Disease Spreading on Ecological Multiplex Networks

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Abstract

Processes in which diseases spread and sustain inside of different types of populations were always interesting subjects for epidemiologists. In recent years, ecologists draw the attention to the fact that not only hosts but also the other species in the community can affect the process of disease spread and in that way influence the result of the infection. In nature, we find many parasites and infectious agents with complex life cycles and which can be transmitted in the ecological communities through contact interaction or through feeding interaction. Multiplex networks are layered networks that can represent different types of interactions between agents. In this networks the agents are part of all the layers, but the structure of their interaction is distinct in each layer. We recognise multiplex networks approach as a way in which we can question the importance of the different forms of transmission for the disease spread in the ecosystems. Our results show that for *Trypanosoma cruzi*, a parasite that can be transmitted through arthropod vectors or through feeding on infected prey, the infection is more widespread when considering both layers in the process. When considering the multiplex structure, species that are not connected through the food web can be affected because of the inclusion of the vectorial layer. The frequency of vectors in the community also influenced the infection spread, increasing the speed of infection in the hosts. We conclude that the multiplex approach is extremely powerful when dealing with different types of interactions and that non-trivial results can be found when the multilayered structure of the process is considered.
I. INTRODUCTION

Organisms differ greatly in how they feed on other species. Trophic interactions, such as grazing, predation, and parasitism, differ fundamentally on how consumers attack their victims. This includes whether they kill their victims, how long they remain to feed on a single victim before killing it or leaving it, and how many victims they feed upon during their lifetimes [23, 41]. Food webs are maps of the trophic interactions between species and they are usually represented as networks of species and the energy links between them [24, 29, 42]. They have been used to identify key linkages between species and processes that govern ecosystem stability, functioning, and biological diversity [8, 15]. However, food web studies have historically not included micro-species or parasite interactions. Recently, there have been efforts to incorporate all the trophic links in food web analysis [8, 21, 22]. These include how parasites change descriptive parameters of food webs, such as connectivity, looping, and linkage density [16, 21]. More importantly, parasites appear to play unique topological roles in food webs. As resources, they have close physical intimacy with their hosts and thus they are concomitant resources for the same predators. As consumers, they can have complex life cycles and inverse consumer-resource body-size ratios, in contrast to many free-living consumers [16].

One of the main challenges in community ecology is to include important elements of the complex life cycles of parasites and understand their effect in the structure and dynamics of food webs [21, 39]. During their life cycles, parasites can have several free-living and parasitic stages. They may infect multiple hosts from diverse trophic levels and use intermediary hosts or vectors to be transmitted to other destination hosts. Several studies have reported that free-living stages of parasites or ectoparasites may be subject to predation and that predators can feed on parasites together with their infected prey [19]. Predation also represents a key transmission pathway, and predators often acquire trophically transmitted parasites from their prey [19]. Until now accommodating such diverse types of links in the same food web remained an open question [21].

Here, we propose a new approach to study complex life cycles and transmission modes in food webs. In this approach, each interaction type between species gives rise to a different layer in a multi-layered complex network structure called multiplex [9]. Multiplex or multi-relational networks are multi-layered graphs where nodes appear on all the layers but are connected according to different topologies on each level [3, 9]. Studies of disease spreading over multiplex structures have appeared only very recently within the scientific literature [7]. However, to the best of our knowledge, multiplexity has never been applied to the analysis of disease spreading in real ecological scenarios. Nonetheless, this multiplex approach would be particularly useful when dealing with multihost parasites that can be transmitted to their hosts via multiple routes. For example, many protozoa and helminths use intermediary hosts or vectors to be transmitted from one host to the other, but can also be transmitted trophically when a predator feeds on an infected prey or vector.

In this dynamical model we are particularly interested in understanding the spatial epidemiology of multihost parasites that are trophically transmitted throughout the food web and also vector-borne via blood-sucking invertebrates. Important human diseases such as
Chagas disease, Leishmaniosis, and Malaria are caused by parasites that have such a complex life cycle [38].

