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Programa de Pós-Graduação em Ecologia

Coevolução em redes de interação antagonista: estrutura e dinâmica

Cecilia Siliansky de Andreazzi

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Para meus pais, Fátima e Marco .

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"Nothing is more central to a dialectical understanding of nature than the realization that the conditions necessary for the coming into being of some state of the world may be destroyed by the very state of nature to which they gave rise. "[1]

Lewontin & Levins

[1] Nada é mais importante para uma compreensão dialética da natureza do que a percepção de que as condições necessárias para a vinda à existência de algum estado do mundo podem ser destruídas pelo próprio estado de natureza a que deu origem (Tradução livre).

Resumo

As pressões seletivas impostas por interações ecológicas são uma das forças que moldam a adaptação por seleção natural em populações. Entre os resultados possíveis das pressões seletivas impostas por interações está a coevolução, isto é, mudanças evolutivas recíprocas que ocorrem nas populações das espécies que interagem. Um dos principais desafios para a ecologia evolutiva é entender se e como o processo coevolutivo ocorre quando espécies interagem com muitas outras espécies formando redes de interações. Nesta tese desenvolvi, com a ajuda de colaboradores, modelos que descrevem a coevolução entre espécies que interagem de forma antagonista. Interações antagonistas são interações ecológicas interespecíficas que resultam em consequências negativas para a aptidão de indivíduos de uma das espécies envolvidas e positivas para indivíduos da outra espécie. Busquei uma melhor compreensão sobre os mecanismos ecológicos e evolutivos responsáveis pela formação, manutenção e evolução das redes de interação antagonista. Em primeiro lugar, encontrei que a assimetria da seleção influenciou a dinâmica evolutiva em antagonismos. A dinâmica coevolutiva gerou corridas armamentistas quando a intensidade da seleção foi maior sobre as vítimas do que sobre os exploradores. Por outro lado, os valores dos fenótipos flutuaram quando a intensidade da seleção foi maior sobre os exploradores do que sobre as vítimas. No entanto, a dinâmica coevolutiva dependeu da estrutura das redes formadas por antagonistas. Redes aninhadas favoreceram a evolução de resistência em vítimas atacadas por exploradores generalistas. A dinâmica evolutiva também reorganizou as redes de interação e, especialmente em cenários nos quais a seleção favoreceu forte acoplamento fenotípico, formou módulos de espécies interagentes. Em segundo lugar, encontrei que regras de interação baseadas no acoplamento fenotípico ou em barreiras fenotípicas reproduziram a estrutura de redes antagonistas empíricas, mas as duas relações funcionais entre fenótipos e aptidão tenderam a subestimar o aninhamento e superestimar a modularidade das redes empíricas. No entanto, a evolução das características foi diferentemente moldada por essas relações funcionais, sendo mais flutuante no modelo de acoplamento fenotípico e mais direcional no modelo de barreiras fenotípicas. Portanto, a coevolução mediada por diferentes relações funcionais resultou em diferentes dinâmicas coevolutivas mas não teve impacto sobre a organização das redes de interação antagonistas. Em terceiro lugar, estudei como variações nas abundâncias e nos fenótipos estão relacionadas e encontrei que a coevolução rápida mediada por forte pressões seletivas impostas por interações ecológicas pode resultar em uma baixa variabilidade nas abundâncias das populações e alta variabilidade fenotípica. Em contraste, em cenários nos quais a seleção imposta por interações é fraca, encontrei uma alta variabilidade nos tamanhos populacionais e baixa variabilidade fenotípica. Portanto, a rápida resposta evolutiva reduziu as flutuações nos tamanhos populacionais, reduzindo extinções devido a flutuações demográficas. Porém, este resultado foi influenciado pela estrutura da rede: a modularidade aumentou a estabilidade das interações enquanto que o aninhamento esteve associado a maior flutuação demográfica. Por fim, estudei espalhamento de um parasita que infecta diferentes espécies de hospedeiros e que pode ser transmitido por meio da predação de um hospedeiro infectado ou por meio de vetores biológicos. Combinei as diferentes redes antagonistas formadas a partir das interações mediadas por cada mecanismo de transmissão em uma rede de interação múltipla espacialmente explícita. Por meio de um modelo matemático, obtive que a transmissão do parasita é maximizada quando ambos os mecanismos de transmissão são considerados ao mesmo tempo e quando os processos ocorrem com probabilidade semelhante. A análise da cartografia da rede múltipla aliada a simulações de imunização de diferentes tipos de hospedeiros mostraram que a

estrutura da rede múltipla pode indicar o papel que cada espécie de hospedeiro desempenha na transmissão do parasita em um determinado ecossistema.

Abstract

The selective pressures imposed by ecological interactions are one of the forces shaping adaptation by natural selection in populations. One of the possible outcomes of the selective pressures imposed by interactions is the coevolution, *i.e.* reciprocal evolutionary change occurring in populations of species that interact. A major challenge for evolutionary ecology is to understand if and how the coevolutionary process occurs when species interact with many other species forming networks of interactions. In this thesis I developed, with the help of collaborators, models that describe the coevolution between species that interact antagonistically. Antagonistic interactions are interspecific ecological interactions that result in negative consequences for the fitness of individuals of one species and positive to individuals of other species. I sought a better understanding of the ecological and evolutionary mechanisms responsible for the assemblage, maintenance and evolution of antagonistic interaction networks. First, I found that the selection asymmetry influenced the evolutionary dynamics in antagonisms. The coevolutionary dynamics led to coevolutionary arms races when the intensity of selection was stronger on victims than on exploiters. On the other hand, traits fluctuated when the intensity of selection was stronger on exploiters than on victims. However, the coevolutionary dynamics depended on the structure of the antagonistic networks. Nested networks favored the evolution of resistance in victims attacked by generalist exploiters. The evolutionary dynamics also reorganized the interaction networks, especially in scenarios in which selection favored a strong trait matching, forming modules of interacting species. Second, I found that interaction rules based on trait matching or exploitation barriers reproduced the structure of empirical antagonistic networks, but the two functional relationships between species traits and fitness tended to underestimate nestedness and overestimate modularity of empirical networks. However, trait evolution was differently shaped by these functional relationships, being more fluctuating in the trait matching model and more directional in the exploitation barriers model. Therefore, coevolution mediated by different functional relationships resulted in different coevolutionary dynamics but had no impact on the organization of antagonistic interaction networks. Third, I studied how species abundances and trait variations are related and I found that the rapid coevolution mediated by strong selective pressures imposed by ecological interactions might result in a low variability in the abundance of populations and high trait variability. In contrast, I found a high variability in population sizes and low trait variability in scenarios in which selection imposed by interactions was weak. Therefore, rapid evolutionary response reduced fluctuations in population sizes, reducing extinctions due to demographic fluctuations. However, this result was influenced by the structure of the interaction network: modularity increased stability of the interactions while nestedness was associated with increased population fluctuation. Finally, I studied the spreading of a parasite that infects different host species and can be transmitted by the predation of an infected host or through biological vectors. I combined the different antagonistic networks formed by the interactions mediated by each transmission mechanism in a spatially explicit network of multiple interactions. By means of a mathematical model, I found that the transmission of the parasite is maximized when both transmission mechanisms are considered at the same time and when the processes are equally likely. The multiplex cartography analysis combined with immunization of different types of hosts simulations showed that multiple network structure could indicate the role each host species plays in parasite transmission in a given ecosystem

Sumário

Introdução geral

A sobrevivência e reprodução de todo organismo vivo depende de indivíduos de outras espécies. As interações entre espécies contribuem para a aptidão dos indivíduos porque fornecem recursos ou riscos que não estariam presentes em uma única espécie (Thompson 2013) e por isso são fundamentais para o processo evolutivo. Diferentes tipos de interações ecológicas resultam em pressões seletivas potencialmente conflitantes que influenciam a evolução e diversificação de características fenotípicas (Strauss & Irwin 2004, Siepielski & Benkman 2010, Raimundo *et al.* 2014). Por isso, as interações ecológicas podem ser consideradas um dos principais mecanismos relacionados ao processo de adaptação por seleção natural (Wade 2007). A adaptação resultante das pressões seletivas impostas por interações pode ser gerada por coevolução: o processo pelo qual ocorrem mudanças evolutivas recíprocas entre as espécies que interagem (Thompson 1994). Um número crescente de estudos tem reportado padrões temporais e espaciais na adaptação recíproca entre espécies que interagem, o que reanimou o debate sobre a importância do processo coevolutivo para a ecologia evolutiva (Thompson 2005, Wade 2007, Schoener 2011).

Um dos principais desafios relacionados ao entendimento processo coevolutivo é que, em geral, as espécies interagem com muitas outras espécies (Thompson 1999). Comunidades ecológicas são compostas por diversas espécies em um determinado local que dependem de outras para alimentação, proteção ou reprodução, podendo interagir de várias formas. Por exemplo, muitas espécies de plantas tem suas folhas consumidas por insetos fitófagos, que por sua vez podem ser predados ou parasitados por indivíduos de muitas outras espécies. Essas mesmas plantas podem depender de abelhas polinizadoras e vertebrados dispersores de sementes para sua reprodução, formando, por conseguinte, redes de interações. A idéia de uma rede de interações entre espécies é tão antiga quanto a contemplação do emaranhado *("entangled bank"*) de Darwin, mostrando a potencial importância das redes em ecologia e evolução (Bascompte 2009).

Estrutura de redes antagonísticas e a dinâmica coevolutiva

Antagonismos são interações que resultam em consequências negativas para a aptidão de indivíduos de uma das espécies envolvidas e positiva para indivíduos da outra espécie e

exemplos incluem parasitismo, predação e herbivoria. Já existe um corpo teórico sólido sobre a dinâmica coevolutiva esperada para pares ou grupos pequenos de espécies antagonistas, prevendo dinâmicas coevolutivas como antagonismo atenuado, polimorfismo flutuante, seleção disruptiva, intensificação das características de ataque e defesa e alternância no ataque a potenciais vítimas (Brown & Vincent 1992, Abrams 2000, Thompson 2005, Gavrilets 1997, Gandon & Michalakis 2002). Essas previsões estão geralmente associadas aos diferentes pressupostos dos modelos, como a natureza das características das espécies, os mecanismos que intermediam a interação e a existência de pressões seletivas adicionais. É possível classificar a previsão desses modelos em ocorrência ou não de ciclos nos tamanhos populacionais e/ou ciclos nas características de ataque e defesa das espécies que interagem (Abrams 2000). Ciclos populacionais e/ou ciclos fenotípicos estão geralmente relacionados à seleção de características fenotípicas que podem ser distribuídas ao longo de eixos bidirecionais, ou seja, características que podem sofrer seleção direcional para valores maiores ou menores que a média (Abrams 2000). A existência de outras pressões seletivas sobre a característica, como seleção estabilizadora, também está relacionada a ocorrência de ciclos (Abrams 2000, Nuismer *et al.* 2005). Já a ausência de ciclos está relacionada à seleção de características distribuídas ao longo de eixos unidirecionais, isto é, características que apresentam um limiar e só podem sofrer seleção direcional para valores maiores que o limiar. A ausência de pressões seletivas adicionais sobre as características também reduz a chance de ocorrer ciclos (Abrams 2000).

Em interações antagonistas é esperada uma grande variação temporal e espacial no grau de complementaridade fenotípica entre espécies que interagem (Thompson 2005, Penczykowski *et al.* 2016). Por exemplo, quando uma espécie de explorador interage com duas ou mais espécies de vítimas, o alvo preferencial do explorador pode variar conforme o nível relativo de defesa das vítimas. Esta variação temporal pode ser gerada por alternância coevolutiva. A alternância coevolutiva é o mecanismo proposto para a coevolução em pequenos conjuntos de espécies e está relacionada a exploradores que apresentam hierarquias de preferências entre as vítimas (Thompson 2005). Neste caso, a seleção natural favorece indivíduos de exploradores que prefiram vítimas com menos defesas. Uma maior pressão de ataque sobre as vítimas com menos defesas, por sua vez, impõe seleção para o aumento das defesas nas espécies de vítimas preferidas, enquanto a seleção para a redução de custos com defesas favorece a diminuição nas defesas das espécies de vítimas que são menos atacadas. A alternância coevolutiva prossegue por meio de repetidos ciclos de mudança evolutiva nas preferências dos exploradores e nos níveis relativos de defesa das vítimas (Davies & Brooke

1989, Nuismer & Thompson 2006, Thompson 2005, Thorogood & Davies 2013). Alternância com intensificação das características pode ser observada em situações onde a evolução das preferências dos exploradores ocorre mais rapidamente do que a resposta evolutiva das vítimas. A alternância com intensificação das características resulta em uma manutenção dos níveis de defesa das vítimas menos atacadas e em um aumento global das características de ataque e defesa (Nuismer & Thompson 2006, Thompson 2005). Em geral, as expectativas teóricas são apoiadas por evidências empíricas, tanto experimentais (Gaba & Ebert 2009, Brockhurst & Koskella 2013) quanto em populações naturais (Martiny *et al.* 2014, Thompson 2005, Thorogood & Davies 2013, Benkman & Parchman 2013, Soler *et al.* 2001, Davies & Brooke 1989, Vikan *et al.* 2011). Por exemplo, aves parasitas de ninho como o cuco podem apresentar alternância entre as espécie de aves hospedeiras nos locais onde ocorre (Soler 2014). A alternância coevolutiva é, portanto, uma dinâmica possível em antagonismos multiespecíficos. Um próximo passo no estudo da dinâmica coevolutiva em antagonismos envolve compreender como a estrutura das interações multiespecíficas molda as mudanças evolutivas recíprocas e altera as dinâmicas esperadas para sistemas formados por poucas espécies.

Interações antagonistas tendem a ser organizadas em compartimentos (ou módulos) internamente conectados e pouco conectados entre si (Olesen *et al.* 2007, Guimarães, Rico-Gray, *et al.* 2007, Thébault & Fontaine 2010). A ocorrência de compartimentos em sistemas antagonistas pode estar relacionada às forças seletivas divergentes características desses sistemas, dessa forma representado unidades coevolutivas restringidas pela história evolutiva pregressa e pela alta especificidade e intimidade das interações (Thébault & Fontaine 2010, Pires & Guimaraes 2012). Além disso, os efeitos indiretos da competição aparente parecem ser prevalentes em simulações descrevendo a dinâmica ecológica em redes antagonistas, o que restringiria o compartilhamento de parceiros de interação e consequentemente promoveria baixa conectância e alta modularidade nas redes (Thébault & Fontaine 2010). Um elevado grau de modularidade aumentaria a estabilidade e resiliência destas redes, já que a teoria prediz que cascatas de extinções se propagariam mais lentamente através de uma rede organizada em módulos do que em uma rede não-modular (Olesen *et al.* 2007, Tylianakis *et al.* 2010).

Padrões aninhados de interações também são observados em alguns antagonismos (Fontaine *et al.* 2011, Pires & Guimaraes 2012). Em redes aninhadas observam-se algumas propriedades, como a presença de um núcleo coeso de espécies, formado por exploradores e vítimas generalistas que interagem entre si; e especialização assimétrica, caracterizada pela

tendência de especialistas a interagirem com as espécies generalistas (Vazquez *et al.* 2005). Um padrão aninhado de interações pode ser gerado caso a combinação de conjuntos de espécies antagonistas seja moldada pelas diferenças interespecíficas em abundâncias ou habilidades de colonização (Lewinsohn *et al.* 2006, Krishna *et al.* 2008, Canard *et al.* 2012, 2014). O aninhamento é característico e bem documentado em interações mutualistas (Guimarães, Machado, *et al.* 2007), embora um número crescente de trabalhos venha documentando este padrão em redes antagonistas (Vázquez *et al.* 2007, Graham *et al.* 2009, Fortuna *et al.* 2010, Flores *et al.* 2011, Pires & Guimaraes 2012). A presença de um núcleo coeso de espécies torna o sistema mais redundante à perda de interações ou espécies (Bascompte & Jordano 2007, Bascompte 2009, Vázquez *et al.* 2007).

Efeitos da dinâmica coevolutiva sobre a estrutura

As redes de interação entre espécies não só são agentes no processo coevolutivo como também são produto dele. Espécies e interações evoluem de forma dialética (Levins & Lewontin 1985) em consequência de sua interdependência. Processos ecológicos e evolutivos como a diversificação, adaptação e extinção atuam na constante transformação das redes, podendo alterar tanto a evolução adaptativa quanto a coexistência de espécies (Post & Palkovacs 2009, Loeuille 2010, Odling-Smee *et al.* 2013). Entender como as redes ecológicas são estruturadas pela seleção natural recíproca e como elas persistem ao longo do tempo e espaço, dado que podem estar sob constante e rápida mudança coevolutiva, também constituem um grande desafio da atualidade (Thompson 2013).

A diversificação mediada pela coevolução entre espécies que interagem de forma antagonista tende a gerar redes com estruturas aninhadas e modulares (Beckett & Williams 2013). Apesar das redes apresentarem estruturas que são consistentes ao longo do tempo, as identidades das espécies e das interações tendem a ser temporalmente variáveis em redes empíricas (Olesen, Stefanescu, *et al.* 2011, Petanidou *et al.* 2008, Olesen *et al.* 2008, Pilosof *et al.* 2013, Krasnov *et al.* 2006). Vários atributos biológicos influenciam a organização das redes ecológicas, como por exemplo a distribuição das abundâncias (Canard *et al.* 2014) e a heterogeneidade do habitat (Schleuning *et al.* 2012, Flores *et al.* 2013). Além disso, características fenotípicas das espécies como morfologia e fenologia podem modular a organização das redes, determinando a probabilidade de uma interação (Santamaría & Rodríguez-Gironés 2007, Stang *et al.* 2006) ou restringindo a interação entre espécies que poderiam potencialmente interagir (Olesen, Bascompte, *et al.* 2011).

No contexto de interações multiespecíficas, diferentes mecanismos de interação podem estar relacionados ao surgimento de aninhamento e modularidade (Santamaría & Rodríguez-Gironés 2007). Estruturas aninhadas podem emergir devido ao forrageamento ótimo dos indivíduos e a variação intrapopulacional da escolha dos recursos (Pires *et al.* 2011, Lemos-Costa *et al. in press*, Araújo *et al.* 2010). Em contraste, as limitações do mecanismo de acoplamento fenotípico devem restringir interações, promovendo assim o aparecimento de módulos (Krasnov *et al.* 2012). As pressões seletivas exercidas por parceiros antagonistas dependem da biologia da interação (Nuismer & Thompson 2006). Se a probabilidade de ataque está relacionada com a similaridade entre os fenótipos dos exploradores e das vítimas, então a seleção natural favorece o acoplamento fenotípico dos exploradores com suas vítimas e vítimas com fenótipos menos similares aos dos exploradores apresentam maior aptidão. As interações entre antígeno e anticorpo de parasitas e hospedeiros são exemplos de interações mediadas por este mecanismo, assim como as entre cucos parasitas de ninho e aves hospedeiras (Krüger 2007, Vikan *et al.* 2011). O mecanismo de acoplamento fenotípico resulta em um eixo bidirecional de vulnerabilidade (Abrams 2000). Diferentemente, quando as interações são mediadas por barreiras à exploração, o que determina a probabilidade de ataque é a capacidade do explorador superar as defesas da vítima. Por exemplo, em alguns tipos de interações predador-presa o tamanho corporal do predador está relacionado ao tamanho das presas que ele consome, sendo que em geral predadores tem maior sucesso ao atacarem presas que são menores que os próprios. Da mesma forma, predadores que se deslocam mais rapidamente que suas presas tendem a ter maior probabilidade de capturá-las. O mecanismo de barreira de exploração resulta em um eixo unidirecional de vulnerabilidade e está relacionado com intensificação das características ou corridas armamentistas (Abrams 2000). Visto que tanto os mecanismos de interação quanto a coevolução influenciam a organização das interações antagonistas, explorar quais são as estruturas de redes geradas pela ação recíproca entre diferentes mecanismos de interação e a coevolução entre as espécies pode ser fundamental para avançarmos nosso entendimento sobre a estrutura e dinâmica dessas interações.

Retroalimentação entre ecologia e evolução

A cada momento, a seleção natural está modificando a composição genética das populações em resposta às condições ambientais. No entanto, à medida que os organismos evoluem, o processo de modificação das espécies também gera mudanças concomitantes no

próprio ambiente. Dessa forma, tanto organismo como ambiente são causa e efeito em um processo coevolutivo (Lewontin 2001). Na última década tem havido um crescente reconhecimento de que a compreensão da estrutura, dinâmica e evolução de comunidades ecológicas requer o aprofundamento de uma perspectiva que combine os processos ecológicos e evolutivos e a retroalimentação entre eles (Odling-Smee *et al.* 2013, Pelletier *et al.* 2009). Interações ecológicas podem influenciar as pressões da seleção natural e sexual e estão subjacentes a muitos processos evolutivos, como a evolução fenotípica (Ackerly 2003), coevolução (Thompson 1999, Strauss *et al.* 2005) e especiação (Schluter 1996). Da mesma forma, sabe-se que processos evolutivos são importantes para compreender a dinâmica das populações (Yoshida *et al.* 2003, Coulson *et al.* 2006, Pelletier *et al.* 2007), formação e composição de comunidades (Loeuille & Loreau 2005, Emerson & Gillespie 2008) e dinâmica de metacomunidades (Urban *et al.* 2008). Há também evidências crescentes de que a evolução pode ser rápida e que processos ecológicos e evolutivos podem ocorrer na mesma escala de tempo de forma interdependente, assim chamada de dinâmica eco-evolutiva (Hairston *et al.* 2005, Fussmann *et al.* 2007, Schoener 2011).

Dinâmicas eco-evolutivas são especialmente comuns em interações antagonistas (Shertzer *et al.* 2002, Yoshida *et al.* 2007, 2003, Morran *et al.* 2011, Mougi & Iwasa 2011). A dinâmica das doenças é, por exemplo, determinada pelos ciclos de retroalimentação eco-evolutiva, de forma que parasitas só são capazes de infectar hospedeiros cuja resistência eles superam por meio de adaptações (Penczykowski *et al.* 2016). O forrageamento adaptativo também está associado a dinâmica eco-evolutiva e estudos têm documentado que mudanças evolutivas rápidas podem influenciar a estrutura interações interespecíficas em comunidades naturais (Thompson 1998, Whitham *et al.* 2006, Fussmann *et al.* 2007). Portanto, qualquer questão evolutiva relacionada a características que mediam interações requer uma compreensão sobre a ecologia das interações presentes na comunidade e de como as pressões seletivas estão estruturadas (Fussmann *et al.* 2007). Da mesma forma, modelos de dinâmica de comunidades precisam considerar que a estrutura das interações varia e que as forças de interação podem evoluir (Thompson 1998).

Objetivos

Esta tese é formada por três capítulos com o objetivo de investigar como a seleção natural atua sobre a estrutura e dinâmica de interações antagonistas. Para isso, nós combinamos modelos matemáticos com a abordagem de redes complexas e dados empíricos.

Seguindo a lógica apresentada na introdução do tema, primeiramente investigamos como a estrutura das interações influencia a coevolução. Posteriormente estudamos como processos coevolutivos mediam a interação entre espécies e quais estruturas de rede emergem desses processos. Finalmente, integramos os processos ecológicos e evolutivos das interações antagonistas a fim de investigar a dinâmica temporal das populações e características fenotípicas e a retroalimentação entre esses processos.

No primeiro capítulo desenvolvemos um modelo para descrever a coevolução de características de ataque e defesa entre espécies que interagem. Investigamos (i) como a relação entre a seleção ambiental e seleção pelos parceiros influencia a evolução das características das espécies; (ii) como as diferentes estruturas de rede influenciam a evolução das características das espécies; e (iii) que estruturas de rede são mais estáveis, i.e menos modificadas pela coevolução das características.

No segundo capítulo modificamos o modelo inicial de forma a permitir que as interações entre espécies mudem em função da probabilidade de interação entre elas. As probabilidades de interação foram determinadas por dois mecanismos de interação, representados como o mecanismo de acoplamento fenotípico e o de barreira de exploração. Nós (i) comparamos os padrões estruturais favorecidos pelos diferentes mecanismos de interação, explorando qual mecanismo melhor prevê a estrutura das redes empíricas; (ii) investigamos como os diferentes mecanismos influenciam a evolução das características de ataque e defesa; e (iii) exploramos a retroalimentação entre a estrutura da rede e a evolução das características das espécies.

No terceiro capítulo nós integramos as dinâmicas ecológicas e evolutivas das espécies por meio de uma abordagem eco-evolutiva. Nós estudamos (i) como as dinâmicas ecológicas e evolutivas são inter-relacionadas; e (ii) como a retroalimentação entre ecologia e evolução influencia a estrutura e dinâmica das comunidades.

Na seção de perspectivas para o estudo das dinâmicas ecológica e evolutiva em redes de interação antagonista, desenvolvemos um modelo para estudar a dinâmica de transmissão de parasitas que infectam diferentes espécies de hospedeiros por meio de múltiplos mecanismos de transmissão. Combinamos as diferentes redes antagonistas formadas a partir das interações mediadas por cada mecanismo de transmissão em uma rede de interação múltipla espacialmente explícita. Nós exploramos (i) qual a importância de cada mecanismo de transmissão para a velocidade em que o parasita é transmitido no ambiente; (ii) como a composição da comunidade de hospedeiros e a frequência relativa dos vetores influencia a

taxa de transmissão do parasita; e (iii) qual o impacto de diferentes estratégias de imunização dos hospedeiros para a taxa de transmissão do parasita.

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Capítulo 1

Network structure and selection asymmetry drive coevolution in species-rich antagonistic interactions

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running title: coevolution in antagonistic networks

key-words: alternation, antagonism, arms-races, coevolution, network

Abstract

One of the current challenges in evolutionary biology is to understand how ecological interactions shape the evolution of interacting species and how trait evolution feedbacks affects the structure of antagonistic networks. The addition of even one species can change the evolutionary dynamics of a pairwise interaction. As a consequence coevolution in species-rich assemblages that form networks of interacting species may ultimately depend on how the distribution of interactions within these networks. We combined an adaptive network framework and evolutionary modeling to study how network organization and patterns of selection affect and are affected by coevolution in antagonisms. We explore how selection imposed by interactions within ecological networks shapes the evolution of attack and defense traits by combining trait evolutionary models and empirical information on 31 local assemblages of antagonistic interactions, such as parasites and hosts, predators and prey and herbivores and plants. In our simulations, coevolution in antagonisms are affected by the intensity and asymmetry of the selection imposed by the interacting partners. Transient escalation in attack and defensive traits was the most prevalent pattern of change, and was especially favored by networks with a highly modular structure. Fluctuating evolution of traits was observed when the intensity of selection was higher in exploiters than in victims and was especially favored in networks with high nestedness. We also observed contrasting evolutionary dynamics at species level. Highly connected species experienced higher temporal variation in selection in all types of network, which resulted in high trait mismatching with their partners. These mismatching patterns may explain the emergence of modularity in antagonisms in which selection is stronger on exploiters than on their victims. Therefore, nestedness shaped the coevolutionary dynamics while modularity emerged as the result of the coevolutionary dynamics

Introduction

Coevolution is phenotypic change driven by reciprocal selection between interacting species (Thompson 1994), and it plays a key role in producing and maintaining species diversity and interactions (Thompson 2005, Yoder & Nuismer 2010). A solid body of theoretical and experimental studies focusing on pairwise antagonistic interactions predicts a suite of evolutionary dynamics, such as selective sweeps due to new mutants (Gandon & Michalakis 2002, Ebert 2008), arms races (Brockhurst & Koskella 2013), and negative frequency-dependent phenotypic selection (Gavrilets 1997). A long lasting challenge in the study of evolution of ecological interactions is to understand if and how the coevolutionary process is changed when more than two species are involved (Fox 1988; Iwao & Rausher 1997; Strauss & Irwin 2004). Multiple hypotheses have been proposed suggesting patterns in how groups, rather than pairs, of species coevolve (Thompson 2013, Barraclough 2015). In this context, tools derived from network science present a promising approach to investigating coevolution in multispecies interactions and distinguishing among alternative hypotheses (Guimarães *et al.* 2007, Nuismer *et al.* 2013, Loeuille & Loreau 2005).

In species-rich networks, evolutionary and coevolutionary changes are intrinsically interwoven through the cascading effects of selection acting on each species (Guimarães *et al.* 2011). Variation in the network organization of ecological interactions may alter the structure of selection, which may impact the focal species as well as other species in the network (Strauss & Irwin 2004, Guimarães *et al.* 2011, Nuismer *et al.* 2013). Therefore, the characterization of the structure of ecological networks may allow inferences on coevolutionary dynamics in species-rich assemblages. Among the most widely observed structural patterns in ecological networks are nestedness and modularity (Fortuna *et al.* 2010). Nestedness is a pattern of interactions in which specialist species tend to interact with subsets of species that interact with more generalist species (Bascompte *et al.* 2003). Modularity occurs when groups of species within a network interact more with each other than with other groups (Olesen *et al.* 2007). Levels of nestedness and modularity vary widely across ecological networks. For instance, antagonistic interactions between plants and gall-making insects form highly modular, non-nested networks, whereas interactions between mammalian predators and their prey form highly nested, non-modular networks (Pires & Guimaraes 2012). The great structural diversity found in antagonistic networks may shape the rate of evolution and coevolution in different ways, molding specialization and patterns of

overlap in interactions between exploiters and victims (Nuwagaba *et al.* 2015, Beckett & Williams 2013). In fact, theory and empirical work suggest that antagonistic interactions may show rapid and diverse ecological and evolutionary dynamics (Abrams 2000, Woolhouse *et al.* 2002, Thompson 2013).

Species-rich networks can favor coevolutionary alternation (Thompson 2005), in which reciprocal selection leads to temporal fluctuation in the specialization of exploiters to particular victim species and the relative levels of defense of victims against exploiter (Davies & Brooke 1989, Nuismer & Thompson 2006). Coevolutionary alternation is expected to emerge if (1) exploiter preference hierarchies are inherited, (2) victim defenses are costly, (3) selection favors exploiters that preferentially attack the currently most vulnerable victims, and (4) prey defenses evolve faster than exploiter preference hierarchies. In contrast, escalating arms races can occur in multi-specific interactions if the preference hierarchies of exploiters evolve faster than victim defenses (Nuismer & Thompson 2006, Davies & Brooke 1989). Empirical work supports assumptions and predictions of coevolutionary alternation and escalating arms races. Selection experiments have shown genetic variation of exploiter preference hierarchies at population level (Strauss & Irwin 2004; Nylin *et al.* 2005) and rapid evolution of defenses within populations (Trussell & Smith 2000, Rausher 2001, O'Steen *et al.* 2002, Yoshida *et al.* 2003, Brockhurst *et al.* 2004, Koskella *et al.* 2012). These studies provide evidence that multiple antagonistic species alter the outcomes of pairwise interactions and reorganize patterns of interaction at community level (Barraclough 2015). The next challenge in the analysis of coevolutionary dynamics in multispecies interactions is to understand the specific roles of the feedback between trait evolution and the structure of antagonistic networks in shaping trait evolution of interacting species.

Here, we combine a model for single-trait evolution with a network analysis of 31 empirical antagonistic assemblages to study how network organization and patterns of selection affect and are affected by coevolution in antagonisms. These networks encompass a large sample of the diverse natural history of antagonistic interactions, including, those between insect and mammalian herbivores and plants, fish and their parasites, and mammalian prey and their predators. We show that the joint effects of selection imposed by partners and network structure shape species evolutionary and coevolutionary dynamics in predictable ways, favoring either escalation, coevolutionary alternation, or both. If selection

on explorers is stronger than on victims, coevolutionary alternation is favored by nested organization, creating asymmetry in specialization between exploiters and victims and generating a hierarchy of preferred victims among exploiters. Coevolution is not only affected by network structure but also reshape network organization: higher modularity arise in antagonistic networks as an outcome of the coevolutionary process, emerging as the product of stronger selective pressures acting on exploiters.

Methods

Model

We combined an adaptive network framework (Gross & Blasius 2008) and evolutionary modeling to study how network organization and patterns of selection affect and are affected by coevolution in antagonisms. The adaptive network approach allowed us to track trait changes, quantify the degree of trait matching among species, and measure network structural change. We modeled evolution of a single trait in victims and exploiters (Figure 1). We assumed that natural selection favored exploiters whose attack traits matched the defenses of victims in ways that increased the chance of a successful attack, and that selection favored victims whose defense traits mismatched the exploiters in ways that allowed escape from attack (Hanifin *et al.* 2008, Nuismer & Thompson 2006). We modeled the evolution of the mean trait value of a population of species i as a real number, z_i, representing a defensive trait if species i is a victim or an attack trait if i is an exploiter. The value of z_i was initially sampled from a normal distribution $\mathcal{N}(0,0.1)$.

At each time step, trait values of each species were updated in response to selection imposed by the environment and antagonistic partners. We assumed environmental selection favors a fixed trait value, θ_i, which is defined as $\theta_i = z_{i\ (t=0)}$ for simplicity. The partial selection differential caused by environmental selection was defined as:

$$S_{i(t)} = \xi_{s_i}(\theta_i - z_{i(t)}) \tag{1}$$

in which ξ_s is the intensity of the environmental selection, $0 < \xi_s < 1$. We assumed that the fitness consequences of the interaction to a victim i and exploiter j depended on the trait matching, here defined as $z_{ij} = |z_i - z_j|$. Selection by victims on exploiters favors matching

and the partial selection differential is described by:

$$M_{ij(t)} = \xi_{d_i} p_{ij(t)} (z_{j(t)} - z_{i(t)}) \tag{2}$$

in which ξ_{d_i} is the selection intensity imposed by interacting species, $0 < \xi_{d_i} < 1 - \xi_s$, and p_{ij} weights the selection imposed by victims on exploiters according to exploiter preference, b, and trait matching:

$$p_{ij(t)} = \frac{f_{ij} e^{-b(z_{j(t)} - z_{i(t)})^2}}{\sum_{k=1;k\neq i}^{R} f_{ik} e^{-b(z_{k(t)} - z_{i(t)})^2}} \tag{3}$$

in which f_{ij} is an element of **F** and describes if the interaction can occur ($f_{ij} = 1$) or not ($f_{ij} = 0$, "forbidden link", Jordano et al. 2003) and R is the total species richness in the network. Most ecological interactions are modulated by multiple traits and **F** allows us to implicitly introduce the effect of other traits that prevent an interaction to occur. Thus, **F** sets a limit on the extent to which network structure can change.

For victims, selection favored trait mismatches (large z_{ij}). We assumed that there is a critical mismatch, ε, in such way that if $|z_{i(t)} - z_{j(t)}| > \varepsilon$ the exploiter has a negligible effect on victim fitness, becoming a commensal species. The partial selection differential caused by selection imposed by exploiters i on victims j was defined as:

$$M_{ji(t)} = \xi_{d_i} u_{ji} (z_{i(t)} \pm \varepsilon - z_{j(t)}) \tag{4}$$

where u_{ji}=1 if $|z_{i(t)} - z_{j(t)}| \leq \varepsilon$, or u_{ji} =0 if $|z_{i(t)} - z_{j(t)}| > \varepsilon$. Because the trait axis is bidirectional, selection favors increasing trait values for the victim, $z_{i(t)} + \varepsilon$ if $z_{j(t)} > z_{i(t)}$ and decreasing trait values, $z_{i(t)} - \varepsilon$ if $z_{j(t)} < z_{i(t)}$. Combining equations (1) to (4) to the breeder's equation results in a general equation describing trait evolution for both explorers and victims:

$$z_{i(t+1)} = z_{i(t)} + h_i^2 (S_{i(t)} + \frac{\sum_{j=1;j\neq i}^{R} f_{ij} M_{ij(t)}}{k_{i(t)}}) \tag{5}$$

where h_i^2 is the heritability of trait z_i, $\frac{\sum_{j=1;j\neq i}^{R} f_{ij}M_{ij(t)}}{k_{i(t)}}$ is the combined effects of the partial selection differentials caused by interacting species, and $k_{i(t)}$ is the number of potential partners of species i at time t, $k_{i(t)} = \sum_{j=1;j\neq i}^{R} f_{ij}$.

Characterizing evolutionary patterns

Coevolutionary escalation predicts sustained directional selection on attack and defense traits, whereas coevolutionary alternation predicts fluctuating selection on those traits. We characterize the evolutionary dynamics using three evolutionary patterns: the directionality of trait change in a given species, the temporal variation in trait matching of interacting species, and degree of trait disparity across species in the network. We characterized directionality in trait change in a given species as:

$$\delta_i = \frac{\left|z_{i(t=10{,}000)} - z_{i(t=0)}\right|}{\sum_{t=0}^{t=10{,}000} \left|z_{i(t+1)} - z_{i(t)}\right|} \tag{6}$$

in which $\left|z_{i(t=10{,}000)} - z_{i(t=0)}\right|$ is the directional trait change after 10,000 time steps and $\sum_{t=0}^{t=10{,}000} \left|z_{i(t+1)} - z_{i(t)}\right|$ is the total amount of trait change across all time steps. We used 10,000 time steps because it allows the asymptotic behavior of the model. Coevolutionary escalation predicts values close to one, characterizing directional trait evolution with little fluctuation over time. In contrast, coevolutionary alternation predicts values close to zero, indicating lack of sustained directional evolution due to fluctuating selection. We investigated the effects of network structure on the trait change directionality by calculating the mean directionality for the entire network, $\langle\delta\rangle = \frac{\sum_{i=1}^{R}\delta_i}{R}$.

We characterized how trait matching varied across time using

$c_{ij} = \sum_{t=1}^{t=10,000} f_{ij} \left| z_{ij(t)} - z_{ij(t-1)} \right|$. Higher c_{ij} values indicate higher temporal variation in trait matching between interacting species *i* and *j*. We estimated trait disparity by describing the breadth of the trait space occupied by all species in the community using $\max_{i \neq j} \left| z_i - z_j \right|_{t=10,000}$.

Do selection intensity and selection asymmetry affect multi-specific coevolution?

We explored the effects imposed by selection intensity and selection asymmetry on multi-specific antagonistic coevolution by performing simulations using five distinct parameterizations (scenarios) of the model. In Scenario 1, selection imposed on exploiters by victims was stronger than the selection imposed on victims by exploiters (ξ_{d_i} = 0.5; ξ_{d_j} = 0.99). This scenario simulates is rooted on the natural history of a number of obligatory parasite-host interactions, in which the parasite depends fully on the host to survive and reproduce, whereas some hosts may have only slightly to moderately reduced lifespan or reproduction (e.g., Haraguchi and Sasaki 1996). Scenario 2 (ξ_{d_i} = 0.99; ξ_{d_j} = 0.5) simulates the "life-dinner principle" (Dawkins & Krebs 1979), which states that selection pressure on prey defensive traits is stronger than that on predator efficiency. The symmetric moderate (scenario 3, $\xi_{d_i} = \xi_{d_j}$ = 0.5) and symmetric strong (scenario 4, $\xi_{d_i} = \xi_{d_j}$ = 0.99) represent benchmarks that separate the effects of intensity and asymmetry captured in scenarios 1 and 2. Finally, scenario 5 is a null scenario in which interaction selection is symmetric and very weak for exploiters and victims ($\xi_{d_i} = \xi_{d_j}$ = 0.01). Each simulation had 10,000 time steps, and we ran 100 simulations per combination of network and scenario. We fixed the remaining parameters at h^2 = 0.25, b = 10 and ε = 0.5 since sensitivity analysis showed their effects were weaker than selection intensity imposed by interacting partners (Figures S1-S4 in Supporting Information). We assigned the same values for the parameters of all species in the network (h^2, b, ε, ξ_{d_i} and ξ_{d_j}) for simplicity. We then analyze how the resulting evolutionary patterns vary across scenarios (see below).

Does network structure affect and is affected by coevolutionary dynamics?

We used 31 two-mode antagonistic networks to parameterize the coevolutionary

model, exploring the effects of network structure on coevolutionary dynamics. The networks range from small networks of mammalian predators and their prey (22 species) to large networks formed by leaf miner herbivores and plants including almost 300 species (Table S1). Different interactions are often associated with particular evolutionary scenarios, *e.g.*, life-dinner principle in predator-prey interactions. However, in this study we use only the information on the network structure to parameterize **F** (Table S1), simulating the five evolutionary scenarios described above. Later, we discuss the outcomes of the model using as reference the evolutionary ecology of particular types of antagonisms.

We calculated four network descriptors: (i) species richness (*R*), (ii) connectance, (iii) nestedness, and (iv) modularity (Supporting Information). We used the metric NODF (Almeida-Neto *et al.* 2008) to estimate nestedness, computed using ANINHADO (Guimarães & Guimarães 2006). Because asymmetries pervade nested networks (Bascompte *et al.* 2003, Guimarães *et al.* 2006), we hypothesized that nestedness may generate a hierarchy of preferred victims among exploiters, favoring coevolutionary alternation. The degree of modularity was estimated using a simulated annealing algorithm to optimize the metric *M* (Newman & Girvan 2004, Guimerà & Amaral 2005) calculated using the software MODULAR (Marquitti *et al.* 2014). We expected that modularity would favor coevolutionary escalation because of the limited number of potential partners within modules.

Nestedness and modularity are affected by other network properties. We controled these confounding factors by using Z-scores to compare levels of nestedness and modularity across different networks, $NODF_{rel} = \frac{NODF - \overline{NODF_{null}}}{\sigma_{null}}$, and $M_{rel} = \frac{M - \overline{M_{null}}}{\sigma_{null}}$ (Fortuna *et al.* 2010), where $\overline{NODF_{null}}$ and $\overline{M_{null}}$ are the metrics mean values obtained with the null model and σ_{null} are their standard deviations, respectively. We controlled the effects of heterogeneity in interaction number, connectance and species richness on network properties by using null model 2 of Bascompte *et al.* (2003), which considers the probability of drawing an interaction as proportional to the number of interactions of both the exploiter and victim species.

We also investigated if patterns of interaction of particular species affect its trait evolutionary dynamics by using species-level descriptors: (i) normalized degree, which is the

number of interactions of the species divided by the number of species in the other trophic level (Martín González *et al.* 2010); (ii) contribution to nestedness (Almeida-Neto *et al.* 2008); (iii) standardized within-module degree; and (iv) among-module connectivity (Guimerà & Amaral 2005), which characterizes how interactions of a given species are distributed within and among modules, respectively (see SI for further details). We also categorized each species as part of the network core or periphery (Díaz-Castelazo *et al.* 2010) using a categorical core–periphery analysis for bipartite graphs (SI). Core species are connected to other core species as well as certain peripheral species, although peripheral species are not interconnected.

We used a combination of analyses to evaluate how the evolution of traits, the number of interactions between species, and network structure are directly and indirectly interrelated. We first used general linearized models (GLMs) to explore how directional trait evolution, $\langle\delta\rangle$, disparity of final traits, and number of lost interactions ($u_{ji(t=10,000)} = 0$, given that $f_{ij} = 1$) relate to the structure of ecological networks. These models allowed us to evaluate the direct effects of nestedness, modularity, and connectedness and richness on trait evolution and changes in specialization.

Because structural descriptors of networks often affect each other we then performed a path analysis (Shipley 2004). We assumed that richness and connectance have a direct effect on the directionality of evolutionary change, $\langle\delta\rangle$, and also on degrees of nestedness and modularity, which in turn can also affect $\langle\delta\rangle$. We tested the entire causal structure of the path model simultaneously by estimating model parameters via maximum likelihood (Shipley 2004). We cross-validated our results by exploring the correlated effects of network structure with an alternative approach based on principal components analysis (see SI).

We next used the directionality of evolutionary change for a species, δ_i, and magnitude of directional change in trait value to characterize the evolutionary dynamics and measure trait net change at the species level. We investigated the effects of patterns of interaction of species (species normalized degree, contribution to nestedness, standardized within-module degree and among-module connectivity) on the species evolutionary dynamics with GLMs using network identity as covariate. We also performed a PCA to synthesize the different aspects of patterns of interaction of species (SI).

