: A:-1.5; B:-1; C:-0.5; D:0; E:0.5; F:1; G:1.5. Blue regions show the trait space that generates a specialist singularity. Blue and black arrows shows the direction of evolution for specialists and generalists, respectively. $\sigma_1^2 = \sigma_2^2 = \sigma^2$ in all panels. a) z^ϕ is both an ESS and a CSS ($\sigma^2 = 1.1$); b) z^ϕ is a CSS but not a ESS, forming branching points ($\sigma^2 = 0.93$); c) z^ϕ is neither an ESS nor a CSS, forming a repellor ($\sigma^2 = 0.91$). Initial conditions: $C = 0.5$, $R_1 = 1$, $R_2 = 1$, $p = 0.5$. Parameter values are: $x_1 = -1$, $x_2 = 1$, $a = 1$, $d = 0.4$, $r_1 = r_2 = 1$, $l = 0.25$, $\tau = 2$.

The bottom row of figure2 shows the fitness surfaces produced by consumers with different trait values (the dots over the lines). Note that, if the trait value is not $z = z^\phi$ (the central filled dot marked by letter D), the surface produced indicates that the population is not at the peak. This means that selection would push the population in the direction of increasing its fitness. However, consumers adopt alternative foraging efforts p depending on their trait value. Then, optimal foraging effort inserts a dynamical property to the fitness surface, dampening its slope. Such a dampening effect continues until the population reaches the generalist singularity. This however, only occurs if the singularity exists (the update function converges). If the population have a trait value withing the blue region, foraging optimization would lead to specialization. As a consequence, the singularity would occur at the specialist phenotypes. Due to this dynamical property, evolutionary trajectories converges towards the singularity (if it exists) even when they are repellors. This means that, in our model, repellors may function as evolutionary branchings.

Discussion

We have studied the role of behavioral dynamics to the ecological and evolutionary stability of generalism (polyphagy). In the model, generalism can be ecologically stable even when consumers are optimal specialists ($z = x_i$). This depends on whether the optimal specialist falls within the region for which system trajectories are attracted by the generalist interior equilibrium. This suggests that, depending on system parameters, specialists can behave as ecological generalists. The counter-intuitive idea that phenotypic specialists can behave as generalists is not new. While studying the diet composition of cichlid fishes, Liem (1980) noticed individuals with morphological features that are compatible with specialist consumption still incorporates a wide variety of other items in their diet. This lack of congruence between trait specialization and foraging behavior was known as the 'Liem's paradox' (Liem 1990, Binning et al. 2009). Robinson & Wilson (1998) proposed a solution to this paradox by arguing that specialists may benefit of non-optimal resources that are "easy to use", and thus behave as generalists in certain circumstances. Such an explanation can be evoked as an interpretation of our results. In our model, as the phenotype-specialist consumer depletes its preferred resource, the relative abundance of the non-preferred resource increases. In well-mixed models such the one analyzed here, shifts in relative abundances can be interpreted as shifts in encounter rates (Morozov & Poggiale 2012). Hence, an increase in the abundance of non-preferred resource also increases the chance that consumers encounter them. Consumers then increase their overall energy intake by balancing their foraging behavior towards a generalist consumption strategy. This suggests that optimal specialists may behave as generalists when opportunity is presented (increase in relative abundance) and the payoff is worthy (net-effect energy intake).