As a case study, we modelled the trophic and vectorial transmission of *Trypanosoma cruzi*, the etiological agent of Chagas disease. We used data on *T. cruzi* sylvatic hosts prevalence in two different regions in Brazil [18, 33]. *T. cruzi* is a multi-host parasite found in more than 100 mammalian species [27]. Human infections are generally associated with infected triatomine bugs (biological vectors), but most recent outbreaks in Brazil were caused by oral transmission, which is probably the most ancient route of infection among wild animals [27, 40]. We investigated the effect of each mode of transmission on the epidemiology of *T. cruzi* and the relationship between the structural characteristics of host species in the multiplex and its epidemiological importance. The effect of different community structures, such as the relative frequency of vectors in the environment was also investigated. We were able to identify key species for disease spreading and the importance of each transmission mode in different community structure scenarios.

II. DATASET

Epidemiological studies of *T. cruzi* infection in wild hosts in two different areas [18, 33] were used to estimate the probabilities of species interaction and disease transmission in the model. For the trophic layer, we first built the qualitative food webs based on the diet of the animals in the areas [1, 6, 11, 12, 30–32]. As there were no species-level classification of the biological vectors present in the areas, we grouped all the species in a compartment called ”vectors”. In this procedure, we classified the interaction with “1” if that item was present in the diet of the animal, and with a “0” if not. We used this binary food web to constrain the possible interactions between species, i.e., if the interaction is equal to zero in the binary network it is also zero in the probabilistic network. To estimate the probabilities of interaction between species in the food webs expected by their body sizes we used the probabilistic niche model [46]. The species mean body weights were obtained from the literature [18, 33, 34, 36]. We used the [17] method based on linear model fitting to estimate the species niche parameters: niche position $n_i$, feeding niche optimum $c_i$ and range $r_i$ of suitable preys around that optimum. We then calculated each interaction probability with the niche model [46].

We estimated the probabilities of interaction in the vectorial layer using the prevalence of species *T. cruzi* [18, 33]. We used the proportion of individuals with positive serology for *T. cruzi* as a proxy for the probability of interaction between host species and vectors. Because the trophic route infection tends to be more severe than the vectorial route infection [17], we assumed that vectors infect animals with positive serology for *T. cruzi*. We are not aware of any data on these interaction probabilities. In addition, we used the proportion of individuals with positive parasitological diagnostics as a proxy for the probability of disease transmission from the host to the vector, since only the individuals with positive parasitaemia are able to transmit the parasite.
III. MULTIPLEX NETWORKS

A. An Introduction to Multiplexity

Network theory investigates the structural and dynamical properties of a set of entities, each one represented as a node or vertex; the nodes take part in pairwise interactions, represented as links or edges. In the last twenty years, the network framework has been successful in modelling many complex systems, throughout the physical, biological, social, and technological sciences [26]. In fact, it allowed for an interpretation of many features of real-world networked systems, such as the emergence of hubs, the “small-world” property and the organisation in hierarchical structures [2, 26, 45]. Recently, the increasing amount of available data brought the scientific community to generalise the complex network formalism, for instance by taking into account also the heterogeneity of real-world interactions [9]. Examples include multi-modal transportation networks in metropolitan areas [25], where each location can be connected to another by different modes of transport. Analogously, countries interrelate via various political and economical channels or proteins interact with each other according to different regulatory mechanics [9, 14]. Also social systems exhibit different kinds of ties, organised and structured on different levels of social environments, as have been observed by sociologists since the 1930s [3, 14]. It is in the context of social network analysis that such representations emerged as multi-relational networks or multiplex networks [44], i.e. multi-layered graphs where nodes appear on all the layers, but they can be connected according to different topologies on each layer. Each multiplex layer contains edges of a given type, e.g. co-location, kinship, commercial, or collaboration relationships between individuals in a social environment. A visual example of a multiplex is depicted in Figure 1.

FIG. 1. Example of a multiplex with two layers only (i.e. a duplex). All the nodes are replicated on all the layers but they are connected differently on each layer.
B. Analysis of the Trophic Layer

In our study we analysed two different kinds of interactions that are related to the transmission of *T. cruzi*. This parasite can be transmitted through both trophic and vectorial routes. We considered each of those transmission route as a layer in our multiplex. Therefore we consider only duplexes: a multiplex network consisting of two layers.