We used the final matrix of trait matching ($\mathbf{Z}_{t=10,000}$) to characterize net evolutionary change and the matrix of cumulative change in trait matching (**C**) to estimate the dynamics of interaction strengths between species. We compared the values of final trait matching and average change in trait matching (c_{ij}) for interactions: (a) between species that are in the same module or in different modules and (b) between core and periphery species using GLMs that included network identity as a covariate.

Finally, we explored the feedback of coevolution on the structure of interactions by analyzing how coevolutionary dynamics affects three descriptors of network organization. We first computed the proportion of interactions that were lost because the victim escaped the attack. Then, we also compared how the levels of nestedness and modularity changes due to the loss of interactions, by computing the NODF and *M* for all for all replicates of each network. We tested potential differences between initial and final structures using paired t-tests.

Results

The emergence of coevolutionary alternation or coevolutionary escalation was affected by the interplay between network structure and asymmetries in selection imposed by species interactions on victims and explorers. Overall, directional trait evolution was the prevalent outcome of the evolutionary dynamics (Figure 2). Fluctuating selection, evidenced by fluctuating trait change, more frequently occurred when the selection intensity imposed by exploiters on victims was weaker than the selection imposed by victims on exploiters (scenario 1, Figure 2). In the scenario in which selection is stronger on exploiter (scenario 1), network structure affected coevolution, with nestedness significantly decreasing the directionality of trait evolution, $\langle\delta\rangle$ (slope (b) = -0.05, F = 17.27, p < 0.0001, df = 29, Fig. 3) and modularity not presenting a significant effect (b = -0.006, F = 0.04, p = 0.84, df = 29). Species richness and connectance had a small negative effect on directionality of trait evolution, indicating that fluctuating selection was more likely to occur in species-rich and highly connected networks (Figure 4A). PCA cross-validated these results, suggesting nestedness is the main structural driver allowing the emergence of fluctuating selection (SI). When the selection intensity on victims was stronger and asymmetric (scenario 2, life-dinner principle), directional selection was the prevalent dynamics (Figure 3). In scenario 2,

directional evolution emerges independent of network structure and higher directionality was not associated with nestedness (b = -0.0015, F = 0.551, p = 0.464, df = 29) or modularity (b = -0.0031, F = 0.682, p = 0.415, df = 29; Figure S5). Similar directional coevolution were observed in both scenarios assuming symmetric selection intensities (scenario 3 and 4, Figure 3). Weaker selection (scenario 5) reduced the magnitude of directional trait change, without qualitatively change the directionality of coevolutionary dynamics (Figures 2 and 3).

The effects of network structure were more pervasive on the other outcomes of coevolutionary dynamics (Figure S6). Whenever selection imposed by victims and exploiters was non-negligible (all scenarios but scenario 5) nestedness affected trait disparity (Figure S7). The higher the degree of nestedness the lower was the trait disparity (negative correlations between nestedness and trait disparity all scenarios but scenario 5, Table S4, Figure S7). In contrast, modularity increased trait disparity only in scenarios in which selection intensity was intermediate or negligible (scenario 3 and 5, Table S4, Figure S8). If victims and/or explorers face strong selection, modularity did not affected trait disparity (scenarios 1, 2, and 4, Table S4, Figure S8). PCA analysis cross-validated these results (SI).

The coevolutionary dynamics also reorganized network structures if victims evolved efficient defenses against exploiters ($\left|z_{i(t)} - z_{j(t)}\right| > \varepsilon$). The proportion of interactions lost was higher when the selection intensity acting on victims was higher (Figures S9 and S10). Higher nestedness resulted in a higher proportion of lost interactions in all scenarios (Table S5, Figure S9). Nestedness amplified the loss of interactions by enabling exploiters to specialize in poorly defended victims (higher trait matching), which in turn allowed other victims to escape the attack. In contrast, modularity had no effect on the proportion of lost interactions (Figure S10).

We focused on the scenario in which more variable coevolutionary dynamics occur (scenario 1, in which selection is stronger on explorers) to explore how the loss of interactions affects network organization. At the end of simulations, the networks had become significantly more modular (t = 8.06, p < 0.0001, df = 30, Figure 5a) and less nested (t = -6.08, p < 0.0001, d. f. = 30, Figure 5a) compared with the original network structure. The temporal decay of nestedness was positively related to initial nestedness (b = 0.26, F = 118.2, d. f. = 29, p < 0.0001, Figure 5b), whereas the increase in the levels of modularity was negatively related to initial network modularity (b = -0.118, F = 13.69, d. f. = 29, p < 0.0001,

Figure 5c). Thus, highly nested networks tend to become more modular, but the final structure of modular networks remains unchanged.

We next evaluated how network organization shapes the trait evolution for species within networks. Trait evolution for each species depended on the number of other species to which it was connected within the network. On average, highly connected species had higher fluctuating selection on traits (smaller δ_i). These highly connected species were those with a high network degree value (higher normalized degree, b = -0.21, F = 31.12, df = 2380 for all species analysis, $p < 0.0001$), connected distinct modules (higher among-module connectivity, b = -0.197, F = 85.731, $p < 0.0001$), contributed more to nestedness (b = -0.004, F = 122.873, $p < 0.0001$), and were part of the core of the network (b = -0.019, F = 13.817, p = 0.0002) The number of species interactions within modules did not affect δ_i (within-module degree, b = 0.006, F = 1.945, p = 0.163).

The network structure itself modulated the relationship between species patterns of interaction and coevolutionary dynamics, as indicated by the analyses of how the coefficients of regression between a species' structural metrics and δ_i vary across networks. As the absolute value of the regression coefficient increased, the average effect of species pattern of interactions on the directionality of evolution became stronger. In nested networks, the negative relationships between the number of interactions and contribution to nestedness of a given species and the directionality of selection, δ_i, were weak or inexistent (Figure 6), indicating that any species can experience fluctuating selection, no matter its structural role. In contrast, these negative relationships were stronger in non-nested networks (Figure 6). Accordingly, in modular networks, the negative relationship between number of interactions of species with different modules (among-module connectivity) and δ_i was stronger (Figure 6c). Again, the number of interactions of a species within modules shows no clear relationship with δ_i (Figure 6d).

Directional evolution leading to an escalation of traits was more common among peripheral species (b = -0.033, F = 234.389, $p < 0.0001$) with a lower number of interactions (normalized degree, b = -0.167, F = 98.61, $p < 0.0001$), contribution to nestedness (b = -0.002, F = 205.274, $p < 0.0001$), and number of interactions with species in modules (among-module connectivity. b = -0.114, F = 143.253, $p < 0.0001$) or the same modules (within-module degree, b = -0.007, F = 12.681, p = 0.0004). The results of PCA cross-validate the

conclusion that species that connect modules are more likely to experience fluctuating selection (SI).

We have shown that network modularity had no effect on the species coevolutionary dynamics. However, modular structures emerged as an important outcome of the coevolutionary process. Modules were found to be stable coevolutionary units and modularity had a key role in how trait matching was distributed within networks. Mean trait matching was higher for species interacting within the same module than between species belonging to different modules ($F = 27.1224$, $p < 0.0001$, Figure S11a). Similarly, when comparing the trait matching among core-core, core-peripheral and peripheral-peripheral species, trait matching was higher between peripheral species, which form modules in networks ($F = 20.9427$, $p < 0.0001$, Figure S11b). Temporal change in trait matching was more frequent in interactions involving highly connected species. Moreover, the cumulative change in trait matching (c_{ij}) indicates that the strength of selection of interacting species belonging to different modules was more variable over time than of interacting species within the same module ($F = 14.0536$, $p = 0.0009$, Figure S12a). Similarly, interactions between core species frequently changed over time, whereas peripheral-peripheral interactions presented a more constant selection strength ($F = 7.3466$, $p = 0.0014$, Figure S12b).

Discussion

We integrated basic approaches derived from evolutionary theory with network-based modeling and empirical data to contribute to the emerging field of evolutionary ecology of ecological networks (Loeuille & Loreau 2005, Gómez *et al.* 2010, Santamaría & Rodríguez-Gironés 2007, Gómez & Verdú 2012, Nuismer *et al.* 2013, Guimarães *et al.* 2011). We identified three central points related to the mechanism of evolutionary change within species-rich antagonistic networks.

First, we show that coevolutionary alternation is a possible outcome of the coevolutionary dynamics mediated by trait matching if selection asymmetry between victims and exploiters occur associated to particular network patterns. Coevolutionary alternation in systems with few species is supported by both theory (Sasaki 2000, Agrawal & Lively 2002) and empirical work (Gómez & Buckling 2011, Hall *et al.* 2011), in which fluctuating selection occurs if ecological conditions reduce selection for costly defenses, such as in

phage-bacteria interactions in nutrient-limited environments (Gómez & Buckling 2011, Hall *et al.* 2011). In species-rich networks, fluctuating evolution is favored by the nested structure of some antagonisms, in combination with asymmetries in selection. Therefore, our results support the notion that coevolution in species-rich antagonisms is neither a simple combination of pair-wise interactions nor unstructured selection due to multiple species interactions. Rather, networks favor coevolutionary dynamics that cannot be predicted from pairwise interactions (Guimarães *et al.* 2011). The exploration of the effects of network organization led to similar insights into the dynamics of disparate systems, such as the spread of disease in human and animal societies (Craft *et al.* 2009, Keeling & Eames 2005), flow of information in technological networks (Watts *et al.* 1998), the persistence of species in fragmented landscapes (Urban *et al.* 2009), and the demographic changes in present and past ecological networks (Thébault & Fontaine 2010, Yeakel *et al.* 2014).

By favoring coevolutionary alternation, asymmetries in selection and nested network organization lead to deviations from the prevalent tendency of coevolutionary dynamics, which favors directional evolution in antagonistic networks. Directional evolution is also common in pair-wise interactions or antagonisms involving a few species, including weevils and camellias (Toju 2011), crossbills and lodgepole pine (Benkman *et al.* 2003), and bacteria and phages (Weitz *et al.* 2013). Directional trait coevolution is the result of selection favoring higher levels of defense and counter-defense as well as by the emergence of local specialization by exploiters in a few partners (Thompson 2005, Nuismer & Thompson 2006). However, costs prevent the levels of defense and counter-defense from escalating indefinitely (Rigby and Jokela 2000, Zuk and Stoehr 2002, Koskella et al. 2012, but see Cogni et al. 2012). Therefore the degree of trait disparity observed in antagonisms is limited by environmental selection in species-poor systems. Again, the existence of a network of antagonisms adds an additional effect on coevolutionary dynamics: escalation is also limited by distinct regimes of conflicting selection imposed by multiple partners.

Second, coevolution due to asymmetries in selection and network organization reshape the organization of antagonistic networks. This coevolutionary feedback indicated that the coevolutionary dynamics in large multi-specific networks are an interactive process of ever-changing interactions (Thompson 2013). If fitness consequences of an interaction are higher for exploiters than for victims, highly nested networks allow exploiters to temporally specialize in victims with whom they have better trait matching. Nested structures also

enabled more victims to escape from the attack, thus increasing network modularity. Assuming some degree of nestedness will occur even if interactions among species are ecologically neutral (Krishna *et al.* 2008), we hypothesize that species-rich antagonisms start as nested networks and stronger selection acting on exploiters than victims at the individual level will lead to specialization and low overlap among exploiters at the population level, which in turn will drive network organization from nested to modular. Once modularity is achieved, environmental and biotic selective forces will favor the evolutionary persistence of the modular organization. Available theory suggests that modular networks of antagonisms are also ecologically stable and naturally emerge from ecological dynamics of non-modular antagonisms (Thébault & Fontaine 2010, Allesina & Tang 2012). Here we suggest modular networks are also a stable consequence of coevolutionary dynamics.

Our theoretical results predict that modularity should be more common in antagonisms in which the fitness consequences of the interaction are assumed to be higher for the exploiters than for the victims. This prediction is also corroborated by empirical evidence. Interactions between host plants and gall-inducing insects, leaf miners, and endophagous seed predators are often highly modular (Pires & Guimaraes 2012). Conversely, nestedness is often observed in interactions showing higher fitness consequences for the victims than for the exploiters, which occurs in interactions between mammalian herbivores and their predators and between plants and grazing grasshoppers (Pires & Guimaraes 2012). Our theoretical findings may also explain the variation in network organization in other consumer-resource relationships, such as mutualisms (Pires *et al.* 2011). Modularity in mutualistic networks is also associated with stronger selective pressure on the consumers than on the resource, which is illustrated by interactions between myrmecophytes and ants (Fonseca & Ganade 1996), plants and floral parasite pollinators (Hembry *et al.* 2013) and anemones and anemonefish (Ricciardi *et al.* 2010). In contrast, nestedness is associated with similar or higher fitness consequences for the resource organisms, as in interactions among plants and most pollinators and seed dispersers (Bascompte *et al.* 2003). Future work should investigate if indeed the differences in fitness consequences of interactions for partners provide a general explanation for the structural variation of ecological networks.

Third, directional evolution and trait matching is not only affected by network organization but also by the interaction patterns of the species within the network. Peripheral species in nested networks have higher trait matching in all selection regimes, suggesting that

specialized interactions may achieve fixed and high levels of trait matching even in nested antagonistic networks. In contrast, coevolutionary alternation dynamics were observed in interactions among species on different modules or between core species, in which we observed variable levels of trait matching (indicated by higher cumulative trait change). Highly connected species – the super-generalists – are expected to coordinate coevolution in mutualistic networks, promoting convergence and trait complementarity (Guimarães *et al.* 2011). In antagonistic networks, super-generalists experience higher fluctuating selection, which may influence the dynamics of the entire network depending on the degree of nestedness. Super-generalists may alternate their interacting partners according to the relative defenses found in the victim community (Thompson 2005). Coevolutionary alternation was proposed to explain the coevolutionary dynamics among highly diverse thick-shelled marine invertebrates and their shell-crushing predators (Leighton 2002), geographically variable interactions between cuckoos and their hosts (Davies & Brooke 1989, Soler *et al.* 2001), and chemically and geographically complex interactions between butterflies and ants (Nash *et al.* 2008). Our work provide a theoretical expectation for the specific set of conditions in which coevolutionary alternation shapes the evolution of preferences and defenses in species-rich interacting assemblages of antagonisms.

To sum up, modularity had a negligible effect on coevolutionary dynamics. However, our results indicate that modular structures arise in antagonistic networks as an outcome of the coevolutionary process, emerging as the product of conflicting selective pressures acting on antagonistic networks. Nestedness sustains fluctuating trait evolution, and it may occur in interactions in which there is an asymmetry in the selection experienced by interacting exploiters and victims. Therefore, the fitness consequences of interactions and asymmetries in those consequences may determine the patterns of species interactions and shape the structure of ecological networks. The generality of these expectations should be investigated for other types of consumer-resource interactions. Experiments and field studies that consider the complex structure of ecological interactions and geographic mosaic of variation in such interactions are necessary to understand coevolution in natural communities and test our predictions. To advance our understanding of coevolution in antagonistic systems, it is also imperative to consider the interplay between ecological and evolutionary dynamics of species interacting in species-rich assemblages. By now, we hypothesize different selection regimes favors distinct coevolution mechanisms and that the structure of selection in multispecific antagonistic interactions can be traced by the network organization.

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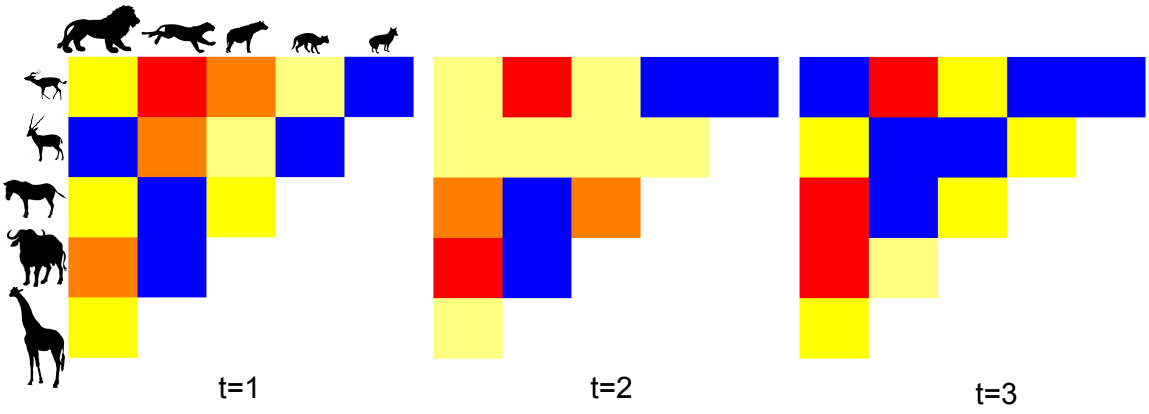

Figure 1: The coevolutionary model. In this hypothetical example, predator-prey interactions are organized as an interaction matrix. The colors of matrix elements depict the selective pressure of predators on prey. Hotter colors indicate interactions that have higher trait matching and higher selection imposed by the interaction partner but specifically blue cells indicate interactions with the highest trait matching in that time step. As the coevolutionary process continues, exploiters can alternate their selected victim, leading to changes in selective pressures and network organization.

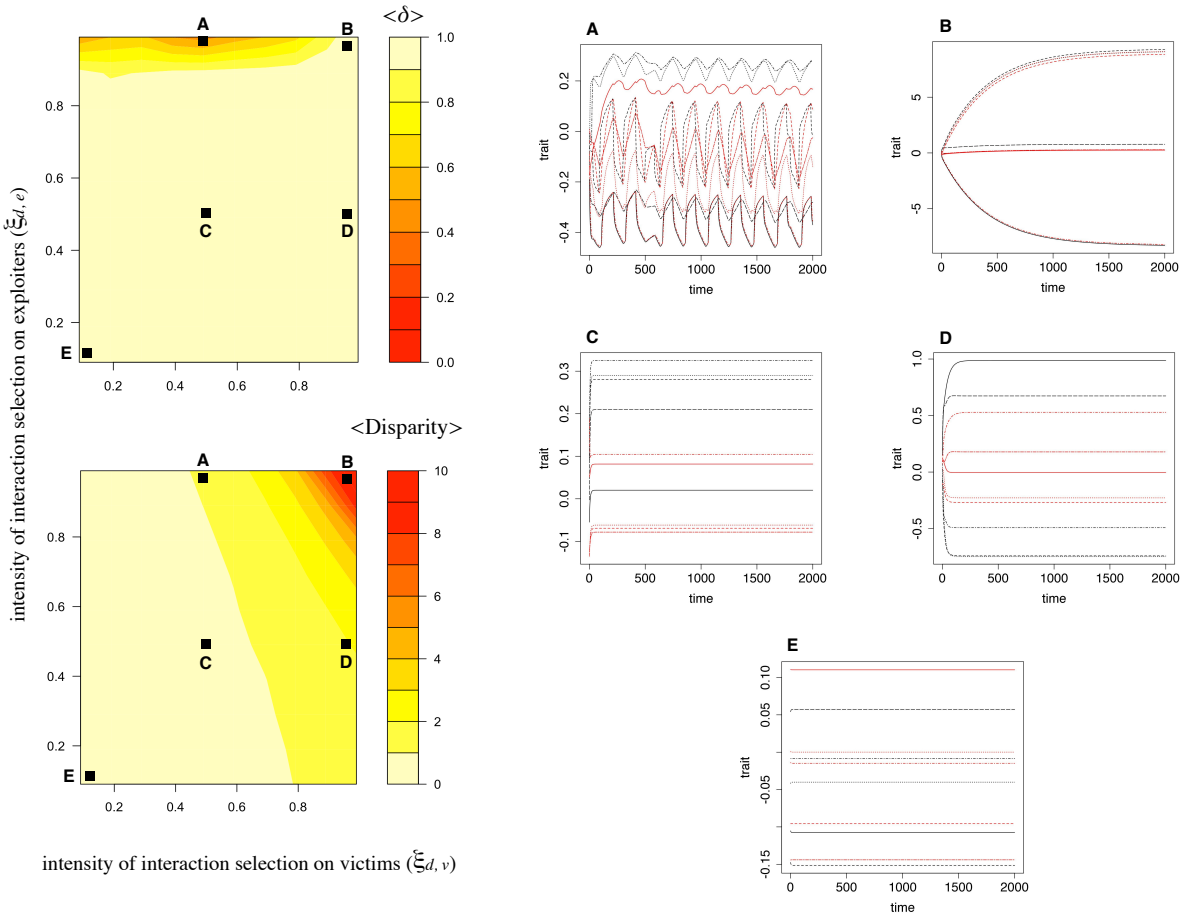

Figure 2: Left: Average directionality (top) and disparity of trait change (bottom) among 100 replicates in the 31 antagonistic networks for all combinations of victim and exploiter interaction selection intensity. Right: an example of the dynamics of species traits found in scenarios 1 (A), 2 (B), 3 (C), 4 (D) and 5 (E) for the 5 x 5 hypothetical network shown in Fig. 1. Black and red lines represent the victim and exploiter trait dynamics, respectively.

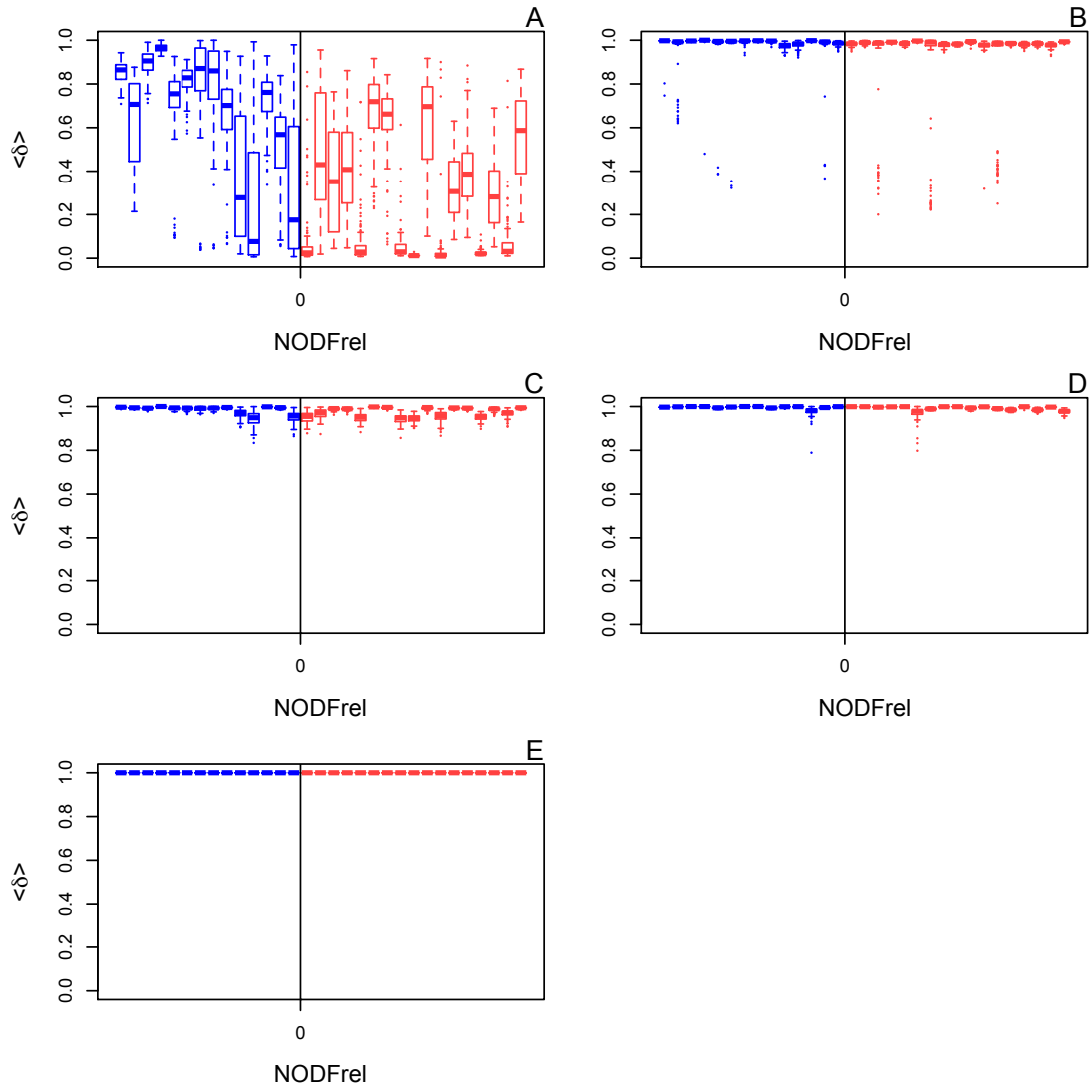

Figure 3: Relationship between network nestedness z-score (NODFrel) and the mean directionality of coevolutionary dynamics, $\langle\delta\rangle$, in each network per simulation replicate. NODFrel values lower than zero (blue) represent networks that are less nested than expected according to the null model, whereas NODFrel values higher than zero (red) represent networks that are more nested than expected according to the null model. A: results from scenario 1, in which the intensity of interaction selection is higher in exploiters ($\xi_{d_i} = 0.5 < \xi_{d_j} = 0.99$). B: results from scenario 2, in which the intensity of interaction selection is higher in victims ($\xi_{d_i} = 0.99 > \xi_{d_j} = 0.5$). C, D and E are the results from scenarios 3, 4 and 5, respectively, in which the intensity of interaction selection is equal between exploiters and victims. In C, both are medium ($\xi_{d_i} = \xi_{d_j} = 0.5$); in D, both are strong ($\xi_{d_i} = \xi_{d_j} = 0.99$); and in E, both are weak ($\xi_{d_i} = \xi_{d_j} = 0.01$).

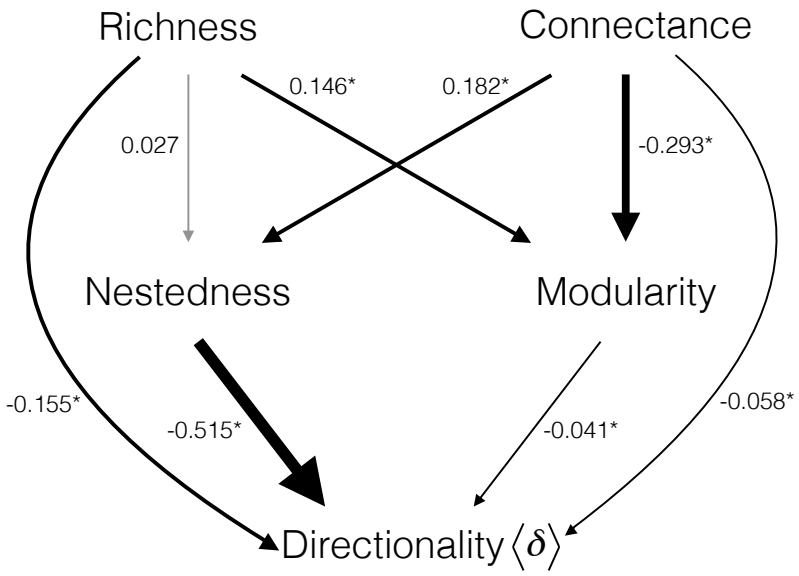

Figure 4: Summary diagram of the effects of different network architectural patterns on the directionality of coevolutionary dynamics of antagonistic networks, $\langle\delta\rangle$. The thickness of the arrows is scaled to standardized coefficients from the path analysis and illustrates the relative effect strength. Significant effects are represented in black and have an asterisk, and non-significant effects are represented in gray. The effects of connectance and richness are split between direct effects and indirect effects through changes in modularity and nestedness. The strength of the indirect effects was calculated as the product of the coefficients along the path. For example, connectance has a direct effect on strength (-0.058), an indirect effect through modularity on strength (0.012; –0.293 × –0.041), and an indirect effect through nestedness on strength (-0.094; 0.182 × – 0.515), which leads to an overall connectance effect of -0.14.

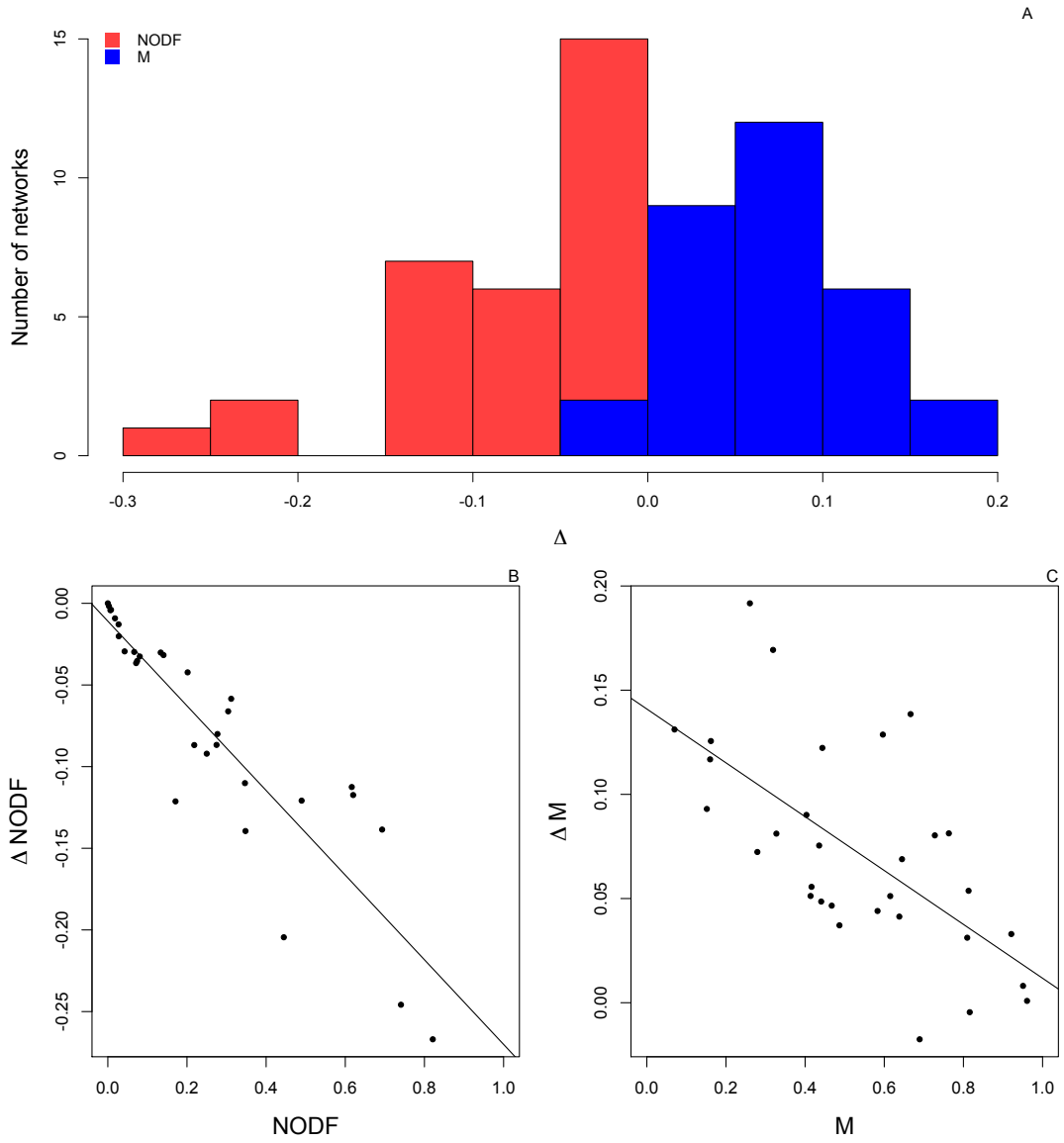

Figure 5: Changes in network nestedness and modularity at the end of the simulations. A: histogram of the difference (Δ) between the initial and final network nestedness (NODF, in red) and modularity (M, in blue). B: difference between the final and initial network NODF depending on the initial NODF value. Higher network NODF values correspond with greater differences between the initial and final values. C: same as in B but for M values. Networks with low M values presented a greater increase in final M values than networks with initially high M values.

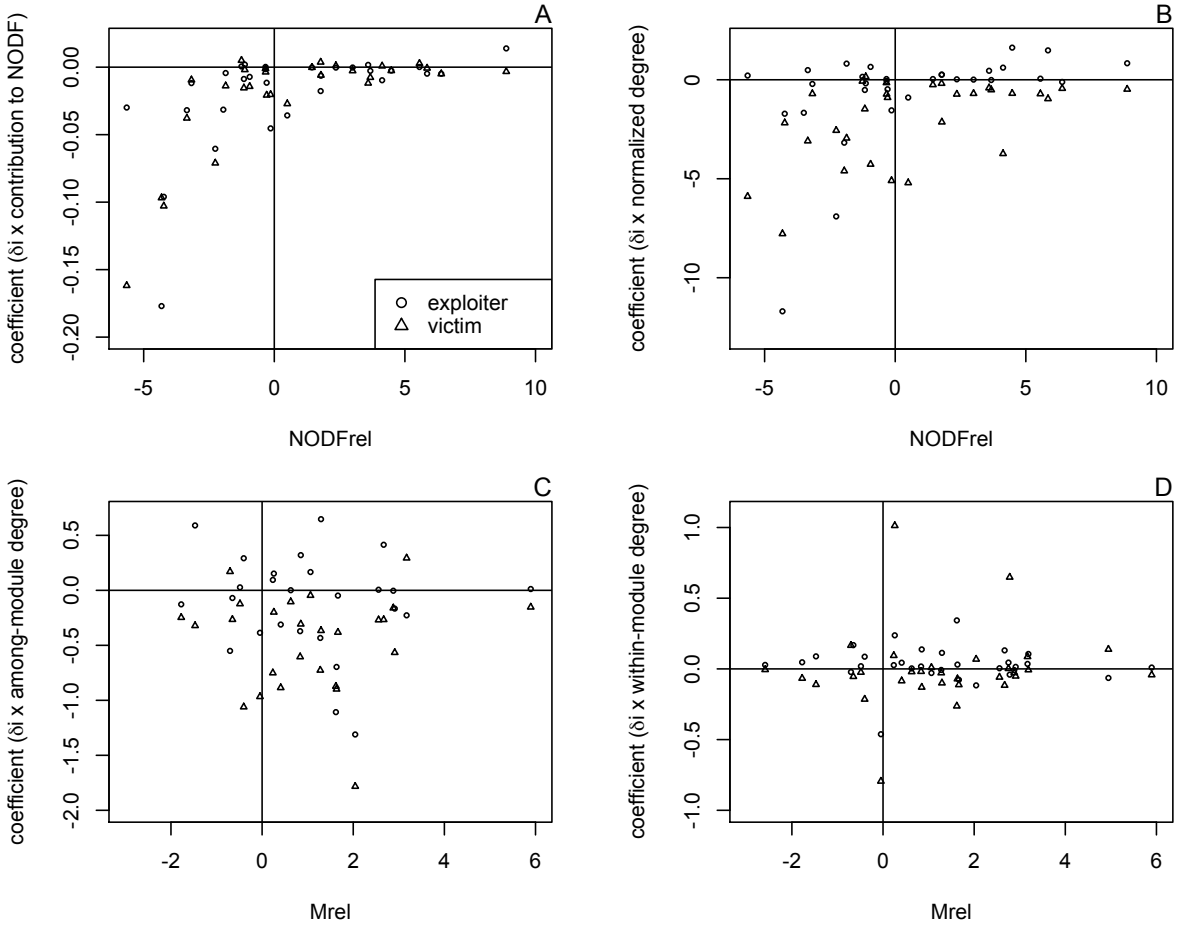

Figure 6: Relationship between the structural roles of victims and exploiters and directionality of their coevolutionary dynamics, δ_i. A and B: in networks that are less nested than expected (negative $NODF_{rel}$ values), there is a negative relationship between δ_i and nestedness contributions and normalized degree, whereas in networks that are more nested than expected (positive $NODF_{rel}$ values), this relationship is lost because trait changes are propagated throughout the entire network. C: among-module degree is negatively related to δ_i and independent of network structure. D: relationship between within-module degree and δ_i.

Table 1: List of the variables and parameters values used in the coevolutionary model

Notation	Definition	values
z_i	Quantitative trait value of species *i*	Sampled from a Normal distribution, mean=0, sd=0.1
Z	Matrix of trait matchings between species *i* and *j* containing all the z_{ij} values	variable
R	Total number of species in the network	variable
F	Empirical matrix of species interactions, in which f_{ij} =1 if species *i* interact with *j* or f_{ij} =0 if *i* do not interact with *j*	given by the data
h_i^2	Heritability of trait z_i	0.25
$k_{i(t)}$	Number of species with which *i* interacts. In the case of victims, if the trait difference is higher than the threshold ($\lvert z_{ij(t)} \rvert > \varepsilon$), then this interaction will be subtracted from its interaction partners	Initially given by the data. For victim species can vary during the simulation.
S_i	Partial selection differential caused by environmental selection	variable
M_{ij}	Partial selection differential caused by selection imposed by the interaction partners	variable
θ_i	Trait value of species *i* (z_i) favored by stabilizing selection	$\theta_i = z_{i(t=1)}$
ξ_s	Intensity of environmental selection	a value between 0 and 1

ξ_d	Intensity of selection imposed by the interaction partners	a value between 0 and 1
p_{ij}	Relative preference of exploiter species *i* on victim species *j*	variable
b	Degree of exploiter preference	10
u_{ji}	Inform if victim species *j* will respond to selection pressure exerted by exploiter *i*	u_{ji}=1 if $\|z_{ij(t)}\| \leq \varepsilon$; u_{ji}=0 if $\|z_{ij(t)}\| > \varepsilon$
ε	Maximum $\|z_{ij(t)}\|$ value that induces evolutionary response on victim *j*	0.5
C	Matrix of with the the cumulative change in trait matching between species *i* and j containing all the c_{ij} values	variable
δ_i	Directionality of species *i* trait change	variable
$<\delta>$	Average directionality of species trait change in a network	variable

Network structure and selection asymmetry drive coevolution in species-rich antagonistic interactions

Supporting Information

1. Networks used in the study

Table S1. Dataset[1] used to parameterize the matriz **F**, including the type of interaction and network basic characteristics (N_v = number of victims; N_e = number of exploiters, C = Connectance).

Data set	Type	N_v	N_e	C	Reference
herb1	Grasshoppers– plants	52	22	0.16	Joern 1979
herb2	Mammalian grazers – plants	10	17	0.6	Hansen et al. 1985
herb3	Grasshoppers – plants	54	24	0.14	Joern 1979
herb4	Leaf-chewing beetles – plants	10	32	0.14	Basset & Samuelson 1996
herb5	Gallers – host plants	10	17	0.12	Dawah & Hawkins 1995
herb6	Leaf-chewing catterpilars – plants	53	24	0.06	Henneman & Memmott 2001
herb7	Leaf-chewing catterpilars – plants	49	26	0.06	Henneman & Memmott 2001
herb8	Mining insects – host plants	204	94	0.01	Lewis et al. 2002
herb9	Sap-feeding insects – plants	42	35	0.05	Loye 1992
herb10	Mining insects – host	50	87	0.02	Memmott et al. 1994

	plants				
herb11	Sap-feeding insects – plants	26	25	0.07	Muller et al. 1999
herb12	Fruit-flies - plants	53	30	0.05	Novotny et al. 2005
herb13	Flower-feeders - plants	81	34	0.06	Prado & Lewinsohn 2004
herb14	Gallers – host plants	10	16	0.12	Tscharntke et al. 2001
herb15	Grasshoppers – plants	43	14	0.3	Ueckert & Hansen 1971
herb16	Phasmids– plants	38	13	0.11	Blüthgen et al. 2006
herb17	Leaf-chewing catterpilars – plants	40	49	0.05	Coley et al. 2006
herb18	Gallers – host plants	29	27	0.04	Cuevas-Reyes et al. 2007
par1	Fish – parasites	07	29	0.38	Arthur 1976
par2	Fish – parasites	10	40	0.22	Leong & Holmes 1981
par3	Fish – parasites	14	51	0.16	Arai and Mudry 1983
par4	Fish – parasites	17	53	0.18	Arai and Mudry 1983
par5	Fish – parasites	33	97	0.1	Bangham 1955
par6	Fish – parasites	06	25	0.35	Chinniah & Threlfall 1978
pred1[2]	Mammalian predator – prey	16	8	0.52	Baskerville et al. 2011
pred2	Mammalian predator – prey	22	5	0.8	Owen-Smith & Mills 2008
pred3	Seed-predators – plants	98	11	0.01	Janzen 1980
pred4	Seed-predators – plants	21	30	0.17	Nakagawa et al. 2003
pred5	Seed-predators – plants	15	37	0.17	Nakagawa et al. 2003
pred6	Mammalian predator – prey	18	4	0.66	Radloff & du Toit 2004

[1] These datasets are available online at the Interaction Web Database (www.nceas.ucsb.edu/interactionweb/index.html) and compiled in Pires & Guimarães (2013)

[2] Subnetwork depicting predator-prey interactions between mammals weighing > 5kg

2. Methodology

Hereafter the formulas used for computing nestedness (NODF); modularity (*M*); and species-level network metrics: each species contribution to nestedness; standardized within-module degree, and among-module connectivity are specified. All these metrics were applied to binary matrices of interaction, i.e., matrices of presence (1) or absence (0) of interaction among pairs of species.

2.1 Nestedness

Nestedness of the studied interaction matrices was accessed by computing NODF - *Nestedness metric based on Overlap and Decreasing Fill* (Almeida-Neto, Guimarães, Loyola, & Ulrich, 2008), a metric that also permits estimates of each species contribution to nestedness. NODF computing is as follows:

$$NODF = \frac{\sum N_{paired}}{\left[\frac{n(n-1)}{2}\right] + \left[\frac{m(m-1)}{2}\right]},$$

where N_{paired} is a degree of nestedness among pairwise rows and columns (see Almeida-Neto et al., 2008 for further details); *n* is the number of rows and *m* is the number of columns of the interaction matrix.

2.2 Species contribution to nestedness

The contribution of each matrix element (species) to nestedness is based on the metric NODF (Almeida-Neto et al., 2008), which considers the decreasing number of presences (1's) between successive rows and columns in a binary matrix and the percentage of presences that occur at identical positions between pairs of rows and columns. As with NODF, the species contribution to nestedness was developed based on the concept that a nested matrix presents decreasing fill and paired overlap of presences (1's) and its calculation

takes into account pairs of rows and pairs of columns. We describe in detail the procedure for calculating contribution to nestedness for the species represented in the rows, and the same is applied to columns.

First, assume a matrix of interactions with m rows and n columns. The number of presences (1's) in each row is contrasted to all the other rows and if the row considered (species *i*) presents equal or fewer number of interactions than the species with which it has been compared (species *j*), then the value zero (0) is attributed to the species *i* in relation to *j*. This value zero represents the lack of contribution of the pair of species *ij* in generating nestedness, as there is no decreasing fill between them. Then, this value is plotted in a square matrix (*m* x *m*), in which the *m* species are represented in both rows and columns and the value zero is the element a_{ij}. On the other hand, if the species *i* establishes more interactions than species *j*, its necessary to calculate the proportion of 1's of the species *j* that is exactly at the same column position to the 1's observed in the species *i*. This proportion is the value to be attributed to species *i* in relation to *j*, representing the power of species *i* in predicting the column occurrence of 1's in the species *j*. In this case, the element a_{ij} would be this proportion.

These pairwise comparisons generate a matrix, *m* x *m*, in which each row contains the proportions of 1's of the species represented in the row correctly predicted by the other species and each column represents the proportions of correct predictions made by the species represented in the column on the occurrences of 1's of the other species. The species contribution to nestedness depends on both proportions, and the average of these proportions (*Average1* and *Average2*) is calculated for each species:

Columns: $Average1 = \left(\sum_{i=1}^{i=m} a_{ij} \right) / (m-1)$

Rows: $Average2 = \left(\sum_{j=1}^{j=m} a_{ij} \right) / (m-1)$

Therefore, the species contribution to nestedness is the sum of *Average1* and *Average 2*, taking into account both the power of prediction and predictability of each species, quantifying its importance in generating nestedness on the matrix as a whole.