In addition to the ecological stability of generalists, we have demonstrated that generalism can be also an evolutionary attractor even for phenotypic specialists. Mainly, this relates to the dynamical nature of the fitness surface when adaptive behavior is considered. As natural selection drives the population towards the fitness peak, behavioral dynamics accommodates consumer foraging effort to the availability and profitability of each resource. Such an accommodation weakens selection. This weakening continues until the consumer population finds itself in the evolutionary singularity of generalism (z^{ϕ}). In a sense, this process is similar to the one described in the seminal paper of Abrams et al. (1993). The paper points out how frequency-dependent selection (FDS) promotes a dynamical surface that traps the population in a region of fitness minima. FDS occurs whenever individual fitness depends on the frequency composition of genotype/phenotype within the current population (Ayala & Campbell 1974, Rueffler et al. 2006, Brisson 2018). As stated in Abrams et al. (1993), FDS "reverses the direction of increasing fitness" whenever the population is not at the stable minima. Here, adaptive behavior functions in a similar way. Behavior causes a rapid change in the fitness surface so that the direction of fitness increase reverts after the population passes through the generalism singularity. Worth note that, here, generalism may be either a fitness minima (z^{ϕ} is a branching point or a repellor) or a fitness maxima. It is, however, always a master-of-none when compared to specialists. This, associated with the fact that generalism attracts trajectories even when consumers are optimal specialists, suggests that adaptive behavior can promote the evolution in the specialism$\longrightarrow$generalism direction.

The dynamism of fitness surfaces promoted by foraging behavior indicates that behavior can be view as both a cause and a consequence of trait evolution. At one hand, the fitness surface experienced by consumers is shaped by the environmental context, which is generated by its current behavior. This produces a set of selective pressures that gives selective advantage to phenotypes that increase individual fitness. This means that behavior plays a causal role in evolution. On the other hand, consumers' with different trait values behave differently. Thus, as the population moves across the fitness surface, different behaviors may be displayed. In this perspective, behavior is a consequence of trait evolution. This is similar to the behavioral-environment feedback loop proposed by Duckworth (2009). In such a feedback, individual behavior is a consequence of the environment. In turn environment is modified due to a given behavioral pattern. The new environment, then, may require alternative behaviors, closing the feedback loop. It is worthy noting that, although changes in trait due to evolution may also represent shifts in environment (Baalen & Huneman 2014), changes in environment does not necessarily drive evolution (Pfennig 2021). As we have shown, the Gaussian function that we have chosen vanishes with the effect of population dynamics in the singularity. This singularity is the same as if an eco-evolutionary feedback loop were considered, suggesting the occurrence of an evo-behavioral feedback loop. In this sense, an evo-behavioral feedback loop is more accurate to describe the role of behavioral in the evolutionary dynamics of our model. This type of feedback is evident in the behavioral update function of our model.

A core conundrum about the role of behavior is whether it promotes or stagnates evolution (Duckworth 2009). In our model, this can be evaluated by iterating the behavior update function $\zeta(z^o)$. At one hand, if z^ϕ is not the fixed point of $\zeta(z^o)$, our analysis shown that behavioral dynamics leads to resource specialization. On the other hand, situations in which z^ϕ is the fixed point implies that natural selection vanishes for the generalist trait value. Hence, behavior promotes evolution if the initial z is a specialist and the generalism singularity exists, or if the initial z is a generalist and the singularity does not exist. Conversely, behavior stagnates evolution if the initial z is a specialist and the singularity does not exist, or if the initial z is a generalist and the singularity exists. In a study, Huey et al. (2003) observed that thermogulatory behavior is likely to hinder physiological adaptations in *Anolis cristatellus* lizards. They themed the phenomena the ”Bogert effect” after Charles Bogert, which started the discussion on the inhibitory role of behavior in evolution (Bogert 1949). Likely in our model, the Bogert effect is mainly discussed as the outcome of the interplay between physiological traits and behavioral dynamics, but it can be extended to morphological properties (Muñoz 2022). The main idea is that behavior buffers the evolving trait against the selective pressures. In our model, this is represented by the dynamism of the fitness landscape that is promoted by foraging behavior. In the case of a generalist that is evolving towards a specialism, this dynamism stagnates consumers in the generalism singularity.

