Parasite Spreading in Spatial Ecological Multiplex Networks

Massimo Stella,1,Cecilia S. Andreazzi,2,3,† Sanja Selakovic,4 Alireza Goudarzi,5 and Alberto Antonioni6,7,8

1Institute for Complex Systems Simulation, University of Southampton, Southampton, UK
2Departamento de Ecologia, Universidade de São Paulo, São Paulo, SP 05508-900, Brazil
3Fundação Oswaldo Cruz, Rio de Janeiro, RJ, 22713-375, Brazil
4Faculty of Veterinary Medicine, Utrecht University, Utrecht, The Netherlands
5Department of Computer Science, University of New Mexico, Albuquerque, NM 87131-0001
6Faculty of Business and Economics, University of Lausanne, 1015 Lausanne, Switzerland
7Grupo Interdisciplinar de Sistemas Complejos (GISC), Departamento de Matemáticas, Universidad Carlos III de Madrid, 28911 Leganés, Madrid, Spain
8Instituto de Biocomputación y Física de Sistemas Complejos (BIFI), Universidad de Zaragoza, 50018 Zaragoza

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Abstract

Several parasites may be transmitted among their hosts through different mechanisms at the same time, which challenges the modelling of the parasite spreading process. Multiplex networks are a particular kind of multi-layer graphs where the same set of 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.

Keywords: Ecological multiplex networks, multi-host parasites, spatial networks, SI dynamics.

*Corresponding author: massimo.stella@inbox.com
†Corresponding author: candreazzi@fioecruz.br
I. INTRODUCTION

Pathogens and parasites (“parasites” hereafter) are one of the most widespread and diverse life form [1]. 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 [1]. 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, the consumption of an infected prey, and congenitally through the placenta [2]; *Cryptosporidium* directly infects its hosts via sexual contact or via fecal-oral transmission [3]; *Trypanosoma cruzi* can be transmitted by triatomine vectors (stercorarian transmission), the consumption of an infected prey, and also congenitally through the placenta [4, 5]. 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 [6].

Contact networks explicitly can be used to understand the epidemiological consequences of complex host interaction patterns [7–12]. 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 as an edge (or link). Interactions can also be embedded in space [11–14] where 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 [413]. 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 [15].

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 as well [16, 17]. Examples include multi-modal transportation networks in metropolitan areas [18, 19], or proteins that interact with each other according to different regulatory mechanism [20, 21]. Ecological systems are also characterised by multiple types of relationships among biological entities, organised and structured on different temporal and spatial scales [17, 22]. Such representations can be described as “multiplex networks” [22, 23]. They are a particular kind of multi-layer networks 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 network 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 [22]. 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 [24, 25].

Descriptions of ecological multiplex networks [22] and studies of infection spreading over
multiplex structures [24, 25] 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 explored. We propose a spatial multiplex-based framework to model multi-host parasite transmission through multiple transmission mechanisms. In this framework, blood meal of interspecific interaction can be represented in a different layer of the multiplex network structure. Our model is inspired by the complex 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 [26]. The main infection route to humans involves the triatomine vectors. blood meals from an infected host can transmit the parasite to the triatomine vector, while defecation by an infected vector on the host following the blood meal 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 [27]. Infection by the oral route occurs when a mammal ingests infected triatomine feces, food contaminated with the parasite or by preying on infected vectors or mammals [5].

We used a Susceptible-Infected (SI) model to describe parasite transmission dynamics in spatially embedded multiplex networks. 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 network 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. 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.

Preliminary studies [27–29] used mean-field methods 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 [27]. 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 complex empirical data.
II. METHODS

A. 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 vector species and their 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) \[18\], which is constructed by randomly placing the populations in a metric space and connecting two nodes by a link if their distance is in a given range defined by neighbourhood radius $R$. All interactions are allowed on this level.

The construction process for a multiplex network (Figure 1) with $N$ nodes, radius $R$, species frequencies $\{f_i\}_{1 \leq i \leq m}$ 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.

- For a node $i$ on the contact layer, a directed edge $(i,k)$ is created with any node $k$ where toroidal distance from $i$ is $d_{ik} < R$. The edge $(i,k)$ is replicated 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.