As mentioned above, the same methods used for species in rows were applied to the

species represented in columns, generating a square matrix of dimensions *n* x *n*, upon which all the other calculations must be made.

2.3 Modularity

The level of modularity (*M*) of each studied network was estimated using a simulated annealing optimization procedure (Guimerà & Amaral, 2005; Newman & Girvan, 2004). For a given division of network nodes into distinct modules, network modularity is computed as follows:

$$M = \sum_{s=1}^{N_M} \left[I_s/I - \left(k_s/2I\right)^2 \right],$$

where N_M is the number of modules in the network, I_s is the number of interactions among all the species within the module *s*, I is the total number of interactions in the network, and k_s is the sum of the number of interactions of all the species inside module *s*.

The simulated annealing algorithm computes different topological properties of each node in the network: the standardized within-module degree, which is a standardized measure of the extent to which each species is connected to the others in its own module and the among-module connectivity, which describes how well each species is connected to species in other modules. The formulas for computing these metrics are bellow.

2.4 Standardized within-module degree (mz_i)

$$mz_i = \frac{k_{is} - \overline{k_s}}{SD_{ks}},$$

where k_{is} is the number of interactions among i and the other species in its own module *s*, $\overline{k}_s$ and SD_{ks} are average and standard deviation of the number of within-module degree of all species in module s.

2.5 Among-module connectivity (mc_i)

$$mc_i = 1 - \sum_{x=1}^{N_M} \left(\frac{k_{ix}}{k_i} \right)^2 ,$$

where k_i is the number of interactions of species *i,* and k_{ix} is the number of interactions from *i* to species in module *x* (including *i*'s own module).

2.6 Core-periphery

We identified core and periphery species for each network using a categorical core–periphery analysis for bipartite networks (Borgatti and Everett 2000). Categorizing a species as part of the core or periphery relies on the sorting of exploiter and victim species in such a way that the connectance among core species is maximized while the connectance among peripheral species is minimized (Díaz-Castelazo et al. 2010). Thereafter, core species are connected to other core species as well as certain peripheral species, although peripheral species are not interconnected.

3. Sensitivity analysis and parameter combinations.

The sensitivity analysis was conducted via latin hypercube sampling with *R package pse*.

In this sensitivity analysis we evaluated the parameter space of the five parameters in the model (heritability (h^2), victim distance (ε), preference (b) and the intensity of the interaction selection (ξ_{d_i} for victims and ξ_{d_j} for exploiters). All networks had similar patterns and to illustrate the results we selected a highly nested and a highly modular network. The scatterplots (Figs S1 and S2) show the pairwise relationship between the response variable (the directionality of evolution, $\langle \delta \rangle$) and the parameters. Figures S3 and S4 show the partial rank correlation coefficients for each network and it can be observed that the intensity of interaction selection had higher coefficients.

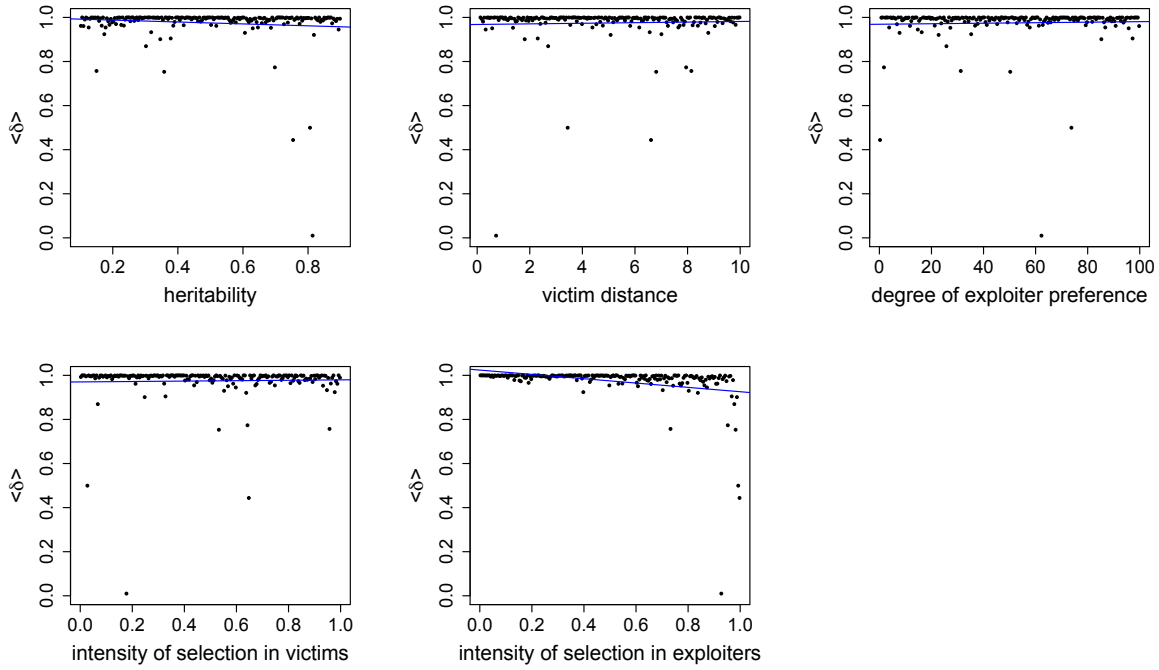

Figure S1: Relationships between the amount of directional evolution, $\langle \delta \rangle$, and the model parameters: heritability (h^2), victim distance (ε), degree of exploiter preference (b), intensity of interaction selection in victims (ξ_{d_i}) and intensity of interaction selection in exploiters (ξ_{d_j}) in pred1 (see Table S1), a highly nested network (NODF = 0.82, M = 0.16).

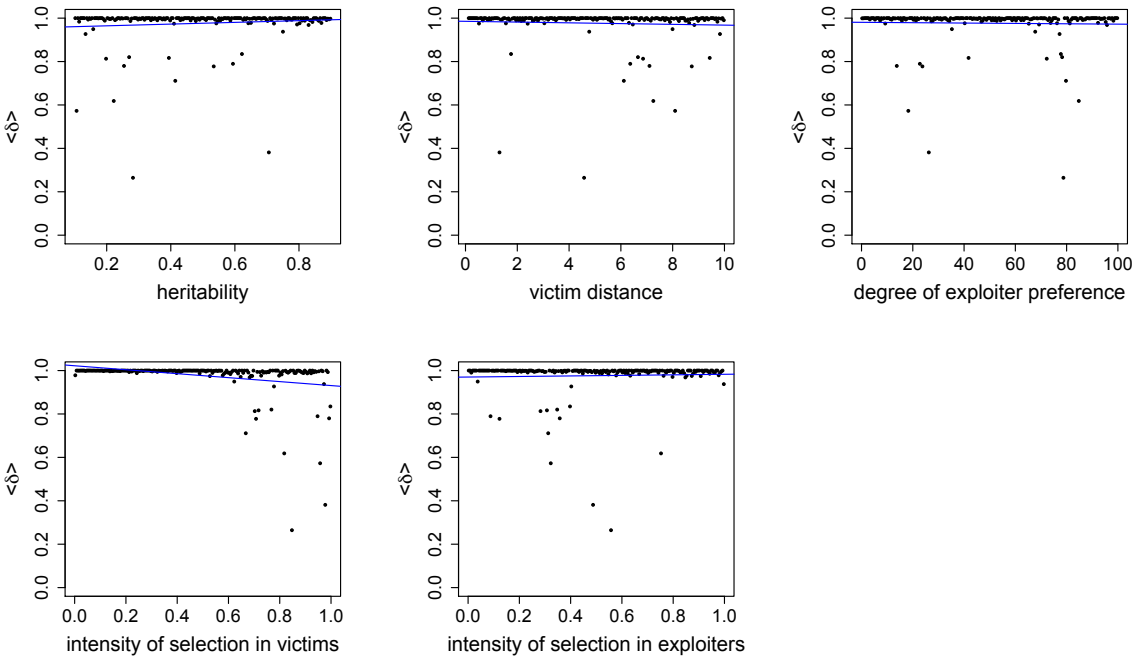

Figure S2: Relationships between the amount of directional evolution, $\langle \delta \rangle$, and the model parameters: heritability (h^2), victim distance (ε), degree of exploiter preference (b), intensity of interaction selection in victims (ξ_{d_i}) and intensity of interaction selection in exploiters (ξ_{d_j}) in herb18 (see Table S1), a highly modular network (NODF = 0, M = 0.96).

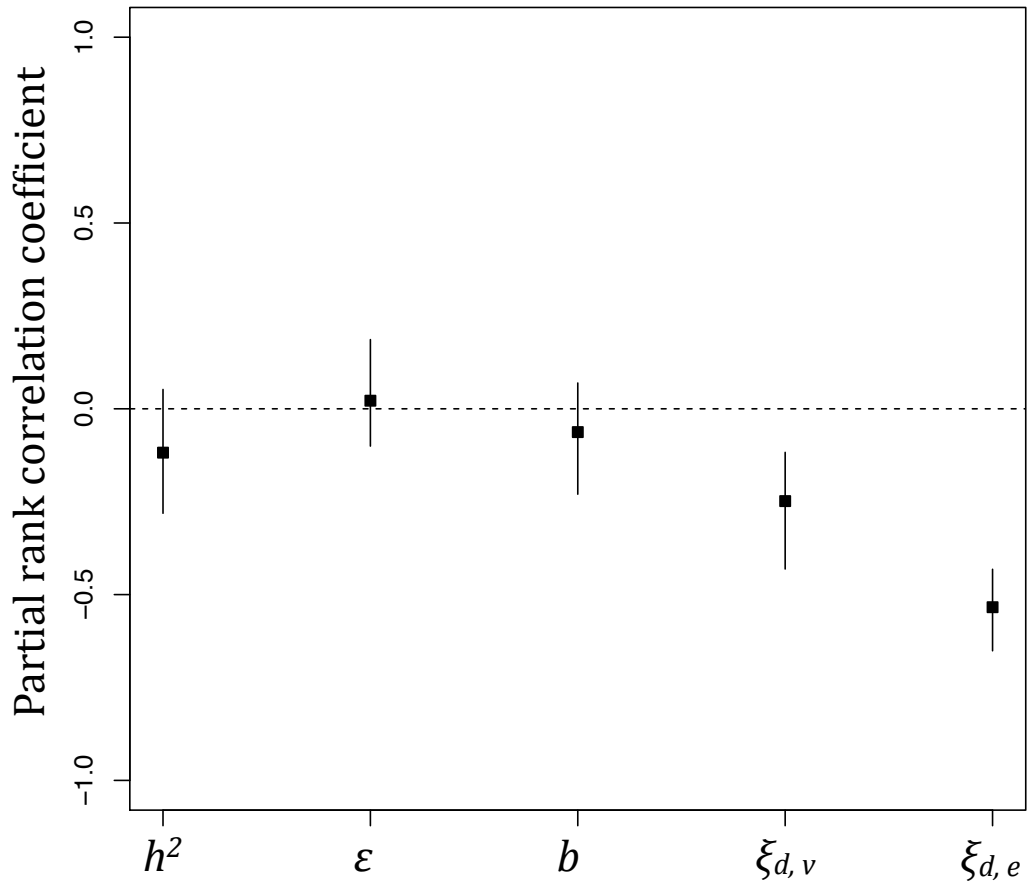

Figure S3: Partial rank correlation coefficients of the parameters: heritability (h^2), victim distance (ε), degree of exploiter preference (b), intensity of interaction selection in victims (ξ_{d_i}) and intensity of interaction selection in exploiters (ξ_{d_j}) in pred1 (see Table S1), a highly nested network (NODF = 0.82, M = 0.16). The partial rank correlation coefficient measures how strong are the linear associations between the result and each input parameter, after removing the linear effect of the other parameters. The confidence intervals were generated by bootstraping.

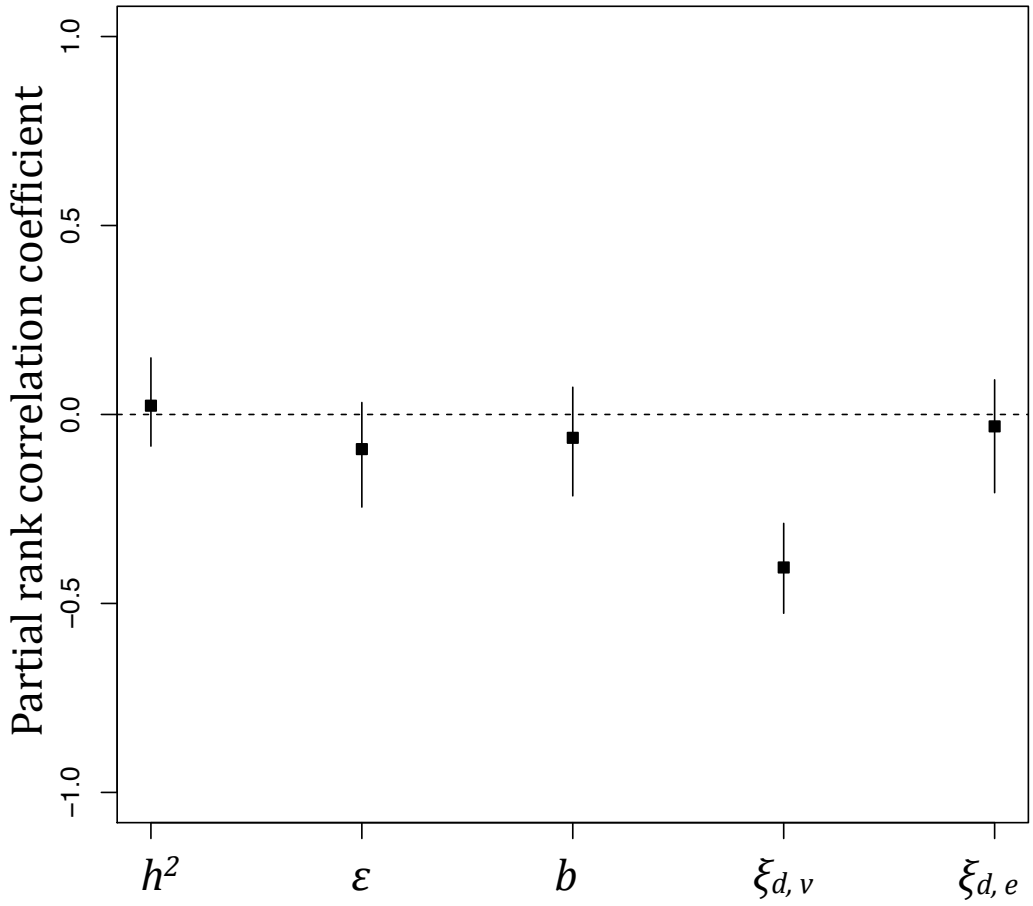

Figure S4: Partial rank correlation coefficients of the parameters: heritability (h^2), victim distance (ε), degree of exploiter preference (b), intensity of interaction selection in victims (ξ_{d_i}) and intensity of interaction selection in exploiters (ξ_{d_j}) in herb18 (see Table S1), a highly nested network (NODF = 0, M = 0.96). The partial rank correlation coefficient measures how strong are the linear associations between the result and each input parameter, after removing the linear effect of the other parameters. The confidence intervals were generated by bootstraping.

4. Additional results for the main model

4.1. Network level results

We synthesized the different aspects of network structure using a principal component analysis (PCA; see Sazima *et al.* 2010 for a similar approach) and used GLMs to analyze the effects of the first and second principal components (PC1 and PC2, respectively) on network and species'coevolutionary dynamics and. When synthesized in a PCA, PC1 and PC2 explained 50% and 24.4% of the variance in structural patterns across networks, respectively. The metrics richness and connectance had the highest coefficients in the PC1 eigenvector, with richness having a positive effect and connectance having a negative effect (Table S2), whereas the metric nestedness had the highest positive coefficient in the PC2 eigenvector (Table S2). Considering the directionality of evolution, only PC2 presented a significant negative effect (b = -0.18, F = 17.68, p = 0.0002), with PC1 not presenting an effect on $\langle\delta\rangle$ (b = 0.007, F = 0.04, p = 0.85). These results are consistent with the path analysis results and indicate that nestedness is the structural pattern with the highest negative effect on the directionality of evolution. PC2 also had a negative effect on the disparity of final traits (b = -0.09, F = 12.02, p < 0.0001) and a positive effect on the proportion of lost interactions (b = 8.98, F = 31.99, p < 0.0001). Finally, PC1 positively affected the disparity of final traits (b = 0.09, F = 36.94, p < 0.0001) but not the proportion of lost interactions (b = 0.97, F = 0.37, p = 0.55).

Table S2: Eigenvalues of the first (PC1) and second (PC2) principal components for the network metrics.

	PC1	PC2
Richness	0.60524	0.09617
Connectance	-0.62285	-0.01448
NODFrel	-0.18307	0.96636
Mrel	0.46068	0.2381

Consistent with δ_i, PC1 had a negative effect on the magnitude of directional change in species trait values (b = -0.033, F = 106.84, p < 0.0001), whereas PC2 had a positive effect (b = 0.017, F = 127.1, p < 0.0001).

Table S3: Eigenvalues of the first (PC1) and second (PC2) principal components for the species metrics.

	PC1	PC2
Normalized degree	0.5653	-0.09709
Contribution to nestedness	0.55762	-0.27791
Among-module connectivity	0.55231	-0.05849
Within-module degree	0.25387	0.9539

Table S4: General linear models results for the effects of nestedness and modulatity on trait disparity on the five selection scenarios. Scenario 1: intensity of interaction selection is higher in exploiters ($\xi_{d_i} = 0.5 < \xi_{d_j} = 0.99$), scenario 2: intensity of interaction selection is higher in victims ($\xi_{d_i} = 0.99 > = \xi_{d_j} 0.5$), scenarios 3: symmetric and intermediate interaction selection ($\xi_{d_i} = \xi_{d_j} = 0.5$), scenario 4: symmetric and strong interaction selection ($\xi_{d_i} = \xi_{d_j} = 0.99$) and scenario 5: symmetric and weak interaction selection ($\xi_{d_i} = \xi_{d_j} = 0.01$).

		F	**slope (b)**	**p**
Nestedness	scenario 1	25.24	-0.03	< 0.0001
	scanario 2	4.106	-0.031	0.05
	scenario 3	14.1	-0.014	< 0.0001
	scenario 4	3.817	-1.196	0.06
	scenario 5	0.158	-0.001	0.694
Modularity	scenario 1	2.06	0.02	0.16
	scanario 2	3.633	0.056	0.07
	scenario 3	4.216	0.017	0.049
	scenario 4	2.69	1.933	0.112
	scenario 5	4.187	0.009	0.049

Table S5: General linear models results for the effects of nestedness on the proportion of lost interactions on the five selection scenarios. Scenario 1: intensity of interaction selection is higher in exploiters ($\xi_{d_i} = 0.5 < \xi_{d_j} = 0.99$), scenario 2: intensity of interaction selection is higher in victims ($\xi_{d_i} = 0.99 > = \xi_{d_j}$ 0.5), scenarios 3: symmetric and intermediate interaction selection ($\xi_{d_i} = \xi_{d_j} = 0.5$), scenario 4: symmetric and strong interaction selection ($\xi_{d_i} = \xi_{d_j} = 0.99$) and scenario 5: symmetric and weak interaction selection ($\xi_{d_i} = \xi_{d_j} = 0.01$).

		F	**b**	**p**
Nestedness	scenario 1	19.7	2.18	< 0.05
	scanario 2	13.73	0.04	< 0.05
	scenario 3	7.203	0.001	< 0.05
	scenario 4	42.42	0.053	< 0.05
	scenario 5	16.445	0.006	< 0.05

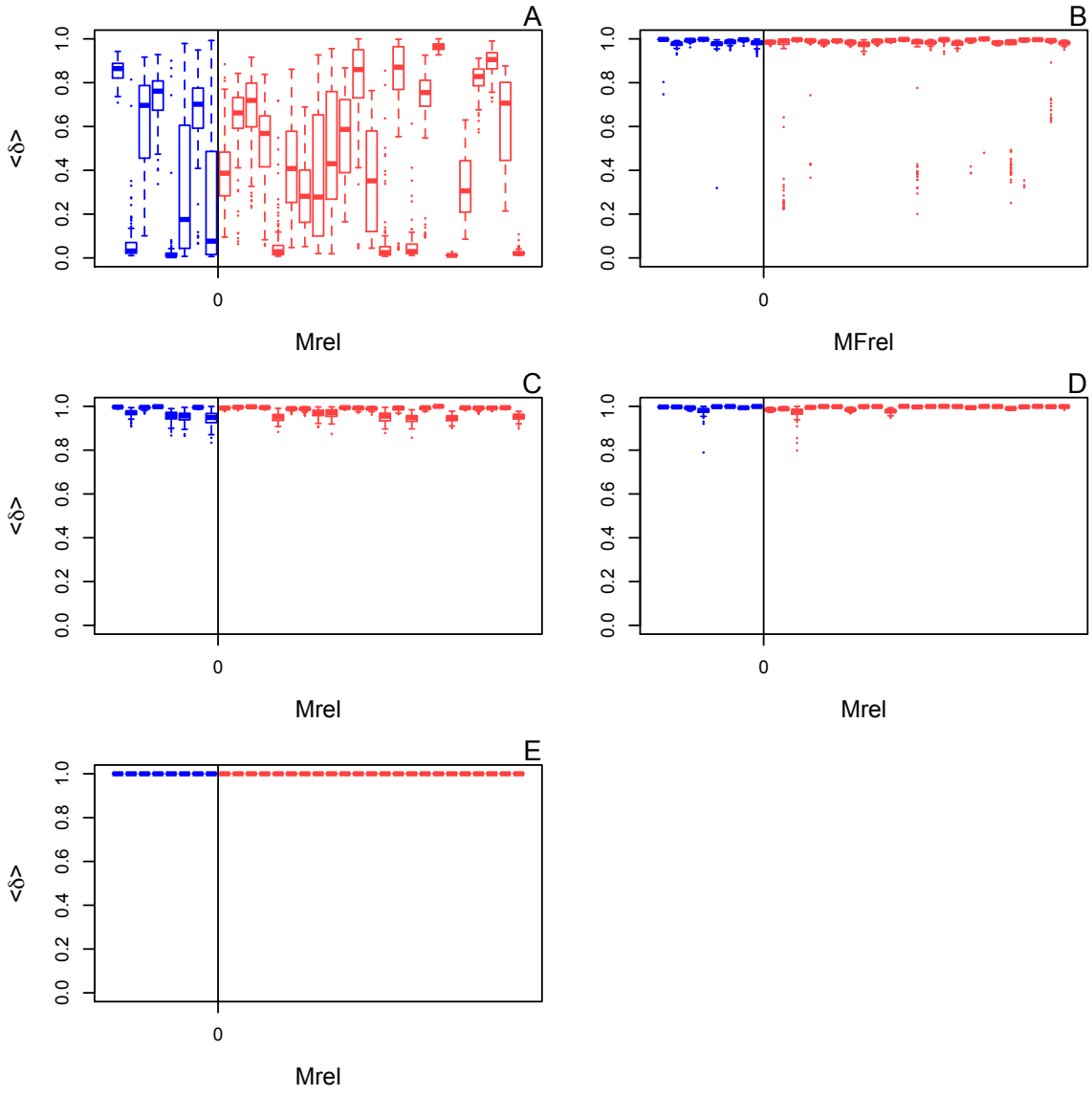

Figure S5: Relationship between network modularity z-score (Mrel) and the mean directionality of coevolution, $\langle \delta \rangle$, on each network per simulation replicate. Mrel values lower then zero (blue) represents networks that are less modular than the expected by the null model, whereas Mrel values higher than zero (red) represents networks that are more modular than the expected by the null model. A: results from scenario 1, on which the intensity of interaction selection is higher in exploiters ($\xi_{d_i} = 0.5 < \xi_{d_j} = 0.99$), B: results from scenario 2, on which the intensity of interaction selection is higher in victims ($\xi_{d_i} = 0.99 > = \xi_{d_j}$ 0.5). C, D and E are the results from scenarios 3, 4 and 5, respectively, on which the intensity of interaction selection is equal between exploiters and victims. In C both are medium ($\xi_{d_i} = \xi_{d_j} = 0.5$), in D both are strong ($\xi_{d_i} = \xi_{d_j} = 0.99$) and in E both are weak ($\xi_{d_i} = \xi_{d_j} = 0.01$).

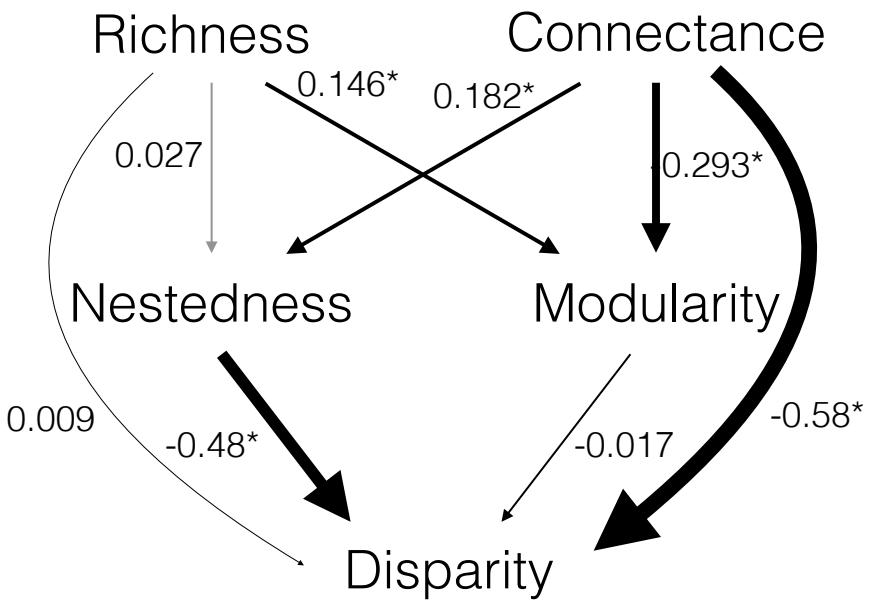

Figure S6: Summary diagram of the effects of different network architectural patterns on trait disparity. The thickness of the arrows is scaled to standardized coefficients from the path analysis and illustrates the relative effect strength. Significant effects are represented in black and have an asterisk, and non-significant effects are represented in gray. The effects of connectance and richness are split between direct effects and indirect effects through changes in modularity and nestedness.

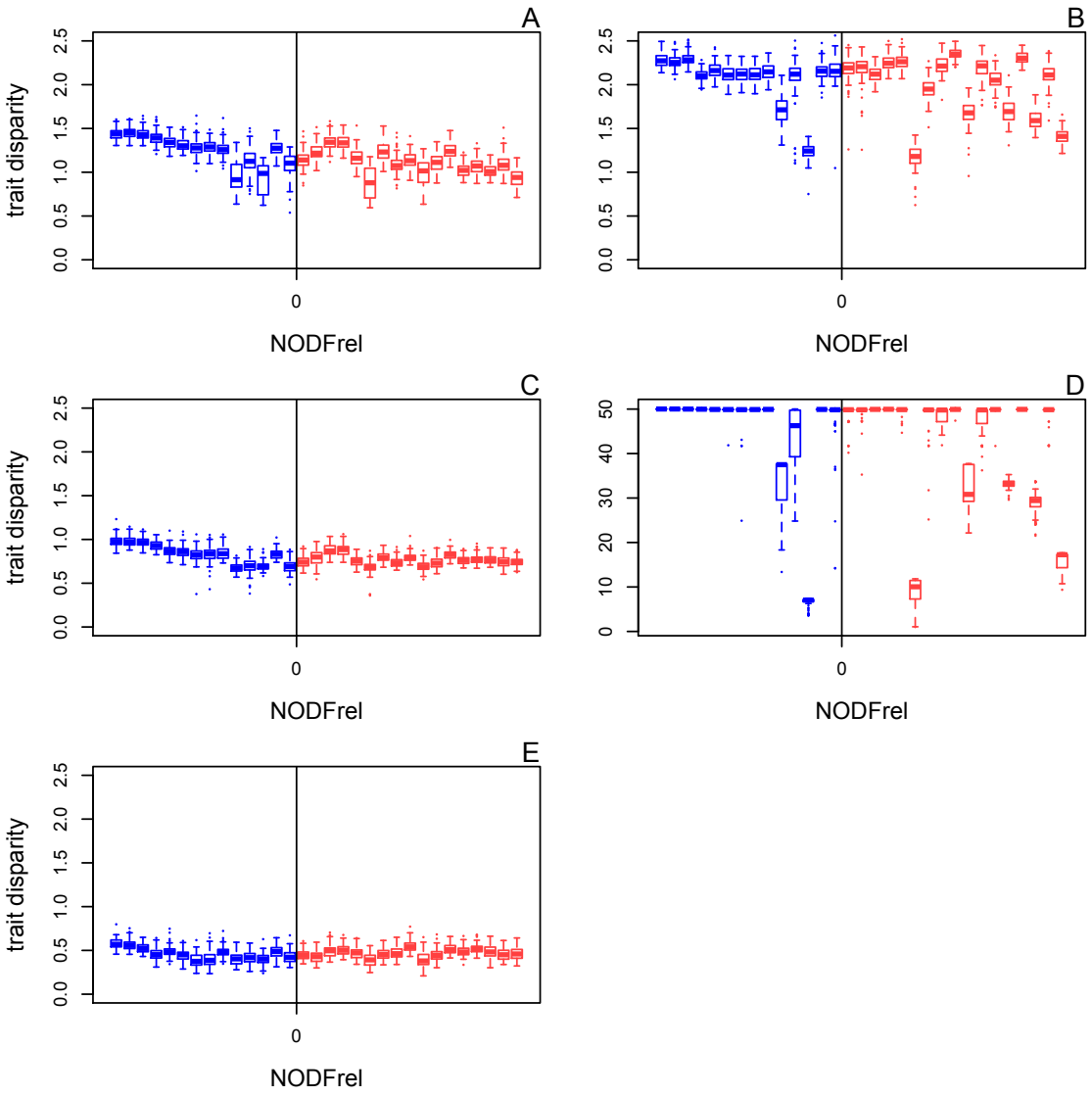

Figure S7: Relationship between network nestedness z-score (NODFrel) and trait disparity on each network per simulation replicate. A: results from scenario 1, on which the intensity of interaction selection is higher in exploiters ($\xi_{d_i} = 0.5$; $\xi_{d_j} = 0.99$), B: results from scenario 2, on which the intensity of interaction selection is higher in victims ($\xi_{d_i} = 0.99$; $\xi_{d_j} = 0.5$). C, D and E are the results from scenarios 3, 4 and 5, respectively, on which the intensity of interaction selection is equal between exploiters and victims. In C both are medium ($\xi_{d,v} = \xi_{d_j} = 0.5$), in D both are strong ($\xi_{d_i} = \xi_{d_j} = 0.99$) and in E both are weak ($\xi_{d_i} = \xi_{d_j} = 0.5$).

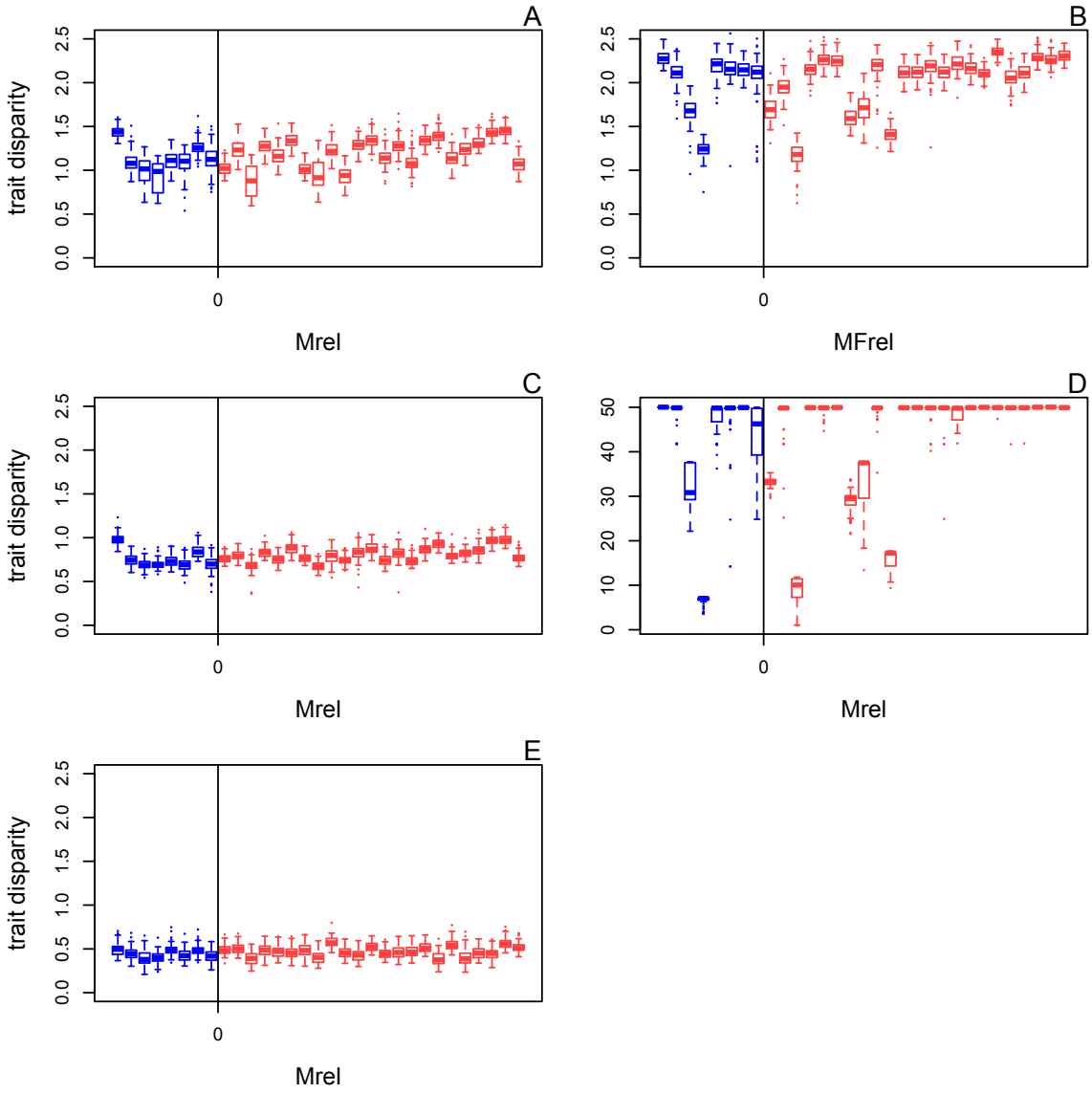

Figure S8: Relationship between network modularity z-score (Mrel) and the trait disparity on each network per simulation replicate. Mrel values lower then zero (blue) represents networks that are less modular than the expected by the null model, whereas Mrel values higher than zero (red) represents networks that are more modular than the expected by the null model. A: results from scenario 1, on which the intensity of interaction selection is higher in exploiters ($\xi_{d_i} = 0.5$; $\xi_{d_j} = 0.99$), B: results from scenario 2, on which the intensity of interaction selection is higher in victims ($\xi_{d_i} = 0.99$; $\xi_{d_j} = 0.5$). C, D and E are the results from scenarios 3, 4 and 5, respectively, on which the intensity of interaction selection is equal between exploiters and victims. In C both are medium ($\xi_{d,v} = \xi_{d_j} = 0.5$), in D both are strong ($\xi_{d_i} = \xi_{d_j} = 0.99$) and in E both are weak ($\xi_{d_i} = \xi_{d_j} = 0.5$).

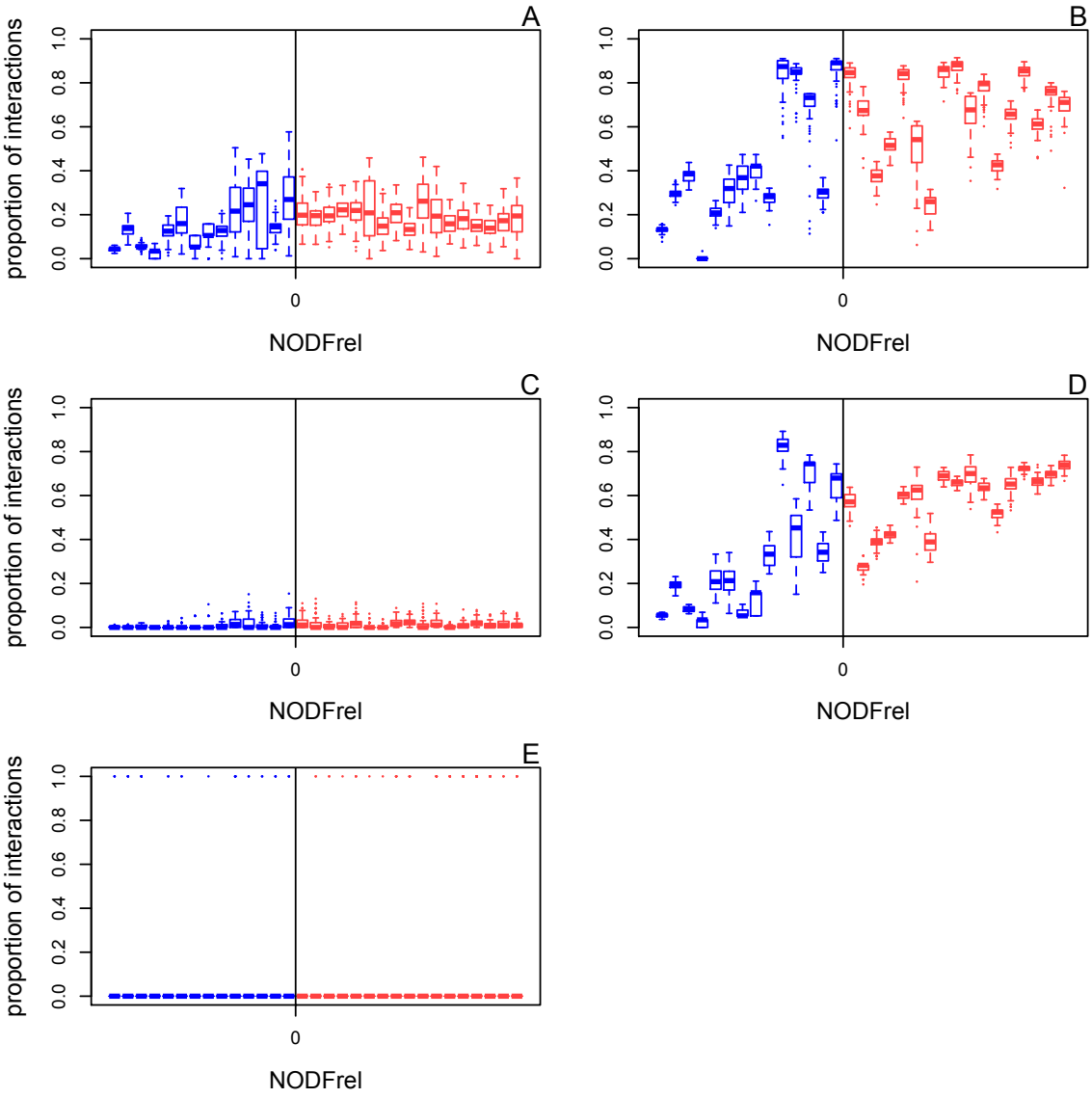

Figure S9: Relationship between network nestedness z-score (NODFrel) and the proportion of interactions victims "escaped" ($|z_i - z_j| > \varepsilon$) on each network per simulation replicate. A: results from scenario 1, on which the intensity of interaction selection is higher in exploiters ($\xi_{d_i} = 0.5$; $\xi_{d_j} = 0.99$), B: results from scenario 2, on which the intensity of interaction selection is higher in victims ($\xi_{d_i} = 0.99$; $\xi_{d_j} = 0.5$). C, D and E are the results from scenarios 3, 4 and 5, respectively, on which the intensity of interaction selection is equal between exploiters and victims. In C both are medium ($\xi_{d,v} = \xi_{d_j} = 0.5$), in D both are strong ($\xi_{d_i} = \xi_{d_j} = 0.99$) and in E both are weak ($\xi_{d_i} = \xi_{d_j} = 0.5$).

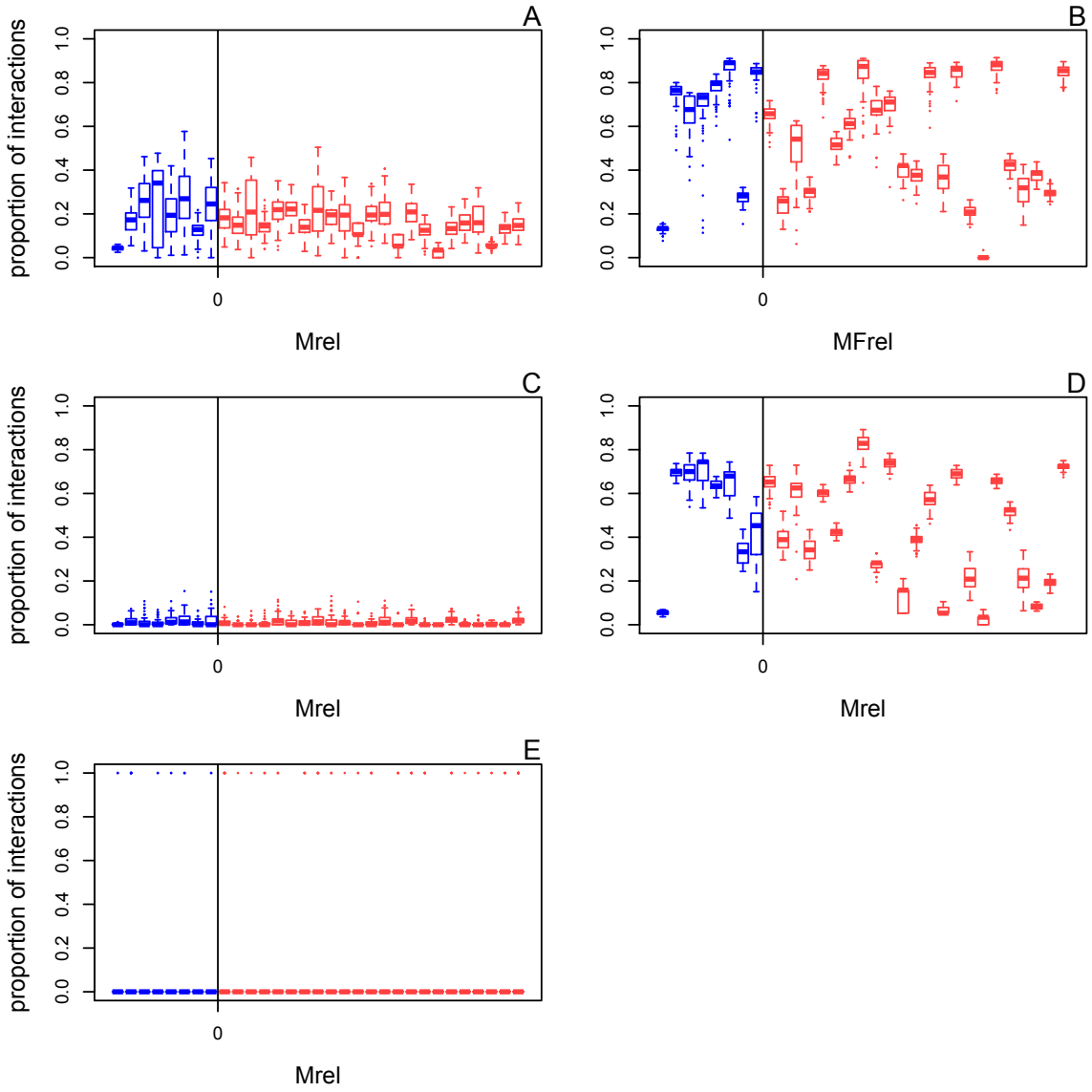

Figure S10: Relationship between network modularity z-score (Mrel) and the proportion of interactions victims "escaped" ($|z_i - z_j| > \varepsilon$) on each network per simulation replicate. Mrel values lower then zero (blue) represents networks that are less modular than the expected by the null model, whereas Mrel values higher than zero (red) represents networks that are more modular than the expected by the null model. A: results from scenario 1, on which the intensity of interaction selection is higher in exploiters ($\xi_{d_i} = 0.5$; $\xi_{d_j} = 0.99$), B: results from scenario 2, on which the intensity of interaction selection is higher in victims ($\xi_{d_i} = 0.99$; $\xi_{d_j} = 0.5$). C, D and E are the results from scenarios 3, 4 and 5, respectively, on which the intensity of interaction selection is equal between exploiters and victims. In C both are medium ($\xi_{d,v} = \xi_{d_j} = 0.5$), in D both are strong ($\xi_{d_i} = \xi_{d_j} = 0.99$) and in E both are weak ($\xi_{d_i} = \xi_{d_j} = 0.5$).