The fact that adaptive behavior allows the existence of singularities that corresponds to generalism implies that, at this point, polyphagous consumers experience no natural selection in despite of the fact that specialism promotes a higher fitness values (the jack-of-all-trades is a master-of-none). If selection is relatively weak, the generalism singularity can be both convergence and evolutionarily stable. In such cases, a consumer population that is monomorphic for the generalist singularity cannot be invaded by any other alternative strategy that maintains the ecological stability of generalists (z^{ϕ} cannot be invaded by any other z' for which criteria eq.4 is maintained). Conversely, Rueffler et al. (2007) found that selective behavior (polyphagous foraging) always leads to specialization. However, this results is a consequence of the non-stable polyphagous behavior of their model. This is not the case in our model since that selective behavior is stable under asymmetrical consumption. This is a property of the adaptive behavior framework that we used in association with the coupled consumer-resource equations. As explained in Uchida et al. (2007), a generalist behavior can only be achieved in asymmetrical cases ($p \neq 0.5$) if resource dynamics is coupled with consumer dynamics (as oppose to constant resource availability). In another study, Peacor et al. (2006) found that plasticity may increase the evolutionary stability of residents. This indicates that plasticity may dampen natural selection. Although behavioral dynamics is a special case of plasticity (see Duckworth 2009), they are analogous mechanisms in our model.

Detecting evolutionary branching in simple ecological models is one of the core advantages of the adaptive dynamics framework (Geritz et al. 1997, Dieckmann & Doebeli 1999). Branching points are not only trait values for which phenotypic divergence occurs, but they are also evolutionary attractors. This means directional selection drives trait adaptation towards the singularity and, once the population reaches it, selection becomes disruptive and pushes the population away from the singularity (Waxman & Gavrilets 2005). Here, we have shown that generalism may represent evolutionary branching points under moderate selection. This happens when generalism is convergence stable (condition 12b is met) but not evolutionarily stable (condition 12a is reversed). As we already discussed, behavior dynamics can promote evolution in the specialism $\longrightarrow$ generalism direction. However, if generalism is a branching point, the direction may be reversed (generalism $\longrightarrow$ specialism direction). A similar phenomena is described by Janz & Nylin (2008) as the *oscillation hypothesis*. This hypothesis proposes a mechanisms for the diversification of phytophagous insects (and further applied to pathogens; Brooks et al. 2019) that is based on the continuous expansion (generalist strategies) and contraction (specialist strategies) of their host-ranges. Given the fact that geographical expansion is a core element in the oscillation hypothesis, it is probably more appropriate for processes of parapatric speciation (although it can be also evoked to explain sympatry). The process of evolutionary branching such as the suggested in our model, is often related to sympatric speciation (Waxman & Gavrilets 2005). If, nonetheless, we replace the geographic expansion of the oscillation hypothesis by behavioral change, both processes may become very similar. But whether adaptive behavior can generate resource range oscillations must be evaluated by implementing a few model extensions, which we discuss bellow.

From our analysis, we already know that generalism is a CSS (z^{ϕ} is convergence stable) when the population is monomorphic for specialism. In order to understand whether oscillations of the type specialism$\longleftrightarrow$generalism indeed arise in our model, one must evaluate whether the disruptive selection that occurs at the branching point drives the population towards the specialist phenotype ($z = x_i$). If so, this would indicate that evolutionary branching leads the population to the initial point (specialism), and then, as we have shown, is again attracted towards generalism, closing the oscillation dynamics (Dennis et al. 2011). The theory of Adaptive Dynamics (AD) (Abrams 2005) is an analytical framework that relies on several simplifying assumptions (e.g. mutations are rare, evolution is slow, populations are monomorphic). For this reason, evaluating the dynamical outcome of the predictions provided by the AD must usually be verified with numerical simulations of analogous models. To this end, one may need to either incorporate a mutation rate so that the population can evolve by small mutational steps. This can be either incorporated in the current model (Lion 2017), or by building an analogous model with individual based models (Doebeli & Ispolatov 2010). Another possibility is to write a replicator-based polymorphic version of our model. This would incorporate shifts in the composition of a polymorphic population without the necessity of mutation events (Cressman et al. 2017, Lion 2017). Then, it would be possible to numerically analyze the evolution of consumers by tracking the change in the frequency composition. An oscillation may emerge if the frequency of specialist and generalist phenotypes shifts in cyclic dynamics.