In this study we consider networks of $N = 10,000$ populations (nodes) and radius of interaction $\rho = 0.056$. The radius of interaction has been tuned according to our node density in order to obtain connected networks, in which there is at least one path connecting each pair of nodes in the network. Therefore, we avoided limitations to parasite spreading due to complete isolation of populations. We study two multiplex networks: the three-species model with $s = 3$ (reference), 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 the free parameter of the model. The other species frequencies are defined as $2f_{\text{predator}} = f_{\text{prey}}$ and under the constraint that $(\sum f_i = 1)$. In the Canastra food web we kept the same proportions as in the three species model ($2f_{\text{predator}} = f_{\text{prey}}$), with the constraints that all different species of 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.
B. 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, $\pi N r_0^2 \approx 28.2$ 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. If node $i$ is infected and the edge $(i, j)$ exists in the vectorial layer, node $j$ becomes infected as well (vectorial layer infection transmission).

4. If node $i$ is infected and the edge $(i, j)$ exists in the trophic layer, node $j$ becomes infected as well (trophic layer infection transmission).

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 in 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.

C. Immunisation

In an immunisation simulation we study the dynamics of parasite spreading when predator and 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 $1, 2, \ldots, m$, we set $p_1 = p_2 = \cdots = p_s$. 
D. Canastra Food Web

We used data from an epidemiological study of *T. cruzi* infection in wild hosts in Southeast Brazil [30] 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 [31–36]. As there was no species-level classification of the biological vectors present in the area, we considered the vectors one single species. We used species prevalence, measured through positive parasitological, to estimate the possible interactions in the vectorial layer [30]. 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 [5]. The Canastra multiplex network has a total of 20 species: 7 predators, 12 prey and 1 vector (Supplementary Information).

III. RESULTS

Our results focus on: (i) highlighting the topological features of our spatial multiplex network through a multiplex cartography [37], (ii) investigating parasite spreading across the multi-layered structure at different values for the vector frequency $f_v$ 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 network, respectively.

A. 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 $f_v$ (Figure 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 network 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 2i): (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 network (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 in the system is low ($f_v = 0.1$), infection transmission across the multiplex network 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 on 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 pandemics
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 $\langle r \rangle_{\text{max}}$ 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 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 predator or prey 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 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 spreading 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 network, 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.

**B. Canastra ecosystem**

The cartographies reported in Figure 2 (e-h) represent snapshots of the Canastra multiplex network 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 network (Figure 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 is because the Canastra multiplex network has one predator species that can be contaminated by vectorial transmission, while the others have links only on 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 network 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 increase their participation in the multiplex network 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 contaminative interactions with vectors on the vectorial layer display participation coefficient close to 1 (Figure 2h). For \( f_v = 0.75 \) and there is also a gap among predators that interact with vectors and those that do not, which was absent in the three-species multiplex network (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 thus there is a higher species evenness, parasite diffusion is optimised when vectorial and trophic transmission mechanism have similar importance. As a comparison, the three-species multiplex network exhibited a minimum in the 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’s higher connectivity on the trophic layer, the parasite ratio increases behave similarly to the three-species system. The parasite spreading propagates much slower on the trophic layer alone than on the full multiplex struc-
ture, 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 on the Canastra multiplex network. When the spreading deceleration 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 for the $p_v = 0.6$ but the deceleration phase takes longer for $p_v = 0.8$ then for the $p_v = 0.6$ and thus $p_v = 0.8$ has a higher global infection time compared to $p_v = 0.6$.

The Canastra multiplex network also displayed a phase transition in the emergence of a global epidemic, similarly to what happened for the three-species multiplex network. 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) to 0.04.

Unlike the three-species multiplex, the Canastra multiplex network displayed different cartographies patterns 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 network, immunising preys does not always hamper the parasite spreading more than immunising predators. From the cartography (Figure 2) 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 network, 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 vector contaminate mostly preys in the Canastra multiplex network. Having a higher vectorial layer importance means that the multiplex structure becomes predominant and the populations that participate the most in the multiplex network, such as preys, can promote the infection spread.

IV. DISCUSSION

It is only recently that network scientists started addressing the multiplex structure of real-world systems such as ecological and epidemiological systems [16]. They used the multiplex framework for modelling different ecological and epidemiological systems. Multiplex networks were used in ecological systems to approach different interaction types [22, 38, 39] and levels of organisation [40, 41]. In epidemiological systems multiplexes were used to describe parasite spreading with Susceptible-Infected-Susceptible dynamics [42, 43],

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susceptible-infected-recovered dynamics [46–48], and multiple types of interactions between random layers [21, 25, 49]. 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 the 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 model 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 heterogeneity 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 the species in the system.