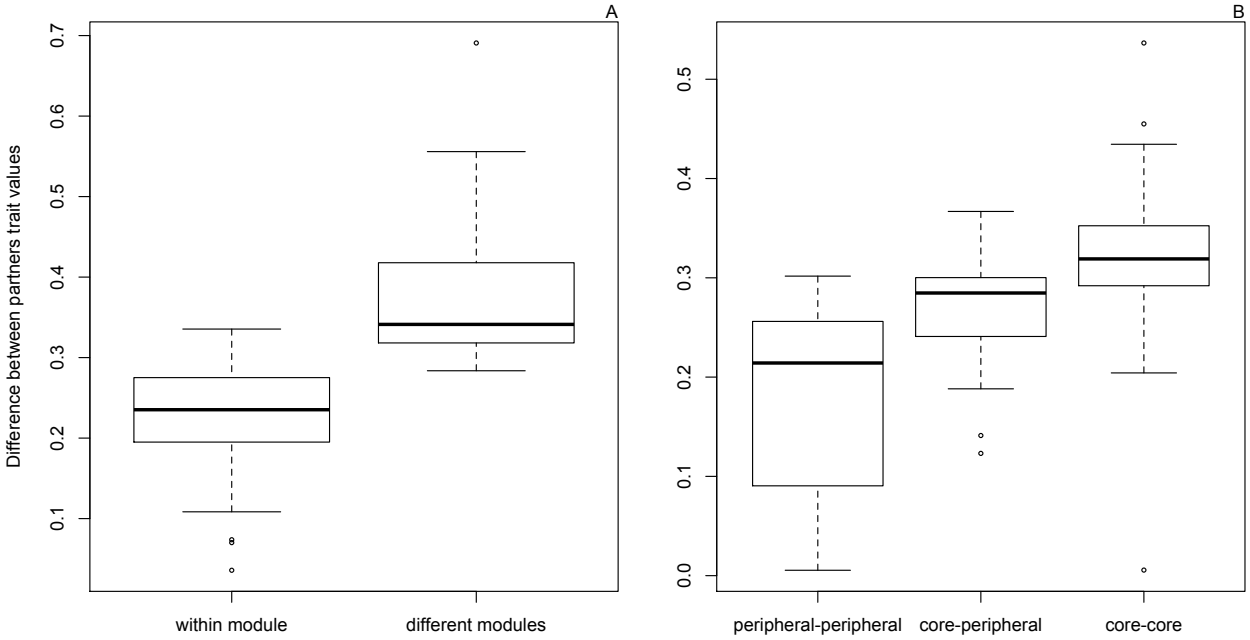

Figure S11: Network mean final trait matching between victim and exploiter interacting species belonging to the same or different modules (A) and among peripheral, core-peripheral or core-core partners (B) in the 31 antagonistic networks.

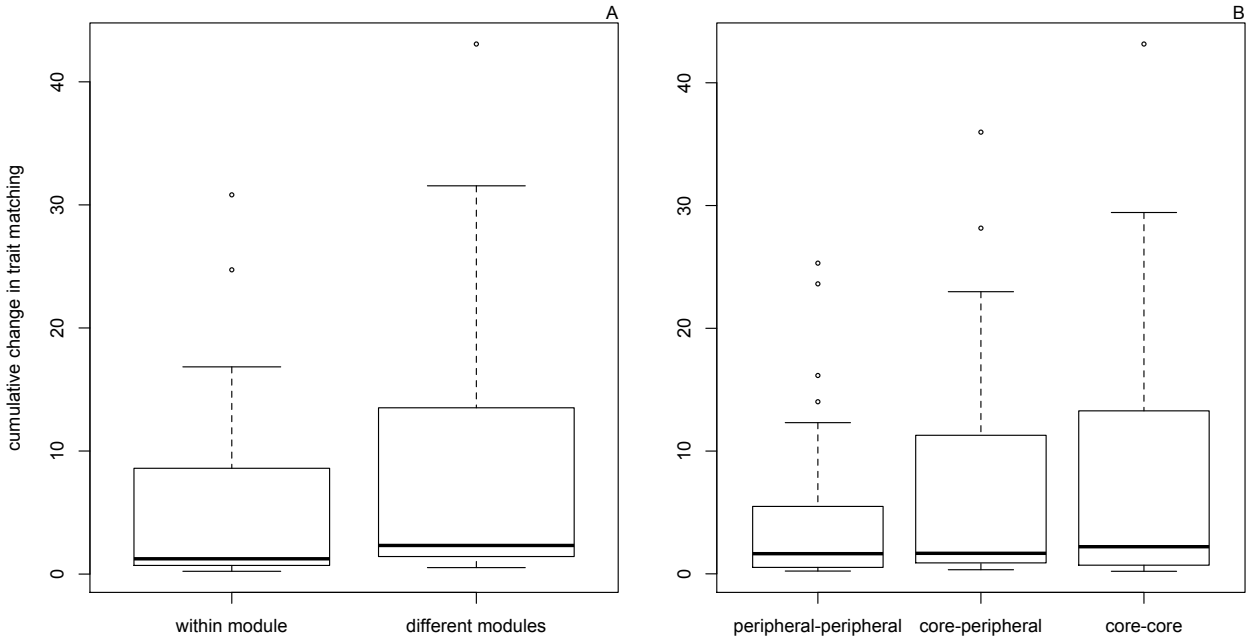

Figure S12: Network mean cumulative change in trait matching between victim and exploiter interacting species belonging to the same or different modules (A) and among peripheral, core-peripheral or core-core partners (B) in the 31 antagonistic networks.

5. Restricting the reciprocal evolutionary response to a subset of interacting species

In our main model, we assumed that all the species that interact exerts an evolutionary pressure on its partners, except in the case when the victims escape the interaction. To break the assumption of reciprocal selection in all the interactions, we simulated scenarios where only a percentage of species show reciprocal response to their partners. In this case, we initially sampled the percentage of species on each group (victims and exploiters) that are going to respond to their partners and the species that are not going to respond only experience stabilizing selection. We selected scenarios where: 100%, 75%, 50%, 25% and 0% of exploiter species are under selection to match their victims traits whereas 0%, 25%, 50%, 75% and 100% of victims are under selection to mismatch their exploiters. We run those simulations for 10,000 time steps, 100 replicates for each network. We used the same parameters used in scenario 1 of the main text, that is, $\xi_{d_i} = 0.99$; $\xi_{d_j} = 0.5$; $h^2 = 0.25$; $b = 10$ and $\varepsilon = 0.5$. The mean directionality of evolution found for each network in each scenario is summarized in Fig. S5. The 1:1 scenario, where all the species are experiencing interaction and stabilizing selection is exactly the same as the scenario 1 of the main text.

Comparing the different scenarios (Figure S5) we can observe that the situation where all the species are experiencing selection by its interaction partners (1:1) is the one where we can have a higher variability among networks in the mean directionality of evolution. In the other cases the directionality is high in all networks. This indicate that the network structure only influences the species evolutionary dynamics when all the species in the community are experiencing reciprocal selection. When the selection is not reciprocal the evolutionary dynamics is highly directional in all the networks.

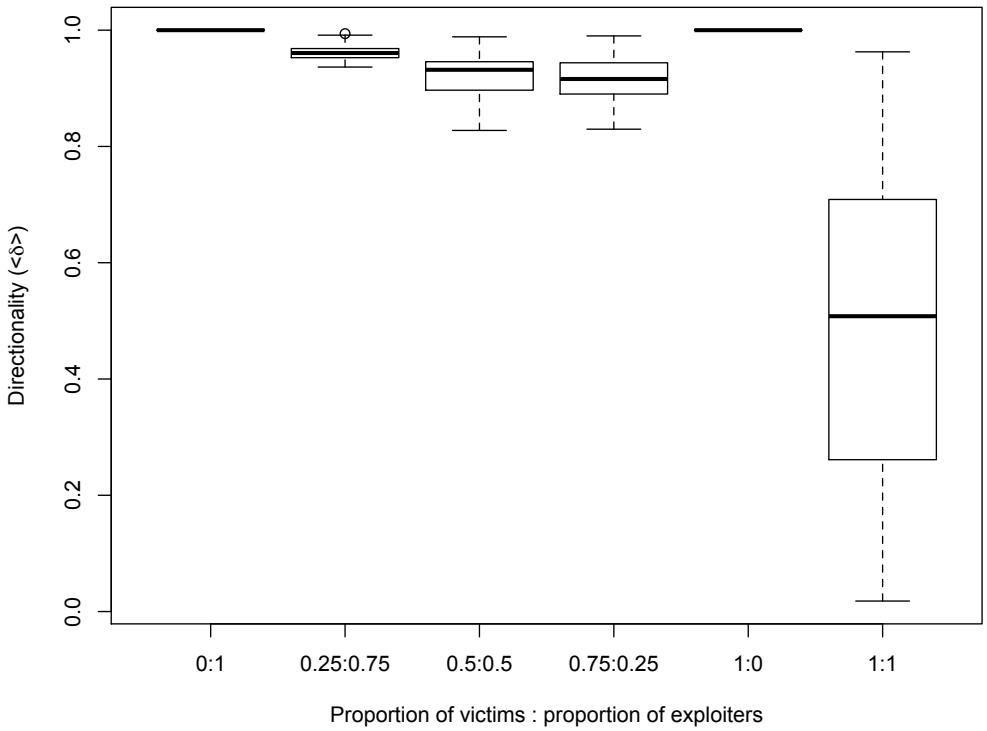

Figure S5: Average directionality $\langle \delta \rangle$ values among 100 replicates in the 31 antagonistic networks for the scenarios where: 100%, 75%, 50%, 25% and 0% of exploiter species are under selection to match their victims traits whereas 0%, 25%, 50%, 75% and 100% of victims are under selection to mismatch their exploiters.

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Capítulo 2

Trait matching and exploitation barriers differently drive evolution in antagonistic networks

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Abstract

A central problem in the study of ecological networks is to understand the underlying ecological and evolutionary processes shaping the observed structural patterns. Phenotypic traits are likely to shape the organization of ecological networks by modulating the likelihood of an interaction or by constraining the interaction between otherwise potentially interacting species We modeled single-trait evolution in victim and exploiter species interacting in antagonistic networks assuming two different functional effects of traits: (i) matching of defense and attack traits or (ii) exploitation barriers imposed by traits. Both trait matching and exploitation barriers reproduced more than 50% of the empirical network structures and the remaining networks had an overestimated modularity and underestimated nestedness. However, these functional effects affected trait evolution in different ways. For example, the exploitation barrier model led to more directional and less temporally variable trait evolution. Trait mediated interactions and coevolution also predicted turnover of interactions, a pattern observed in empirical networks. Therefore, coevolution differently drove the evolution of species traits when interactions were mediated by distinct functional relationships, but this had no impact on the way the antagonistic interactions were organized. Combining trait information with ecological network studies is the current challenge for linking evolutionary and ecological dynamics of species interactions and our results contribute to the understanding of how species may coevolve in time.

Introduction

Biodiversity is organized into complex ecological networks of interacting species (Bascompte 2009, Thompson 2009, Pimm *et al.* 1991). The network organization of interactions among species may affect different aspects of ecological and evolutionary dynamics, from community stability (Allesina & Tang 2012, Thébault & Fontaine 2010, Rohr *et al.* 2014) to ecosystem functioning (Díaz *et al.* 2013, Lavorel *et al.* 2013) and coevolution (Guimarães *et al.* 2011, Nuismer *et al.* 2013). At the same time, a central problem in the study of ecological networks is to understand the underlying ecological and evolutionary processes shaping the observed structural patterns. Two pervasive network patterns that have been widely observed in ecological networks are nestedness and modularity. Nestedness is the extent to which specialist species tend to interact with proper subsets of the species that interact with more generalists ones. Modularity is the compartmentalization of the network into semi-independent groups or modules (Newman 2006). Nestedness is hypothesized to buffer communities against extinctions or temporal fluctuations in the abundance of specialist species (Rohr *et al.* 2014, Bascompte *et al.* 2006, Suweis *et al.* 2013) and to increase biodiversity by promoting species coexistence (Bastolla *et al.* 2009, but see Allesina & Tang 2012). Modularity is hypothesized to increase the stability of interaction networks since disturbances are less likely to spread across different modules that are weakly connected (Thébault & Fontaine 2010, Allesina & Tang 2012). Several factors influence the organization of ecological networks, as for example the distribution of species abundance (Lewinsohn *et al.* 2006, Krishna *et al.* 2008, Canard *et al.* 2014), habitat heterogeneity within communities (Schleuning *et al.* 2011, Flores *et al.* 2013), shared evolutionary history (Rezende *et al.* 2007, 2009, Eklof *et al.* 2012) and species phenotypic traits (Woodward *et al.* 2005, Stang *et al.* 2006, Donatti *et al.* 2011).

Phenotypic traits, such as body size, morphology, chemical composition, physiology, temporal activity, feeding preferences, and behavior are likely to shape the organization of ecological networks by modulating the likelihood of an interaction (Williams & Martinez 2000, Eklöf *et al.* 2013, Rezende *et al.* 2007, Pires *et al.* 2011, Blüthgen *et al.* 2008, Santamaría & Rodríguez-Gironés 2007, Stang *et al.* 2006) or by constraining the interaction of otherwise potentially interacting species (Mouillot *et al.* 2008, Olesen, Bascompte, *et al.* 2011). The role of traits in shaping pairwise interactions is well documented in antagonistic interactions, in which the outcome results in a negative effect on the fitness of one of the

interacting partner and a positive effect for the other (Toju 2011, Benkman *et al.* 2003). At the network level, the functional effect of traits on pairwise interactions may influence the interaction patterns of individuals leading to both nested and modular structures. For instance, variation in body size explains nested patterns in predator-prey interactions among African mammals (Sinclair & Arcese 2011). Nestedness can also emerge from behavioral feeding choices. In fact, optimal diet theory predicts that an adaptive consumer utilizes all the resources above a specific threshold quality (MacArthur & Pianka 1966, Kondoh 2003), which in association with variation in resource use may lead to nestedness, at least within populations (e.g., Lemos-Costa *et al. in press*). Optimal diet choice also implies that nested patterns can emerge as a result of interspecific hierarchy in resource quality combined with consumers that have varying exploitation threshold levels/barriers (Cohen *et al.* 2003, Pires *et al.* 2014). In contrast, trait matching constraints may restrict interactions, thereby increasing compartmentalization (Cattin *et al.* 2004, Rossberg *et al.* 2006, Yeakel *et al.* 2012) and promoting the emergence of modules (Lewinsohn *et al.* 2006, Krasnov *et al.* 2012, Rezende *et al.* 2009). Therefore, the functional effects of traits that mediate species interactions and species fitness may affect overall community interaction patterns. Since network structure modulates the selection pressures imposed by interacting species and may promote the coevolution of traits that mediate interactions (Thompson 2013), species traits and network structure are both determinants and outcomes of ecological and evolutionary processes acting on species interactions (Guimarães *et al.* 2011, Nuismer *et al.* 2013).

Network structure potentially drives the structure of selection among interacting species (Guimarães *et al.* 2011, Nuismer *et al.* 2013). Nestedness favors the occurrence of evolutionary cascades in mutualistic networks (Guimarães *et al.* 2007) and phenotypic convergence and complementarity among mutualistic partners (Guimarães *et al.* 2011, Nuismer *et al.* 2013). In antagonistic networks nestedness is related to prey susceptibility levels and a hierarchy in prey preference by consumers. This hierarchical relationship could lead to coevolutionary alternation in defense and counter-defense traits (Nuismer & Thompson 2006). Modularity would favor trait convergence among unrelated species interacting with similar partners (Krasnov *et al.* 2012) and arms races between interacting species (Thompson 2013). Thus, network structure can favor distinct coevolutionary dynamics. In this regard, understanding the functional effects of traits on how coevolution and network structure is a fundamental step to link the ecological and evolutionary dynamics of coexisting species (Thompson 2013).

In this study we explored how species coevolution driven by distinct functional relationships between traits and interaction probabilities shape the structure of antagonistic networks. Using two simple models, we investigated whether coevolution mediated by linkage rules representing trait matching and exploitation barriers can reproduce the topological properties of empirical antagonistic networks. In the trait matching model, the similarity between the traits of exploiters and victims determines whether individuals from a given pair of species can interact. For example a high similarity between cuckoo and host eggs decreases the probability of egg rejection (Krüger 2007, Vikan *et al.* 2011). In the barrier traits model, what determines whether species pairs interact is not their similarity but rather the ability of the exploiter to overcome victims defense. Interactions between seed predators and host plants such as the camellia weevils are a good example of exploitation barriers (Toju 2011). In this case, weevils successfully infect camellia seeds only if their rostra are longer than camellia fruit pericarp. Otherwise, the camellia seeds are protected from predation (Toju 2011). We used these two models to (i) compare the structural patterns favored by trait matching and exploitation barrier functional relationships, exploring which mechanism better predicts the structure of empirical networks; (ii) investigate if distinct mechanisms differently drive species trait evolution; and (iii) explore the feedbacks between network structure and species trait evolution. Our results indicate that different functional effects of traits predicted equally well the nestedness and modularity of empirical antagonistic networks. However, these functional effects affected trait evolution in different ways. The exploitation barrier model led to more directional and less temporally variable trait evolution while the trait matching model led to more fluctuations in trait evolution.

Methods

The evolutionary models

We combined an adaptive network framework (Gross & Blasius 2008) and evolutionary modeling to study how different functional relationships affect species trait evolution and network organization. The adaptive network approach combines the evolutionary dynamics on network with the dynamics of the network itself. Combining these two dynamics allows us to explore how the network topology influences species evolutionary process and simultaneously, how species coevolution consequently re-shapes the network.

This approach allowed us to track trait changes, quantify the expected species trait distribution in the community, and measure interaction turnover and network structural change.

In our model interaction networks were initially assembled according to the probabilities determined by functional relationships of the mechanism of interaction linkage. Species traits evolved in response to the selection pressures imposed by antagonistic interactions and the environment. Trait evolution redefined the probabilities of interactions, which could reshape network structure in a feedback loop. Species are characterized as victims or exploiters and their interactions are represented as two-mode networks, in which exploiters interact with victims but there are no interactions among species that belong to the same trophic level. We modeled the mean trait value of a population of species i as a real number, z_i, which represented a defensive trait if species i is a victim or an attack trait if i is an exploiter. The mechanism mediating interactions among exploiters and victims defines the functional relationship between species trait and fitness and therefore the selection pressures. The value of z_i was initially sampled from a uniform distribution ranging from 0 to 2, simulating a situation in which there is initial diversity in species traits. At each time step, z_i was updated in response to selection imposed by the environment and antagonistic partners. We assumed environmental selection favors a fixed trait value, θ_i, which was defined as $\theta_i = z_{i\,(t=0)}$ for the trait matching model and as $\theta_i = 0$ for the trait barrier model. The partial selection differential caused by environmental selection was defined as:

$$S_{i(t)} = \xi_S(\theta_i - z_{i(t)}) \tag{1}$$

in which ξ_s is the intensity of the environmental selection, $0 < \xi_s < 1$.

The network of species interactions was assembled at each time step according to two different mechanisms that determined the probability of species interactions: trait matching and trait barrier. The total number of species and interactions in the simulated networks were parameterized using the number of species and interactions observed in empirical antagonistic networks. We next describe how the different mechanisms were modeled.

Trait-matching model

In the trait-matching model, the similarity of species traits determined the probability that two species interacted in a given time step:

$$p_{ij(t)} = e^{-\alpha(z_{i(t)} - z_{j(t)})^2} \tag{2}$$

in which α measures how sensitive the probability of interaction is to the difference in species traits. Selection on exploiters i favors matching with victim's j trait and the partial selection differential ($M_{ij(t)}$) was calculated as described in Eq. 3a. For victims j, selection favors trait mismatches and we assumed a critical mismatch, ε, in such way that if $\left|z_{i(t)} - z_{j(t)}\right| > \varepsilon_j$ the exploiter has a negligible effect on victim fitness ($u_{ji(t)} = 0$). The partial selection differential caused by selection imposed by the exploiter i on victim j ($M_{ji(t)}$) was defined by Eq. 3b.

$$M_{ij(t)} = \xi_d a_{ij(t)}(z_{j(t)} - z_{i(t)}) \tag{3a}$$

$$M_{ji(t)} = \xi_d u_{ji(t)} a_{ji(t)}(z_{i(t)} - z_{j(t)} \pm \varepsilon_j) \tag{3b}$$

In Eqs. 3a and 3b ξ_d is the selection intensity imposed by interacting species relative to environmental selection, $0 < \xi_d < 1 - \xi_s$, $u_{ji(t)} = 1$ if $\left|z_{i(t)} - z_{j(t)}\right| \le \varepsilon_j$, or $u_{ji(t)} = 0$ if $\left|z_{i(t)} - z_{j(t)}\right| > \varepsilon_j$. Selection favors larger trait values for the victim, $z_{i(t)} + \varepsilon_j$, if $z_{j(t)} > z_{i(t)}$ and smaller trait values, $z_{i(t)} - \varepsilon_j$, if $z_{j(t)} < z_{i(t)}$. The relative degree of trait matching at time t, $a_{ij(t)}$, weights the selection imposed by interaction partners according to the relative degree of trait matching at time t:

$$a_{ij(t)} = \frac{l_{ij(t)} e^{-\alpha(z_{i(t)} - z_{j(t)})^2}}{\sum_{k=1;k\neq i}^{R} l_{ik(t)} e^{-\alpha(z_{i(t)} - z_{k(t)})^2}} \tag{4}.$$

In which l_{ij} define the observation ($l_{ij(t)} = 1$) or not ($l_{ij(t)} = 0$) of the interaction and it is an element of the binary matrix $\mathbf{L}_{(t)}$ at time t and R is the total number of species in the network. The interactions were updated in $\mathbf{L}$ at each time step according to the probability of species interactions, $p_{ij(t)}$ (Eq. 2), keeping the observed number of interactions in the empirical network fixed.

Barrier model

For the trait barrier functional relationship, the probability of an interaction between two species depended on how larger is the exploiter trait, z_i, relative to victim trait, z_j, and was calculated as:

$$p_{ij(t)} = \frac{1}{1+e^{-\alpha(z_{i(t)}-z_{j(t)})}} \tag{5}$$

in which α measures how sensitive the probability of interaction is to the difference in species traits.

For the trait barrier model, selection on exploiters favors a larger trait value that overcomes the barrier determined by the victim's trait value. We assumed that the trait of exploiter i, $z_{i(t)}$, needs to be larger than $z_{j(t)} + \beta_i$ for a successful attack of victim j. The partial selection differential for exploiters ($B_{ij(t)}$) is described Eq. 6a. For victims j, selection favors traits that are sufficiently large in order to avoid attack in such way that if $z_{j(t)} \geq z_{i(t)}$ the exploiter has a negligible effect on victim fitness ($q_{ji(t)} = 0$). The partial selection differential caused by selection imposed by exploiters on victims was defined by Eq. 6b.

$$B_{ij(t)} = \xi_d t_{ij(t)}(z_{j(t)} - z_{i(t)} + \beta_i) \tag{6a}$$

$$B_{ji(t)} = \xi_d q_{ji(t)} t_{ji(t)}(z_{i(t)} - z_{j(t)}) \tag{6b}$$

In Eqs. 6a and 6b ξ_d is the intensity of the interaction selection, $0 < \xi_d < 1 - \xi_s$, and $t_{ij(t)}$ weights the selection imposed by each interaction partner according to the relative probability of species interactions at time t (Eq. 7). In Eq. 6b, $q_{ji(t)} = 1$ if $z_{j(t)} < z_{i(t)}$, or $q_{ji(t)} = 0$ if $z_{j(t)} \geq z_{i(t)}$.

$$t_{ij(t)} = \frac{l_{ij(t)}\left(\frac{1}{1+e^{-\alpha(z_{i(t)}-z_{j(t)})}}\right)}{\sum_{k=1;k\neq i}^{R} l_{ik(t)}\left(\frac{1}{1+e^{-\alpha(z_{i(t)}-z_{k(t)})}}\right)} \tag{7}$$

Combining the partial selection differentials caused by environmental selection (Eq. 1) and interaction selection (Eqs 3 and 6) we had the general equations describing mean trait evolution for each species. Eqs. 8 and 9 describes mean trait evolution of species i (exploiters

or victims) interacting with species *j* (which are victims in the case of *i* being an exploiter or *vice-versa*) for the trait matching or exploitation barrier model, respectively.

$$z_{i(t+1)} = z_{i(t)} + h_i^2 (S_{i(t)} + M_{ij(t)}) \quad (8)$$

$$z_{i(t+1)} = z_{i(t)} + h_i^2 (S_{i(t)} + B_{ij(t)}) \quad (9)$$

where h_i^2 is the heritability of trait z_i,

Model performance

We characterized the accuracy of the structural fit between the empirical estimates of nestedness or modularity and their counterparts in the coevolved theoretical networks by computing the normalized model error (NME, see Pires *et al.* (2011) for similar approach) between these estimates. The normalized NME of a given metric can be defined as the absolute difference between the model's median value and the empirical value divided by the difference between the model's median value and the metric value at the 2.5% or 97.5% quantiles, depending on whether the empirical value is lower or larger than the model's median, (Williams & Martinez 2008, modified by Pires *et al.* 2011) By using this approach, we did not make particular assumptions about the distribution of property values generated by the food web model. The empirical structural metric is considered significantly different from the distribution of modeled structures if -1 > NME > 1, where positive NME values outside this range indicate overestimation of a property value by the model, and negative NME values indicate outside this range underestimation. We evaluated model performance by comparing the NME values for each model and by comparing the percentage of networks that reproduced (|NME| < 1) the empirical structures in each model.

Exploring the coevolutionary dynamics in antagonistic networks

We explore the differences between trait matching and exploitation barrier mechanisms on multi-species antagonistic coevolution under scenarios of strong (high α) and weak (α) sensitivity to trait similarity. In the weak sensitivity scenario (α = 0.1), trait differences had a small effect on the probability of species interactions. In the strong

sensitivity scenario (α= 10), trait differences had a large effect on the probability of species interactions.

We used two null scenarios as theoretical benchmarks to improve our understanding of the effects of species traits and network organization on coevolution. In null scenario 1 trait values evolved without affecting network organization. In this null scenario, the interactions were not determined by the trait-based functional relationships. The probability of species interaction did not depend on traits and was equal for all species ($p_{ij} \sim 1/N_V N_E$), therefore there was no sensitivity to trait similarity (α) in this model. We used the null scenario 2 to understand how networks would be assembled by trait-based mechanisms in the absence of coevolutionary feedback. In the null scenario 2 interactions were determined by fixed traits equal to the environmental optimum (θ_i) and the probabilities of interactions were computed as in the trait matching and trait barriers models (p_{ij} in Eqs. 2 and 5). For this second null scenario we also considered the two degrees of sensitivity to trait similarity (α). Each simulation was performed until 5,000 time steps, allowing the characterization of the simulated evolutionary dynamics. We ran 100 replicates per empirical network on each model and scenario. The remaining parameters were determined at: ξ_d = 0.5, h^2 = 0.25, ε_i = U[0.4, 0.6], β_i = U[0.4, 0.6].

Parameterizing the model

We used species richness of exploiters and victims and the connectance of 89 bipartite empirical networks to parameterize the matrix **L** in our simulations aiming to explore the effects of network structure on coevolutionary dynamics. This dataset encompasses a broad range of antagonisms, including parasitism, predation and various plant-herbivore interactions (Table S1). These networks range from very small networks of bacterias and phages that include 5 species to large networks of fishes and their parasites including almost 240 species (Table S1).

Trait dynamics

We used two measures to characterize species trait dynamics: the magnitude of

directional trait change, $MTC = \left| z_{i(t=5{,}000)} - z_{i(t=0)} \right|$ and the temporal trait change, $TTC = \sum_{t=0}^{t=5{,}000} \left| z_{i(t+1)} - z_{i(t)} \right|$. We calculated the average values of *MTC* and *TTC* across species for each network to investigate the effects of network structure on trait evolutionary dynamics.

We performed a two-way ANOVA to compare the trait dynamics outcomes predicted by the trait matching and trait barrier models in the two scenarios of trait sensitivity (α). The response variables were the magnitude of directional trait change (*MTC*) and temporal trait change (*TTC*) and the predictor nominal variables were the mechanism mediating interactions (trait matching or trait barrier), and the level of trait sensitivity (strong, weak and null - in the case of null model 1). We also explored if network characteristics such as species richness and connectance affected the trait dynamics outcomes predicted by the trait matching and trait barrier models in the different scenarios of trait sensitivity by using two-way ANCOVA. We did not report *p*-values because for simulation data statistical significance is not useful to determine the relative importance of effects of the various predictors (White *et al.* 2014). We rather used the effect sizes and sign of the estimated regression parameters to investigate the importance of different predictors (White *et al.* 2014).

Network dynamics

In both evolutionary models we assumed that the probability of species interaction ($p_{ij(t)}$) depended on species traits. Therefore, at each time step this probability was recalculated according to species traits and then species interactions were re-assigned following the actualized interaction probabilities. Initial species richness and connectance were maintained at each time step. To create these binary networks we first assigned interspecific interactions according to species-specific $p_{ij(t)}$, requiring that each species had at least one interaction. The remaining interactions were sampled according to $p_{ij(t)}$ and distributed among the unfilled cells, so that connectance in the empirical and coevolved networks was the same. This allowed the occurrence of interaction turnover among species, which potentially changed network structure over time. At each time step we calculated the (i) proportion of interaction turnover, which was defined as

$$T = \frac{l_{ij(t)}(1 - l_{ij(t-1)}) + l_{ij(t-1)}(1 - l_{ij(t)})}{E} \quad (10)$$

in which E is the total number of interactions, (ii) nestedness, computed using the metric NODF (Almeida-Neto *et al.* 2008) and (iii) modularity, computed using the metric Q (Newman & Girvan 2004). The degree of modularity was estimated using the fast greedy modularity optimization algorithm for finding community structure (Clauset *et al.* 2004), which combines fast computing time with adequate performance for characterization of networks as large as ecological networks (Marquitti *et al.* 2014).

We characterized the temporal dynamics of species interactions using the temporal mean proportion of interaction turnover and the coefficient of variation of *NODF* or Q values along each simulation replicate (n = 1,000 time steps, 89 networks, 100 simulation replicates per network and scenario). We compared the effects of the mechanisms of interaction, species sensitivity and presence of coevolutionary dynamics using three-way ANOVA and Tukey HSD post-hoc tests. We explored the extent to which interaction turnover explained the coefficient of variation in network structure using two-way ANCOVA. Finally, we performed correlations to explore the relationships between the average proportion of interaction turnover and the magnitude of directional selection (*MTC*) to understand the feedback between species coevolution and network dynamics.

Results

Model performance

The percentage of networks whose metrics were reproduced by each model varied between 22% and 54% (Table 1). The coevolutionary models performed differently in reproducing both the nestedness and modularity of the empirical antagonistic networks (Table 1). The trait matching model with either levels of sensitivity to differences in species traits predicted the structural properties of about half of empirical antagonistic networks (Table 1). Similar figures were obtained using the barrier model with strong sensitivity (Table 1). The trait matching model with a strong sensitivity also had smaller normalized model error in predicting empirical network NODF and Q (Figure 1). Empirical network nestedness was underestimated and modularity was overestimated by both functional relationships, in average (Figure 1).

Trait dynamics

The trait matching model predicted a much higher temporal change in species traits (*TTC*) than when interactions were assembled by the barrier mechanism ($R^2 = 0.66$, Figure 2). An increased sensitivity to trait difference decreased temporal trait change in the trait matching model but had no effect in the barrier models (Figure 2). Under the exploitation barrier mechanism the temporal change in species traits was not different than that predicted by the null model 1 (Figure 2), in which the probability of interaction between species was independent of their traits and equal to all species pairs. Species net evolutionary change also varied between mechanisms: the exploitation barrier model predicted a much higher magnitude of directional change (*MTC*) in trait values than did the trait matching model ($R^2 = 0.99$, Figure 3). The sensitivity to trait difference had no effect in the magnitude of directional change in trait values and this magnitude was always higher than the directional change predicted by the null model 1 in the exploitation barrier model (Figure 3). In contrast, the magnitude of directional change under the trait-matching mechanism varies with the sensitivity of interactions to trait differences. A weak sensitivity in the trait matching model predicted a higher magnitude of directional change in trait values than did the null model 1 (Figure 3), whereas stronger sensitivity reduced the magnitude of directional change, which was not different than the expected by the null model 1 (Figure 3).

Network structure differently affected species trait dynamics in each model. Species richness increased the temporal change in species traits in the trait matching model ($R^2 = 0.67$, Figure 4a, no matter the degree of sensitivity) but had no effect in the barrier model (Figure 4b), as it was not different than the expected by the null model 1 in the barrier model (Figure 4b). Connectance decreased the temporal change in species traits ($R^2 = 0.89$, Figures 4c and 4d) in both models. In the trait matching model connectance had no effect when the probability of interactions was not related to trait values (null model 1, Figure 4c). Connectance had a weaker effect on trait change when interactions were assembled according to the exploitation barrier mechanism than when it was not related to trait values (null model 1, Figure 4d). Species richness decreased the magnitude of directional change in species traits only when interactions were mediated by trait matching with a strong sensitivity ($R^2 = 0.99$, Figure 5a). In the other scenarios richness had no effect (Figures 5a and 5b). Connectance decreased the magnitude of directional change when interactions were mediated by trait matching with a weak sensitivity but increased when the probability of interactions were not

related to trait values (Figure 5c). In the other scenarios connectance had no effect (Figures 5c and 5d)

Network dynamics

Both functional relationships lead to interaction turnover, which ranged from 0% to 50% of the interactions. Exploitation barrier and trait matching with weak sensitivity predicted 38% of interaction turnover, which was not different than the expected by the two null models (R^2 = 0.33, Figure 6). The average interaction turnover in the trait matching model with strong sensitivity was much lower than in the other scenarios. Coevolution had no effect on interaction turnover because there was no difference between the model with no coevolutionary response (null model 2) and models with coevolutionary dynamics (Figure 6). Richness decreased interaction turnover (R^2 = 0.53) when interactions were mediated by trait matching with weak sensitivity but had no effect in the trait matching model with strong sensitivity (Figure 7a). Richness also reduced interaction turnover in the exploitation barrier model no matter the sensitivity (Figure 7b). Network connectance had a positive quadratic effect on interaction turnover for both trait matching and exploitation barrier models (R^2 = 0.88, Figures 7c and 7d).

In average, networks had a small temporal variation in nestedness. This variation was not related to the functional relationship between species trait and fitness, trait sensitivity or coevolution (R^2 = 0.04, Figure 8). Nestedness was slightly more variable in time when considering the exploitation barrier mechanism of interaction with a strong sensitivity to trait differences (Figure 8). The temporal variation in nestedness was not different than the expected by the null models for the trait matching and weak barrier interaction rules (Figure 8). Both trait-based linkage mechanisms equally decreased temporal variation in modularity when compared to randomly assembled interactions (null model 1) no matter the sensitivity to trait differences (R^2 = 0.09, Figure 9). However, coevolution had no effect on the modularity temporal coefficient of variation because it was not different than the expected by the null model 2 (Figure 9). Interaction turnover had a very weak positive effect on the coefficient of variation of nestedness (R^2 = 0.06, Figures 10a and 10b) and modularity (R^2 = 0.25, Figures 10c and 10d), which indicates that network structure is relatively robust to interaction turnover.

Interaction turnover was not correlated to temporal changes in species traits when the interactions were mediated by the exploitation barrier mechanism (Figures 11b and 11d) and this relationship was not different than the expected by neutral interactions (null model 1). For the trait matching mechanism with weak sensitivity there was a negative trend between interaction turnover and temporal trait change that differed from the null models (Figure 11a). When the sensitivity was stronger this relationship was quadratic, with a positive relationship for low values of trait change and negative relationship for higher values (Figure 11a). The magnitude of directional change was not related to interaction turnover for both mechanisms (Figures 11c and 11d).

Discussion

We found that the nested and modular structures of antagonistic networks are well predicted by trait-based mechanisms of interaction linkage because both models reproduced the structures of more than 50% of the empirical networks. However, there was no particular network structure related exploitation barrier or trait matching mechanism and both models tended to overestimate modularity and underestimate nestedness. Coevolution differently drove the evolution of species traits when interactions were mediated by distinct functional relationships, but this had no impact on the way the antagonistic interactions were organized. Thus, our results suggest that it may be not possible to infer the coevolutionary process based only in the structure of antagonistic networks. In contrast, network structure did markedly affect phenotypic evolution. We were able to identify three central points about how coevolution shapes the structure of antagonistic networks and the trait evolution of the interacting species.

First, the models that assumed a strong sensitivity to trait differences significantly predicted the structure of a higher percentage of networks. The higher fit of the strong sensitivity models indicates that species traits play an important role in determining the architecture of antagonistic networks. The importance of neutral and niche processes in structuring communities are central in ecological theory and several studies have already explored the effect of these processes using distinct approaches (Leibold & McPeek 2006, Gravel *et al.* 2006, Kembel 2009). In the context of ecological networks there is a growing effort in trying to understand the processes that account for the patterns of nestedness and

modularity found in empirical networks (Canard *et al.* 2014, 2012, Vázquez *et al.* 2007, Nuwagaba *et al.* 2015, Krishna *et al.* 2008). The neutral expectation assumes that species pairwise probability of interaction should be proportional to the product of their relative abundances (Vázquez *et al.* 2007). Networks assembled under the neutral assumptions tend to overestimate nestedness and underestimate modularity when compared to the structures found in real networks (Canard *et al.* 2014, Krishna *et al.* 2008). In contrast, niche approaches assume that species traits determine the probability of species interactions (Blüthgen *et al.* 2008). Interestingly, networks assembled assuming trait-based mechanisms tend to overestimate network modularity and underestimate nestedness (Nuwagaba *et al.* 2015, Santamaría & Rodríguez-Gironés 2007). Our results point to the same direction because both trait matching and exploitation barrier mechanisms of interaction assemblage tended to underestimate nestedness and overestimate modularity. This indicates that real network structures are in between these scenarios and that a complete explanation for the structure of ecological networks should combine neutral and niche approaches.

Second, coevolution proceeds in distinct ways depending on how traits affect the probability of interactions. Differences between trait matching and exploitation barrier mechanisms were much more pronounced when considering the coevolutionary dynamics. Coevolution of interactions mediated by trait matching predicted a high fluctuating trait dynamics while the barrier mechanism favored escalating traits. The occurrence of cyclic dynamics was already found for a variety of pairwise predator-prey coevolutionary models (Gavrilets 1997, Abrams & Matsuda 1997) and the main assumption that underlies these results is the use of a bidirectional axis of prey vulnerability, which is a trait matching mechanism (Abrams 2000). A unidirectional axis of vulnerability tends to stabilize species traits and a similar pattern was found with the exploitation barrier mechanism. In the barrier model species traits may increase until they reach a maximum value that is a compromise between interaction and environmental selection. More interestingly, the relationship between network structure and trait dynamics was stronger in the trait matching model. Species traits fluctuation decreased in more connected networks. Because we assumed that one trait described species defense and attack strategies, species traits were distributed along a single axis. In this situation, a higher number of interactions among species generated conflicting selection pressures on each species. This conflicting selection pressures found in highly connected networks reduce the potential for adaptive trait change (Barraclough 2015). Other studies have argued that a greater number of traits would increase the chances of victims

escaping the attack (Abrams 2000, Gilman *et al.* 2012). With our findings we suggest that natural selection would favor the evolution of a greater number of attack and defense traits when species interact in highly connected networks, allowing the populations to adapt to otherwise conflicting selective pressures imposed by partners. High interaction turnover also decreased the fluctuation in species traits when interactions were mediated by trait matching, which indicates that adaptive change of interacting partners can be an alternative mechanism driving coevolution in antagonistic interactions mediated by trait matching. Fluctuation in traits was less likely when interactions were mediated by exploitation barrier mechanism and antagonistic coevolution was less affected by community structure.

Network connectance was the best predictor for the frequency of interaction turnover and there were no difference in the interaction turnover observed for the exploitation barrier and low sensitivity trait matching mechanisms. A uniform probability of interaction (null model 1) also predicted the same pattern of interaction turnover, which indicates that species traits have a weak effect on the probability of interaction mediated by the barrier mechanism, even when species sensitivity is high. A strong sensitivity to trait matching increased the temporal consistency of species interactions. Very few studies have documented the temporal dynamics of species interactions in natural environments but available data shows that interaction turnover can vary from 28% (Petanidou *et al.* 2008) to 66% (Olesen *et al.* 2008) of the interactions in mutualistic systems and are around 40% in antagonistic systems (Pilosof *et al.* 2013). These studies also found that although interaction identity can be highly plastic, network structural properties (e.g. degree centralization, connectance, nestedness, modularity, average distance and network diameter) show low temporal variation (Petanidou *et al.* 2008, Olesen, Stefanescu, *et al.* 2011, Olesen *et al.* 2008, Díaz-Castelazo *et al.* 2010, Pilosof *et al.* 2013, Saavedra *et al. in press*). Our estimates of interaction turnover and the low coefficient of variation in nestedness and modularity agree with these empirical results. We go further and predict that we should observe a higher temporal variation in network nestedness when interactions are mediated by an exploitation barrier mechanism and species have a strong sensitivity to trait differences. We also suggest that a low variation in modularity would be observed when communities are structured based on species traits.

Third, coevolution had a very weak effect on interaction turnover and temporal variability of nestedness and modularity no matter the functional relationship. Higher nestedness variability was found only in the exploitation barrier model with strong

sensitivity. Therefore, similar network structures and dynamics can emerge as the result of distinct functional relationships between species trait and fitness with or without coevolution. Because the coevolutionary process does not necessary result in specific network structures, the effects of coevolution on species ecology and evolution might be underestimated (Thompson 2013). Neutral interactions based on the distribution of species abundances have been pointed out as a mechanism to explain the high interaction turnover found in ecological networks (Poisot *et al.* 2012, Canard *et al.* 2012). However, our results revealed that trait mediated interactions and coevolution can provide plausible explanations for the considerable turnover observed for antagonistic interactions. Neutrality could occur within modules (Kondoh *et al.* 2010) while niche differentiation may act between modules, which could explain why nestedness and modularity metrics were so weakly explained by neutral interactions in previous studies (Canard *et al.* 2012, 2014). Niches would determine potential links, among which abundance would filter out unrealizable links and determine the strength of interactions.