Searching for absolute reasons to call an organism a generalist will undoubtedly lead to claims against the existence of such a strategy. Claims such as ”evolution always leads to specialism” (Loxdale & Harvey 2023) are misleading for at least two reasons. The first is that adaptation is not evolution and natural selection is only one of the forces driving evolutionary changes (Lande 1976). Hence, even if adaptation is considered synonym of specialization there is no reason to believe that specialization is the inevitable outcome of long-term evolution. Second, natural selection vanishes for any population residing at the evolutionary singularity, this serves for both specialists and generalists. As we have shown, adaptive behavior plays a relevant role in generating these singularities. Given the fact that behavioral change occurs much faster then evolutionary dynamics, behavior accommodations can dampen (and even vanish) with the selective pressures over adaptive traits. We are aware of the simplicity of our model an thus our conclusions must be interpreted with cautiousness. More complex environments may indeed hinder the stability of generalism. Yet, evolutionary singularities may appear in the generalist region whenever behavioral optimization generates a dynamical fitness surface. That is not to say that generalism is as common in nature as specialism. Rather, we argue that generalism can be an adaptation that is both evolutionarily and convergence stable, and it does not necessarily depends on environmental fluctuations. In fact, it may only require resource diversity and behavioral accommodation, two factors that are relatively ubiquitous in nature.

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Appendix I

Let $f_i(t) = R_i(t)/(\sum_{i=1}^{n} R_i(t))$ be the frequency of resource i in the community pool of resources. Then, by the chain rule from calculus, one can easily show that the temporal dynamics of f_i is described by the following replicator type equations:

$$\begin{aligned} \frac{df_i}{dt} &= f_i \left[\frac{dR_i}{R_i dt} - \sum_{i}^{n} f_i \frac{dR_i}{R_i dt} \right] \\ &= f_i \left[r_i - lR_i - \sum_{i}^{n} f_i \left(r_i - lR_i \right) \right]. \end{aligned} \tag{AI.1a}$$

Then, at the point where $df_i/f_i dt = 0$, the f_i is

$$f_i = 1 - \frac{\sum_{j \neq i}^{n} f_j (r_j - lR_j)}{r_i - lR_i}. \tag{AI.1b}$$

Now, if we replace $R_j = K_j = r_j/l$ in the above, it can be shown that $f_i = 1$. There is, the relative abundance of i is unity in despite of the fact that j is not extinct. However, since that j has no effect on either i or in the specialist consumer, it becomes isolated from the rest of the community.

This indicates that consumers behaving as generalists can maintain the flow of information throughout the interacting species within the community. This is clear in the fact that, if consumers behave as generalists, the actual frequency of each resource can be calculated by replacing $R_1 = fR$ and $R_2 = (1-f)R$, setting equation 1d to zero and solving for f, which leads to the following quantity for the interior equilibrium of f:

$$\hat{f} = \frac{e_2}{e_1 + e_2}. \tag{AI.2}$$

Then, replacing $R_1 = fR$ and $R_2 = (1-f)R$ in the consumer dynamics (eq. 1a), setting it to zero and solving for the overall resource dynamics R leads to

$$\hat{R}(f,p) = \frac{d}{a\left[e_1 f p + e_2 (1-f)(1-p)\right]} \tag{AI.3}$$

Next, we set eq. 1b to zero and solve for C:

$$\hat{C}(R,f,p) = \frac{r_1 - lRf}{ap}. \tag{AI.4}$$

To find the $p = \hat{p}$ for which the consumers behave as generalist we first write the temporal dynamics of the overall density of resources $R = R_1 + R_2$ considering that $R_1 = fR$:

$$\begin{aligned} \frac{dR}{dt} &= \frac{dR_2}{dt} + \frac{dR_2}{dt} \\ &= R\left[\bar{r} - lR\left(f^2 + (1-f)^2\right) - Ca\left(fp + (1-p)(1-f)\right)\right]. \end{aligned} \tag{AI.5}$$