In the Canastra dataset we have 20 species, each of which is represented by a node. The nodes engage in directed trophic interactions and parasitic interactions. A connection is directed from species A to species B if "A is eaten by B". In the trophic layer, the two nodes having higher in-degree (representing top predators) are the *Leopardus pardalis* and the *Chrysocyon brachyurus*, predating 15 and 14 species, respectively. The trophic height of a species is the average trophic position of a species in all food chains it belongs to and it indicates its role as top predator in the food web [13]. In Canastra, the species with the highest trophic height were also *L. pardalis* and *C. brachyurus*. In addition, the species with higher out-degree (representing the most preyed species) are the *Calomys* and the *Oligoryzomys*, each being predated by 8 species. Furthermore, together with the vectors, these last two species also have the highest radiality centrality of the network, in other words they play a locally prominent role in the food web (at a scale comparable to the web diameter itself). The radiality centrality [20] of node *i* in a network with *n* nodes is defined as

\[ r_i := \frac{1}{n-1} \sum_j (D - d_{ij} + 1) \] (1)

where *d_{i,j}* is the geodesic distance between nodes *i* and *j* (i.e. the minimum number of hops necessary to go from *i* to *j* on the network) while *D* is the network diameter (i.e. the maximum geodesic distance between two network nodes). Intuitively, in the context of food webs, with directed trophic interactions, the radiality centrality can be thought of as an indicator of the importance of preys for the food web itself (Pearson’s coefficient: 0.99, p-Value ≤ 10^-7). This correspondence between an ecological property of species and a network theoretic measure is further evidence for the appropriateness of network science in the study of ecological systems. Another species that plays an important role in the trophic layer is the *Didelphis albiventris*, which has a betweenness centrality [20] of 1. Betweenness centrality is the ratio of paths that go through a given node over the total number of paths in a given networks. Therefore, the *D. albiventris* is present on every trophic pathway in the food web, and it may therefore play a prominent role in the spreading of Chagas disease. Figure 3 gives a visual representation of the trophic and vectorial layers in the Canastra dataset.

In the Pantanal dataset we have 19 species. *L. pardalis*, also present in this food web, is the species with the highest trophic height, followed by *Cerdocyon thous* and *Nasua nasua*. Similar to the Canastra dataset, the vectors in Pantanal are the most predated species (predated by 15 other species). The other species are predated by less than 5 species. Furthermore, the most predated species are those with higher radiality centrality in the network. A betweenness centrality analysis does not reveal any interesting pattern,
FIG. 2. Visual representation of the multiplex structure, comprising the trophic layer (left) and the vectorial layer (right) for the Canastra dataset. Note that all the species are present in both layers but they are connected differently.

C. Analysis of the Multiplex Structure

Following [3], let us define a multiplex $M$ as a formal collection of a finite number $m$ of single-layered networks. Let us denote with $A^\alpha$ the adjacency matrix of the network in the $\alpha$-th layer. Each entry $a^\alpha_{ij}$ is equal to 1 if nodes $i$ and $j$ are connected on layer $\alpha$ or 0 otherwise. Let us define the adjacency matrix $A$ of the multiplex $M$ as the set $A^\alpha \alpha$.

Furthermore, we define the edge overlap $o_{ij}$ of edge $(i, j)$ between layers $\alpha$ and $\beta$ as $a^\alpha_{ij} + a^\beta_{ij}$. In other words, the edge overlap quantifies the number of times that a given connection is present in the multiplex structure, across different layers and therefore $\leq o_{ij} \leq m$. In our case, for the Canastra dataset only three edges have an edge overlap of 2 in the whole duplex (i.e. a multiplex with $m = 2$ layers only) while there are 14 repeated connections in the Pantanal dataset. Given the very small numbers in play, we cannot quantify correlations that explain this difference in overlap in a statistically significant way.

As the simplest characterisation of the multiplex structure, let us introduce also the aggregated adjacency matrix $P$, whose entries satisfy $p_{ij} = 1$ if nodes $i$ and $j$ are connected
in at least one of the \( m \) layers:

\[
p_{ij} := \prod_{\alpha} a_{ij}^\alpha.
\]  

(2)

The aggregated matrix \( P \) represents the adjacency matrix of the single-layer network obtained by “projecting” all the layers onto a single network. This matrix cannot encapsulate the whole information contained in the multiplex structure [3, 4], nonetheless it represents a simple approximation when analysing multiplexity.