We have evaluated the network structures and temporal dynamics of interactions assembled based on two distinct trait-based mechanisms of linkage: trait matching and exploitation barriers. The models performed well in predicting the nestedness and modularity of empirical antagonistic networks and estimated realistic values for the frequency of interaction turnover observed in real systems. Coevolution did not affect network structure and dynamics but had significant effects on species trait dynamics. Thus, looking for spatial and temporal trait patterns of interacting species would provide crucial cues about the coevolutionary process. Combining trait information with ecological network studies is the current challenge for linking evolutionary and ecological dynamics of species interactions and understanding how species coevolve in space and time.

Acknowledgements

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Figures

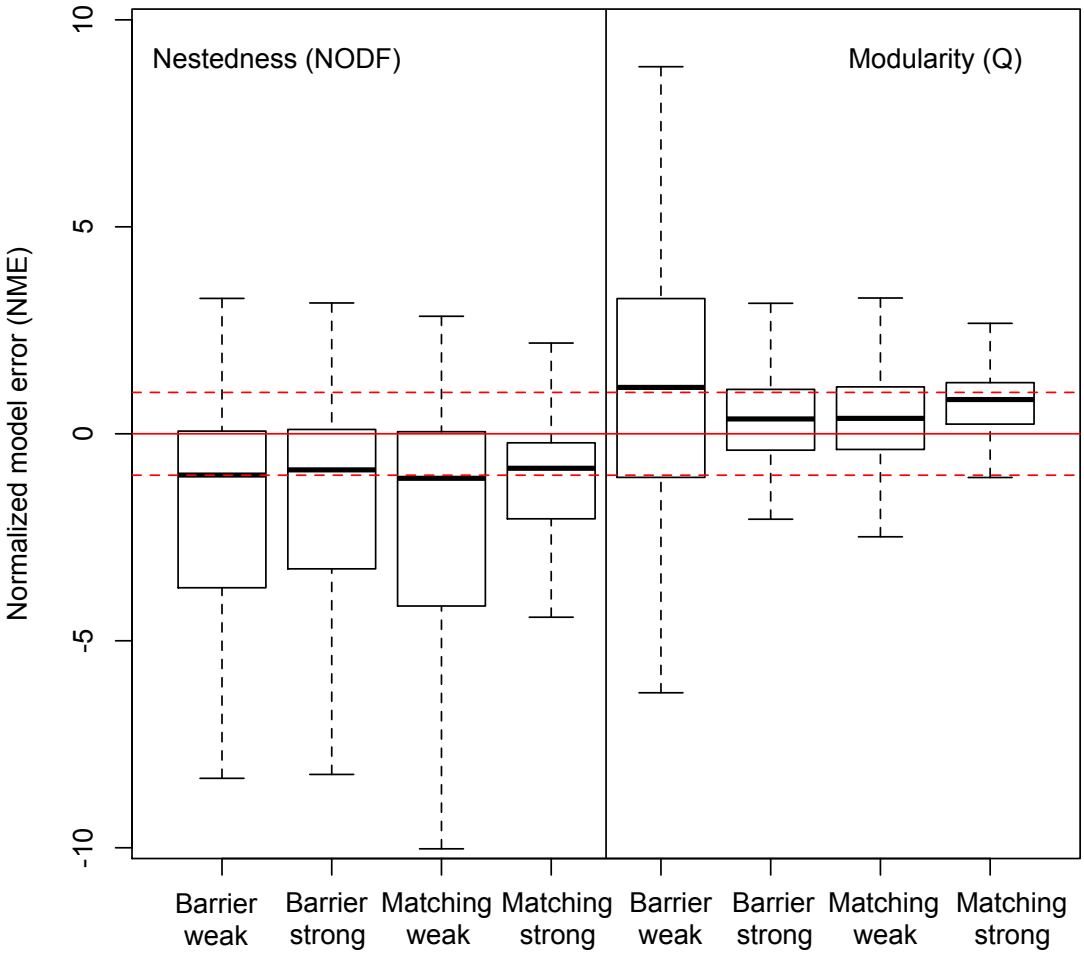

Figure 1: Normalized model error of nestedness and modularity metrics predicted by trait matching and trait barrier models with weak (α = 0.1) and strong sensitivity (α = 10). Black lines within boxes represent median values for the 89 networks. Upper and lower limits of boxes represent 1st and 3rd quartiles, respectively. The red lines highlight the NME values between -1 and 1, which are considered not significantly different from the empirical network structures.

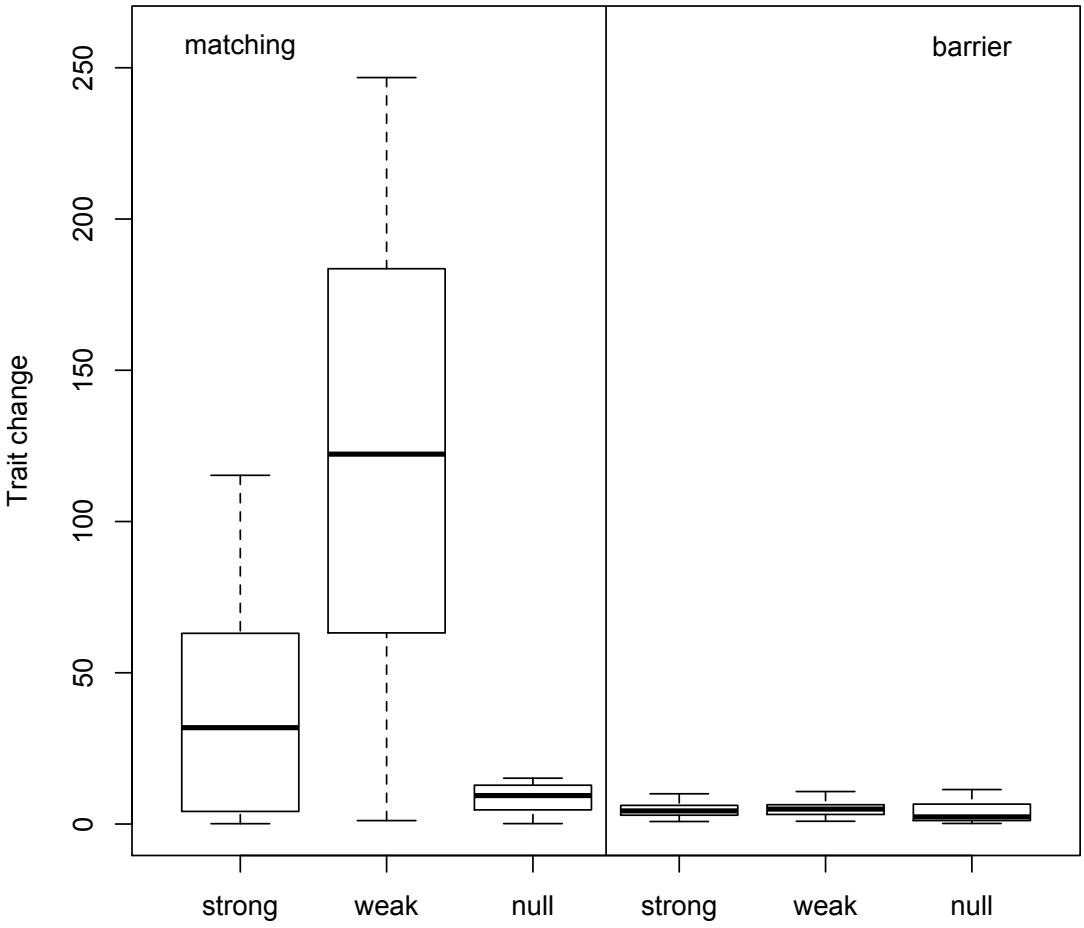

Figure 2: Average temporal trait change (*TTC*) for the trait matching and trait barrier models in the trait sensitivity scenarios. Black lines within boxes represent median values for the 89 networks. Upper and lower limits of boxes represent 1st and 3rd quartiles, respectively.

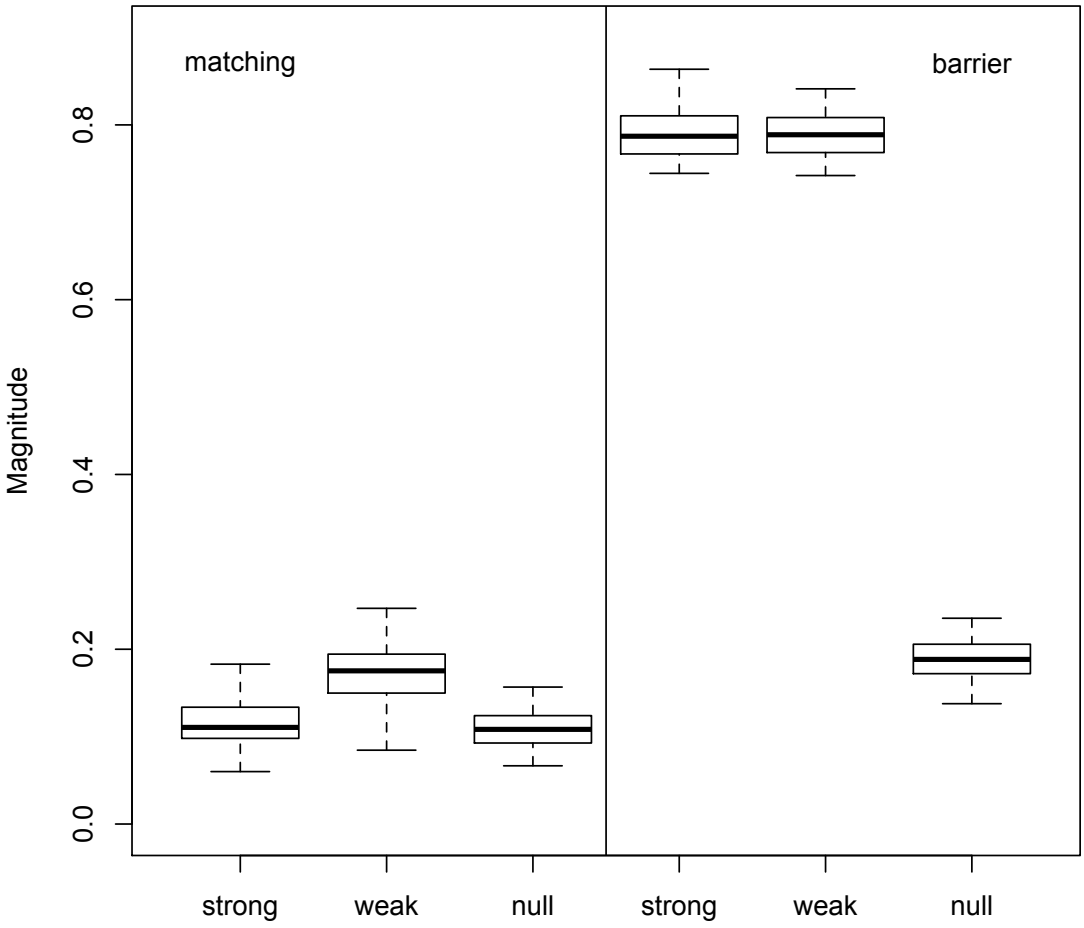

Figure 3: Average magnitude of directional trait change (*MTC*) for the trait matching and trait barrier models in the trait sensitivity scenarios. Black lines within boxes represent median values for the 89 networks. Upper and lower limits of boxes represent 1st and 3rd quartiles, respectively.

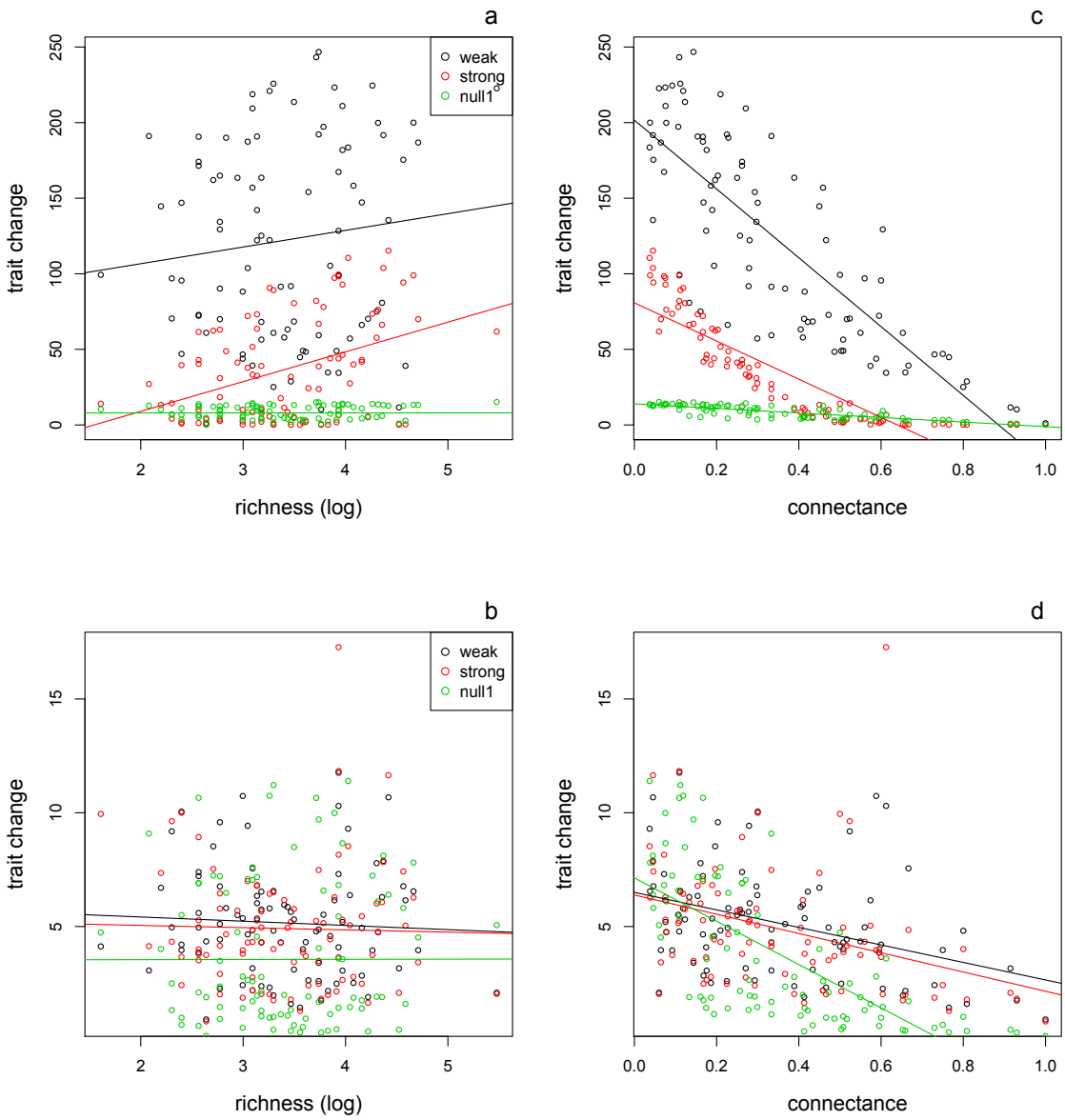

Figure 4: Effect of species richness (a and b) and connectance (c and d) in the average temporal trait change (*TTC*) for the trait matching (a and c) and trait barrier (b and d) models in the trait sensitivity scenarios. Each dot represents mean values among simulation replicates for the 89 networks.

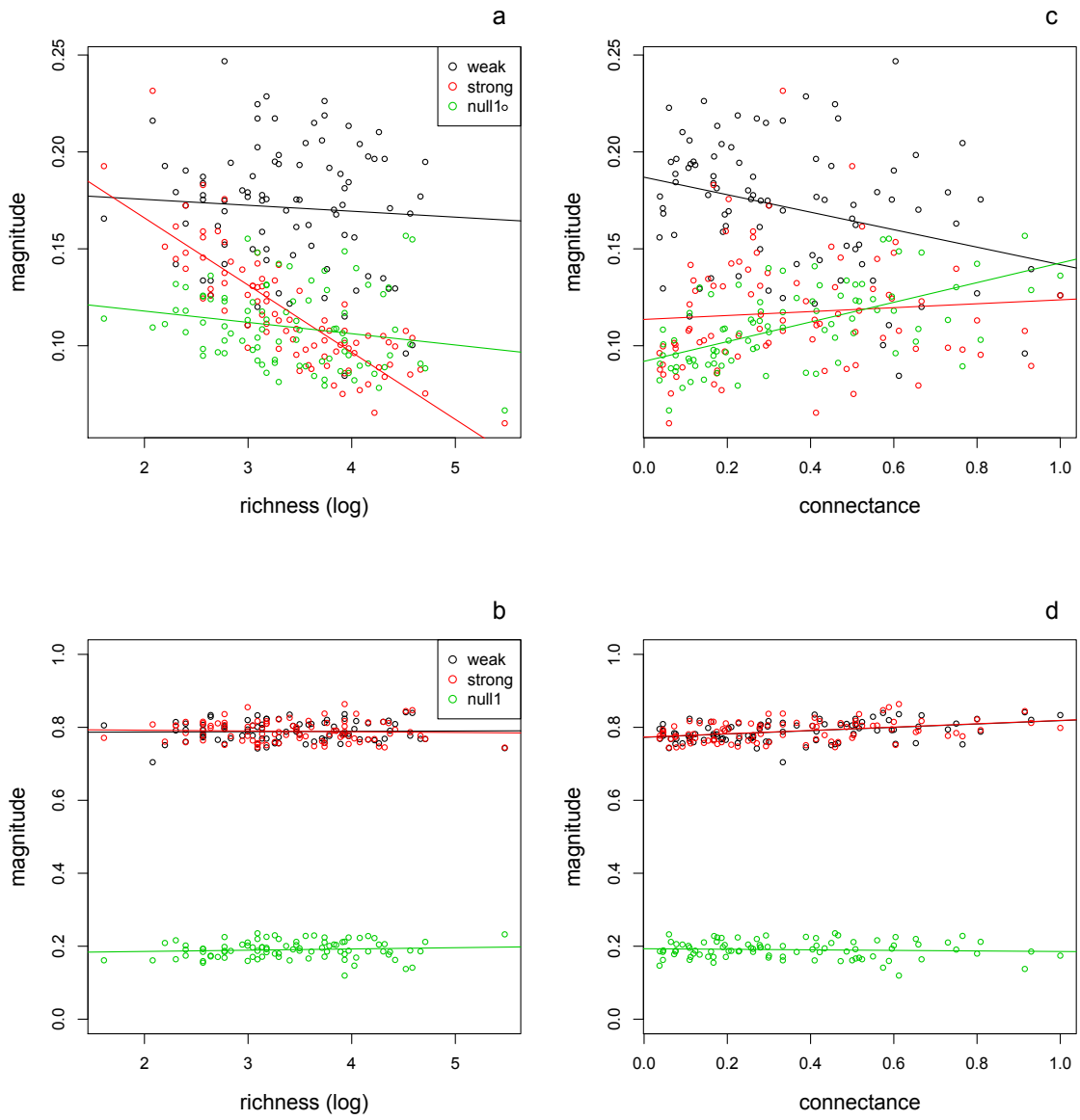

Figure 5: Effect of species richness (a and b) and connectance (c and d) in the average magnitude of directional trait change (*MTC*) for the trait matching (a and c) and trait barrier (b and d) models in the trait sensitivity scenarios. Each dot represents mean values among simulation replicates for the 89 networks.

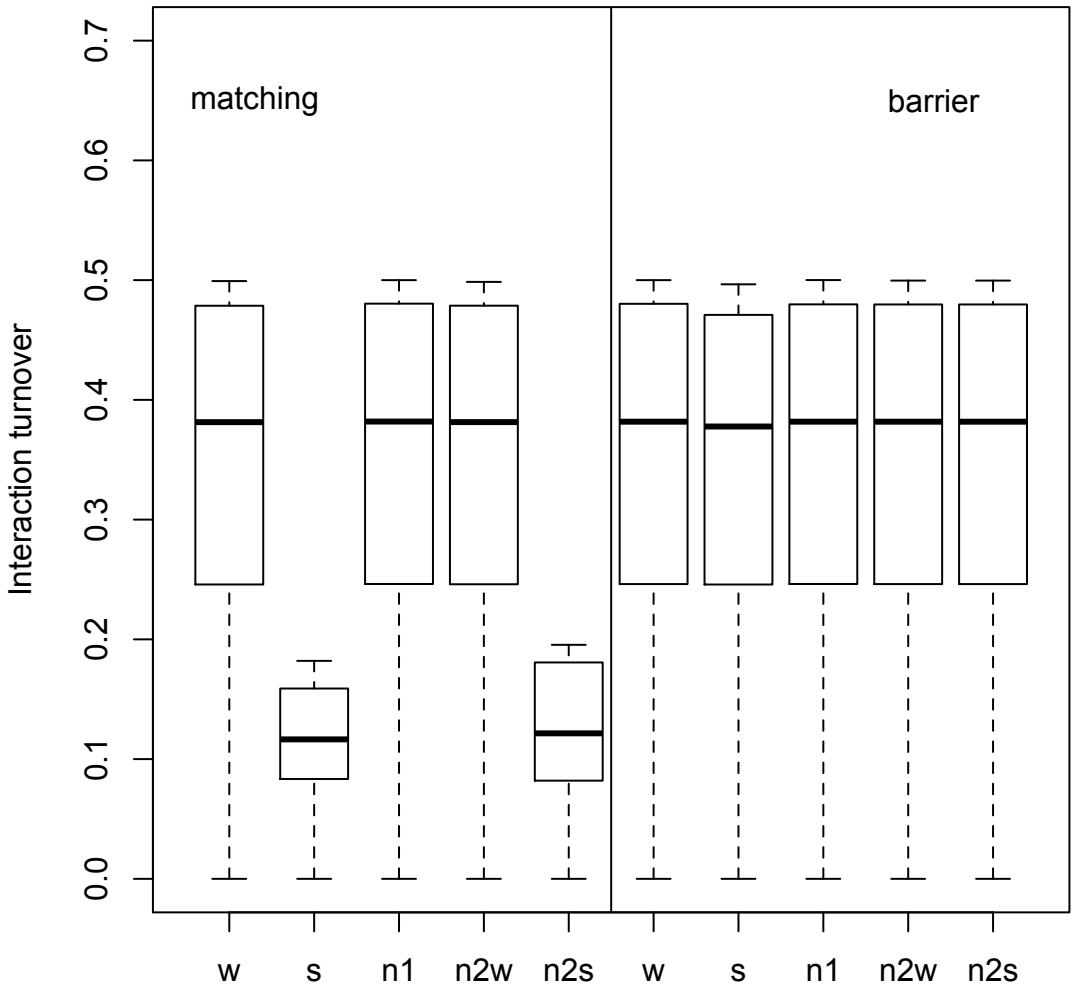

Figure 6: Average proportion of interaction turnover for the trait matching and trait barrier models. Black lines within boxes represent median values for the 89 networks. Upper and lower limits of boxes represent 1st and 3rd quartiles, respectively. w is the scenario with weak sensitivity ($\alpha = 0.1$), s is the scenario with strong sensitivity ($\alpha = 10$), n1 is null model 1, n2s is null model 2 with weak sensitivity ($\alpha = 0.1$) and n2s is null model 2 with strong sensitivity ($\alpha = 10$).

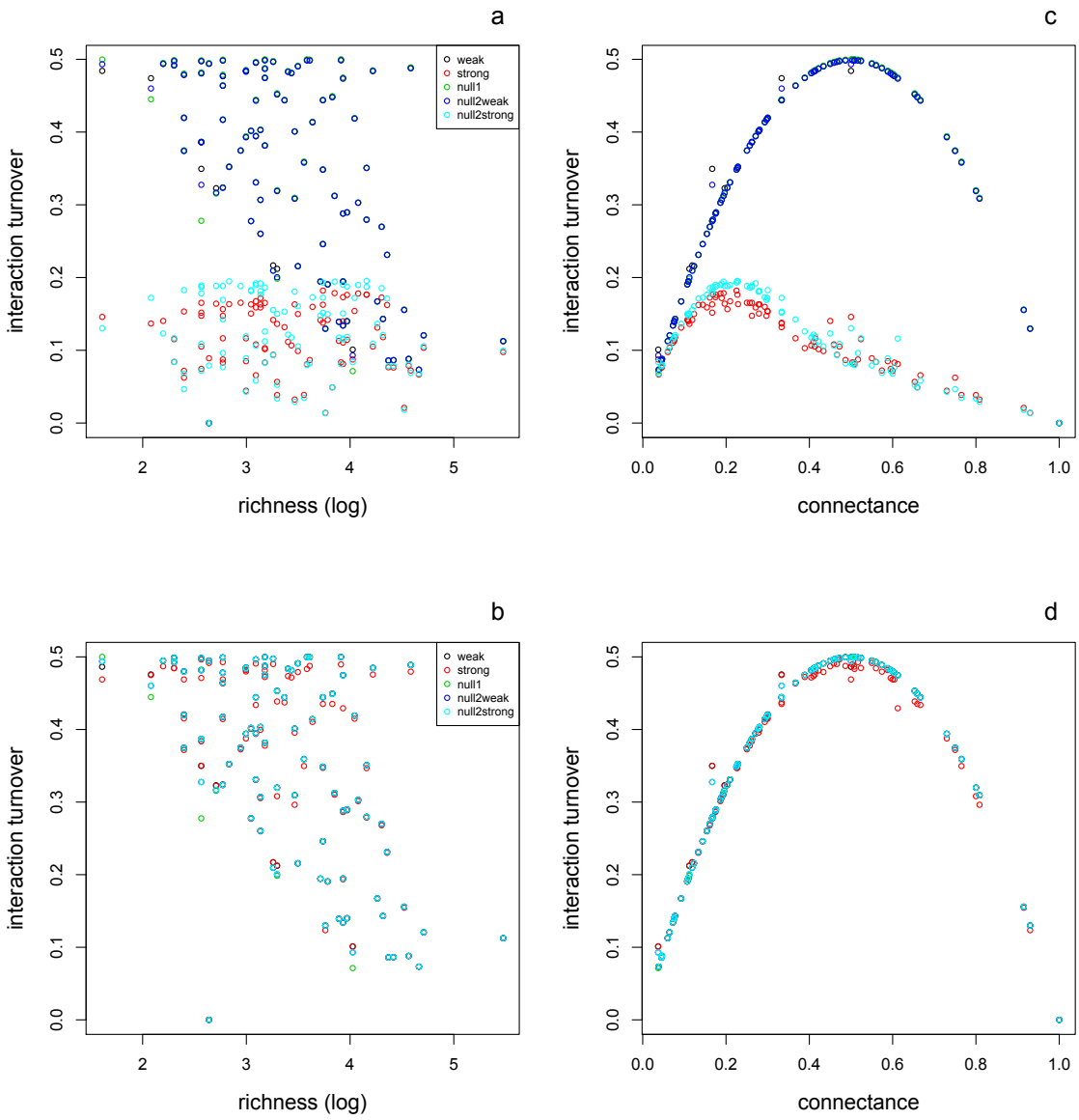

Figure 7: Effect of species richness (a and b) and connectance (c and d) in the proportion of interaction turnover for the trait matching (a and c) and trait barrier (b and d) models in the trait sensitivity scenarios. Each dot represents mean values among simulation replicates for the 89 networks.

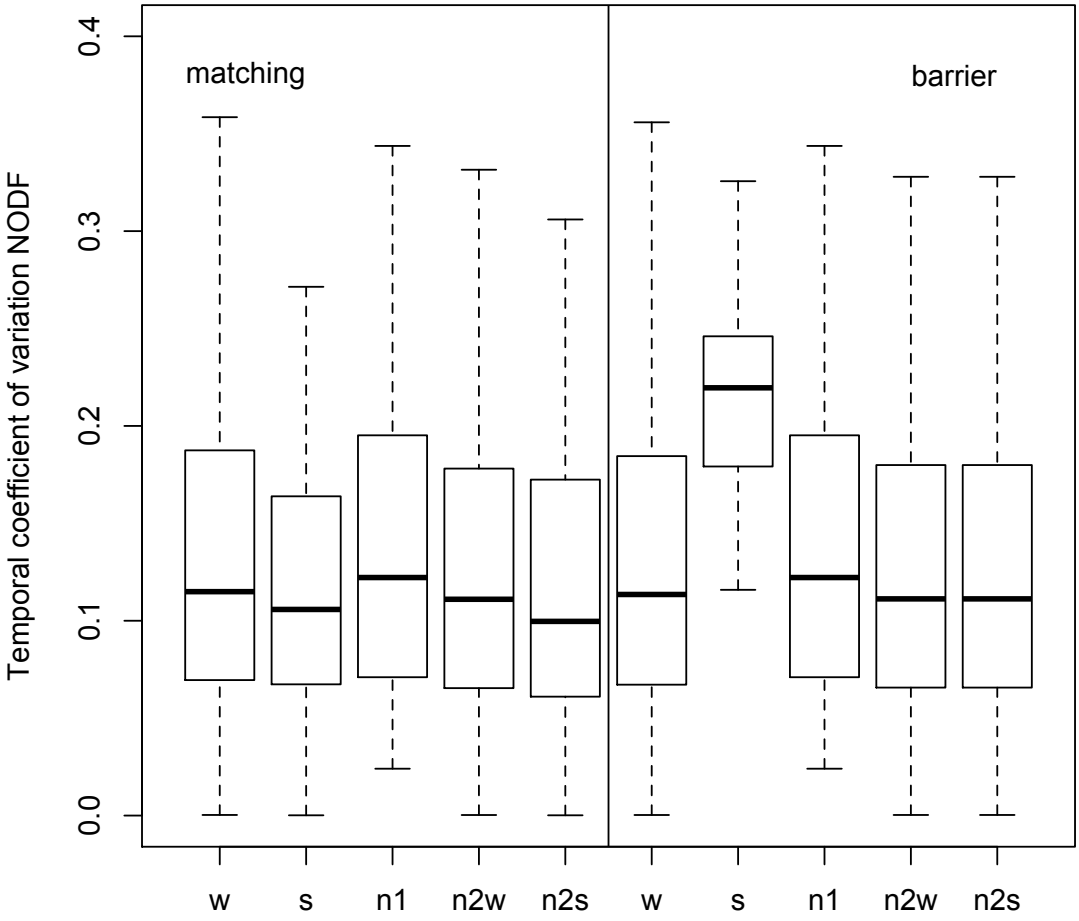

Figure 8: Average coefficient of variation in nestedness (NODF) for the trait matching and trait barrier models. Black lines within boxes represent median values for the 89 networks. Upper and lower limits of boxes represent 1st and 3rd quartiles, respectively. w is the scenario with weak sensitivity (α = 0.1), s is the scenario with strong sensitivity (α = 10), n1 is null model 1, n2s is null model 2 with weak sensitivity (α = 0.1) and n2s is null model 2 with strong sensitivity (α = 10).

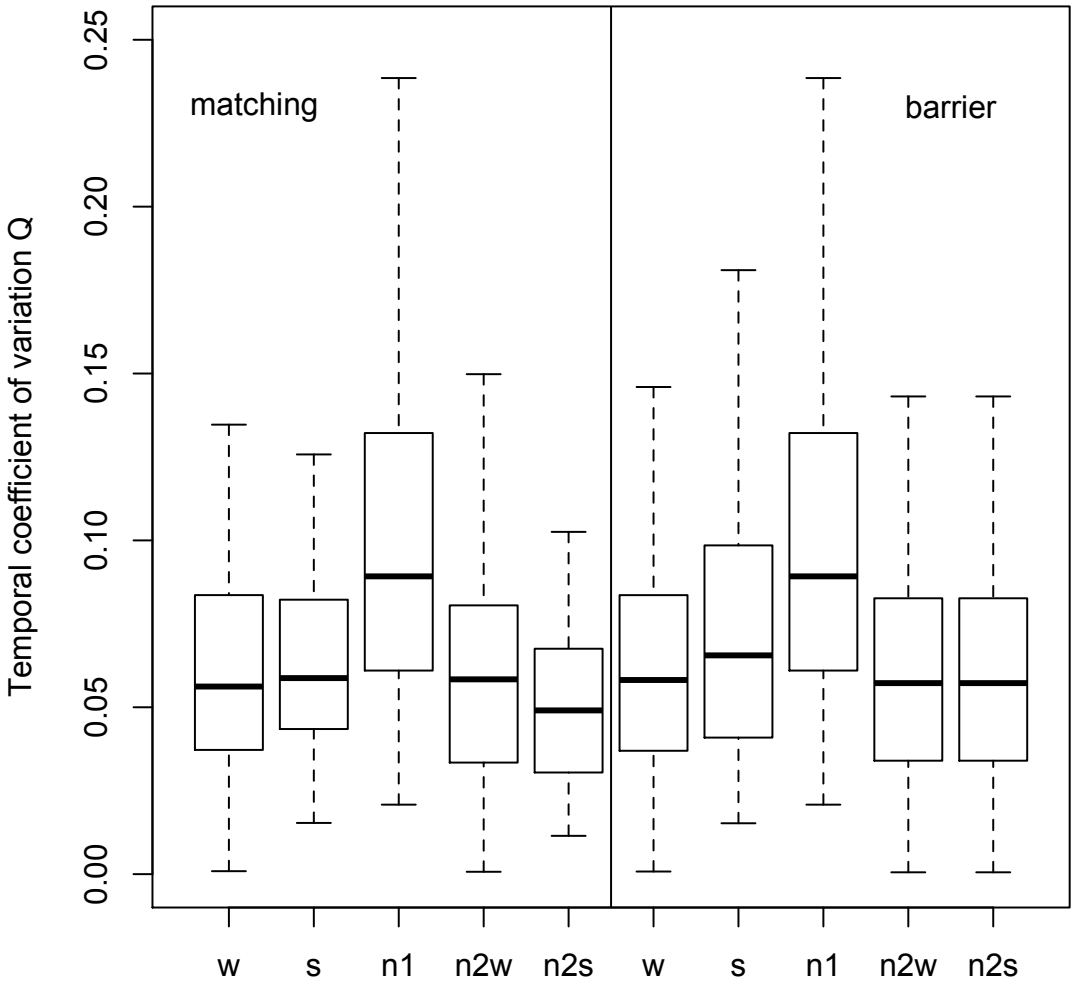

Figure 9: Average coefficient of variation in modularity (Q) for the trait matching and trait barrier models. Black lines within boxes represent median values for the 89 networks. Upper and lower limits of boxes represent 1st and 3rd quartiles, respectively. w is the scenario with weak sensitivity ($\alpha = 0.1$), s is the scenario with strong sensitivity ($\alpha = 10$), n1 is null model 1, n2s is null model 2 with weak sensitivity ($\alpha = 0.1$) and n2s is null model 2 with strong sensitivity ($\alpha = 10$).

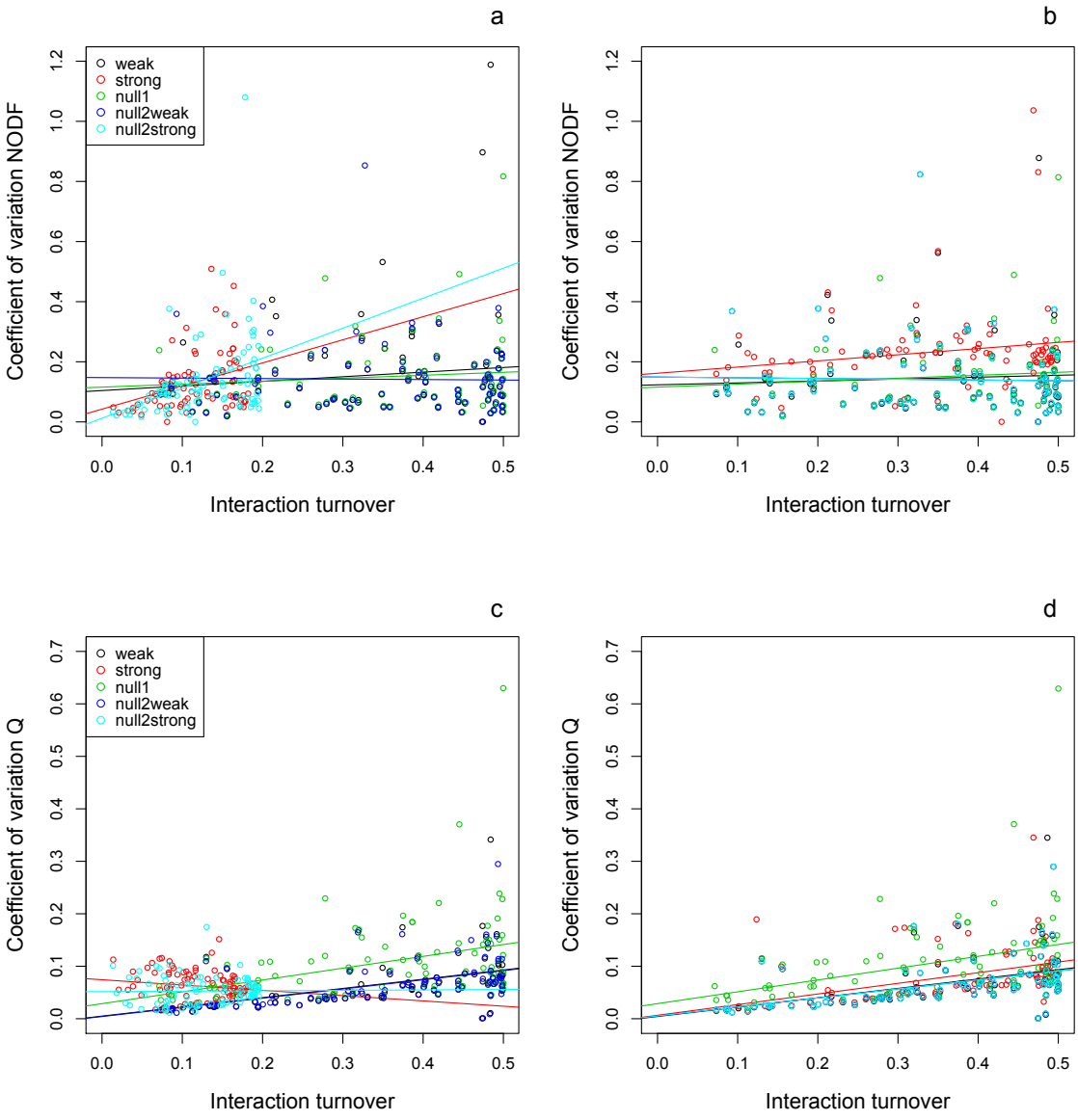

Figure 10: Effect of the proportion of interaction turnover in the coefficient of variation of nestedness (a and b) and modularity (c and d) for the trait matching and trait barrier models, respectively in the trait sensitivity scenarios. Each dot represents mean values among simulation replicates for the 89 networks.

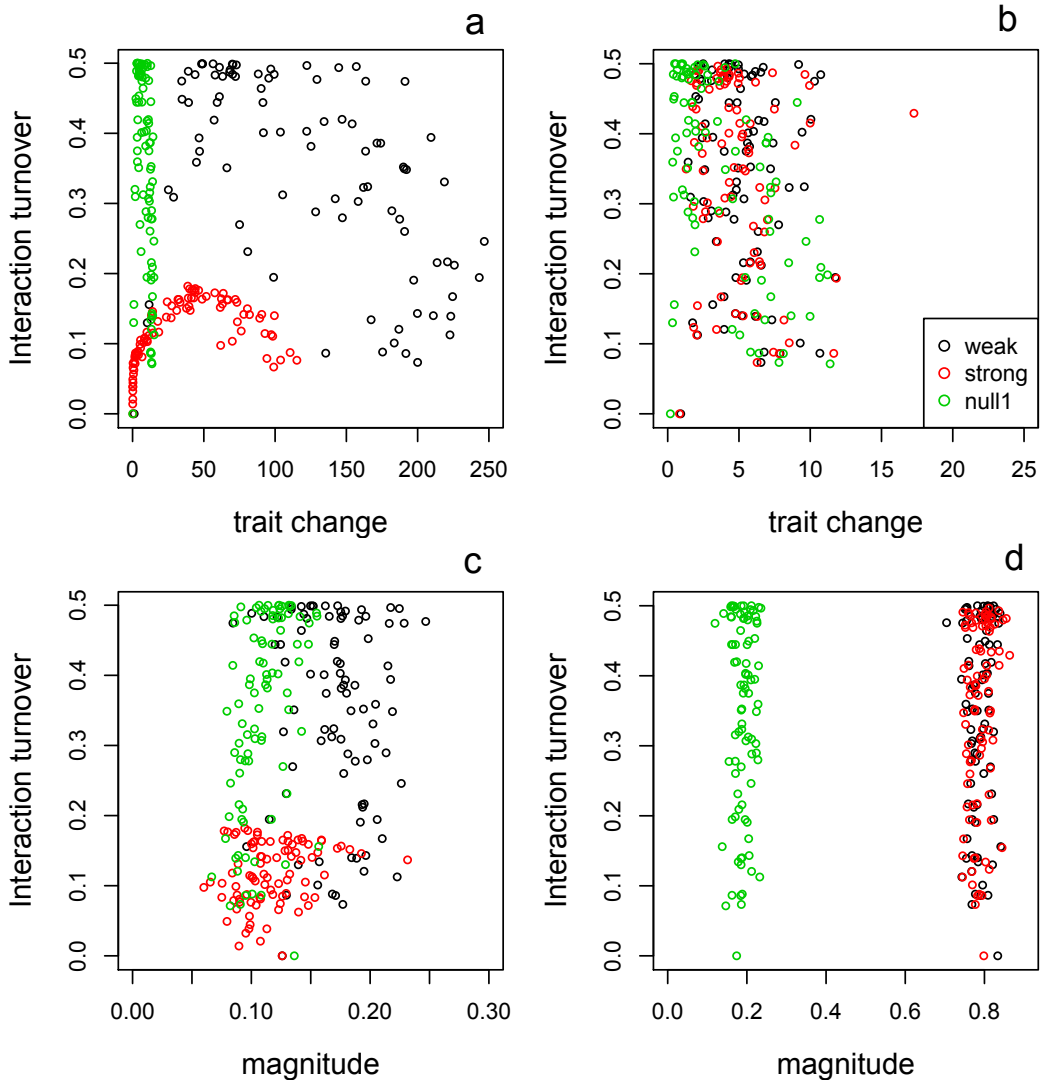

Figure 11: Relationship between the average proportion of interaction turnover and the average temporal trait change (a and b) and the average magnitude of directional trait change (c and d) for the trait matching and trait barrier models, respectively, in the trait sensitivity scenarios. Each dot represents mean values among simulation replicates for the 89 networks.

Table 1: Number (and percentage) of networks that had their nestedness and modularity metrics significantly predicted by trait matching and trait barrier models in the weak and strong sensitivity scenarios.

	Nestedness (NODF)		Modularity (Q)	
	matching	barrier	matching	barrier
weak	36 (40%)	34 (38%)	43 (48%)	20 (22%)
strong	42 (47%)	35 (39%)	48 (54%)	45 (51 %)

Trait matching and exploitation barriers differently drive evolution in antagonistic networks.

Supporting Information

1. Networks used in the study

Table S1. Dataset[1] used to parameterize species richness and connectance of the matriz **L**, including the type of interaction and network basic characteristics (N_V = number of victims; N_E = number of exploiters, C = Connectance).