Where $\bar{r} = \sum_i^n f_i r_i = f r_1 + (1-f) r_2$. Then, solving AI.5 for p yields

$$\hat{p}(R,C,f) = \frac{1}{f-(1-f)}\left[\frac{\bar{r} - l\hat{R}(f,p)[f^2 + (1-f)^2]}{a\hat{C}(R,f,p)} - (1-f)\right] \tag{AI.6}$$

Now, we do the following: (1) replace $\hat{f}$ in $\hat{R}(f,p)$, (2) $\hat{R}(f,p)$ and $\hat{f}$ in $\hat{C}(R,f,p)$, and (3) plugin them all in $\hat{p}(R,C,f)$ leads to

$$\hat{C} = \frac{r_1 + r_2}{a}\left[1 - \frac{ld(e_1+e_2)}{ae_1e_2(r_1+r_2)}\right], \tag{AI.7a}$$

$$\hat{R}_1 = \frac{d}{ae_1}, \tag{AI.7b}$$

$$\hat{R}_2 = \frac{d}{ae_2}. \tag{AI.7c}$$

$$\hat{p} = \frac{e_2(e_1ar_1 - ld)}{e_1e_2a(r_1+r_2) - ld(e_1+e_2)}. \tag{AI.7d}$$

Which is the interior equilibrium of the generalist behavior.

Appendix II

Assuming population homogeneity allows us to describe individual fitness by the per capita rate of increase of the consumer dynamics. Thus, the fitness of resident consumers is given by

$$F(z,p,\mathbf{R}) = \frac{dC}{Cdt} = e_1(z)paR_1 + e_2(z)(1-p)aR_2 - d \tag{AII.1a}$$

Then, assuming that rare mutants emerge after the ecological and behavioral system reaches its interior equilibrium, we write the following expression for the invasion fitness:

$$\mathcal{F}(z,z') = e_1(z')\hat{p}(z)a\hat{R}_1(z) + e_2(z')[1-\hat{p}(z)]a\hat{R}_2(z) - d. \tag{AII.1b}$$

The natural selection experienced by rare mutants can be captured by the gradient of selection, which is the first derivative of the invasion fitness in relation to the mutant trait z':

$$\frac{\partial\mathcal{F}(z,z')}{\partial z'} = a\left[\hat{p}(z)\frac{(x_1-z')}{\sigma_1^2}e_1(z')\hat{R}_1(z) + (1-\hat{p}(z))\frac{(x_2-z')}{\sigma_2^2}e_2(z')\hat{R}_2(z)\right]. \tag{AII.2a}$$

Which, evaluated at the generalist singularity ($z = z^{\phi}$) yields

$$\left.\frac{\partial\mathcal{F}(z,z')}{\partial z'}\right|_{z'=z=z^{\phi}} = \frac{d}{\sigma_2^2}\left[\frac{e_2(z^{\phi})(e_1(z^{\phi})ar_1 - ld)[(x_1-z^{\phi})\sigma_2^2 - (x_2-z^{\phi})\sigma_1^2]}{\sigma_1^2[e_1(z^{\phi})e_2(z^{\phi})a(r_1+r_2) - ld(e_1(z^{\phi})+e_2(z^{\phi}))]} + x_2 - z^{\phi}\right]. \tag{AII.2b}$$

Now, to evaluate the curvature of the invasion fitness surface we calculate the second derivative of the invasion fitness with respect to the mutant trait:

$$\frac{\partial^2\mathcal{F}(z,z')}{\partial z'^2} = a\left[\hat{p}(z)\frac{e_1(z')\hat{R}_1(z)}{\sigma_1^2}\left(\frac{(x_1-z')^2}{\sigma_1^2} - 1\right) + [1-\hat{p}(z)]\frac{e_2(z')\hat{R}_2(z)}{\sigma_2^2}\left(\frac{(x_2-z')^2}{\sigma_2^2} - 1\right)\right]. \tag{AII.3a}$$

