Chapter 4
Interconnecting Networks: The Role of Connector Links

Javier M. Buldú, Ricardo Sevilla-Escoboza, Jacobo Aguirre, David Papo, and Ricardo Gutiérrez

Abstract Recently, some studies have started to show how global structural properties or dynamical processes such as synchronization, robustness, cooperation, transport or epidemic spreading change dramatically when considering a network of networks, as opposed to networks in isolation. In this chapter we examine the effects that the particular way in which networks get connected exerts on each of the individual networks. We describe how choosing the adequate connector links between networks may promote or hinder different structural and dynamical properties of a particular network. We show that different connecting strategies have consequences on the distribution of network centrality, population dynamics or spreading processes. The importance of designing adequate connection strategies is illustrated with examples of social and biological systems. Finally, we discuss how this new approach can be translated to other dynamical processes, such as synchronization in an ensemble of networks.

J.M. Buldú (✉)
Laboratory of Biological Networks, Center for Biomedical Technology, UPM, Pozuelo de Alarcón, 28223 Madrid, Spain

Complex Systems Group, Universidad Rey Juan Carlos, 28933 Móstoles, Madrid, Spain
e-mail: jmbuldu@gmail.com

R. Sevilla-Escoboza
Centro Universitario de los Lagos, Universidad de Guadalajara, Enrique Díaz de Leon, Paseos de la Montaña, Lagos de Moreno, Jalisco 47460, México
e-mail: sevillaescoboza@gmail.com

J. Aguirre
Centro Nacional de Biotecnología (CSIC). C/ Darwin 3, 28049 Madrid, Spain
e-mail: jaguirre@cnb.csic.es

D. Papo
Laboratory of Biological Networks, Center for Biomedical Technology, UPM, Pozuelo de Alarcón, 28223 Madrid, Spain
e-mail: papodav@gmail.com

R. Gutiérrez
Department of Chemical Physics, The Weizmann Institute of Science, Rehovot 76100, Israel
e-mail: rcd.gutierrez@gmail.com

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4.1 Introduction

During more than a decade, the application of Complex Networks Theory to real systems has given fruitful results in the understanding of how networked systems organize, interact and evolve [1–4]. Initially, the main motivation was to characterize the topology of real systems (randomness, heterogeneity, modularity, etc.) and its connection with structural problems such as resilience, robustness or navigability [5–9]. Then, attention was devoted to how dynamical processes such as synchronization [10, 11], spreading [12–15] or congestion [16–18] were constrained by the network structure [19]. In a further development, the interplay between structure and dynamics was interpreted as a closed loop, wherein the structural properties of networks could be understood as a consequence of an adaptative process influenced by the dynamics and vice versa [20].

More recently, the idea that a network is, in many real cases, a *network of networks* (NoN), has emerged [21, 22]. In many cases, component networks of a NoN can be interpreted as modules of a unique modular network. While the detection and analysis of modules inside a network has been deeply studied [23, 24], the influence of intranetwork structures on dynamical processes remains largely unexplored. For instance, as shown in Ref. [21], interconnections between networks may play a crucial role in processes such as percolation, eventually leading to dramatic first order transitions. Other example is epidemic spreading, where it was shown that the creation of links between the most central nodes of two communities can enhance the propagation of a disease through the whole network [25].

In this chapter we focus on the competition taking place when two initially separated networks are coupled with one or more *connector links* to form a unique *ensemble network*. In particular, we examine how one or both networks can be better off according to some criterion depending on the connecting strategy that is adopted. To determine which network is benefitting the most from the interaction, we make use of the eigenvector centrality [4]. The eigenvector centrality is a measure of node importance that is obtained by calculating the eigenvector associated to the largest eigenvalue of the connectivity matrix, which, as we will see, depends on the dynamical process occurring in the network. Next, the centrality captured by each competing network is obtained as the sum of the centrality of all its nodes. The whole problem can then be framed as a competition for limited resources, since an increase of centrality for one network necessarily entails a corresponding decrease in that of its competitors.

The advantage of such a way of analyzing network competition is that, in addition of being a measure of node importance, the eigenvector centrality is related to a series of dynamical processes, such as disease spreading, diffusion processes, evolution of genotypes, rumor and opinion formation (see Ref. [4] for a review). In these cases, the transient or final state of the system depends directly on the eigenvector $\mathbf{u}_1$ associated to the largest eigenvalue λ_1 of the connectivity matrix.

We will describe how the eigenvector $\mathbf{u}_1$ of two isolated networks is modified when certain connections between them are created, leading to an interconnected network [21, 22].

In the remainder of this chapter we first analyze how the eigenvector $\mathbf{u}_1$ of a NoN can be obtained from the spectral properties of the networks forming the ensemble. We then identify the optimal strategies that a network can follow when connecting to other networks and apply this methodology to population dynamics and epidemic spreading. We finally discuss the main concepts introduced in this chapter and point to possible problems to be tackled in the future.

4.2 The Influence of Interconnectivity on the Spectral Properties of an Interconnected Network

In this section we give analytical expressions for the spectral properties associated to a generic connectivity matrix $\mathbf{M}$, resulting from the connection of two initially isolated networks A and B [26]. The connectivity matrix is a weighted version of the classical adjacency matrix $\mathbf{A}$, where the component M_{ij} measures the strength of the connection between nodes i and j (and $M_{ij} = 0$ if i and j are not connected to each other). The aim is to gain a priori knowledge of the main spectral properties of the interconnected network by inspecting the structure of A and B, and the link(s) connecting both networks. Specifically, we are interested in: (i) the highest eigenvalue of the connectivity matrix and (ii) its associated eigenvector.