Data set	Type	N_V	N_E	C	Reference
bac1	Bacteriophage - bacteria	11	4	0.5	Abe & Tanji 2007
bac2	Bacteriophage - bacteria	14	6	0.3	Barrangou et al. 2002
bac3	Bacteriophage - bacteria	18	3	0.56	Braun-Breton & Hofnung 1981
bac4	Bacteriophage - bacteria	9	5	0.31	Campbell et al. 2006
bac5	Bacteriophage - bacteria	18	8	0.37	Capparelli et al. 2010
bac6	Bacteriophage - bacteria	23	4	0.18	Caso et al. 2006
bac7	Bacteriophage - bacteria	5	15	0.39	Ceyssens et al. 2009
bac8	Bacteriophage - bacteria	30	13	0.39	Comeau et al. 2005
bac9	Bacteriophage - bacteria	32	16	0.23	Comeau et al. 2006
bac10	Bacteriophage - bacteria	5	17	0.46	DePaola et al. 1998
bac11	Bacteriophage - bacteria	15	10	0.27	Doi et al. 2003
bac12	Bacteriophage - bacteria	12	12	0.26	Duplessis & Moineau 2001
bac13	Bacteriophage - bacteria	6	7	0.21	Gamage et al. 2004
bac14	Bacteriophage - bacteria	93	2	0.32	Goodridge et al. 2003
bac15	Bacteriophage - bacteria	34	12	0.36	Hansen et al. 2007
bac16	Bacteriophage - bacteria	23	46	0.4	Holmfeldt et al. 2007
bac17	Bacteriophage - bacteria	32	12	0.9	Kankila & Lindstrom 1994

bac18	Bacteriophage - bacteria	11	10	0.66	Krylov et al. 2006
bac19	Bacteriophage - bacteria	22	3	0.5	Kudva et al. 1999
bac20	Bacteriophage - bacteria	66	9	0.17	Langley 2003
bac21	Bacteriophage - bacteria	8	7	0.32	McLaughlin & King 2008
bac22	Bacteriophage - bacteria	25	27	0.47	Meyer (unpub)
bac23	Bacteriophage - bacteria	11	24	0.77	Middelboe et al. 2009
bac24	Bacteriophage - bacteria	24	14	0.21	Miklic & Rogelj 2003
bac25	Bacteriophage - bacteria	8	4	0.66	Mizoguchi et al. 2003
bac26	Bacteriophage - bacteria	102	4	0.79	Pantucek et al. 1998
bac27	Bacteriophage - bacteria	100	5	0.53	Paterson et al. 2010
bac28	Bacteriophage - bacteria	24	24	0.19	Poullain et al. 2008
bac29	Bacteriophage - bacteria	20	11	0.4	Quiberoni 2003
bac30	Bacteriophage - bacteria	17	14	0.29	Rybniker et al. 2006
bac31	Bacteriophage - bacteria	24	6	0.21	Seed & Dennis 2005
bac32	Bacteriophage - bacteria	28	22	0.56	Stenholm et al. 2008
bac33	Bacteriophage - bacteria	21	44	0.16	Sullivan et al. 2003
bac34	Bacteriophage - bacteria	7	9	0.17	Suttle & Chan 1993
bac35	Bacteriophage - bacteria	16	16	0.81	Synnott et al. 2009
bac36	Bacteriophage - bacteria	18	7	0.09	Wang & Chen 2008
bac37	Bacteriophage - bacteria	18	27	0.1	Zinno et al. 2010
herb1	Grasshoppers– plants	52	22	0.16	Joern 1979
herb2	Mammalian grazers – plants	10	17	0.6	Hansen et al. 1985
herb3	Grasshoppers – plants	54	24	0.14	Joern 1979
herb4	Leaf-chewing beetles – plants	10	32	0.14	Basset & Samuelson 1996
herb5	Gallers – host plants	10	17	0.12	Dawah & Hawkins 1995

herb11	Sap-feeding insects – plants	26	25	0.07	Muller et al. 1999
herb12	Fruit-flies - plants	53	30	0.05	Novotny et al. 2005
herb14	Gallers – host plants	10	16	0.12	Tscharntke et al. 2001
herb15	Grasshoppers – plants	43	14	0.3	Ueckert & Hansen 1971
herb16	Phasmids– plants	38	13	0.11	Blüthgen et al. 2006
herb18	Gallers – host plants	29	27	0.04	Cuevas-Reyes et al. 2007
par1a	Fish – parasites (complex life cycle)	07	19	0.47	Arthur 1976
par1b	Fish – parasites (simple life cycle)	07	10	0.23	Arthur 1976
par2a	Fish – parasites (complex life cycle)	10	32	0.22	Leong & Holmes 1981
par2b	Fish – parasites (simple life cycle)	8	8	0.3	Leong & Holmes 1981
par3a	Fish – parasites (complex life cycle)	14	39	0.18	Arai and Mudry 1983
par3b	Fish – parasites (simple life cycle)	9	12	0.17	Arai and Mudry 1983
par4a	Fish – parasites (complex life cycle)	17	42	0.18	Arai and Mudry 1983
par4b	Fish – parasites (simple life cycle)	12	11	0.18	Arai and Mudry 1983
par6a	Fish – parasites (complex life cycle)	06	18	0.39	Chinniah & Threlfall 1978
par6b	Fish – parasites (simple life cycle)	06	07	0.26	Chinniah & Threlfall 1978

par8a	Fish – parasites (complex life cycle)	46	18	0.23	Kirjušina & Vismanis 2007
par8b	Fish – parasites (simple life cycle)	24	6	0.4	Kirjušina & Vismanis 2007
par9a	Fish – parasites (complex life cycle)	37	42	0.04	Arthur & Ahmed 2002
par9b	Fish – parasites (simple life cycle)	6	7	0.17	Arthur & Ahmed 2002
par10	Fish – parasites (complex life cycle)	39	200	0.06	Bellay et al. 2011
par11a	Fish – parasites (complex life cycle)	10	28	0.29	Violante-González et al. 2007
par11b	Fish – parasites (simple life cycle)	10	6	0.36	Violante-González et al. 2007
par12a	Fish – parasites (complex life cycle)	6	16	0.27	Bensley et al. 2011
par12b	Fish – parasites (simple life cycle)	3	5	0.33	Bensley et al. 2011
par13a	Fish – parasites (complex life cycle)	19	52	0.09	Azevedo et al. 2010
par13b	Fish – parasites (simple life cycle)	20	33	0.07	Azevedo et al. 2010
par14a	Fish – parasites (complex life cycle)	12	21	0.12	Székely & Molnár 1997
par14b	Fish – parasites (simple life cycle)	10	13	0.15	Székely & Molnár 1997
par19	Fish – parasites (complex	12	29	0.1	Muzzall & Whelan 2011

	life cycle)				
par21a	Fish – parasites (complex life cycle)	8	11	0.25	Morozińska-Gogol 2007
par21b	Fish – parasites (simple life cycle)	7	3	0.52	Morozińska-Gogol 2007
par22a	Fish – parasites (complex life cycle)	11	12	0.28	Choudhury et al. 2004
par22b	Fish – parasites (simple life cycle)	8	8	0.20	Choudhury et al. 2004
par23a	Fish – parasites (complex life cycle)	44	62	0.04	Chemes & Takemoto 2011
par23b	Fish – parasites (simple life cycle)	18	31	0.08	Chemes & Takemoto 2011
par24a	Fish – parasites (complex life cycle)	36	75	0.06	Arthur & Te 2006
par24b	Fish – parasites (simple life cycle)	45	51	0.04	Arthur & Te 2006
par28a	Fish – parasites (complex life cycle)	25	50	0.08	Muzzall & Whelan 2011
par28b	Fish – parasites (simple life cycle)	3	2	0.5	Muzzall & Whelan 2011
par30	Fish – parasites (simple life cycle)	13	8	0.28	Violante-González et al. 2007
par31a	Fish – parasites (complex life cycle)	6	5	0.3	Jalali & Barzegar 2006
par31b	Parasites - fish host (simple life cycle)	7	15	0.20	Jalali & Barzegar 2006

pred1[2]	Mammalian predator – prey	16	8	0.52	Baskerville et al. 2011
pred2	Mammalian predator – prey	22	5	0.8	Owen-Smith & Mills 2008
pred4	Seed-predators – plants	21	30	0.17	Nakagawa et al. 2003
pred6	Mammalian predator – prey	18	4	0.66	Radloff & du Toit 2004

[1] These datasets are available online at the Interaction Web Database (www.nceas.ucsb.edu/interactionweb/index.html) and compiled in Pires & Guimarães (2013), Flores et al. (2011) and Bellay et al. (2013)
[2] Subnetwork depicting predator-prey interactions between mammals weighing > 5kg

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Capítulo 3

Eco-evolutionary feedbacks promote fluctuating selection and long-term stability of species-rich antagonistic networks

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running title: eco-evolutionary feedbacks in antagonistic networks

key-words: antagonism, coevolution, network, population dynamics, trait matching, trait dynamics.

Abstract

In exploiter-victim interactions coevolutionary and ecological dynamics may interact in feedback loops. The connection between population dynamics and evolution of traits is crucial to understanding long-term stability in ecological networks. Here we investigate how coevolving interactions can shape phenotypic traits and drive population dynamics in species networks. We used an eco-evolutionary model parameterized with information about 23 empirical antagonistic networks to show that long-term stability of antagonistic networks rely upon rapid fluctuating selection of traits and interactions. Our results also indicate that the feedback loops between ecological and evolutionary processes lead to predictable patterns of species specialization and distribution of abundance in antagonistic networks. Eco-evolutionary feedbacks significantly increase mean abundance and decrease abundance variation. The strength of trait matching between exploiters and victims was on average 100 times more variable in time when interaction selection was strong. Our results present a theoretical link between the study of ecological networks and the fluctuating selection hypothesis of community stability, pointing out the ways by which coevolution may decrease the vulnerability of antagonistic networks.

Introduction

Ecology affects evolution and natural selection is one of the mechanisms that bridge ecological and evolutionary processes in biological systems (Schoener 2011). Ecological interactions among species may cause natural and sexual selection pressures that drive many evolutionary processes, such as phenotypic evolution (Ackerly 2003), coevolution (Strauss *et al.* 2005, Thompson 2012, 1999) and speciation (Schluter 1996). Similarly, evolutionary processes are important for understanding population dynamics (Yoshida *et al.* 2003, Pelletier *et al.* 2007, Coulson *et al.* 2006), community composition and assembly (Emerson & Gillespie 2008, Loeuille & Loreau 2005) and metacommunity dynamics (Urban *et al.* 2008). Eco-evolutionary dynamics posits that both directions of effect, from ecology to evolution and from evolution to ecology, are substantial in their influence (Schoener 2011), and in some cases result in feedback loops between ecological and evolutionary processes (Post & Palkovacs 2009). Feedbacks between evolutionary processes and ecological community dynamics can alter both the adaptive evolution and coexistence of species (Odling-Smee *et al.* 2013, Loeuille 2010, Post & Palkovacs 2009).There is also mounting evidence that evolution can be very rapid and that ecological and evolutionary change may be compatible in time and may interact in a feedback loop (Fussmann *et al.* 2007, Hairston *et al.* 2005, Schoener 2011, Thompson 2013). Rapid evolutionary changes are especially common in predator-prey and host-parasite systems (Shertzer *et al.* 2002, Yoshida *et al.* 2003, 2007, Mougi & Iwasa 2011, Morran *et al.* 2011). A classical example is the rapid evolution of beak and body size on a Galápagos ground finch seed predator in response to changes in ecological conditions (Grant & Grant 2006).

Phenotypic changes driven by evolutionary processes may influence population dynamics of victims and their natural enemies by affecting interaction strengths (Abrams 2010, Yoshida *et al.* 2003, Palkovacs & Post 2009, Becks *et al.* 2010). For instance, in parasite-host interactions the infection dynamics occurs through eco-evolutionary feedback loops with parasites infecting only hosts whose resistances have evolved to overcome (Penczykowski *et al.* 2016). Evolution of host and/or parasite traits can occur within the course of an epidemic, shaping the host and parasite densities (Penczykowski *et al.* 2011). Many studies have documented rapid evolutionary change that affects the interspecific interactions and genotypic structure within natural communities (see reviews by Thompson 1998, 2013; Whitham *et al.* 2006; Fussmann *et al.* 2007). For example, the study by Johnson

& Agrawal (2005) experimentally generated selection on biomass, life history, and herbivore resistance of the primrose *Oenothera biennis* and the resulting evolutionary changes affected abundance and diversity of the associated arthropods. Therefore, the evolution of interaction strengths may change community structure, which may increase or decrease community stability (Loeuille 2010, Rooney & McCann 2012).

It has been shown that evolution of body size (Loeuille & Loreau 2005) or adaptive foraging (Kondoh 2003, Beckerman *et al.* 2006) affect structural attributes of ecological communities, such as connectance; *i.e.* the fraction of all possible links that are realized in a network, or the number of trophic levels of a food web. Ecological changes may in turn modulate the strength of selection and drive subsequent evolution in the system. This broad range of theoretical predictions indicates that rapid trait evolution could contribute significantly to the short-term dynamics of community interaction structure. Hence, considering some aspects of the community ecology such as the structure of species interactions would increase the understanding and predictive potential of evolutionary questions (Fussmann *et al.* 2007). In the same way, models of community dynamics assuming that the structure of interspecific interactions could evolve over short periods may lead to new insights about the mechanisms that promote and maintain biological diversity (Chapin *et al.* 2000, de Mazancourt *et al.* 2008, Loeuille *et al.* 2013)

The outcomes of eco-evolutionary dynamics between pairs of antagonistic species are generally related to the strength of selection imposed by the interaction (Abrams 2000). Reciprocal evolution can result in escalating attack and defense traits or in coevolutionary cycles (Abrams 2000). In diverse communities, pairwise coevolution might be constrained by the network of multiple interactions. For mutualistic interactions theory predicts that coevolution might occur among multiple interactions, perhaps even propagating evolutionary responses through the network (Guimarães *et al.* 2011). Here, we investigate how community structure drives species ecology, evolution and their feedbacks in species-rich antagonistic interactions. We model population dynamics and trait evolution of several interacting victim and exploiter species to explore the eco-evolutionary feedbacks in antagonistic networks and whether the persistence of interactions and the stability of communities are promoted by ongoing rapid evolution that fine-tunes interactions.

Specifically, we aim to investigate how coevolving interactions may shape phenotypic traits and drive population dynamics over time. For that, we parameterize an eco-

evolutionary model with data from 23 empirical antagonistic networks. We measure network and species topological characteristics to explore how network structure affect eco-evolutionary dynamics of communities, and how species interaction patterns within the networks drive population and trait temporal dynamics. To further understand the effect of eco-evolutionary feedbacks on long-term ecological network stability, we compare outcomes with population dynamics with and without trait evolution. Our results support fluctuating selection hypothesis of community stability (Thompson 1998), which states that long-term stability of communities would rely significantly upon rapid fluctuating selection of traits and interactions (Thompson 1998). Our results also indicate that the feedback loops between ecological and evolutionary processes lead to predictable patterns of species specialization and abundance distribution.

Methods

The Model

We model the eco-evolutionary dynamics of victim and exploiter species interacting within antagonistic networks. Interactions between species are mediated by matching of defense and attack traits (Hanifin *et al.* 2008). Trait matching assumes that the probability of interaction depends on the similarity between victim's and exploiters's traits and it has been shown to be important in coevolving interactions such as among brood parasites and their hosts (Vikan *et al.* 2011) and wild parsnip and parsnip webworms (Zangerl & Berenbaum 2003). Each network is composed by $N_V + N_E$ species ($1 < i < N_V$ and $1 < j < N_E$), and each victim species i and exploiter species j were characterized by their abundance, V_i and E_j, and a mean trait value, z_i and y_j , respectively. We focus our work in assemblages that are characterized by two trophic interactions and interactions only occur between species in different trophic levels, such as parasite-host or plant-herbivore interactions. Initial V_i and E_j values were sampled from normal distributions (mean $\pm$ SD, V_i: 0.5 ± 0.1; E_j: 0.1 ± 0.1). Initial values of z_i and y_j were sampled from a normal distribution (0 ± 0.1). A victim-exploiter discrete time model with linear functional response describes species population dynamics as follows

$$\Delta V_i = r_{i(t)} V_i - c_i V_i^2 - \sum_{j=1}^{N_E} x_{ij} m_{ij(t)} E_j V_i \qquad (1a)$$

$$\Delta E_j = r_{j(t)}E_j - c_j E_j^2 + \sum_{i=1}^{N_V} x_{ji} m_{ji(t)} E_j V_i \quad (1b)$$

The strength of species interactions, $m_{ij(t)}$, which is an element of matrix **M** depends on the degree of trait matching between species, $m_{ij(t)} = m_{ji(t)} = e^{-\gamma(z_{i(t)} - y_{j(t)})^2}$, where γ determines the interaction sensitivity to deviations from trait matching. The m_{ij} follows a Gaussian where γ defines the variance and maximal m_{ij} ($m_{ij} = 1$) occurs with maximal trait matching, $z_{i(t)} = y_{j(t)}$. The intraspecific competition term of victim species i (c_i) and exploiter species j (c_j) was defined as $c_i = c_j = 1$. Population growth rates $r_{i(t)}$ and $r_{j(t)}$ are defined as $r_{i(t)} = b_i - d_{i(t)}$ and $r_{j(t)} = b_j - d_{j(t)}$, where b_i and b_j are victim and exploiter species intrinsic birth rates, and $d_{i(t)} = 1 - e^{-\alpha(\theta_i - z_{i(t)})^2}$ and $d_{j(t)} = 1 - e^{-\alpha(\theta_j - y_{j(t)})^2}$ are death rates that reduce population growth rates and are affected by the degree of mismatch between population mean trait value and the optimum trait favored by environmental selection, θ_i and θ_j. The effect of mismatch is controlled by the parameter α, which is the sensitivity to deviations from the optimum trait favored by the environment. We assume that $\theta_i = z_{i(t=0)}$ and $\theta_j = y_{j(t=0)}$. The matrix $\mathbf{X} = [x_{ij}]$ is the binary matrix of species interactions and forbidden links, where $x_{ij} = x_{ji} = 1$ if species i interacts with j and $x_{ij} = x_{ji} = 0$ otherwise, *i.e.*, when a forbidden link occurs. We explore a broad range of birth rates, b, and in our analysis we have used the parameter values that allowed a higher proportion of species to persist ($b_i = 1.01$ for victim species and $b_j = 0.3$ for exploiter species).

Species fitness for the victims (W_i) and exploiters (W_j) was defined as the population per capita rate of increase (Saloniemi 1993), here described as:

$$W_{i(t)} = \frac{V_{i(t+1)}}{V_{i(t)}} \approx r_{i(t)} - c_i V_{i(t)} - \sum_{j=1}^{N_E} x_{ij} m_{ij(t)} E_{j(t)} \quad (2a)$$

$$W_{j(t)} = \frac{E_{j(t+1)}}{E_{j(t)}} \approx r_{j(t)} - c_j E_{j(t)} + \sum_{i=1}^{N_V} x_{ji} m_{ji(t)} V_{i(t)} \quad (2b)$$

To estimate the trait values that maximize species fitness at each time step we numerically calculate the fitness gradients for victims ($\frac{\delta W_{i(t)}}{\delta z_{i(t)}}$) and exploiters ($\frac{\delta W_{j(t)}}{\delta y_{j(t)}}$). The traits favored

by selection, $Z_{i(t)}$ and $Y_{j(t)}$, are the global maximum of the species fitness gradients at time t.

Trait evolution is modeled as discrete events of change caused by the joint selective pressures imposed by environment and antagonistic partners. The species mean trait will change in one generation according to the breeder's equation (Lande 1976):

$$\Delta z_{i(t)} = h_i^2 (Z_{i(t)} - z_{i(t)}) \tag{3a}$$

$$\Delta y_{j(t)} = h_j^2 (Y_{j(t)} - y_{j(t)}) , \tag{3b}$$

where h^2 is the heritability of species trait. We assumed constant and equal heritabilities for all species ($h^2 = 0.25$). Species mean traits in the next generation is then calculated as

$$z_{i(t+1)} = z_{i(t)} + \Delta z_{i(t)} \tag{4a}$$

$$y_{j(t+1)} = y_{j(t)} + \Delta y_{j(t)} . \tag{4b}$$

The dynamics expressed by Eq. (4) reflects adaptive changes in species trait resulting from natural selection (Abrams *et al.* 1993). We study scenarios with different environmental and interaction selection strength on species coevolution. The first scenario describes a condition with weak environmental and interaction selection ($\alpha = \gamma = 0.001$). Weak environmental selection may arise when the probability to survive depends weakly on matching the environmental optimum trait. In the same way, weak interaction selection occurs when the probability of interaction depends only weakly on victim and exploiter trait matching. Our second scenario considers strong environmental and strong interaction selection ($\alpha = \gamma = 0.1$). In this scenario the probability to survive and to interact depend strongly on trait values. The other two scenarios describe asymmetric selection regimes, where environmental selection is weak and interaction selection is strong ($\alpha = 0.001$, $\gamma = 0.1$) and environmental selection is strong and interaction selection is weak ($\alpha = 0.1$, $\gamma = 0.001$).

Empirical antagonistic networks and community structure

We use 23 empirical antagonistic networks to parameterize the matrix **X** (see Equation 1), which encompass a broad range of antagonisms, including parasitism, herbivory,

predation and grazing. Species richness ranged from 12 species in communities of bacterias and phages to 31 species in communities of fish hosts and their parasites (Table S1). We did not included larger networks because the simulations were time-consuming. To explore the effects of network structure on eco-evolutionary dynamics, we used four network descriptors: (i) species richness, (ii) connectance, *i.e.,* the proportion of realized interactions, (iii) nestedness, and (iv) modularity. Nestedness is a pattern of interactions in which specialist species tend to interact with subsets of species that interact with more generalist species (Bascompte *et al.* 2003). We used the metric NODF (Almeida-Neto *et al.* 2008) to estimate nestedness, computed using ANINHADO (Guimarães & Guimarães 2006). Modularity characterizes groups of species that have more interactions within groups than among groups (Olesen *et al.* 2007). The network degree of modularity was estimated using a simulated annealing algorithm to optimize the metric *Q* (Newman & Girvan 2004, Guimerà & Amaral 2005) calculated using the software MODULAR (Marquitti *et al.* 2014).

We used z-scores to compare levels of nestedness and modularity across different networks. The nestedness z-score, $NODF_z = \frac{NODF - \overline{NODF_{null}}}{\sigma_{null}}$, and modularity z-score, $Q_z = \frac{Q - \overline{Q_{null}}}{\sigma_{null}}$, measure the degree of nestedness or modularity of each empirical network in relation the mean expected nestedness and modularity under a given null model. $\overline{NODF_{null}}$ and $\overline{Q_{null}}$ are the average *NODF* and *Q* values and σ_{null} are their standard deviations after 1000 runs of the null model. We used *null model 2* of Bascompte *et al.* (2003), which assumes that the probability of drawing an interaction is proportional to the mean number of interactions (degree) of the exploiter and victim species. This null model was used to control the effects of heterogeneity in number of interactions, connectance and species richness on nestedness and modularity (Bascompte *et al.* 2003).

To investigate if species degree of generalization or specialization within the network determines its abundance and trait dynamics, we characterized each species according to the following descriptors: (i) normalized degree, which is the number of interactions normalized by the maximal number of potential partners in the other trophic level (Martín González *et al.* 2010); and (ii) standardized Kullback-Leibler distance (d'), which is a relative entropy measure for partner diversity and ranges from 0 for the most generalized to 1 for the most

specialized species (Blüthgen & Menzel 2006).

Ecological and coevolutionary dynamics

We run each replicate for 3,000 time steps, which was sufficient time to reach convergence, and we ran 100 replicates per network and scenario, totalizing 9,200 simulations. To characterize species ecological dynamics we calculated species mean abundances and variance across time since $t = 0$. We used the abundances coefficient of variation to measure abundance temporal variability and compare the ecological dynamics across species and scenarios. We also calculated the average coefficient of variation of abundances across species to describe the ecological dynamics on each network. To infer stability we assumed that the highest the temporal variability in abundance, the highest the vulnerability of that community to stochastic extinctions (Donohue *et al.* 2013, Loreau & de Mazancourt 2013, Yeakel *et al.* 2014). Similarly, we characterized trait dynamics by the species coefficient of variation in trait values across time. We described the network trait dynamics as the average coefficient of variation across species. A higher variation in trait values indicates a stronger and faster evolutionary response to selective pressures.

We characterized the temporal fluctuation in the interaction strength among species pairs, s_{ij}, as the cumulative change in trait matching following, $s_{ij} = x_{ij} \sum_{t=1}^{t=3,000} \left|(z_{i(t)} - y_{j(t)}) - (z_{i(t-1)} - y_{j(t-1)})\right|$, where $x_{ij} = 1$ if species i and j interact and zero otherwise and $z_{i(t)}$ and $y_{j(t)}$ are the mean trait values of victim species i and exploiter species j at time t, respectively. The mean cumulative change in interaction strength across all species in a network was calculated as $s = \frac{\sum_i \sum_j s_{ij}}{N_V \times N_E}$, where s_{ij} is the cumulative change in trait matching between a victim and exploiter species. Higher s values indicate higher temporal variation in interaction strength among species, which we use as a proxy for fluctuating selection.

We used ANOVA to compare the mean abundances, coefficient of variation in the abundances and coefficient of variation in trait values across scenarios. The effect of network and species metrics on ecological and coevolutionary dynamics was investigated using

general linear models considering each scenario as a covariable. We did not report *p*-values because for simulation data statistical significance is not useful to determine the relative importance or presence/absence of effects of the various predictors in simulation studies (White *et al.* 2014). We rather used the effect sizes and sign of the estimated regression parameters for investigating the importance of different predictors (White *et al.* 2014).

Results

Network dynamics

Mean abundance was larger (coefficients: $\beta_{1(both\ weak)}$=0.06, $\beta_{2(both\ strong)}$=0.06, $\beta_{3(interaction\ stronger)}$=0.075, $\beta_{4(environmental\ stronger)}$=0.06, $F_{9196,3}$=31.31) when interaction selection was stronger than environmental selection (Figure 1). There were no differences in the mean abundances among the scenarios with both strong and both weak strength of interaction and environmental selection, respectively, and when environmental selection was stronger then interaction selection (Figure 1). In contrast, abundances were less variable in time ($\beta_{1(both\ weak)}$=3.86, $\beta_{2(both\ strong)}$=3.85, $\beta_{3(interaction\ stronger)}$=2.51, $\beta_{4(environmental\ stronger)}$=3.85, $F_{9196,3}$=119.5) when interaction selection was stronger than environmental selection (Figure 2). There were no differences in the coefficient of variation in abundances among the scenarios with both strong and both weak strength of interaction and environmental selection, respectively, and when environmental selection was stronger then interaction selection (Figure 2). On the other hand, the coefficient of variation in species trait values was much higher under strong interaction selection ($\beta_{1(both\ weak)}$=5.73, $\beta_{2(both\ strong)}$=7.37, $\beta_{3(interaction\ stronger)}$=22.52, $\beta_{4(environmental\ stronger)}$=0.56, $F_{9196,3}$=55.84). The pattern was reversed when environmental selection was stronger, which made traits less variable in time (Figure 3). There was no difference in trait variation when interaction and environmental selection were both either weak or strong (Figure 3). These results suggest that eco-evolutionary feedbacks are restricted to the scenario with strong interaction selection and weak environmental selection, in which significantly changes mean abundance and higher abundance variation occur. In contrast, eco-evolutionary feedbacks are absent when there is weak interaction and environmental selection or weak interaction and strong environmental selection. In the scenario with strong interaction selection but also strong environmental selection the feedbacks were weaker and the effect of the eco-evolutionary feedback was observed in the trait coefficient of variation only (Figure 3).

Network structure affected the eco-evolutionary dynamics in a consistent way. Connectance and nestedness decreased mean abundance (connectance: F_{90}=74.34, R^2=0.45 and nestedness: F_{90}=18.09, R^2=0.16, Figure 1) and increased abundance variation (connectance: F_{90}=77.28, R^2=0.46 and nestedness: F_{90}=25.51, R^2=0.21, Figure 2). Species richness had a very weak negative effect on mean abundance (F_{90}=5.33, R^2=0.05, Figure 1) and weak positive effect on abundance variation (F_{90}=19.97, R^2=0.17, Figure 2). Species richness also increased trait variation (F_{90}=77.28, R^2=0.46, Figure 3) and connectance and nestedness had a weak positive effect (connectance: F_{90}=6.62, R^2=0.06 and nestedness: F_{90}=3.13, R^2=0.02, Figure 3). Modularity affected eco-evolutionary dynamics in the opposite direction than richness, connectance and nestedness. Modularity increased mean abundance (F_{90}=22.59, R^2=0.2, Figure 1), reduced abundance variation (F_{90}=12.85, R^2=0.12, Figure 2) and had a weak negative effect on trait variation (F_{90}=3.4, R^2=0.03, Figure 3).

The interaction strength between exploiters and victims was on average 100 times more variable in time when interaction selection was stronger than environmental selection ($F_{9196,3}$=2239.0, Figure 4). Fluctuation in interaction strength between interacting species was low in the other selection scenarios (Figure 4). In agreement with the fluctuating selection hypothesis of community stability, which states that long-term stability of communities would rely significantly upon rapid fluctuating selection on the links among species (Thompson 1998), we found a negative relationship between the temporal variability in abundances and the accumulated change in the interaction strength between species partners (β=-0.02, F_{90}=8.31, R^2=0.07). However, network structure had no significant effect on the temporal change in pairwise interaction strengths (Figure 4).

Species dynamics

The eco-evolutionary dynamics was highly variable among species and metrics related to species generalization/specialization within the network were related to their population dynamics (Figure 5). In the same way, trait dynamics was related to species generalization/specialization within the network (Figure 6). In general, more specialized species have larger mean abundances, those with lower normalized degree (β=-0.16, $F_{1876,4}$=43.27, R^2=0.08, Figure 5a) and higher specialization (β=0.3, $F_{1876,4}$=217.9, R^2=0.32, Figure 6a). The temporal variation in abundances did not showed a consistent pattern across species patterns of interaction. The coefficient of variation in abundances had a very weak negative effect on species normalized degree (β=-3.3, $F_{1876,4}$=2.63, R^2=0.004, Figure 5b), but also weak negative effect on specialization degree (β=-14.3, $F_{1876,4}$=39.49, R^2=0.08, Figure

6b). However, generalist species consistently showed higher temporal trait fluctuation because more connected and less specialized species tended to have a higher coefficient of variation in trait values (normalized degree: β=17.9, $F_{1876,4}$=5.07, R^2=0.008, Figure 5c; specialization: β=-14, $F_{1876,4}$=4.56, R^2=0.008, Figure 6c). It is worth emphasizing that these effects were weak because of the high variance among species.

Discussion

In this study, we integrated approaches derived from ecological and evolutionary theory with network-based modeling and empirical data to investigate the consequences of rapid coevolution and eco-evolutionary feedbacks on the stability of antagonistic networks. Our results show that eco-evolutionary feedbacks significantly increase mean abundance and decrease abundance variation compared to the scenario without feedbacks (i.e., weak interaction selection). We also found that the interaction strength between exploiters and victims was on average 100 times more variable in time when interaction selection was stronger than environmental selection. Our results present theoretical evidence for the fluctuating selection hypothesis of community stability and identified three central points related to the mechanism by which coevolution decreases the vulnerability of antagonistic networks to stochastic processes.

First, strong interaction selection between victim and exploiters driving eco-evolutionary feedbacks predicted a positively correlated trend between species richness, connectance and nestedness and the variance in abundance and trait variation. As in other coevolutionary models based in a bidirectional axis of vulnerability (Abrams 2000), we also found fluctuating dynamics in populations and traits in all scenarios. However, the relative magnitude of mean and variance abundance and trait variation depended on the relative strength of the different types of selection and on the network organization. High variability in abundances and low variability in species traits were observed when interaction selection was weaker or equal than environmental selection. Stronger interaction selection reversed this relationship, increasing trait variability and decreasing abundance variability (compare Figure 2 with 3). Previous models in predator-prey systems have shown that population cycles were related to fluctuating selection because rapid trait evolution reciprocally influenced population dynamics (Abrams & Matsuda 1997b, Yoshida *et al.* 2003). Our results indicate that trait and abundance dynamics can be decoupled when the network structure of interactions is considered. In a network of species interactions, environmental

selection equal or stronger than interaction selection constraints exploiter and victim trait coevolution and the numerical response play an important role in community dynamics. On the other side, when interaction selection is stronger than environmental selection, particular network structural patterns favor rapid trait coevolution decreases population fluctuations. In a broader perspective, these theoretical results illustrate how ecological and evolutionary processes may represent alternative routes to how populations respond to changes in interacting assemblages formed by dozens of species (Barraclough 2015).

Second, adaptive trait fluctuations temporally change the interaction strength of interacting partners, which dampens the temporal variability in species abundances. This indicates that adaptive trait fluctuations can stabilize exploiter-victim population dynamics in antagonistic networks, a pattern that was also found in other models using only few interacting species (Abrams & Matsuda 1997a, Abrams 2000, Mougi & Iwasa 2010, Mougi 2012) and also experimentally (Friman *et al.* 2014, Yoshida *et al.* 2007, 2003). However, when different approaches to explore stability such as the analysis of local stability to small perturbations were used it was found that evolution decreased the resilience of highly diverse communities (Loeuille 2010). The magnitude of population temporal variability also depended on the structure of the interactions among exploiters and victims. There is a long-standing debate on whether ecological community complexity increases their stability (May 1972, McCann 2000, Ives & Carpenter 2007, Allesina & Tang 2015), with ecosystems often supporting large numbers of species interacting in highly complex networks of direct and indirect pathways (Ings *et al.* 2009, Bascompte 2010). One mechanism that has been proposed to enhance stability in complex antagonistic communities is fluctuating selection on interaction strengths among species (Thompson 1998), which can arise from adaptive foraging and behavior or evolutionary trait changes (Kondoh 2003, Yamaguchi *et al.* 2011, Nuwagaba *et al.* 2015, Yoshida *et al.* 2003, Johnson & Agrawal 2003). Our results support that the structure of the interactions determines the relative stability of the networks, and that different aspect of network structure affects its stability in opposite ways. Network richness, connectance and nestedness increased abundance variation, whereas modularity stabilized species abundance fluctuations. Additionally, modularity increased overall species mean abundances. A reduced sharing of interacting partners, which is found in modular networks, is supposed to reduce the negative indirect effects of apparent competition and thereby the propagation of direct and indirect negative effects across the network (Thébault & Fontaine 2010, Allesina & Tang 2012). Our results suggest that modular structure interacts with the fluctuating selection mechanism because modularity restricts the number of possible partners

with which a species interacts, which reduces the amount of trait change required to adaptively adjust the strength of interaction with its partners. In addition, the amount of trait change is reduced in highly modular network because species outside modules will seldom trigger adaptive evolution in species within modules (Guimarães *et al.* 2011). When compared to other structural patterns, higher modularity has already been related with higher ecological stability in antagonistic networks (Thébault & Fontaine 2010).

Third, species dynamics and the degree of specialization within networks were not homogeneous and had highly variable responses across species. The degree of species generalization partially predicted their behavior, but much of the variation was still not explained by species normalized degree and specialization (d'). Contrary to the neutral expectation (Hubbell 2005, Vázquez *et al.* 2007, Canard *et al.* 2012), we found that specialized species tented to have higher mean abundances than generalists. If species were ecologically equivalent, abundances would determine the probability of interactions and it is predicted that generalists would be the most abundant species (Vázquez *et al.* 2007, Canard *et al.* 2012). However, when interaction trait constraints are more important than species abundance in determining species interactions we would not expect a positive association between abundance and degree of generalization. We found a negative relationship with the species normalized degree (Figure 5a) and a positive relationship with the specialization level (Figure 6a), which indicates that when the strength of interactions change in time because of species coevolution, the feedbacks between ecological and evolutionary dynamics generate a trade-off between the number of interacting partners and abundance. This trade-off has already been found in parasite-host and parasitoid-host interactions (Fry 1990, Poulin 1998, Poullain *et al.* 2008) and has been related to the high cost of adaptations to multiple victim defense (or exploiter attack) mechanisms (Poulin 1998; but see Poisot *et al.* 2013). Exploiters that specialize on a few victims (or victims that are attacked by a few exploiters) may attain greater abundance because they do not need to invest more than a specific range of adaptations against attack or defense mechanisms. The negative relationship between trait change and specialization corroborate this hypothesis (Figures 5a and 5c).

In addition, we found that species abundances tended to be smaller when environmental selection was stronger or equal to interaction selection. Contrasting selection pressures often reduce the abundances because of trade-offs effects on the mechanisms that contribute to species fitness (Abrams 1995, 2000, Werner & Peacor 2003). For instance, selection for a trait that increases exploiter's attack or victim's defense may decrease its ability to deal with environmental conditions. These secondary effects of trait evolution may

explain why species achieve smaller mean abundances and larger variation when environmental selection had a stronger effect on fitness (Figures 1 and 2).

By introducing an eco-evolutionary framework to the study coevolution in species-rich networks, we found support for the hypothesis that fluctuating selection would lead to long-term community stability (Thompson 1998). However, network structure constrained adaptive change in interaction strengths therefore stabilizing community dynamics. Different approaches based on adaptive foraging already pointed out that modularity increases antagonistic network stability, whereas connectance and nestedness reduces it (Kondoh 2003; Thébault & Fontaine 2010; Allesina & Tang 2012). Our results corroborate this pattern and indicate that modularity also enhances the fluctuating selection mechanism by driving it to a coevolutionary stable unit. The eco-evolutionary feedback loop also generated a trade-off between the number of interacting partners and abundance, which is not expected if strengths of interactions are determined uniquely by species abundances.

Our results suggest that studying eco-evolutionary feedbacks is important because it encourages evolutionary biologists to recognize that organisms can shape their environment in ways that alter the outcome of evolution, and it encourages ecologists to recognize that contemporary evolution creates phenotypic differences that can alter the role of a species in a community or ecosystem at contemporary time-scales (Post & Palkovacs 2009). In this way, the study of eco-evolutionary feedbacks focuses attention on the bidirectional interactions that unify ecology and evolution, and may provide a mechanistic understanding of how communities respond to disturbances and fragmentation, how diseases evolve and reshape communities, how introduced species spread across landscapes and how links among species change even amid normal environmental fluctuations (Thompson 1998, Stockwell *et al.* 2003, Kinnison & Hairston 2007).

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Figures

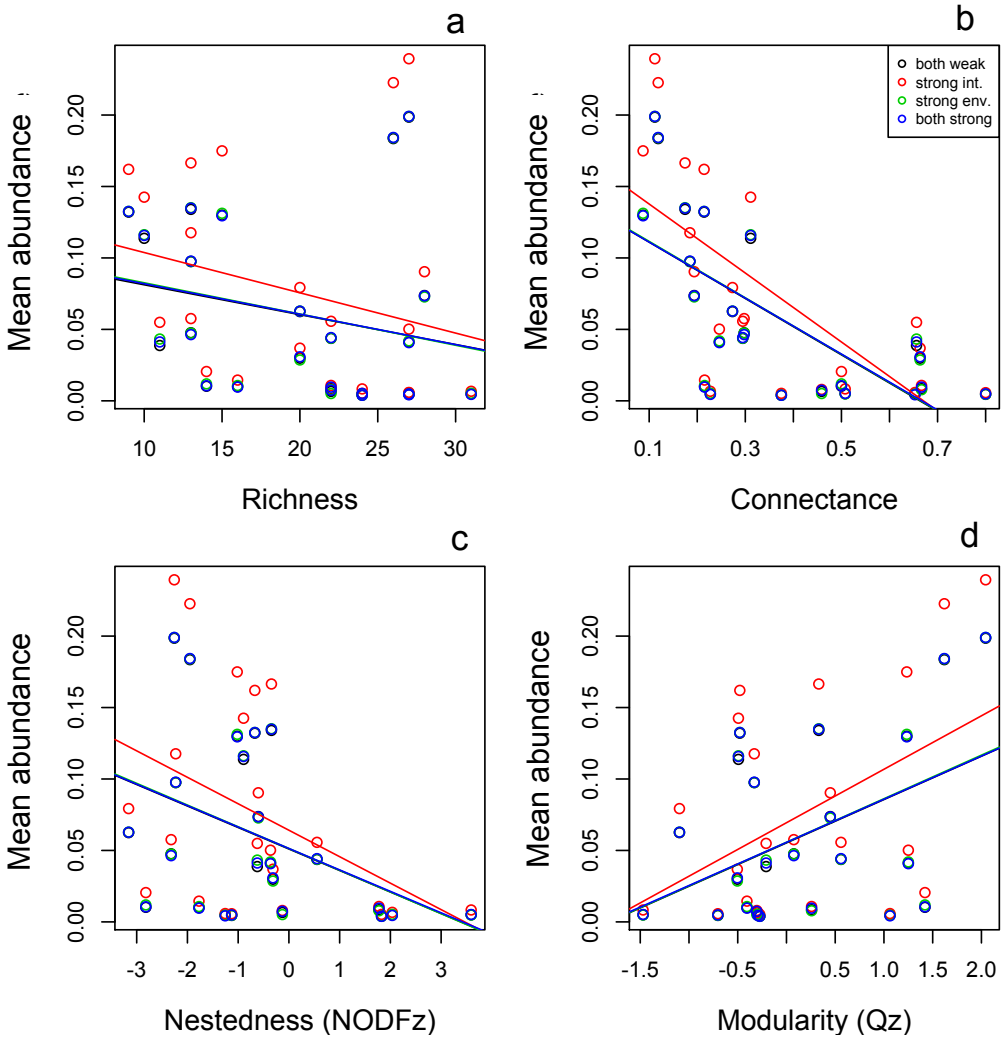

Figure 1: Effect of Richness (a), Connectance (b), Nestedness (c) and Modularity (d) on mean abundance in each empirical network. Each dot represents the network average values after 100 replicates. Results shown for the scenarios with environmental and interaction selection weak (black), strong (blue) and stronger environmental than interaction selection (green).

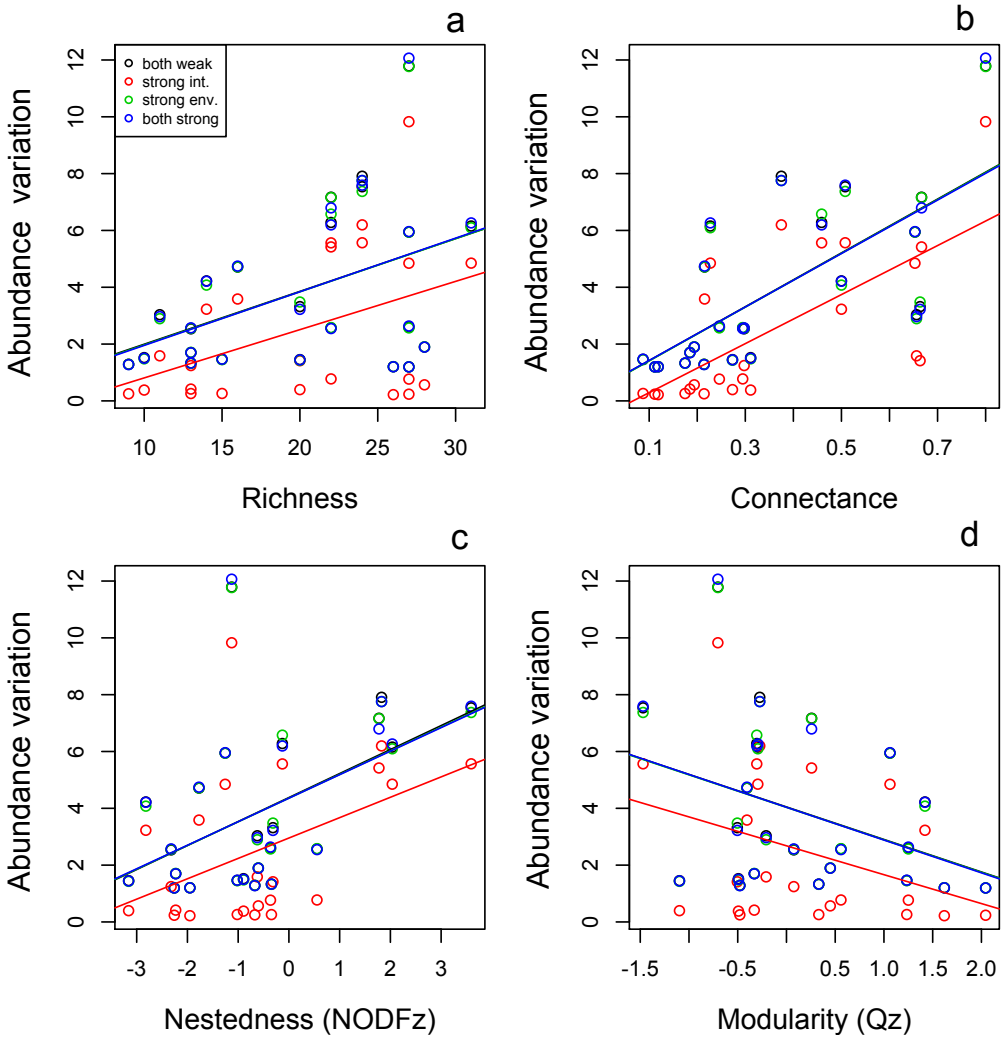

Figure 2: Effect of Richness (a), Connectance (b), Nestedness (c) and Modularity (d) on abundance variation in the four scenarios. Each dot represents the network average values after 100 replicates. Results shown are for the scenarios with environmental and interaction selection weak (black), strong (blue) and stronger environmental than interaction selection (green).