Which, again, evaluated at the generalist singularity ($z = z^{\phi}$) leads to

$$\left.\frac{\partial^2\mathcal{F}(z,z')}{\partial z'^2}\right|_{z'=z=z^{\phi}} = d\left[\hat{p}(z^{\phi})\left[\frac{1}{\sigma_1^2}\left(\frac{(x_1-z^{\phi})^2}{\sigma_1^2} - 1\right) - \frac{1}{\sigma_2^2}\left(\frac{(x_2-z^{\phi})^2}{\sigma_2^2} - 1\right)\right] + \frac{1}{\sigma_2^2}\left(\frac{(x_2-z^{\phi})^2}{\sigma_2^2} - 1\right)\right]. \tag{AII.3b}$$

The last quantity we need to evaluate relates to the criteria for the CSS. That is:

$$\frac{\partial^2 \mathcal{F}(z,z')}{\partial z \partial z'} = a\left[\frac{(x_1 - z')}{\sigma_1^2} e_1' \frac{\partial\left[\hat{R}_1 \hat{p}\right]}{\partial z} + \frac{(x_2 - z')}{\sigma_2^2} e_2' \frac{\partial\left[\hat{R}_2(1-\hat{p})\right]}{\partial z}\right]. \tag{AII.4a}$$

Where the partial derivatives within the brackets of the above can be calculated by the product rule from calculus, yielding:

$$\frac{\partial\left[\hat{R}_1 \hat{p}\right]}{\partial z} = \frac{de_1\left(z - x_1\right)}{a\left(dl\left(e_2 + e_1\right) - r_2 - r_1\right)}\left[\frac{dl + \left(dle_1 - ar_1\right)}{\sigma_1^2} - \frac{dl\left(dle_1 - ar_1\right)\left(\frac{(z-x_2)e_2}{\sigma_2^2} + \frac{(z-x_1)e_1}{\sigma_1^2}\right)}{\left(dl\left(e_2 + e_1\right) - r_2 - r_1\right)\left(z - x_1\right)}\right] \tag{AII.4b}$$

and

$$\frac{\partial\left[\hat{R}_2(1-\hat{p})\right]}{\partial z} = \frac{de_2(z - x_2)}{a}\Big[\frac{dl + \left(dle_2 - r_2 + (a-1)\, r_1\right)}{\sigma_2^2\left(dl\left(e_2 + e_1\right) - r_2 - r_1\right)} \tag{AII.4c}$$

$$- \frac{dl\left(dle_2 - r_2 + (a-1)\, r_1\right)\left(\frac{(z-x_2)e_2}{\sigma_2^2} + \frac{(z-x_1)e_1}{\sigma_1^2}\right)}{\left(dl\left(e_2 + e_1\right) - r_2 - r_1\right)^2\left(z - x_2\right)}\Big]. \tag{AII.4d}$$

Lastly, one need to evaluate at the point

$$\left.\frac{\partial^2 \mathcal{F}(z,z')}{\partial z \partial z'}\right|_{z'=z=z^{\phi}}. \tag{AII.4e}$$

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5 CONSIDERAÇÕES FINAIS

Nesta tese, formalizamos alguns argumentos relativos à evolução da gama de recursos dos consumidores. De forma geral, esses argumentos estão relacionados a como ocorre a diversificação do consumo sem mudanças aparentes no ambiente. Como discutimos durante os capítulos, isso tem algumas implicações interessantes para o surgimento de surtos de doenças. Na natureza, essas emergências podem ser percebidas como inesperadas quando interpretadas sem considerar alguns dos mecanismos que investigamos.