In the Canastra dataset, overlaying together the trophic layer and the vectorial one does not alter the network hubs, with the *Leopardus pardalis* still having an in-degree of 15. As expected, in the aggregated network for both Canastra and Pantanal datasets, obtained by joining the trophic layer with the oriented edges of the vectorial layer, a boost in radiality is registered for the vector. This is a preliminary indication that the multiplex structure will play a predominant role in the spreading of *T. cruzi* infection mediated by the vectors. No significant changes in the betweenness centrality measure have been observed in the aggregated networks.

A more genuine measure of node importance in the multiplex structure is given by the interdependency factor [3]. The interdependency \( \lambda_i \) for node \( i \) in a multiplex is defined as:

\[
\lambda_i := \sum_{j \neq i} \frac{\psi_{ij}}{\sigma_{ij}},
\]  

(3)

where \( \sigma_{ij} \) is the total number of shortest paths from node \( i \) to node \( j \) on the whole multiplex and \( \psi_{ij} \) is the number of shortest paths from node \( i \) to node \( j \) that make use of links in two or more layers. Note that a shortest path on a multiplex can jump between layers at any given node. For instance, the shortest path going from \( i \) to \( k \) on a duplex might use the link \((i,j)\) on layer 1 and then the link \((j,k)\) on layer 2 and therefore be \{[(i,j),1],[j,k),2]\}. The higher the interdependency \( \lambda_i \) the easier it is for node \( i \) to reach other nodes in the multiplex by using connections on different layers. Both the two datasets exhibit a non-negligible average interdependency, with the Canastra dataset having a mean interdependency of \( \lambda_{can} = 0.24 \) and the Pantanal dataset having a lower value of interdependency \( \lambda_{pan} = 0.15 \). As reported in Figure 3, both in the Canastra and Pantanal datasets, the vectors have the highest interdependency value. In other words, the multiplex structure, with its combination of vectorial and trophic interactions, allows the vectors to be more intensively connected with all the other species. Furthermore, the vectors are not the only species having a non-zero interdependency. Additionally, the species interdependency was significantly related to species trophic height \((\beta = -0.14, p < 0.0001, \text{Figure 4})\). Hence, these facts are a strong indication that the multiplex structure will play a prominent role in boosting the effectiveness of the disease spreading.
FIG. 3. Interdependency ratio for the species in the Canastra (orange squares) and in the Pantanal (blue squares) datasets. In both the datasets, the species are ordered based on their body sizes, from the heaviest to the lighter. The vector is always the last species.

FIG. 4. Relationship between species trophic height and interdependency in Canastra (orange dots) and Pantanal (blue dots). The position of the vectors and of the top predator *Leopardus pardalis* is highlighted.
IV. MODEL

A. Multiplex network construction

In this model we consider $S$ different species, each with its corresponding relative frequency $f_i$ (see Appendix section for details). Note that the number of considered species $S$ in the Canastra dataset is 20, while it is 19 in the ecosystem of Pantanal. We applied the metabolic theory of ecology [5] to estimate species frequency such that $f = m^{-3/4}$, where $m$ is species body size. We then normalised the frequencies, i.e., $\sum_{i=1}^{S} f_i = 1$. This estimation has been done following body size datasets in [18, 33].

A node in the network represents an individual of a given species. The attributes of a node $i$ are: the given species $s_i$, its spatial position $(x_i, y_i)$, and its current state $v_i$ that may be susceptible or infected.

The spatial environment is represented as a square of size $L = 1$ and the metric we consider is the Euclidean distance in the two-dimensional space.

The process we have used to obtain ecological multiplex network of about $N$ nodes can be summarised as follows:

1. A number $\lceil N f_i \rceil$ of individuals nodes is randomly distributed in the squared space per each species $1 \leq s_i \leq S$.