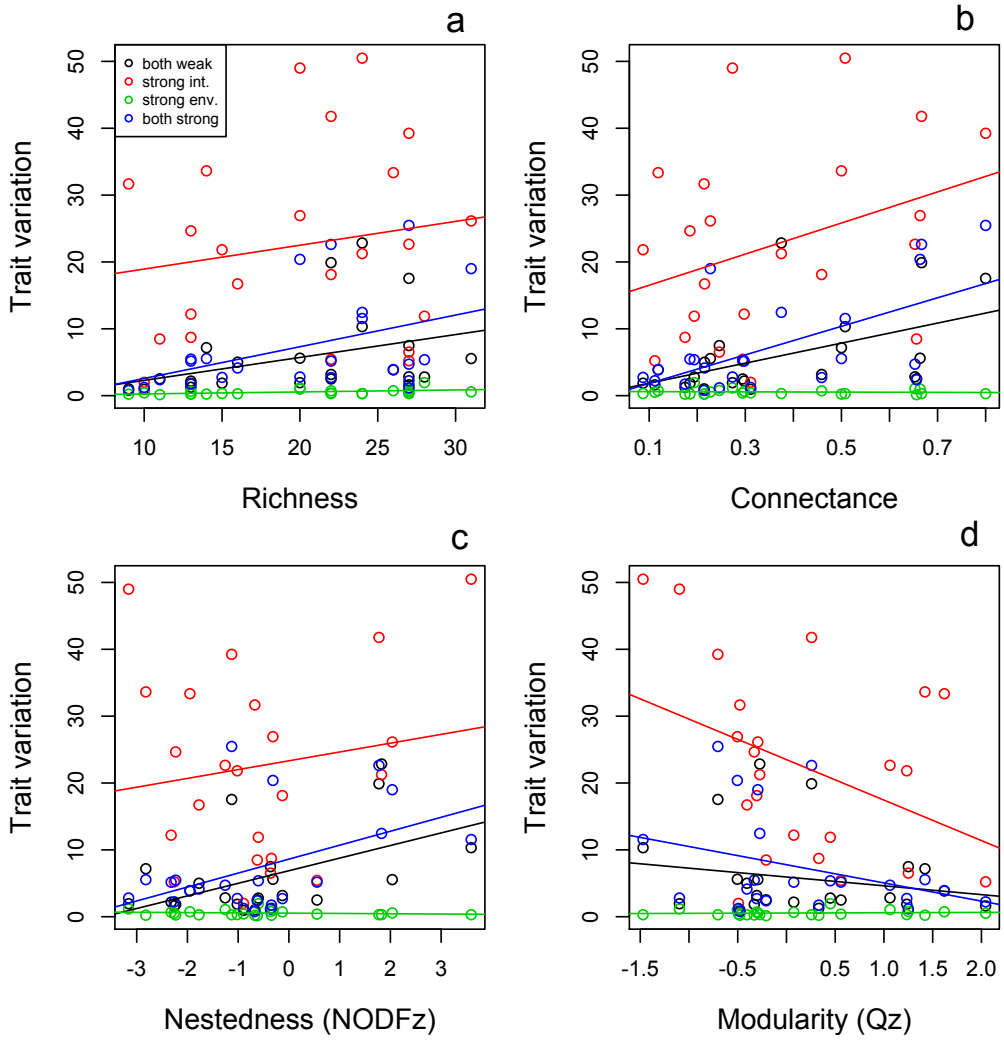

Figure 3: Effect of Richness (a), Connectance (b), Nestedness (c) and Modularity (d) on trait variation in the four scenarios. Each dot represents the network average values after 100 replicates. Results shown are for the scenarios with environmental and interaction selection weak (black), strong (blue) and stronger environmental than interaction selection (green).

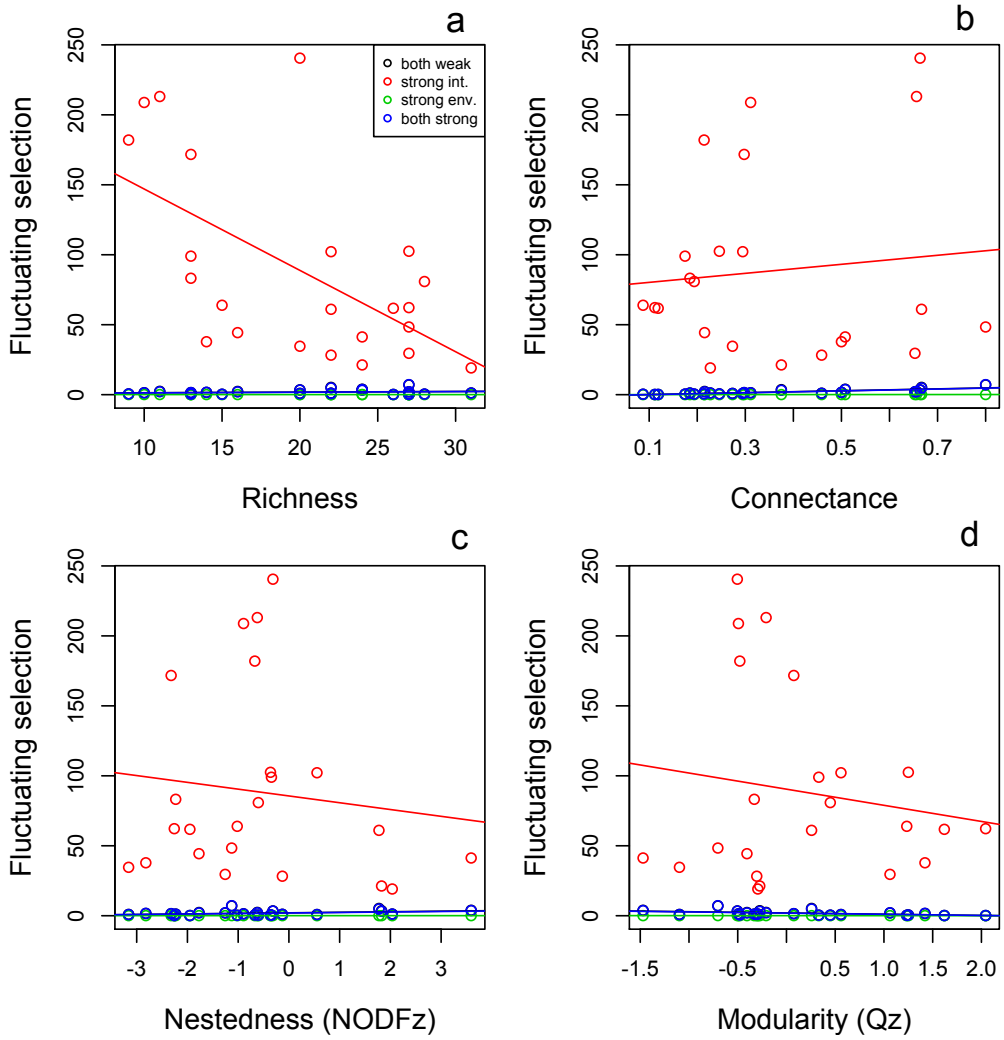

Figure 4: Effect of Richness (a), Connectance (b), Nestedness (c) and Modularity (d) on fluctuating selection. Fluctuating selection was measured as mean cumulative change in interaction strength (*s*) in the four scenarios. Each dot represents the network average values after 100 replicates. Results shown are for the scenarios with environmental and interaction selection weak (black), strong (blue) and stronger environmental than interaction selection (green).

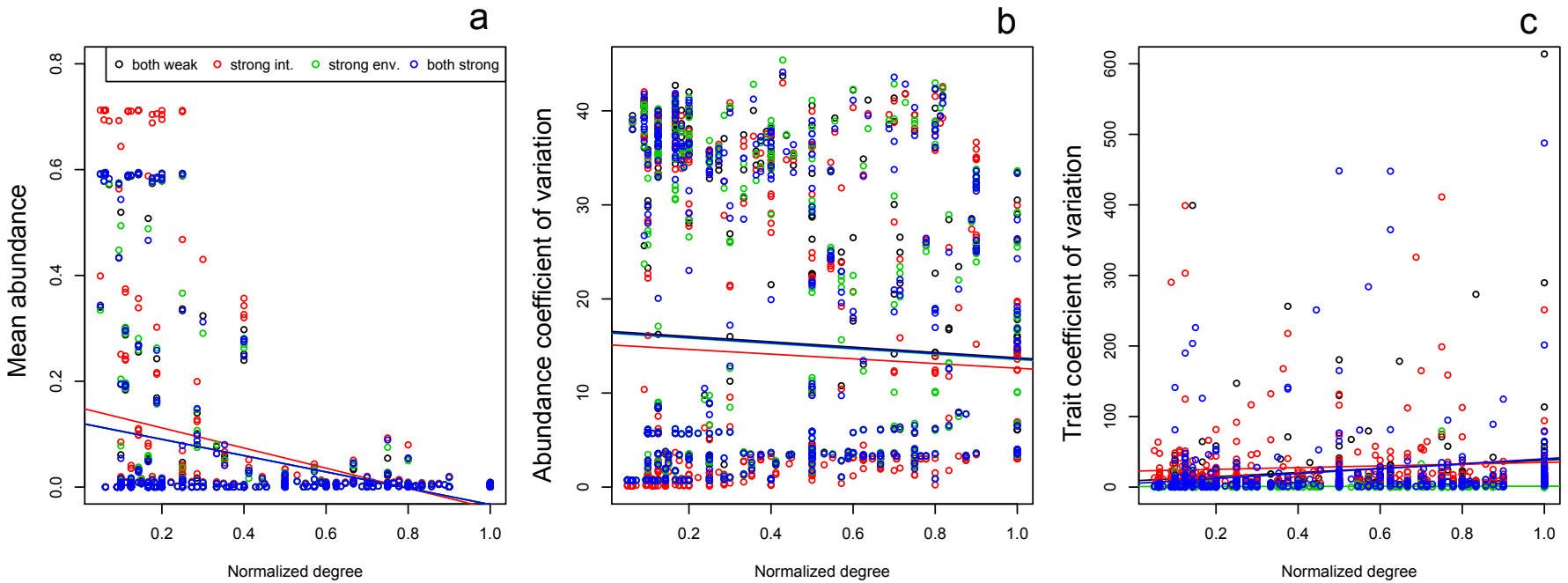

Figure 5: Effect of species normalized degree on mean abundance (a), abundance coefficient of variation (b), and trait coefficient of variation (c) in the four scenarios. Each dot represents the network average values after 100 replicates. Results shown are for the scenarios with environmental and interaction selection weak (black), strong (blue) and stronger environmental than interaction selection (green).

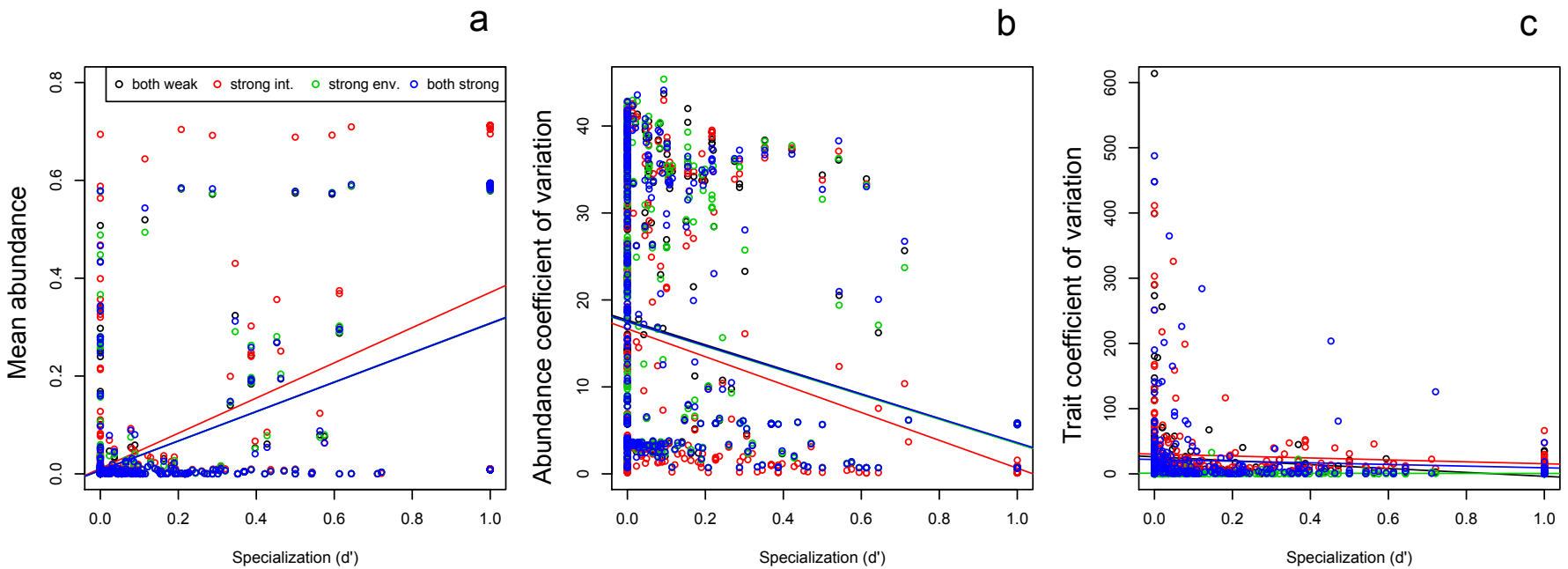

Figure 6: Effect of species Specialization (*d'*) on mean abundance (a), abundance coefficient of variation (b), and trait coefficient of variation (c) in the four scenarios. Each dot represents the network average values after 100 replicates. Results shown are for the scenarios with environmental and interaction selection weak (black), strong (blue) and stronger environmental than interaction selection (green).

Eco-evolutionary feedbacks promote fluctuating selection and long-term stability of species-rich antagonistic networks

Supporting Information

1. Networks used in the study

Table S1. Dataset[1] used to parameterize the matriz **X**, including the type of interaction and network basic characteristics (N_V = number of victims; N_E = number of exploiters, C = Connectance).

Data set	Type	N_V	N_E	C	Reference
herb2	Mammalian grazers – plants	10	17	0.6	Hansen et al. 1985
herb5	Gallers – host plants	10	17	0.12	Dawah et al. 1995
herb14	Gallers – host plants	10	16	0.12	Tscharntke et al. 2001
pred1[2]	Mammalian predator – prey	16	8	0.52	Baskerville et al. 2011
pred2	Mammalian predator – prey	22	5	0.8	Owen-Smith & Mills 2008
pred6	Mammalian predator – prey	18	4	0.66	Radloff & du Toit 2004
bac1	Bacteriophage - bacteria	11	4	0.5	Abe & Tanji 2007
bac10	Bacteriophage - bacteria	5	17	0.46	DePaola et al. 1998
bac11	Bacteriophage - bacteria	15	10	0.27	Doi et al. 2003
bac13	Bacteriophage - bacteria	6	7	0.21	Gamage et al. 2004
bac18	Bacteriophage - bacteria	11	10	0.66	Krylov et al. 2006
bac2	Bacteriophage - bacteria	14	6	0.3	Barrangou et al. 2002

bac25	Bacteriophage - bacteria	8	4	0.66	Mizoguchi et al. 2003
bac31	Bacteriophage - bacteria	24	6	0.21	Seed & Dennis 2005
bac34	Bacteriophage - bacteria	7	9	0.17	Suttle & Chan 1993
bac36	Bacteriophage - bacteria	18	7	0.09	Wang & Chen 2008
bac4	Bacteriophage - bacteria	9	5	0.31	Campbell et al. 2006
bac5	Bacteriophage - bacteria	18	8	0.37	Capparelli et al. 2010
bac6	Bacteriophage - bacteria	23	4	0.18	Caso et al. 2006
par12	Parasites - fish host	6	21	0.25	Bensley et al. 2011
par21	Parasites - fish host	8	14	0.29	Morozińska-Gogol 2007
par22	Parasites - fish host	11	20	0.23	Choudhury et al. 2004
par31	Parasites - fish host	8	20	0.19	Jalali & Barzegar 2006

[1] These datasets are available online at the Interaction Web Database (www.nceas.ucsb.edu/interactionweb/index.html) and compiled in Pires & Guimarães (2013), Flores et al. (2011) and Bellay et al. (2013)
[2] Subnetwork depicting predator-prey interactions between mammals weighing > 5kg

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Capítulo 4

perspectivas para o estudo das dinâmicas ecológica e evolutiva em redes de interação antagonista

Title: Parasite spreading in spatial ecological multiplex networks

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*Equally contributed

running title: parasite spreading in multiplex

key-words: Ecological multiplexes, multi-host parasites, spatial networks, SI dynamics

Abstract

Several parasites may be transmitted among its hosts through different mechanisms at the same time, which challenges the modelling of the parasite spreading process. Multiplex are multi-layer graphs where the same nodes can be connected according to different topologies and mechanisms on each layer. We present a novel spatially-embedded multiplex network framework for modelling multi-host infection spreading through multiple routes of transmission. Our model is inspired by *Trypanosoma cruzi*, a parasite transmitted by trophic and vectorial mechanisms. In our ecological multiplex network, nodes represent species populations interacting through a food web and a vectorial contaminative layer at the same time. We modelled Susceptible-Infected dynamics in two different scenarios: a simple theoretical food web and an empirical one. Our simulations in both scenarios show that the infection is more widespread when both transmission mechanisms are considered at the same time and it is maximised when they have similar importance. This indicates that trophic and contaminative transmission may have additive effects in real ecosystems. We also found that the ratio of vectors-to-host in the community (i) crucially influences the infection spread, (ii) regulates a percolating phase transition in the parasite transmission and (iii) increases the infection rate in hosts. Through the study of the multiplex cartography and immunisation experiments, we show that the multiplex structure can be fundamental in outlining the role that each host species plays in parasite transmission in a given ecosystem. We also show that the time needed to infect all the nodes in empirical ecological scenarios is minimised when both transmission mechanisms have similar importance.

Introduction

Pathogens and parasites ("parasites" hereafter) are one of the most widespread and diverse lifeform (Dobson 2004). Parasites may use different routes of infection in order to maximise their transmission in the host populations. Multi-host parasites include many zoonoses with complex dynamics that challenge infection control and prevention efforts (Dobson 2004). For instance, several multi-host protozoan parasites of public health concern exhibit more than one mode of transmission: *Toxoplasma gondii* can infect its hosts by fecal-oral transmission, consumption of an infected prey, and congenitally through the placenta (Dubey 2004); *Cryptosporidium* directly infects its hosts via sexual contact or via fecal-oral transmission (Fayer et al. 2000); *Trypanosoma cruzi* can be transmitted by triatomine vectors (stercorarian transmission), the consumption of an infected prey, and also congenitally through the placenta (Noireau et al. 2009, Jansen et al. 2015). This complexity of host types and transmission modes challenges the development of models that account for the different sources of variation. The network approach is a promising alternative because it allows accounting for the individual, species-level and spatial sources of heterogeneity (Craft et al. 2011).

Contact networks can be used to understand the epidemiological consequences of complex host interaction patterns (Keeling 2005, Meyers et al. 2005, Bansal et al. 2006, Ferrari et al. 2006, Craft et al. 2009, Dalziel et al. 2014). In a contact network, each species or population is represented as a node and each contact that potentially results in transmission between two nodes is represented by a link. Interactions also occur in space (Craft et al. 2009, Davis et al. 2008, Davis et al. 2015, Dalziel et al. 2014) and the probability of interaction between nodes may depend on the distance between them. The number of contacts of a node is called the degree of the node and the degree distribution is a fundamental quantity in network theory (Barabási & Albert 1999). All epidemiological models make assumptions about the underlying network of interactions, often without explicitly stating them. For example classical mean field models used in epidemiology assume random network of contacts. Contact network models, however, mathematically formalise this intuitive concept so that epidemiological calculations can explicitly consider complex patterns of interactions (Bansal et al. 2007).

Recently, the recognition that real-world networks may include different types of interactions among entities prompted the development of methods that take into account the

heterogeneity of interactions (Boccaletti et al. 2014, Kivela et al. 2014). Examples include multi-modal transportation networks in metropolitan areas (Barthélemy 2011, Morris & Barthélemy 2012), or proteins that interact with each other according to different regulatory mechanism (Cardillo et al. 2013, Cozzo et al. 2013). Ecological systems are also characterized by multiple types of relationships among biological entities, organised and structured on different temporal and spatial scales (Shai et al. 2015, Kivela et al. 2014). Such representations can be described as "multi-layer networks" or "multiplex networks" (Wasserman & Faust 1994, Shai et al. 2015). They are multi-layer graphs where the same nodes appear on all the layers, but they can be connected according to different topologies and mechanisms on each layer. Each multiplex layer contains edges of a given type. In the context of parasites that can be transmitted over multiple transmission modes, multiplex networks can be used to include distinct mechanisms of parasite transmission as interconnected layered networks (Shai et al. 2015). This approach encapsulates the heterogeneity in the transmission of real-world diseases and helps us understand how the interplay between different modes of transmission affects infection dynamics in an ecosystem (Buono et al. 2014, Salehi et al. 2014).

Descriptions of ecological multiplexes (Shai et al. 2015) and studies of infection spreading over multiplex structures (Buono et al. 2014, Salehi et al. 2014) have recently appeared in the literature. However, the consideration of real ecological scenarios in the analysis of parasite spreading through multiple transmission mechanisms has not been exlored. We propose a spatial multiplex-based framework to model multi-host parasite transmission through multiple transmission mechanisms. In this framework, each type of interespecific interaction can be represented in a different layer of the multi-layered complex network structure. Our model is inspired by the ecology of *Trypanosoma cruzi* (Kinetoplastida: Trypanosomatidae) in its multiple host community. *T. cruzi*, the etiological agent of Chagas disease, is a relevant example of a multi-host parasite and a serious disease affecting 6-9 million people (Hotez et al. 2008). The main infection route to humans involves the triatomine vectors. Bloodmeals from an infected host can transmit the parasite to the triatomine vector, while defecation by an infected vector on the host following the bloodmeal can result in stercorarian transmission to the host. In sylvatic hosts the stercorarian transmission may occur when the animal scratches the bite and inadvertently rubs the parasite-contaminated matter into the lesion (Kribs-Zaleta 2006). Infection by the oral route occurs when a mammal ingests infected triatomine feces, food contaminated with the parasite or by preying upon infected vectors or mammals (Jansen et al. 2015).

We used a Susceptible-Infected (SI) model to describe parasite transmission dynamics in spatially embedded multiplexes. The multiplex framework helps us understand how infection spread is related to the multiplex structure and what is the epidemiological importance of vectors and hosts in different ecological scenarios. We studied a theoretical three-species spatial multiplex first to understand the interplay between the multiplex structure and epidemiological dynamics. Then we used empirical data of a local *T. cruzi* host community to model the dynamics of *T. cruzi* multiple transmission routes on its multiple hosts. In the vectorial transmission layer, vectors are contaminated after interacting with infected hosts and transmit the parasite while interacting with non-infected hosts. In the trophic transmission layer hosts acquire the parasite after feeding on infected vector or host. Preliminary studies (Kribs-Zaleta 2006, Kribs-Zaleta 2010, Pelosse & Kribs-Zaleta 2012) used mean-field approaches to model *T. cruzi* transmission among its main sylvatic hosts and vectors. Their results indicate that in a fully connected scenario with no explicit spatial structure, vectorial and oral transmission effects are additive in maintaining and furthering the spread of the infection (Kribs-Zaleta 2006). With the multiplex framework we aim to understand the effect of multiplex topology on spreading dynamics. We explored the relative importance of each transmission route and the coupled effects of both mechanisms for parasite transmission. We also evaluated the effect of host diversity on infection spread by comparing the results of the model with a simple food-web structure with the one derived from empirical data.

Methods

Ecological multiplex network construction

A multiplex network consists of nodes of s types (species), where different types of interaction between the nodes are represented in M network layers ($M = 3$ in this study). In our set-up, node type represents species and nodes represent populations of those species. In our numerical simulations, we consider three kinds of ecosystem layers:

1. **trophic** layer: links represent trophic interactions, directed from populations of prey species to populations of their predators, according to the interactions in the food-web.

2. **vectorial** layer: undirected links represent interaction between populations of the vector species and its hosts.
3. **contact** layer: undirected links represent contacts between populations of any species, given by the spatial distance between two nodes as in a Random Geometric Graph (RGG), which is constructed by randomly placing the populations in the metric space and connecting two nodes by a link if their distance is in a given range defined by the neighborhood radius, R. (Barthélemy 2011). All interactions are allowed on this level.

The construction process for a multiplex network (Figure 1) with N nodes, radius R and species frequencies f_i ($1 \leq i \leq s$), can be summarised as follows:

- The N nodes are uniformly embedded within the unitary two dimensional space $\Omega = [0,1]^2$ with periodic boundary conditions (i.e., a toroidal space). Each node i is assigned to a given species s_i, according to the probability defined by its corresponding frequency f_i. Nodes have the same spatial positions in all the layers.
- Node i is considered in the contact layer. A directed edge (i, k) is created with all the nodes k in such layer where toroidal distance from i is $d_{ik} < R$. The edge (i, k) is created on the other multiplex layers where the interspecific interaction (s_i, s_k) is allowed.
- The connectivity process in the previous step is repeated for all nodes i and j.

In this study we consider networks formed by N = 10,000 populations (nodes) and radius of interaction ρ = 0.056. The radius of interaction has been tuned according to our node density in order to obtain connected networks, in which there are direct or indirect paths connecting any pair of nodes in the network. Therefore, we avoided limitations to parasite spread due to complete isolation of populations. We study two multiplex networks: the three-species model with s = 3 species and the Canastra food web with s = 20. In the reference model, we have three species type: predators, preys and vectors. The frequency of vectors, f_v, is a variable of the model. The other species frequencies were defined as $2f_{predator} = f_{prey}$ and under the constraint that $\sum_{i=1}^{s} f_i = 1$). In the Canastra foodweb we kept the same proportions as in the three species model ($2f_{predator} = f_{prey}$), with the constraint that all the different species within predators and preys have equal frequencies. Connectivity patterns for both models are

represented as Boolean matrices (Appendix). For the definitions of the other multiplex metrics, we refer the reader to Appendix.

Parasite transmission dynamics

We describe the parasite transmission dynamics on the ecological multiplex network. A node, *i.e.* a population of a given species type, can be infected or susceptible and its state is the same in all layers. We start the simulation by infecting all the nodes in a random circle of radius $r_0 = 0.03$, that is, $\pi N r_0^2 \sim 28.2$ nodes are infected at the beginning, on average. Subsequently, the parasite spreading evolves as follows:

1. a random node i is chosen together with one of its adjacent neighbours j in the contact layer within the radius R considered.
2. the vectorial layer is chosen to be considered in the infection transmission for this step with probability p_v, which is a measure of the vectorial layer importance. Step 3 is then performed when the vectorial layer is chosen. Otherwise, Step 4 takes place.
3. (vectorial layer infection transmission) if node i is infected and the edge (i, j) exists in the vectorial layer, node j becomes infected as well.
4. (trophic layer infection transmission) if node i is infected and the edge (i, j) exists in the trophic layer, node j becomes infected as well.
5. Steps 1-4 are repeated until the maximum number of time steps, t, is reached.

The process in stages 1-4 is repeated N (the number of nodes of the network) times for each time step. Each population is randomly chosen each time step and at the end of the transmission process every node is chosen once, on average.

Immunisation

In an immunisation simulation we study the dynamics of parasite spreading when predator or prey species have been immunised. Each node in the network has an immune attribute that is set to true if a node is chosen to be immune. An immune node is not susceptible to the parasite, by any possible route of transmission. The number of immune nodes is determined per species by specifying the probability of immunisation p_i for each species i. To perform the immunisation, members of the species i outside of the initial infection radius r_0 are chosen randomly with probability p_i and are set to be immune.

We consider two immunisation scenarios to investigate the relative role that different trophic levels had in spreading the parasite. In the first scenario only prey species are immunised and in the second scenario only predator species are immunised. For simplicity, the p_i values for all prey and predator species are set uniformly. For instance, in prey-only immunisation, where prey species are sequentially indexed by 1, 2, ..., s, we set $p_1 = p_2 = ... = p_s$.

Canastra food web

We used data from an epidemiological study of *T. cruzi* infection in wild hosts in Southeast Brazil (Rocha et al. 2013a) to estimate the trophic and vectorial networks. For the trophic layer, we built a qualitative potential food web based on the diet of the animals (Bueno et al. 2003, Ramos 2007, Cavalcanti 2010, Amboni 2007, Carvalho Neto & Santos 2012, Reis et al. 2011). As there was no species-level classification of the biological vectors present in the area, we considered the vectors as one single species. We used species prevalence – measured through positive parasitological diagnostics – to estimate the possible interactions in the vectorial layer (Rocha et al. 2013a). We assumed that positive parasitological diagnostics for *T. cruzi* could be used as a proxy vectorial transmission, since only the individuals with positive parasitaemia are able to transmit the parasite (Jansen et al. 2015). The Canastra multiplex has a total of 20 species: 7 predators, 12 prey and 1 vector (Supplementary Information).

Results

Our results focus on: (i) highlighting the topological features of our spatial multiplex through a multiplex cartography (Battiston et al. 2014), (ii) investigating parasite spreading across the multi-layered structure at different values for the frequency of vectors (f_v) and importance of vectorial transmission (p_v), and (iii) quantifying the biological role of different species in the parasite spreading by means of immunisation simulation experiments. The measures adopted to describe the transmission process are defined in Appendix. We first report the results concerning the three-species reference model followed by the Canastra multiplex, respectively.

Three-species reference system

The three-species reference model consists of the simplest epidemiological scenario for the multiplex transmission. It is based on a simple trophic chain in which vectors are consumed by preys and preys are consumed by predators. In the vectorial layer the vector contaminate both preys and predators (Figure 1).

The multiplex cartographies highlight different roles played by each species at different vector frequencies f_v (Figures 2a-d). When vectors are rare in the system ($f_v = 0.01$, Figure 2a), both predators and preys are relatively poorly connected (*i.e.* their standardised overlapping degree is $z < 2$, see Appendix). Also, predators focus most of their links in the trophic layer, thus displaying participation coefficients (Appendix) lower than 2/3. Vectors are hubs ($z > 2$) and they distribute their links uniformly between both trophic and vectorial layers. These vectors are *truly multiplex* nodes with a participation coefficient higher than 2/3. This is because most of the multiplex is composed of prey and predator populations linked to vector populations on both layers. When the frequency f_v of vector nodes increases between 0.1 (Figure 2b) and 0.25 (Figure 2c), vectors still participate uniformly on both layers but their average node degree decreases. Similar behaviour is reported when $f_v = 0.5$ (plot not shown). Increasing the vector frequency up to $f_v = 0.75$ leads to a system where vectors are predominant but each species plays a different role (Figure 2d): (i) preys receive links from vectors on both trophic and vectorial layers and become truly multiplex hubs, (ii) predators are less connected and tend to organise their connections across a wider range of participation coefficients (they are not connected to vectors on the trophic layer), (iii) vectors tend to spread their connections across both layers similarly but are also less connected compared to predators (since vectors cannot interact with each other).

We delved further into the interplay between vector frequency and parasite spreading by computing the *global infection time*, *i.e.* the time at which the infection spreads across the whole connected component of the multiplex (Appendix). Investigating the infection time at different values of vectorial layer importance p_v reveals an interesting interplay between trophic and vectorial routes of infection transmission. As reported in Figure 3, when vector frequency is low ($f_v = 0.1$), infection transmission across the multiplex is minimum for p_v different from either 0 or 1, *i.e.* there is an optimal combination of both trophic and vectorial mechanisms for infecting the same network within the shortest infection time. However, the vector frequency alters such behaviour: when $f_v = 0.25$, 0.5 or 0.75 the infection time

decreases monotonically with the vectorial layer importance p_v. This might be a consequence of the dominant role vectors play in the vectorial layer. Interestingly, increasing the vector frequency from $f_v = 0.1$ to 0.25 or even 0.5 leads to an overall decrease of the infection times, depending on p_v. However, when vector frequency raises up to $f_v = 0.75$, the trophic layer becomes ineffective in infecting the multitude of vector populations in the system, since they can be infected only in vectorial layer. This explains the overall increase in the infection times, reported in the left plot of Figure 3. However, at higher vector frequencies, prey and predator populations become easily infected through vectorial transmission and this explains the monotonically decreasing behaviour of the infection times against p_v.

Analysing the speed at which the parasite spreads across the multiplex structure reveals interesting patterns. As reported in Figure 3, the parasite transmission initially accelerates within the system (when $t < 100$) but only if the vectorial layer importance $p_v > 0$. A power-law fitting procedure retrieved a scaling exponent $\Delta R(t) \propto t^{\alpha}$ with $\alpha \approx 1$ when $t \in$ [50, 100]. If transmission spreads only in the trophic layer it leads to a qualitatively different behaviour, and it becomes increasingly difficult to infect more populations over time (*i.e.* the parasite spreading rate decreases almost monotonically). Behaviour consistent with that (Figure 3; $f_v = 0.75$, $p_v = 0.5$) was also observed for all the other parameter values reported in other plots. The only difference observed was in the order of the peaks of parasite spreading rate, which are reached according to the p_v value (*i.e.* the higher p_v the sooner the peak was reached), only when $f_v > 0.2$.

We also investigated the infection dynamics for very small values of vector frequencies (Figure 4). We found that our model displayed a threshold in the emergence of global epidemics around the critical value $f_v = 0.02$. Very small variations in the abundance of vector populations within the simulated ecosystem leads to dramatic changes in the ratio of infected populations after a suitably long relaxation time of 10,000 iterations (Figure 4). When $f_v < 0.02$, the system is disconnected and the parasite cannot spread over the multiplex structure: even after a long time under the SI dynamics, there is no pandemic registered. Our simulations show that the vectorial layer importance slightly shifts the critical threshold of the phase transition, which occurs across all the different values of p_v (for $p_v = 0$ or $p_v = 1$ plots not reported for clarity). This phase transition marks the beginning of a distinct "phase" of the model ($f_v > 0.02$), for which the parasite can percolate throughout the whole system even when vector frequencies are low.

As indicated by the grey area in Figure 4, the mean infection radius $\langle r \rangle$ at the final state undergoes a phase transition around $f_v = 0.02$. However, $\langle r \rangle$ converges to its maximum value $\langle r \rangle_{\max}$ at a faster rate compared to the ratio of infected population. For instance, when $f_v = 0.04 \approx 70\%$ of the populations in the system are infected while their average distance from the centre is close to the maximum value (see the dashed black line in Figure 4). In other words, even when a considerable proportion of populations are not infected yet, the parasite spreading can still be considered "global". This happens because the infected populations are uniformly scattered across the whole system, which is possibly a consequence of the direct transmission on the trophic layer.

Infection times and parasite ratio increases provide global and time-detailed information about the parasite spreading dynamics on the multiplex structure. In order to quantify the role played by each species in the parasite spreading we also analysed the above infection metrics in immunisation scenarios, where a fraction of populations of a given species was immune to the parasite. Given our previous findings of different species playing different roles within the network cartography (Figure 2d), we focused on the immunisation scenario for a fixed vector frequency $f_v = 0.75$. In fact, it is when $f_v = 0.75$ that preys, predators and vectors occupy different regions in the multiplex cartography. In Figure 5 we report the global infection times when the same absolute number of predators or preys populations are immunised (notice that predators are half as frequent as preys in our model). Our results show that immunising preys over predators leads to a greater increase in the system infection times for all values of vectorial layer importance p_v. The better performance of immunising preys over predators is also reflected in the increase of the parasite ratio $\Delta R(t)$ (Figure 5). In fact, immunising preys not only makes the system experience a pandemic at a later stage but it also significantly slows down the parasite spread in the first accelerating phase (*i.e.*, it lowers the $\Delta R(t)$ when $t < 140$). Even though slowing down the parasite transmission and reaching a pandemic at a later stage might sound equivalent, a closer look at the parasite ratio increase reveals that in the predator immunisation scenario there is a higher diffusion speed in the decelerating infection phase, $t > 140$ (Figure 5). Because of this behaviour, we report on both patterns. This difference could be attributed to the different role played by preys and predators on the trophic layer, *i.e.*, preys are infected before predators because they are directly infected by vectors when feeding on an infected vector. Further numerical experiments indicate that this is not the case: immunisation experiments performed at vector frequency $f_v = 0.25$ show that immunising either 50% of

predators or 25% of prey gives statistically equivalent results in terms of both the parasite spreading times and the propagation rates. Since the "who-eats-who" order is the same in both $f_v = 0.25$ and $f_v = 0.75$ immunisation scenarios, the relative difference in immunisation performances has to be attributed to the role played by each species within the global network topology. Immunising preys is different from immunising predators only if nodes relative to such two species have different roles within the multiplex, *i.e.*, their cartographies are distinct. This computational evidence points to the meaningfulness of the concept of network cartography for the parasite spreading dynamics because at higher vector frequencies $f_v = 0.75$ preys become truly multiplex hub nodes.

Canastra food web

The cartographies reported in Figure 2 (e-h) represent snapshots of the Canastra multiplex with increasing frequencies of vectors. When vectors are in very low frequency ($f_v = 0.01$), predators and preys occupy the same regions of the cartography as in the three-species multiplex (Figures 2a and 2e). With higher frequencies of vectors (Figure 2f, 2g and 2h) there is one predator species that displays a wide variation in the participation coefficient, while the participation coefficients of the other predators is zero. This happened because the Canastra multiplex has one predator species that can be contaminated by vectorial transmission, while the others have links only in the trophic layer. Similar case occurs with the preys, since only half of them have connections on the vectorial layer. Analogously to the three-species system, increasing the frequency of vectors leads to scenarios where some predators and preys display a wide range of participation coefficients. However, at both $f_v = 0.1$ and $f_v = 0.25$ predators are more connected than preys in the Canastra multiplex since they display a higher average overlapping degree. This occurs because predators are at higher trophic levels than preys and thus receive more connections in the trophic layer. Therefore, for values as low as $f_v = 0.1$ the species show varied patterns in the cartography. At $f_v = 0.25$, preys tend to show an increased participation in the multiplex as a sign of increased connectivity in the vectorial layer. When vector populations are highly frequent in the system, $f_v = 0.75$, the cartography reveals some extreme patterns: the preys that have interactions with vectors in the vectorial layer display participation coefficient close to 1 (Figure 2h). For $f_v = 0.75$ there is also a gap among predators that interact with vectors and those that do not, which was absent in the three-species multiplex (Figure 2h).

The time required to infect almost all the populations in Canastra is minimised when the vectorial layer importance p_v is around 0.6, at vector frequencies f_v = 0.1, 0.25 and 0.5 (Figure 6). This decreased infection time indicates that the parasite spreading is optimised when vectors are not so frequent and vectorial and trophic transmission mechanisms have similar importance. As a comparison, the three-species multiplex exhibited a minimum global infection time only when f_v = 0.1, for p_v different from either 0 or 1. In the extreme scenario f_v = 0.75 the infection times decreased monotonically and transmission occurs only via the vectorial layer. In fact, when the system includes many vectors, the bidirectional links in the vectorial layer allow for faster parasite spreading through the infected predator and prey populations. Pandemics are reached faster when the importance of vectorial layer transmission is increased (Figure 6). Despite Canastra has a higher connectance in the trophic layer, the parasite ratio increases behave similarly to the three-species system. The parasite spreading propagates much slower in the trophic layer alone than on the full multiplex structure, indicating an additive effect in the spread of the infection across trophic and vectorial interactions. For $p_v > 0$ the following slow-down phase does not behave independently of p_v, after each infection peak has been reached (see left plot in Figure 6). Therefore, such peaks cannot be considered good proxies of the infection times in the Canastra multiplex. When the spreading desaceleration occurs in different times, it sums up differently to the peak times, thus establishing global infection times that are not straightforwardly related to the peak times. For instance, the peak for p_v = 0.8 is reached sooner than for the p_v = 0.6 but the desaceleration phase takes longer for p_v = 0.8 than for the p_v = 0.6. Because of that, the p_v = 0.8 has a higher global infection time compared to the p_v = 0.6.

The Canastra multiplex also displayed a phase transition in the emergence of a global epidemic, similarly to what happened for the three-species multiplex. However, the different topology of trophic and vectorial layers brought to a slight increase in the critical vector frequency value, from 0.02 (three-species multiplex) to 0.04.

Unlike the three-species multiplex, Canastra displayed different cartographies only at low vector frequencies. Therefore, we investigated immunisation scenarios at f_v = 0.1 and f_v = 0.25. The results for f_v = 0.1 are reported in Figure 7 and are analogous to the f_v = 0.25 case (plots not shown for brevity). Contrary to the three-species multiplex, immunising preys does not always hamper the parasite spreading more than immunising predators. From the cartography (Figure 2f) one would expect predators to play a pivotal role in spreading the

parasite, given their higher connectivity, on average. However, in the same cartography preys display a slightly higher average participation and hence could also play a central role in the epidemics. Therefore, in contrast to the three-species multiplex, it is not possible to make predictions based on the cartography alone. Our immunisation simulations reveal the presence of two scenarios: when the parasite spreads preferentially across the trophic layer, then immunising predators over preys significantly increases the infection times (see the right plot in Figure 7) and slows down the parasite transmission (see the left plot in Figure 7). This is mainly due to the fact that predators are hubs in the trophic layer and hence show a higher overlapping degree in the cartography. However, when the vectorial layer importance p_v increases above 0.3, then immunising preys becomes the most effective immunisation strategy, since the vectors contaminate mostly preys in Canastra multiplex. Having a higher vectorial layer importance means that the multiplex structure becomes predominant and the populations that participate the most in the multiplex, such as preys, can promote the parasite spread.

Discussion

Recently, network scientists have addressed the multiplex aspects of real-world systems such as ecological and epidemiological systems (Boccaletti et al. 2014). Multiplex networks were used in ecological systems to approach different interaction types (Melián et al. 2009, Fontaine et al. 2011, Shai et al. 2015) and levels of organization (Belgrano 2005, Scotti et al. 2013). In epidemiological systems multiplexes were used to describe parasite spread with Susceptible-Infected-susceptible dynamics (Saumell-Mendiola et al. 2012, Sahneh et al. 2013, Granell et al. 2013, Sanz et al. 2014), Susceptible-Infected-Recovered dynamics (Dickison et al. 2012, Marceau et al. 2011, Buono et al. 2014), and multiple types of interactions between random layers (Zhao et al. 2014, Cozzo et al. 2013, Salehi et al. 2014). The modelling of multi-host parasites that are transmitted through multiple mechanisms in the ecosystem can be improved by applying the multiplex framework. We used multiplex networks to study both a simple predator-prey-vector system as a reference case, and to empirical data from host communities of *T. cruzi* in natural habitat (Canastra). Our three-species system as well as our empirical-based system showed that the ecological aspects of vectors, hosts and parasites might be mapped on the multiplex cartography. Considering the node and link heterogeneities in a spatial context allowed us to identify percolation thresholds for parasite spreading. This is particularly interesting because the

Susceptible-Infected dynamics in homogeneous hosts always leads to epidemic waves. In addition, we found that multiplex cartography had important implications in parasite spreading dynamics and that parasite transmission depends on: (i) the relative importance of distinct transmission mechanisms, (ii) the role species play on overall multiplex structure and (iii) the relative frequency of species in the system.