No capítulo I, nosso principal argumento é que mudanças na composição de cepas dentro de uma população de patógenos não requerem mudanças nas condições ambientais nem a ocorrência de novas mutações. Em vez disso, eles podem ser conduzidos pela dinâmica interna dos patógenos (por exemplo, interações intrapopulacionais). Assumindo que diferentes cepas podem ter diferentes capacidades para explorar hospedeiros alternativos, mudanças na composição produzem eventos de expansão e retração de sua gama de hospedeiros. Nesse sentido, mostramos que, quando a dinâmica interna é regida por uma dinâmica de jogo evolutivo, a composição de cepas sofre oscilações que podem ser estáveis (ciclos limites) ou instáveis (ciclos heteroclínicos). De qualquer forma, essas oscilações não podem ser previstas pelo monitoramento da prevalência da doença em populações de hospedeiros reservatórios. Em vez disso, deve-se dar atenção à diversidade e composição de patógenos a fim de entender se suas frequências estão seguindo algum tipo de oscilação determinística. Ao monitorar esse tipo de informação, surtos causados por eventos de transbordamento de patógenos podem ser antecipados e contidos. Além disso, o modelo que desenvolvemos também faz parte da inovação deste capítulo. Nosso modelo aceita qualquer tipo de dinâmica de jogo entre cepas de patógenos. A dinâmica do jogo pode ser facilmente descrita por uma rede de interação. Além disso, incorporamos um parâmetro fenomenológico que descreve se a superinfecção é letal, resulta em exclusão competitiva ou em coinfecção. Como mostramos, o resultado da superinfecção desempenha um papel relevante na geração das oscilações e, portanto, na evolução da gama de hospedeiros do patógeno.

No capítulo II, propomos o que chamamos de hipótese das *taxas de contato heretogêneo* (HCR). Essa hipótese afirma que, isoladamente, a HCR é suficiente para diversificar o meio seletivo vivenciado pelos patógenos. Então, dada a heterogeneidade natural das redes de contato do mundo real, a diversificação de patógenos pela seleção natural pode ser considerada a regra, e não a exceção. O único requisito para a hipótese da HCR é que os patógenos apresentam trade-offs de história de vida.

De acordo com nossas expectativas, nossas simulações indicam que o aumento da heterogeneidade de contato também aumenta a variabilidade de meios seletivos entre as populações hospedeiras. Este efeito não requer variabilidade em nenhum outro parâmetro do hospedeiro. Isto é, mesmo que os hospedeiros sejam clones idênticos e forneçam exatamente o mesmo microambiente, a diversificação ainda ocorre. O principal mecanismo subjacente a este fenômeno é a variabilidade na oportunidade ecológica promovida pela HCR. Ainda neste capítulo, mostramos que um padrão epidemiológico fonte-dreno emerge da estrutura da rede de contatos. Esta propriedade emergente tem implicações para a gestão de surtos de doenças e programas de vacinação em todas as paisagens. Outra sugestão interessante do nosso modelo é que as pressões seletivas locais que promovem a diversificação são o resultado da estrutura da rede. Isso significa que a adaptação local pode não ser impulsionada por pressões que atuam localmente, mas sim como uma propriedade emergente de toda uma paisagem.

No capítulo III, nosso principal argumento é que a dinâmica comportamental promove a estabilidade evolutiva do generalismo (polifagia). Como mostramos por meio de modelos matemáticos, o comportamento do consumidor produz uma superfície de aptidão dinâmica que aprisiona a população em uma região que favorece a evolução do generalismo. Isso indica que os generalistas podem evoluir como resultado da seleção natural em ambientes estáveis. Isso vai contra as perspectivas anteriores de que a evolução generalista requer flutuações ambientais, ou são apenas estados transitórios que invariavelmente evoluem para a especialização. Em nosso modelo, isso não ocorre, pois, em certas circunstâncias, os especialistas ótimos evoluem para o generalismo. De um modo geral, este capítulo enfatiza que a seleção natural pode expandir o repertório de recursos dos consumidores mesmo quando isso significa uma redução na aptidão geral. Isso parece contraditório, mas explicamos que as decisões comportamentais produzem uma superfície de aptidão diferente daquela que prevemos sem considerar o comportamento.

Para concluir, esta tese aponta como os organismos podem lidar com sua luta pela existência aproveitando a oportunidade ecológica. Isso não quer dizer que eles parem de lutar. Em vez disso, sugere que a persistência depende da manifestação de capacidades ocultas. Por esta razão, compreender que os repertórios de recursos são maiores do que os observados e que as superfícies/paisagens de fitness são intrinsecamente dinâmicas é crucial para prever trajetórias evolutivas e seus resultados.

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