2. (vectorial layer step) A link between nodes $i$ and $j$ is created in the vectorial layer with the following probability:

$$p^V_{(i,j)} = V(s_i, s_j)e^{-k_V d(i,j)},$$

where $V$ is the interaction matrix in the vectorial layer (see Appendix section for details), $d(i, j)$ is the physical distance between nodes $i$ and $j$, and $k_V$ is a free parameter that controls the importance of the space in the vectorial layer. This parameter is fixed in order to obtained fairly connected networks on this layer. This creation link process is repeated for all pairs of nodes $(i, j)$ in the network. The trophic layer is undirected and we do not consider links where corresponding nodes lie at distance greater than $d_{max} = 0.2$.

3. (trophic layer step) A link between nodes $i$ and $j$ is created in the trophic layer with the following probability:

$$p^T_{(i,j)} = T(s_i, s_j)e^{-k_T d(i,j)},$$

where $T$ is the interaction matrix in the trophic layer (see Appendix section for details), $d(i, j)$ is the physical distance between nodes $i$ and $j$, and $k_T$ is a free parameter that controls the importance of the space in the trophic layer. This parameter is fixed in order to obtained fairly connected networks on this layer. This creation link process is repeated for all pairs of nodes $(i, j)$ in the network. Moreover, since the trophic layer network is to be considered as directed, the link $(i, j)$ can exist while the link $(j, i)$ is not present and vice versa, $T(s_i, s_j) \neq T(s_j, s_i)$. Again, we do not consider links where nodes are at distance more than $d_{max} = 0.2$. 

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B. Disease diffusion dynamics

Here we describe the disease diffusion dynamics on ecological multiplex networks that are defined in the previous section. A node, or an individual of a given species, in the network can be infected or susceptible and its state must be the same in both layers.

We start the simulation infecting all the individuals of species number $S$ (the last species is the vector species in both datasets) within the circle area of radius $R_0 = 0.05$ and centre $x_0 = 0.5, y_0 = 0.5$, that is, the center of the squared space. Subsequently, the disease diffusion process evolves as it follows:

1. the vectorial layer is chosen to be considered in the disease diffusion for this step with probability $p_{vector}$. Step 2 is then performed when the vectorial layer is considered, Step 3 otherwise.

2. (vectorial layer disease diffusion) A random node $i$ and a random neighbour $j$ in the vectorial layer are selected. If the node $i$ is infected also the node $j$ becomes infected with probability $V_d(s_i, s_j)$.

3. (trophic layer disease diffusion) A random node $i$ and a random neighbour $j$ in the trophic layer are selected. If the node $i$ is infected also the node $j$ becomes infected with probability $T_d(s_i, s_j)$.

4. Steps 1, 2 and 3 are repeated until the maximum number of time steps is reached.

The process in stages 1, 2 or 3 is repeated $N$, the size of the populations, times for each time step. This also means that an individual in the population is chosen once in the diffusion process at each time step, on average. The two matrices of interactions, $V$ and $T$, and the diffusion matrix $V_d$, have been estimated from the two datasets in [18, 33]. On the other hand, we assumed that the diffusion matrix $T_d$ is equal to one in each of its values.

C. Model output analysis

To investigate the effect of each layer on the dynamics of the infection, we simulated the model for diffusion probabilities in the vectorial layer $p_{vector}$ varying from 0 (only the trophic layer) to 1 (only the vectorial layer).

To compare the dynamics of each species per diffusion probabilities situations, we fitted a logistic regression to the average proportions of infected individuals per time. The coefficients of a logistic regression are the steepness parameter of the curves. We then compared the species coefficients per each diffusion probability on different community compositions (with 10%, 20% or 80% of vectors). The relaxation time of the average proportion of total infected individuals was used as a proxy for the velocity of infection spread in the different diffusion probabilities situations.
V. RESULTS

The dynamics of the *T. cruzi* infection in the Canastra and Pantanal multiplexes are summarised in Figures 5 and 6. It is possible to observe that in both multiplexes the proportion of infected individuals of all species was low when we had only the trophic layer (v0). In this situation, there were no infected vectors because vectors are dependent on the vectorial layer to get infected. The top predators were the species that had a higher proportion of infected individuals. Comparing the communities with different relative proportion of vectors (columns), there was a slightly increase in the proportion of infected individuals when there was a higher relative frequency of vectors in the community (a, d and g). When both layers had the same importance for the diffusion of the infection (v0.5), we had a higher proportion of infected individuals in all species. Increasing the relative frequency of vectors in the community decreased the proportion of infected vectors. Once again, the top predators were the species with the higher proportion of infected individuals. When the transmission was only the vectorial layer (v1), the two multiplexes differed in the infection dynamics. In Canastra, only three species (sp2 is *L. pardalis*, sp11 is *Marmorsops incanus* and sp20 is the vector) became infected, independent of the relative frequency of vectors. However, the higher the frequency of vectors in the community, the slower was the infection in the vectors. In the Pantanal multiplex, more species became infected with transmission occurring only in the vectorial layer. More interesting, there was no relationship between the trophic height of the species and the proportion of infected individuals. Increasing the relative frequency of vectors in the community accelerated the infection in all species except for the vectors.