Multiplex cartography (Battiston et al. 2014) provided information on the biological role that different species plays in parasite spreading. Because species interactions were spatially embedded, the resulting multiplex structure was determined by the relative frequency of each species and the interactions they have in both the trophic and vectorial layers. We found the structural role one species plays in parasite spreading was possible to be achieved by the multiplex cartography in both the reference and empirical systems. In the three-species multiplex, a higher frequency of vectors (f_v > 50% of the total population) increased prey connectivity and therefore their participation in multiplex topology. We found different results when considering a more realistic ecological scenario. In Canastra, predators dominated the multiplex because of their higher connectance and higher average overlapping degree. Therefore, multiplex structure not only affected species interaction patterns in the community but also had important consequences for transmission dynamics.

In vector-borne diseases, densities of hosts and vectors as well as the ratio of their densities, have strong implications for parasite transmission (Ross & Thomson 1911, Velasco-Hernandez 1994, Kribs-Zaleta 2010, Pelosse & Kribs-Zaleta 2012). The three-species multiplex showed that higher vector frequency increased vector importance in the vectorial layer, which enhanced preys and predators infection and parasite transmission. This relationship explains why infection times decreased monotonically with increased importance of the vectorial layer, which was more strongly with an increased relative frequency of vectors in the community (Figure 3). On the other hand, if vector frequency is low and parasite spreads only in the trophic layer, it becomes increasingly difficult to infect more populations over time. In this situation the fastest global infection was achieved when both mechanisms of transmission were important for parasite spreading (there was a minimum in the infection time around p_v = 0.6). Moreover, in the Canastra multiplex, we observed an analogous minimum even with higher vector frequencies (Figure 6). This suggests that global infection time is minimised when both mechanisms of transmission have similar importance in more complex ecological scenarios.

Percolation thresholds are spatially explicit tipping points that indicate long-range interactions within a given system (Davis et al. 2008). If a network does not allow for long-range interactions among nodes, *i.e.* if the network is not strongly connected, then the infection will not transmit to the whole system. In our case the connectivity of the multiplex was crucially affected by the frequency of species. For very small frequency of vectors f_v, our model showed a percolation threshold in both the three-species and the Canastra multiplex. The presence of such *"phase transition"* in a simple SI dynamics for a non-zero value of f_v is mainly related to the spatial structure and to directed trophic interactions in the multiplex. In the three-species model the parasite can percolate through the whole system only if $f_v > 0.02$, while in Canastra the critical vector frequency was found to be around $f_v = 0.04$. We conjecture that this increase might be due to a higher diversity of potential hosts: with more species available there is an increased chance that vectors will interact with animals that do not become infected with the parasite. Interestingly, our theoretically computed frequencies agree with previous findings that even a small frequency of vectors in the ecosystem is sufficient to maintain Chagas disease in a human population (Reithinger et al. 2009).

Immunising preys in the reference model dramatically increased global infection time and the rate of disease spreading in the populations. However, immunising preys over predators resulted in different infection times only if these species had distinct cartographies. This result points to the meaningfulness of the network cartography for understanding the parasite spreading dynamics. The multiplex cartography pointed out that preys play an important role in the three-species model and thus could be a better target for immunisation. The immunisation simulations confirmed that imunising preys could hamper the parasite spread in the three-species case. In the Canastra multiplex, predators were the species that attained most of their connections in the multiplex and thus had a higher importance in the cartography. This higher importance in the multiplex suggests that predators are acting as a sink for the parasite and reducing overall transmission in Canastra. Empirical studies had already pointed out the potential importance of predators as parasite bio-accumulators (Rocha et al. 2013b, Jansen et al. 2015). This is mainly due to the fact that predators are hubs in the trophic layer and hence show a higher overlapping degree in the cartography. However, preys also displayed a slightly higher average participation in the Canastra cartography and hence could also play a central role in the epidemics. In fact, when the vectorial layer importance p_v is above 0.3, immunising preys becomes the most effective immunisation strategy. This happened because vectors contaminate mostly preys in the Canastra multiplex. Again, the

roles played by each species in the multiplex cartography depended on the frequency of vectors and is related to their importance for parasite spreading.

For a given vector frequency, the lowest infection times was registered when the parasite spreads on both layers at the same time (*i.e.* for intermediate values of p_v) in both three-species and Canastra multiplex. Therefore, our theoretical network model indicates that vectorial and trophic mechanisms of transmission are additive in sustaining the spread of multi-host parasites such as *T. cruzi*, further agreeing with previous studies (Kribs-Zaleta 2006). In random multiplexes (Saumell-Mendiola et al. 2012) the epidemic process also depends on the strength and nature of the coupling between the layers. This previous study (Saumell-Mendiola et al. 2012) showed that the global epidemic threshold considering all the multiplex layers turned out to be smaller than the epidemic thresholds of two layers separately. Our results indicate that the spread of a parasite that uses multiple mechanisms for transmission may have a higher efficiency then one single mechanism, even when the transmission layers are highly structured and differ in their topologies. The multi-layered transmission, which is observed in many parasites with complex life cycles and multiple mechanisms of infection, seems to be a very efficient strategy for spreading in communities of multiple hosts.

It has to be underlined that the main aim of our multiplex model is not to provide a realistic mechanism for the spreading dynamics of *T. cruzi* in wild hosts. Instead, our approach aims at providing a comprehensive framework for investigating the spreading of multi-host parasites across different transmission mechanisms. Additional information should be taken into account if one would want to study the dynamics of *T. cruzi* in wild hosts and Chagas disease epidemiology. For instance, it is known that the stercorarian transmission results in a much higher probability of parasite transmission from host to vector than from vector to host (Rabinovich et al. 1990). A more realistic model should include these differences. In addition, host physiological and ecological characteristics influence their probability to transmit *T. cruzi*. A higher proportion of insects in host diets increase host probability of infection (Roellig et al. 2009, Rabinovich et al. 2011, Rocha et al. 2013b). Finally, host species that share ecological habitat with vector species are more likely to be exposed to the infection (Jansen et al. 2015).

Many zoonoses, which are infections naturally transmitted between vertebrate animals and humans, have multiple hosts and mechanisms of transmission. Examples of

zoonoses transmitted to humans by arthropod vectors include Malaria, Leishmaniasis, Chagas disease, West Nile virus, plague and Lyme disease (Schmidt & Ostfeld 2001). The multiplex framework presented here could improve our understanding of the epidemiology and evolution of these parasites and help us elaborate more efficient control strategies for reducing disease insidence in humans. Additional layers, such as network of human interactions with its socio-ecological characteristics, could be included to make the model more realistic.

Acknowledgements

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Appendix

In here we report on the different measures we adopted in order to characterise both the structure of the multiplex network and the infection patterns in the parasite spreading.

Multiplex Cartography

A multiplex cartography visually represents the role played by a given node within across different layers according to its topological features (Battiston et al. 2014, Boccaletti et al. 2014). The metrics considered in a multiplex cartography are: the standardised overlapping degree z_i and the participation coefficient P_i of node i. The overlapping degree o_i is defined as (Boccaletti et al. 2014) the sum of all the degrees of node i across the M multiplex layers:

$$o_i = \sum_{\alpha=1}^{M} x_i^{(\alpha)} \tag{1}$$

where x_i is the degree of node i in the layer $\alpha = M$. The overlapping degree o_i represents a proxy of the overall local centrality that a node has within the multi-layer network. The standardised overlapping degree is thus defined as $z_i = \frac{(o_i - \langle o_i \rangle)}{\sigma(o_i)}$. In the scientific literature (Battiston et al. 2014), it is customary to call hubs those multiplex nodes displaying a standardised overlapping degree $z_i > 2$. However, the overlapping degree provide limited information about the way connections are distributed over different layers. The distribution of the connections over the different layers can be expressed via the participation coefficient P_i of node i:

$$P_i = \frac{M}{M-1}\left[1 - \sum_{\alpha=1}^{M}\left(\frac{x_i^{(\alpha)}}{o_i}\right)^2\right] \tag{2}$$

P_i ranges between 0 (for nodes that concentrate all their connections in one level only) and 1 (for nodes that distribute connections over all the M layers uniformly). According to this

measure, it is possible to distinguish among focused nodes ($0 < P_i \leq 1/3$), mixed nodes ($1/3 < P_i \leq 2/3$) and truly multiplex nodes ($2/3 < P_i \leq 1$) (Battiston et al. 2014).

Infection Measures

On a macroscopic scale, we investigated the parasite spreading by computing the *global infection time*, defined as the time step at which the giant weakly connected component of the multiplex is infected. Alternatively, the infection time indicates the time step t_{inf} at which the disease infects the most nodes within the network. If $R(t) = N_{inf}(t)/N$ is the ratio of infected populations/nodes at time t, then $\mathrm{Max}_t\,(R(t)) = R(t_{inf})$.

Infection times represent a global, macroscopic statistics of the parasite spreading. Nonetheless, it is interesting to analyse the transmission process via microscopic statistics, such as the *parasite ratio increases* $\Delta R(t) := R(t+1) - R(t)$, defined as the time-step increase of the ratio of infected populations within the system. The $\Delta R(t)$ is a measure for the rate at which the parasite is spreading within the multiplex.

In order to capture the spatial features of our multiplex model we measured also the *mean radius of infected populations* $\langle r \rangle$ defined as the average distance of the infected nodes from the centre of the embedding square $\Omega := [0,1]^2$ with periodic boundary conditions. Given our hypothesis of uniform spreading of species populations within Ω, it is relatively straightforward to compute an upper bound $\langle r \rangle^*$ for $\langle r \rangle$ as:

$$\langle r \rangle^* = \int\int_0^1 \sqrt{(x-\frac{1}{2})^2 + (y-\frac{1}{2})^2}\, dxdy \approx 0.3826 \qquad (3)$$

Figures

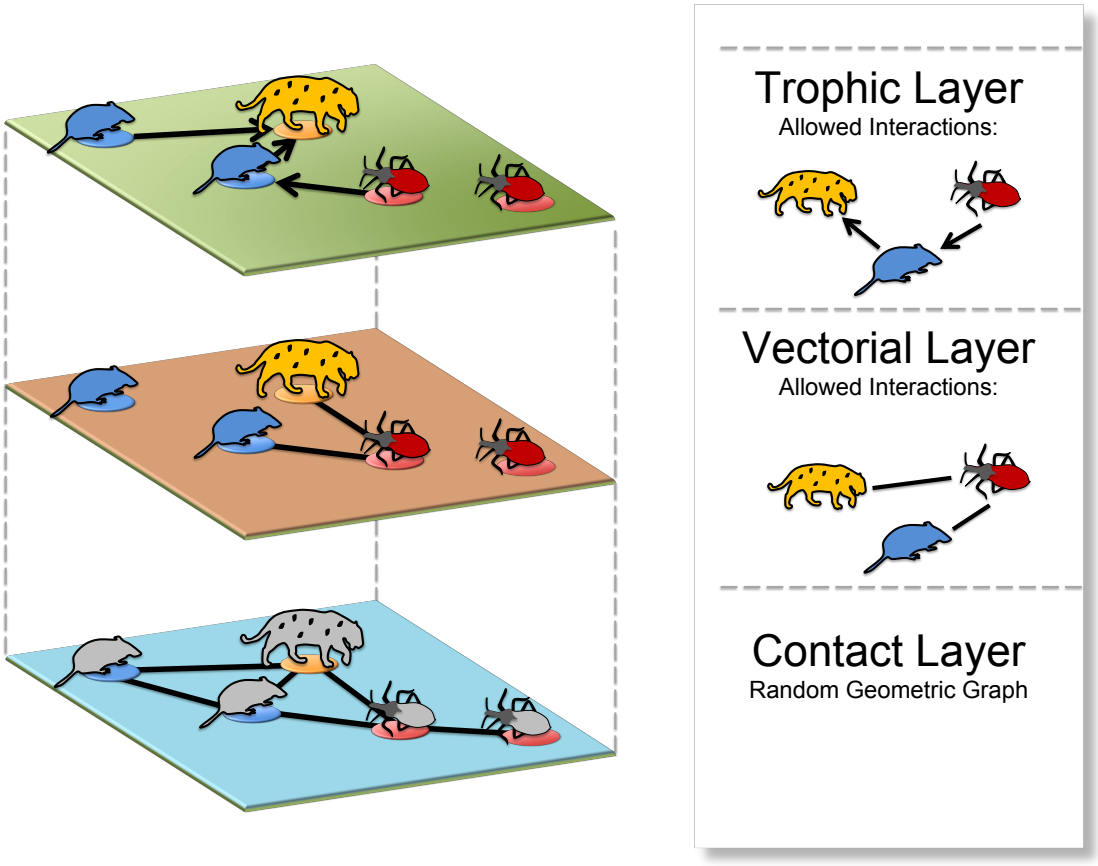

Figure 1: Visual representation of our model over the three layers: a trophic layer, a vectorial layer and their underlying contact layer. Nodes are relative to the three-species example and they are drawn according to their "identities", e.g. "predator", "prey" and "vector". According to such identities, the trophic and vectorial layers allow only for specific interactions to be present within the system. For instance, the allowed interactions in the three-species model are reported on the right. The parasite can spread on both such layers. When a node gets infected in one layer it gets infected on all the others as well.

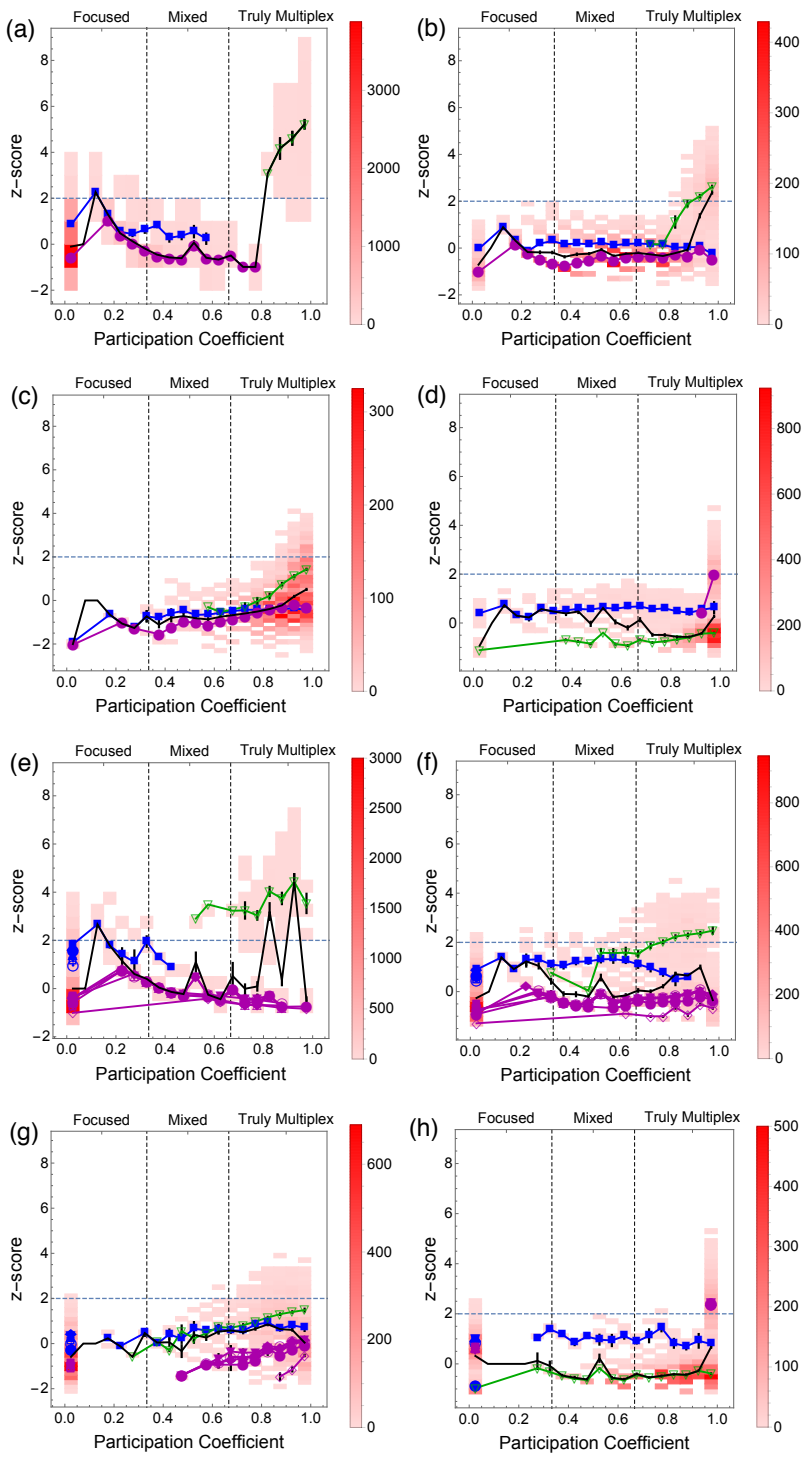

Figure 2: Cartographies as histogram densities for the three-species multiplex (a,b,c,d) and Canastra (e,f,g,h), for vector frequency f_v = 0.01, 0.1, 0.25 and 0.75, respectively. Each histogram tile is colour-coded according to the number of points falling within its ranges: more coloured tiles have the most nodes in them. The black line highlights average values while coloured lines identify individual species: blue squares represent predators, purple circles represent preys and green triangles represent vectors. Notice that in (e,f,g,h) predators and preys are represented with the same colours but different shapes represent different species.

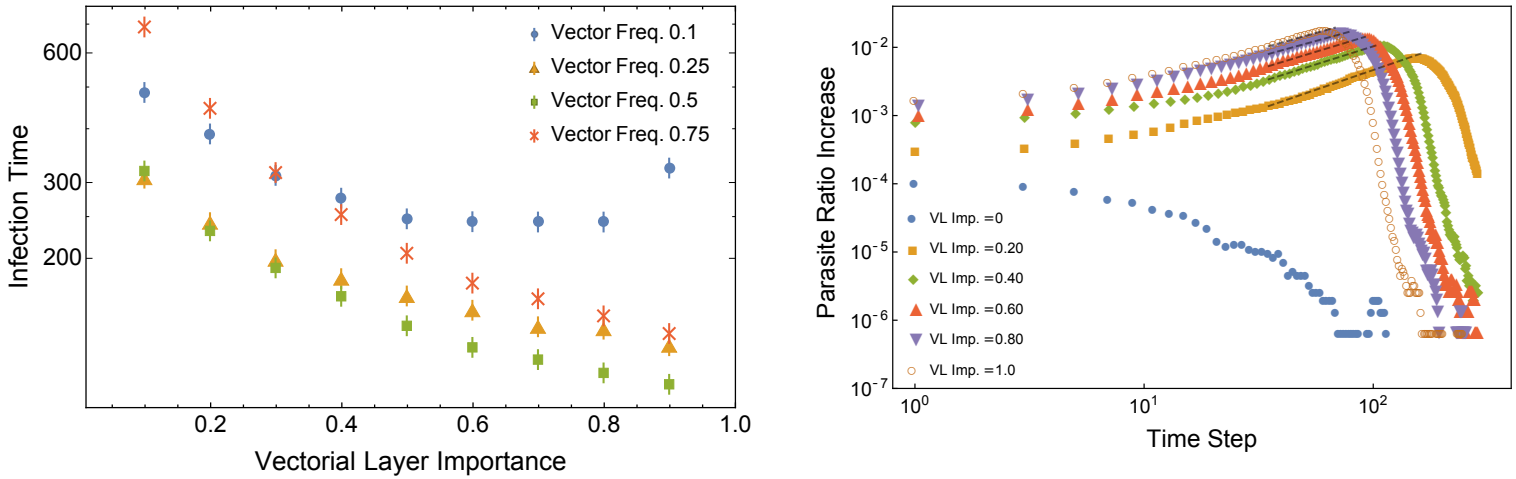

Figure 3: Left: global infection times versus vectorial layer importance p_v for different values of vector frequency f_v = 0.1, 0.25, 0.5 and 0.75 for the three-species multiplex. Right: global infection rate over time for f_v = 0.75 and p_v = 0.5, expressing the speed of parasite spreading within the multiplex. A qualitatively similar behaviour was observed also for other parameter combinations. Results in both plots are averaged over 10 repetitions of the three-species model. The dashed straight lines for time $t \in [40,140]$ are relative to a power-law fitting with exponent $\alpha \approx 1$.

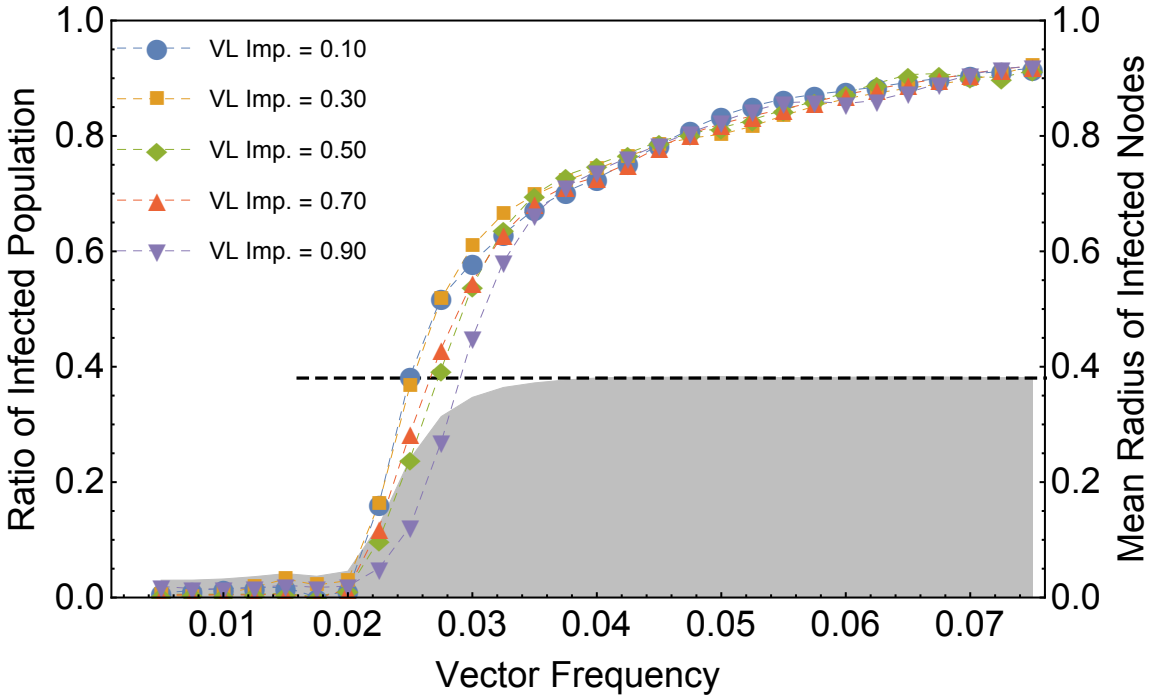

Figure 4: Ratio of global infected populations after 10,000 time steps, sampled at different values of p_v, against vector frequency f_v. When vectors are rare in the system, the model displays a phase transition in the presence/absence of a global pandemic. The critical threshold is localised around $f_v \approx 0.02$, for all the values of the vectorial layer importance. The grey shape represents the mean infected radius and it is averaged over different p_v values. When $f_v > 0.02$ the infection radius saturates faster than the global percentage of infected populations. All curves are averaged over 10 model iterations.

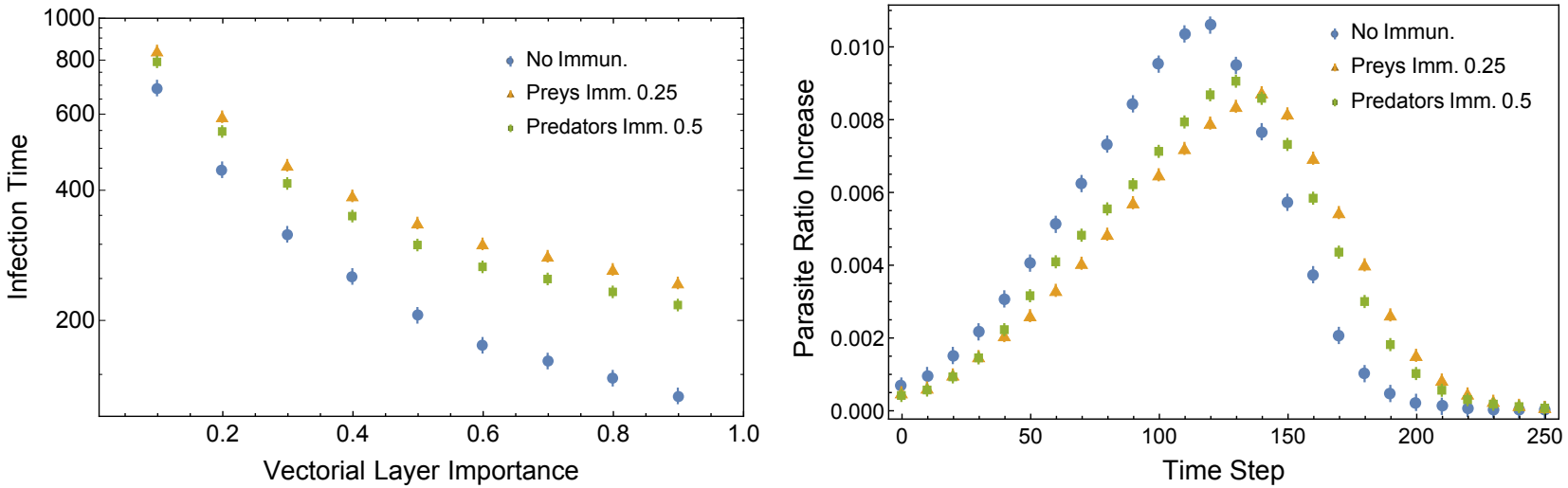

Figure 5: global infection time versus vectorial layer importance p_v for different immunisation experiments with f_v =0.75, where either no immunisation is present (blue dots) or 25% of preys are immunised (golden triangles) or 50% of predators are immunised. Right: global infection rates for different immunisation experiments with f_v =0.75. Error bars are computed over 10 independent experiments. Immunising preys is the best choice in terms of both reducing the global infection time and slowing the infection spread over time.

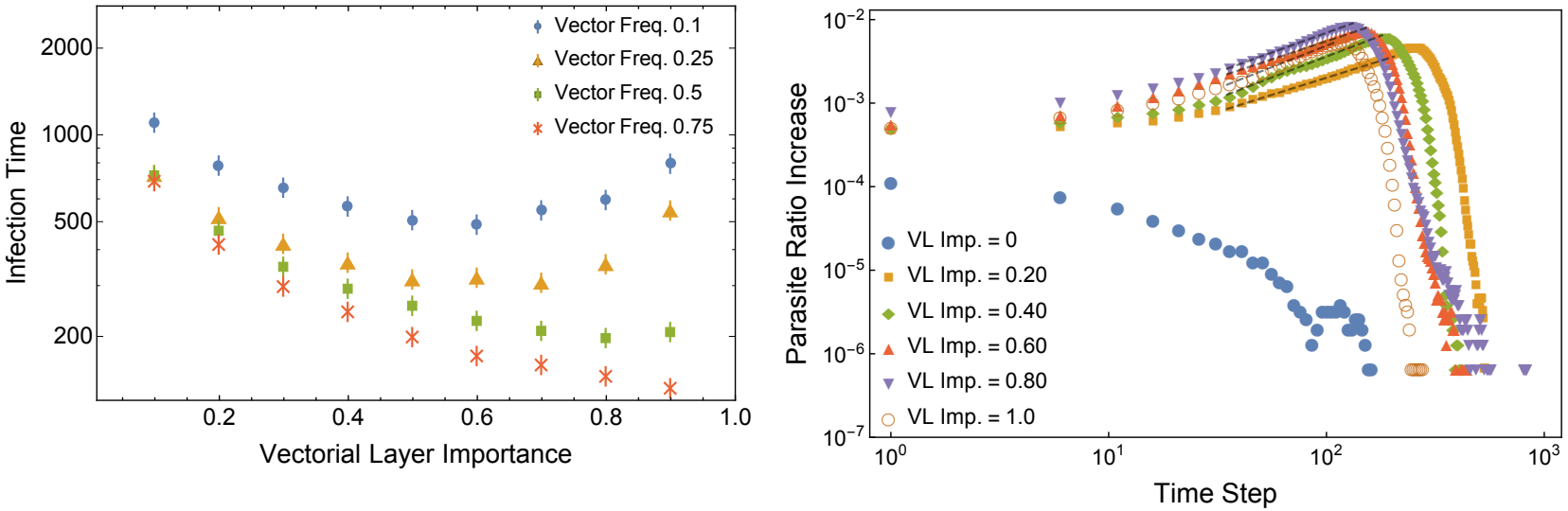

Figure 6: Left: global infection times versus vectorial layer importance p_v for different values of vector frequency f_v = 0.1, 0.25, 0.5 and 0.75 for the Canastra model. Right: global infection rate over time for f_v =0.25 and p_v =0.5, expressing the speed of parasite spreading within the multiplex. A qualitatively similar behaviour was observed also for other parameter combinations. Results in both plots are averaged over 10 repetitions of the three-species model. The dashed straight lines for time $t \in [80,300]$ are relative to a power-law fitting with exponent $\alpha \approx 1$.

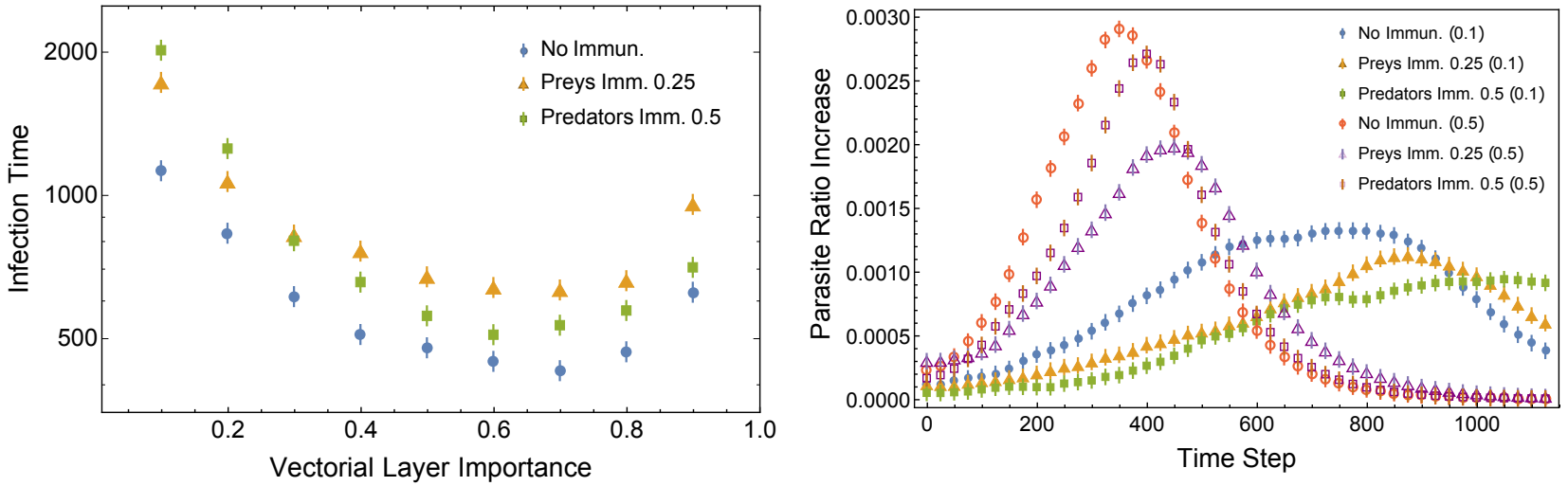

Figure 7: Left: Global infection time of the Canastra model versus vectorial layer importance p_v, for f_v =0.1 and for: (i) no immunisation present (blue dots), (ii) immunisation of 25% of prey (golden triangles) and (iii) immunisation of 50% of predators (green squares). Error bars are computed over 10 model iterations. A similar behaviour was observed also for f_v =0.25 (plots not shown for brevity). Right: global infection rates for the above immunisation experiments (i, ii, iii). When p_v =0.1, immunising prey (golden triangles) is less effective than immunising predators (green squares) in slowing down the parasite spread. The opposite scenario happens when $p_v > 0.2$. An example is reported in the figure: when $p_v = 0.5$ immunising prey (empty purple triangles) is more effective than immunising predators (empty brown squares).

Parasite spreading in spatial ecological multiplex networks

Supplementary Information

Table S1: Interactions between hosts and vectors in the Canastra multiplex trophic layer.

TROPHIC MATRIX

	CHR	LEO	CER	LYC	CON	DID	LUT	CAL	NES	MON	MAR	OXY	CER	NEL	AKM	AKO	GRA	OLI	CAL	VEC
Chrysocyon brachyurus (CHR)	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Leopardus pardalis (LEO)	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Cerdocyon thous (CER)	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Lycalopex vetulus (LYC)	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Conepatus semistriatus (CON)	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Didelphis albiventris (DID)	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Lutreolina crassicaudata (LUT)	0	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Caluromys philander (CAL)	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Nectomys squamipes (NES)	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Monodelphis sp (MON)	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Marmorsops incanus (MAR)	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Oxymycterus delator (OXY)	1	1	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0
Cerradomys subflavus (CER)	1	1	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0
Necromys lasiurus (NEL)	1	1	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0
Akodon montensis (AKM)	1	1	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0
Akodon sp (AKO)	1	1	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0
Gracilinanus agilis (GRA)	0	1	1	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Oligoryzomys spp (OLI)	1	1	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0
Calomys sp (CAL)	1	1	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0
Vector (VEC)	0	0	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	0

Table S2: Interactions between hosts and vectors in the Canastra multiplex vectorial layer.

VECTORIAL MATRIX **(only host-vector interactions are allowed)**

	VEC
Chrysocyon brachyurus (CHR)	0
Leopardus pardalis (LEO)	1
Cerdocyon thous (CER)	0
Lycalopex vetulus (LYC)	0
Conepatus semistriatus (CON)	0
Didelphis albiventris (DID)	0
Lutreolina crassicaudata (LUT)	0
Caluromys philander (CAL)	1
Nectomys squamipes (NES)	0
Monodelphis sp (MON)	0
Marmorsops incanus (MAR)	1
Oxymycterus delator (OXY)	0
Cerradomys subflavus (CER)	1
Necromys lasiurus (NEL)	0
Akodon montensis (AKM)	1
Akodon sp (AKO)	1
Gracilinanus agilis (GRA)	0
Oligoryzomys spp (OLI)	0
Calomys sp (CAL)	1
Vector (VEC)	0

Considerações Finais

Abordagens que integram teorias ecológicas e evolutivas com base em dados empíricos são fundamentais para o avanço da compreensão sobre os mecanismos subjacentes à origem, modificação e manutenção da biodiversidade. Nesta tese desenvolvi, com a ajuda de colaboradores, modelos coevolutivos buscando uma melhor compreensão sobre os mecanismos ecológicos e evolutivos responsáveis pela formação, manutenção e evolução de redes de interação formadas por espécies antagonistas. Nossos resultados indicam que a estrutura das redes de interação pode moldar as pressões seletivas recíprocas entre espécies e influenciar a coevolução. A evolução pode modificar a força da interação entre espécies e assim influenciar os padrões de interação ao longo do tempo. Considerando os modelos desenvolvidos nos três capítulo desta tese, de forma geral estruturas modulares emergiram ou foram mantidas em decorrência do processo coevolutivo. Já estruturas aninhadas promoveram maior flutuação nas forças de interação entre espécies, o que favoreceu uma maior variabilidade temporal nas características e/ou abundâncias das espécies.

Os três capítulos desta tese abordaram diferentes aspectos do processo coevolutivo entre exploradores e vítimas em comunidades ricas em espécies. No capítulo 1 encontramos que a coevolução das características de ataque e defesa depende da intensidade da seleção exercida pelos parceiros. De forma geral, quando a seleção exercida pelos parceiros foi mais intensa que a seleção ambiental, as espécies apresentaram maior quantidade de mudança fenotípica. A evolução dos fenótipos tendeu a ser direcional, gerando corridas armamentistas, quando a intensidade da seleção foi maior para as vítimas do que para os exploradores. Por outro lado, a evolução fenotípica tendeu a ser flutuante quando a intensidade da seleção foi maior para os exploradores do que para as vítimas. Portanto, a alternância coevolutiva foi um possível resultado da dinâmica coevolutiva quando houve assimetria da seleção entre vítimas e exploradores. Encontramos também que a estrutura das redes de interação teve um importante efeito sobre a dinâmica coevolutiva das espécies, principalmente quando a força da seleção foi assimétrica entre exploradores e vítimas. Espécies conectadas a um grande número de parceiros ou a parceiros fenotipicamente diversos tenderam a responder mais intensamente a mudanças evolutivas que ocorreram em um subconjunto das espécies com as quais interagem, Dessa forma, o aninhamento favoreceu uma resposta evolutiva temporalmente especializada. Este mecanismo facilitou a evolução de resistência (ou escape

da interação) em vítimas atacadas por exploradores generalistas. Portanto, as interações entre espécies com fraco acoplamento fenotípico foram acomodadas em estruturas aninhadas, as quais tenderam a ser menos estáveis no tempo, podendo levar ao aparecimento de estruturas modulares. A coevolução entre espécies com forte acoplamento fenotípico formou módulos e a modularidade das redes foi mantida em decorrência do processo coevolutivo.

No capítulo 2 encontramos que regras de interação baseadas nos fenótipos das espécies reproduziram a estrutura de redes antagonistas empíricas. Os cenários nos quais as espécies apresentavam uma alta sensibilidade às diferenças entre os fenótipos foram os que melhor reproduziram a estrutura das redes empíricas. No entanto, não encontramos diferenças nas estruturas de redes previstas pelos mecanismos de barreira ou acoplamento fenotípico. Ambos os mecanismos de interação tenderam a subestimar o aninhamento das redes empíricas e superestimar a modularidade e não foi possível evidenciar uma relação direta entre o mecanismo coevolutivo e a estrutura das redes antagonistas geradas. Apesar de gerarem redes equivalentes do ponto de vista estrutural, as dinâmicas coevolutivas resultantes diferiram entre os dois mecanismos de interação. A coevolução das características de ataque e defesa apresentou maior flutuação quando as interações foram mediadas por acoplamento fenotípico, enquanto que o mecanismo de barreira resultou em maior intensificação das características. A evolução das características das espécies modificou as probabilidades de interação, o que mudou a estrutura das interações no tempo. Ambos os mecanismos previram uma alta taxa de mudança das interações no tempo, mas a dinâmica temporal das interações não influenciou a estrutura geral (aninhamento e modularidade) das redes.

No capítulo 3 encontramos que a coevolução rápida pode resultar em uma relação desacoplada entre as dinâmicas das características fenotípicas e das densidades populacionais das espécies. Observamos uma alta variabilidade na densidade das populações e baixa variabilidade nas características das espécies quando a seleção por parceiros foi mais fraca ou igual a seleção ambiental. Forte seleção por parceiros reverteu essa relação, aumentando a variabilidade das características e diminuindo a variabilidade das densidades. Nossos resultados indicaram que uma rápida resposta evolutiva pode reduzir flutuações nas densidades, o que pode reduzir o risco de extinção devido a processos demográficos. Por isso, a evolução rápida das forças de interação entre espécies pode aumentar a estabilidade das populações a longo prazo. A estrutura das redes também influenciou a dinâmica coevolutiva e a modularidade aumentou a estabilidade demográfica enquanto o aninhamento foi responsável por uma maior flutuação nos tamanhos populacionais. A dinâmica eco-evolutiva entre exploradores e vítimas gerou uma relação conflitante entre o grau de generalismo das

espécie e seu tamanho populacional, o que resultou em uma relação negativa entre essas duas características.

Na seção de perspectivas para o estudo das dinâmicas ecológica e evolutiva em redes de interação antagonista nós utilizamos ferramentas de análise de redes múltiplas para estudar a dinâmica de transmissão de parasitas multi-hospedeiros que são transmitidos por meio de múltiplos mecanismos. Neste estudo encontramos que as características estruturais das espécies na rede múltipla podem indicar a sua importância para processos dinâmicos na rede. Observamos que o parasita se espalha mais rapidamente quando os múltiplos mecanismos de transmissão podem ocorrer com probabilidades semelhantes. A composição da comunidade de vetores e hospedeiros também influenciou a velocidade da transmissão, sendo que maior frequência relativa de vetores no ambiente aumentou a taxa de infecção em hospedeiros. A importância das espécies para a estrutura da rede múltipla, medida pela cartografia da rede, indicou o papel que elas desempenharam para a transmissão do parasita. Por último, mostramos que as espécies com maior importância na cartografia da rede são também as que seriam melhores alvos para programas de imunização de hospedeiros.

Modelos desenvolvidos nesta tese fornecem indícios sobre a importância de processos ecológicos e coevolutivos para a evolução e manutenção das redes de interação entre espécies. As hipóteses propostas abrem possibilidades para investigação mais aprofundada de diversos aspectos relacionados a coevolução entre exploradores e vítimas em comunidades ricas em espécies. Um importante fator a ser estudado é como o número e diversidade de características fenotípicas relacionadas ao ataque e defesa influencia o processo coevolutivo em redes de interação. A inclusão de processos como extinção e especiação poderia aumentar a compreensão sobre a dinâmica temporal das interações e o surgimento de inovações evolutivas.

As previsões teóricas resultantes dos modelos desenvolvidos nesta tese também poderiam ser utilizadas para a formulação de predições que podem ser testadas empiricamente. Estudos que acompanhassem a dinâmica populacional e os níveis relativos de defesa e contra-defesa em comunidades de parasitas e hospedeiros ou predadores e presas poderiam fornecer dados sobre a dinâmica evolutiva dessas interações e a estrutura da seleção. Estudos sobre a dinâmica temporal das interações também forneceriam importantes dados para testar a hipótese da seleção flutuante como um importante mecanismo que promove a coexistência entre espécies e a manutenção das redes de interação.

Um dos objetivos de modelos teóricos é buscar elucidar a importância de mecanismos sobre propriedades gerais dos sistemas de interesse. Inevitavelmente existe um compromisso

entre o grau especificidade de um modelo e a possibilidade de generalização dos resultados. Nesta tese buscamos uma maior generalidade das explicações e previsões dos modelos e por isso não enfocamos em aspectos mais específicos da biologia e história natural das interações antagonistas. A evolução das interações ecológicas é um tema central na biologia e não pretendemos esgotar este tema com os modelos propostos nesta tese. Muitas são as possibilidades de se explorar a coevolução do ponto de vista teórico e pensamos que as ferramentas analíticas derivadas do estudo de redes complexas tem muito a contribuir. O avanço do conhecimento científico se dá pela proposição de novas idéias e reconsideração de conceitos já estabelecidos. Nesse contexto, nossos resultados indicam que pode ser possível estudar e modelar a estrutura da seleção recíproca em comunidades ricas em espécies utilizando como base informações sobre a organização das interações ecológicas. No entanto, é preciso considerar que diversidade, variação e contingência são propriedades dos sistemas biológicos e um dos principais desafios da ecologia é desenvolver teorias que considerem essas propriedades como premissas fundamentais.