Multiplex cartography [37] 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 the 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 network, a higher frequency of vectors ($f_v > 50\%$ of the total population) increased prey connectivity and therefore their participation in the multiplex topology. We found different results when we considering a more realistic ecological scenario. In Canastra, predators dominated the multiplex topology because of their higher connectivity and higher average overlapping degree. Therefore, the 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 [29, 50–52]. The three-species multiplex network showed that higher vector frequency increased vector importance in the vectorial layer, which enhanced prey 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 the vector frequency is low and parasite spreads only on 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 network, we observed an analogous minimum even with higher vector frequencies (Figure 6). This suggests that global infection time in complex ecological scenarios 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 [13]. 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 network 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 network. The presence of such a 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 network. In the three-species model the parasite can percolate through the whole system only if $f_v > 0.02$, while in the Canastra system 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 [53].

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 showed 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 immunising preys could hamper the parasite spreading in the three-species case. In the Canastra multiplex network, the predators were the species that attained most of their connections in the multiplex network and thus had a higher importance in the cartography. This higher importance in the multiplex network suggests that the 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 [5]. 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 is because vectors contaminate mostly preys in the Canastra multiplex network. Again, the roles played by each species in the multiplex cartography depended on the frequency of vectors and is relate to their importance for parasite spreading.

For 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 networks. 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 [27]. In random multiplex networks [42] the epidemic process also depends on the strength and nature of the coupling between the layers. This previous study [42] 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, 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 [55]. A more realistic models 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 [54, 56, 57]. Finally, host species that share ecological habitat with vector species are more likely to be exposed to the infection [5].

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 [58]. 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 incidence in humans. Additional layers, such as network of human interactions with its socio-ecological characteristics, could be included to make the model more realistic.

**Author Contributions**

AA, CSA, SS, MS conceived and designed the study, AA and AG wrote the code, AA, AG and MS performed experiments, CSA analysed the empirical data and MS analysed the data, CSA, SS and MS discussed the results. All authors wrote the paper and gave final approval for publication.

**Supplementary Material**

The vectorial and trophic matrices for the Canastra model have been uploaded as supplementary material.

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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.
A. Multiplex Cartography

A multiplex cartography visually represents the role played by a given node within across different layers according to its topological features \[16, 37\]. 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 \[16\] the sum of all the degrees of node \(i\) across the \(M\) multiplex layers:

\[
o_i = \sum_\alpha x_i^{(\alpha)}, \tag{1}
\]

where \(x_i\) is the degree of node \(i\) in the layer \(\alpha \in (1, \ldots, M)\). The overlapping degree \(o_i\) represents a proxy of the overall local centrality that a node has within the multiplex network. The standardised overlapping degree is thus defined as \[37\] the ratio of \(o_i\) to its expected value:

\[
z_i = \frac{o_i - \langle o_i \rangle}{\sigma(o_i)}.
\]

In the scientific literature \[37\], it is customary to call hubs those multiplex network 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 \((2/3 < P_i \leq 1)\) \[37\].

B. 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 network is infected. Alternatively, the infection time indicates the time step \(t_{inf}\) at which the disease infects the most nodes within the network. If \(R(t) = \frac{N_{inf}(t)}{N}\) if the ratio of infected populations/nodes at time \(t\), then \(Max_t(R(t)) = R(t_{inf})\).

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 increase \(\Delta R(t) \equiv R(t + 1) - R(t)\), 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 network.

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 \(\Omega\), it is relatively...
straightforward to compute an upper bound $\langle r \rangle^*$ for $\langle r \rangle$ as:

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

FIGURES
FIG. 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.
FIG. 2. Cartographies as histogram densities for the three-species model (a,b,c,d) and for the Canasta dataset (e,f,g,h), for vector frequency $f_v = 0.01, 0.1, 0.25$ and $0.75$. 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 prey and green triangles represent vectors. Notice that in (e,f,g,h) predators and prey are represented with the same colours but different shapes.
FIG. 3. Left: global infection times versus vectorial layer importance $p_v$ for different values of vector frequency $f_v = 0.1, 0.25, 0.5, 0.75$ for the three-species model. Right: global infection rate over time for $f_v = 0.75$ and $p_v = 0.5$, expressing the diffusion speed of the disease over time within the multiplex network. 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 $\gamma \approx 1$.

FIG. 4. Ratio of global infected populations after 10,000 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 pandemics. 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.
FIG. 5. Left: 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 prey 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 prey is the best choice in terms of both reducing the global infection time and slowing the infection spread over time.

FIG. 6. Left: global infection times versus vectorial layer importance $p_v$ for different values of vector frequency $f_v = 0.1, 0.25, 0.5, 0.75$ for the Canastra model. Right: global infection rate over time for $f_v = 0.25$ and $p_v = 0.5$, expressing the diffusion speed of the disease over time within the multiplex network. 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 $\gamma \approx 1$. 
FIG. 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 disease 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).