The logistic regression coefficients indicated how steep was the increase in infection for each species. In Canastra, (Figure 7), it was possible to identify different behaviours depending on the species trophic height. In general, top predators had a steeper increase in the proportion of infection compared to lower trophic levels. They were also more affected by the probability of disease diffusion in the vectorial layer $p_{vector}$, that is, the higher the disease diffusion in the vectorial layer, the steeper is the infection in time. However, in very high $p_{vector}$ only sp2 (*L. pardalis*) and sp11 (*M. incanus*) had infection, which is in accordance with the results found in Figure 5. Vectors were differently affected by the $p_{vector}$ then other species. Because their interaction pattern, infection in vectors is dependent on adjacent host getting infected first. This creates a delay in the disease diffusion dynamics.

In Pantanal multiplex (Figure 8), the top predators was also the species that had, in general, steeper increase in infection. However, the relationship between the logistic regression coefficients and the probability of disease diffusion in the vectorial layer ($p_{vector}$) had a bell shape in Pantanal, while in Canastra it was skewed to the right. Vectors were also not affected by the probability of disease diffusion in the vectorial layer ($p_{vector}$).

As reported in Figure 9, our simulation showed that the spreading of the disease on the multiplex depends in a non-monotonic way on the importance of the vector layer. More in details, for different values of the relative frequency of vectors (IV), increasing the probability of diffusion in the vectorial layer initially makes the spread of the disease faster, in other
FIG. 5. Average proportion of infected individuals per time for the initial 500 time steps in Canastra multiplex. Species colours are related to species trophic height in the trophic layer, such as that hot-colored species are the ones with a higher trophic heights (top predators). The vector is sp20. From a to c are the results for a community with 10% of vectors relative frequency, d to f for a community with 20% of vectors relative frequency and g to i for a community with 80% of vectors relative frequency. v0 is for $p_{vector} = 0$, v0.5 is $p_{vector} = 0.5$ and v1 is $p_{vector} = 1$. 
FIG. 6. Average proportion of infected individuals per time for the initial 500 time steps in Pantanal multiplex. Species colours are related to species trophic height in the trophic layer, such as that hot-colored species are the ones with a higher trophic heights (top predators). The vector is sp19. From a to c are the results for a community with 10% of vectors relative frequency, d to f for a community with 20% of vectors relative frequency and g to i for a community with 80% of vectors relative frequency. v0 is for $\mathbf{p}_{\text{vector}} = 0$, v0.5 is $\mathbf{p}_{\text{vector}} = 0.5$ and v1 is $\mathbf{p}_{\text{vector}} = 1$. 
FIG. 7. Logistic regression coefficients for the average proportion of infected individuals per time in Canastra multiplex for the different diffusion probabilities in the vectorial layer $p_{\text{vector}}$. Species colours are related to species trophic height in the trophic layer, such as that hot-colored species are the ones with a higher trophic heights (top predators). The vector is sp20.

(a) (b) (c)

FIG. 8. Logistic regression coefficients for the average proportion of infected individuals per time in Pantanal multiplex for the different diffusion probabilities in the vectorial layer $p_{\text{vector}}$. Species colours are related to species trophic height in the trophic layer, such as that hot-colored species are the ones with a higher trophic heights (top predators). The vector is sp19.

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words, the time step (i.e. relaxation time) at which 90% of the population gets infected decreases. However, in general, the relaxation time increases when the importance of the vectorial layer is higher than a given threshold, as reported also in [V]. This non-monotonic behaviour is not evident for the Canastra dataset, for $IV = 80\%$, at least at our discrete observation scale for the vectorial layer importance. Nonetheless, this phenomenon is intrinsically related to the multiplex structure: when the vectorial layer becomes preponderant the infection diffuses mainly by vector interactions, which involve only a relatively small fraction of nodes, so that the other species on the trophic layer get infected by predator-prey interactions after some characteristic diffusion time. This behaviour is compatible with our results coming from the logistic regression coefficients.
FIG. 9. Relaxation time for the *Canastra* (left) and the *Pantanal* (right) multiplexes on a log-log scale, for different values IV of initial vector abundance. The relaxation time represents the first time step at which at least 90% of the total population in the multiplex is infected. The presence of local minima and non-monotonic behavior is evident from the plots, symptom of the different role played by the trophic and the vectorial layer.

### TABLE I

<table>
<thead>
<tr>
<th>Relative frequency of vectors (IV)</th>
<th>Pantanal</th>
<th>Canastra</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.1</td>
<td>(112, 0.35)</td>
<td>(143, 0.55)</td>
</tr>
<tr>
<td>0.2</td>
<td>(77, 0.50)</td>
<td>(115, 0.7)</td>
</tr>
<tr>
<td>0.4</td>
<td>(75, 0.65)</td>
<td>(135, 0.80)</td>
</tr>
<tr>
<td>0.8</td>
<td>(118, 0.85)</td>
<td>(317, 0.95)</td>
</tr>
</tbody>
</table>

TABLE I. Table of the minimum relaxation time, and relative vector layer importance, versus the relative frequency of vectors for the Pantanal and Canastra datasets, respectively. The minimum relaxation time is a measure of how fast the disease spreads, in terms of simulation time steps, over at least 90% of the total population. In the multiplex, different importances of the vector layer bring to different minimum relaxation times, depending on the initial vector abundance.

### VI. DISCUSSION

We here introduced a promising approach for the study of parasites that have complex life cycles and multiple hosts. The use of multiplex networks to study epidemiological dynamics is incipient and revealed non-expected results when different types of interactions were considered in the infection transmission [37, 43]. Our study showed that the epidemiology of parasites that can be spread via multiple routes of infection have non-trivial dynamics. For the specific case of *T. cruzi* infection, we found that the epidemiological dynamics is largely influenced by the multiplex structure, and that when both layers are considered a much higher proportion of infected individuals is expected. The structural roles played by each species in the multiplex was related to its importance for the infection transmission, which enable us to identify key species for this process.

In random multiplexes it was found that the behaviour of the epidemic process depends on the strength and nature of the coupling between the networks [37]. This previous study found that the global epidemic threshold of the process turned out to be smaller than the epidemic thresholds of the two layers separately. This implies that an endemic state may arise even if the epidemics is not endemic in any of the two layers separately [37]. In our study
we found that the number of species affected and the proportion of infected individuals was much higher when both layers were considered in the transmission. This result indicates that even in multiplexes with highly structured layers that differ in their topologies, the spreading of a disease via multiple routes may have a higher efficiency than when it happens in only one layer. The multilayered transmission, which is observed in many parasites with complex life cycles, seems to be a very efficient strategy for the diffusion on multiple hosts that differ on their probability of infection.

Ecoepidemiological studies of *T. cruzi* transmission in wild hosts have already pointed that the trophic interactions among hosts are important for the disease dynamics [18, 33, 34]. The epidemiological importance of top predators as bioaccumulators of trophic transmitted parasites was observed in the model results as the proportion of infected individuals was related to the trophic height of the species. Other interesting result is that *D. albiventris* was the species with the highest betweenness in the multiplex. Marsupials and specifically *Didelphis* species are recognised as one of the most important *T. cruzi* reservoirs [35]. Its topological importance in the *T. cruzi* transmission multiplex indicates that the structural role played by each species may be related to its importance for the transmission of the parasite. We recognise that the model we simulated is tremendously simplistic face the complexity of the biology and epidemiology of *T. cruzi*. However, even using the most simple SI structure we were able to catch a qualitative dynamics that is consistent with the *T. cruzi* behaviour. Thus, the qualitative predictions for the importance of each layer is still valid.

Many zoonoses, that is, diseases and infections that are naturally transmitted between vertebrate animals and humans, have multiple hosts and complex life cycles. Examples of zoonoses that are transmitted to humans by moderately to highly generalised arthropod vectors include Malaria, Leishmaniasis, Chagas disease, West Nile virus, plague, and Lyme disease [38]. All of them are characterised by at least one layer of transmission among its hosts, which may have complex structures. The multiplex approach presented here could improve our understanding of the epidemiology and the evolution of these parasites and help to elaborate more efficient control strategies for the diseases they cause. More layers as for example the network of human interactions and its socio-ecological characteristic could be included to make the model more realistic.

VII. CONCLUSIONS AND FUTURE WORK

The multiplex approach of our specific system can be further developed and it can question not only the importance of the transmission way of certain diseases in the ecosystem but also to understand many other general processes that are important for the structure, functioning and stability of the ecosystem. Differentiating the susceptible, infectious and recovered/immune individuals could help us to understand the importance of the epidemiological state for the dynamics of the disease through the ecosystem. Further, weighting the nodes and links of the multiplex can lead us to understanding of ecoenergetic flow in the ecosystem and change of the interaction strength between species influenced by the parasite *T. cruzi*. Also, inserting into the model further spatial elements would be critical for the
analysis of the real-world ecosystems.

A first point for future direction would be applying the multiplex analysis to bigger datasets, possibly encompassing more species and more types of interactions.

The multiplex approach may be a powerful tool when dealing with different types of interactions in complex systems that can be represented by complex networks. The important steps considered in the dealing with these kind of systems are the temporal and spatial scales involved in the each class of interactions which form each layer of the multiplex [28]. This idea should be further developed.

ACKNOWLEDGEMENT

All the authors would like to thank the whole SFI CSSS2014 organising team for the greatly rewarding experience of the Santa Fe Summer School 2014. M.S. acknowledges the EPSRC and the DTC in Complex Systems Simulation. A.G. would like to acknowledge the support from the National Science Foundation of the United States of America under grant CDI-1028238 and CCF-1318833. C.S.A is supported by the CNPq, Brazil. S.S would like to acknowledge the support from the Complexity Program of The Netherlands Organisation for Scientific Research (NWO).


Appendix A: Model Parameters

In this section we give the model parameters for the datasets of Canastra and Pantanal.

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</tr>
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<td>Leopardus pardalis (LEO)</td>
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<tr>
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</tr>
<tr>
<td>Lycalopex vetulus (LYC)</td>
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<tr>
<td>Conopatus semistriatus (CON)</td>
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</tr>
<tr>
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<tr>
<td>Vector (VEC) f_vector</td>
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</table>

FIG. 10. Frequencies for species in the dataset of Canastra. The frequency of vector species is a free parameter of the model and all the following frequencies have been re-normalised in the numerical simulations as a function of $f_{vector}$.

<table>
<thead>
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<th></th>
<th>CHR</th>
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<th>DID</th>
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<th>CAL</th>
<th>NES</th>
<th>MON</th>
<th>OXY</th>
<th>NEL</th>
<th>AKM</th>
<th>AKO</th>
<th>GRA</th>
<th>OLTI</th>
<th>CAL</th>
<th>VEC</th>
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<td>Cerdocyon thous (CER)</td>
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FIG. 11. Trophic matrix for the species interactions in the dataset of Canastra, $T_d$. The diffusion matrix, $T_d$, for the trophic layer is equal to one in each of its values.
### FIG. 12
Left: Vector matrix for the species interactions in the dataset of Canastra, $V$. In this layer only vector-host interactions are allowed, that is, all other matrix values are set to be zero.

Right: Vector diffusion matrix, $V_d$, for the dataset of Canastra. Again, all other matrix values are set to be zero.

### FIG. 13
Frequencies for species in the dataset of Pantanal. The frequency of vector species is a free parameter of the model and all the following frequencies have been re-normalised in the numerical simulations as a function of $f_{\text{vector}}$. 
FIG. 15. Left: Vector matrix for the species interactions in the dataset of Pantanal, $V$. In this layer only vector-host interactions are allowed, that is, all other matrix values are set to be zero. Right: Vector diffusion matrix, $V_d$, for the dataset of Canastra. Again, all other matrix values are set to be zero.