Figure 4.1 schematically represents two independent networks A and B, of N_A and N_B nodes and L_A and L_B links respectively, which initially form the disconnected network AB of $N_A + N_B$ nodes and $L_A + L_B$ links. Next, we connect both networks via a set $\{cl\}_{l=1,\ldots,L}$ of L connector links to create a *total interconnected network* T of $N_T = N_A + N_B$ nodes and $L_T = L_A + L_B + L$ links. The adjacency matrix $\mathbf{G_T}$ corresponding to network T is therefore formed by adding to the block diagonal network containing the original adjacency matrices of A and B, $\mathbf{G_{AB}}$, the connector links. For simplicity, let us suppose that $\mathbf{G_T}$ is symmetric, that is, the links of network T are bidirectional (this is tantamount to considering the initially isolated networks A and B to be symmetric and establishing interconnecting links that are bidirectional). Depending on the topological importance of the nodes that act as connectors between networks, four different strategies in the election of a connector link can be adopted: (a) peripheral-peripheral (PP), (b) peripheral-central (PC), (c) central-central (CC) and (d) central-peripheral (CP). Let us call $\lambda_{A,i}$ and $\lambda_{B,i}$ the i eigenvalues of the connectivity matrices $\mathbf{M_A}$ and $\mathbf{M_B}$ respectively, where i goes from 1 to the size of the corresponding network (N_A or N_B) with $i = 1$ corresponding to the largest eigenvalue and the rest following in decreasing order. The relation between connectivity matrices such as $\mathbf{M_A}$, $\mathbf{M_B}$ and $\mathbf{M_T}$ and the adjacency matrices such as $\mathbf{G_A}$, $\mathbf{G_B}$ and $\mathbf{G_T}$ depends on the peculiarities of the process. Let us suppose $\lambda_{A,1} > \lambda_{B,1}$ throughout the chapter, being the *strong* network the one with highest

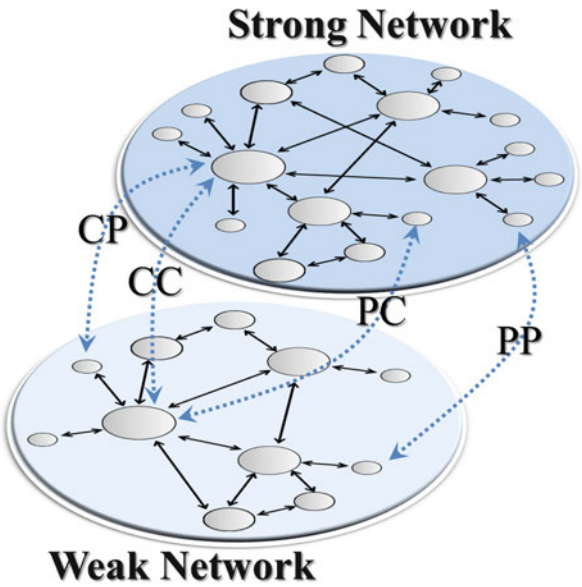

Fig. 4.1 Schematic representation of the different strategies for connecting two networks, according to the centrality of the connector nodes. The strong network is defined as the network with higher λ_1 (first eigenvector of the connectivity matrix **M**). Central nodes C are those with the highest eigenvector centrality, obtained from $\mathbf{u}_1$ (eigenvector associated to λ_1), while peripheral nodes P have the lowest centrality. Initially, the networks remain disconnected and, next, we connect them by adding connector links. According to the centrality of the connector nodes, four different strategies can be followed: (a) peripheral-peripheral (PP), (b) peripheral-central (PC), (c) central-central (CC) and (d) central-peripheral (CP)

λ_1 and the *weak* network the one with the lowest. This way, from now on, network A (B) will be the strong (weak) network.

We call $\mathbf{u}_{A,i}$ the N_A vectors of length N_T where the first N_A elements coincide with the eigenvector i of matrix $\mathbf{M_A}$ and the rest are equal to zero, while $\mathbf{u}_{B,i}$ are the N_B vectors of length N_T where the first N_A elements are zeros and the rest coincide with the eigenvector i of matrix $\mathbf{M_B}$. $\lambda_{T,i}$ and $\mathbf{u}_{T,i}$ are the eigenvalues and eigenvectors of matrix $\mathbf{M_T}$. The main idea of the analytical calculations is to describe the total graph T as a perturbation of graph A by graph B, in a way that the weight of the connector links is $\epsilon \ll 1$. Therefore, as $\lambda_{A,1} > \lambda_{B,1}$ by construction, the maximum eigenvalue $\lambda_{T,1}$ will be a perturbation of $\lambda_{A,1}$ and its associated eigenvector $\mathbf{u}_{T,1}$ will be a perturbation of $\mathbf{u}_{A,1}$. This methodology is inspired by the perturbation theory of matrices presented in [27], and among some other examples it was recently applied in the context of Complex Network Theory to characterize the importance of network nodes and links [28], and for the detection of communities [29]. We give

a fully detailed calculation in the Appendix, which ends up with

$$\mathbf{u}_{T,1} = \mathbf{u}_{A,1} + \epsilon \sum_{k=1}^{N_B} a_k \mathbf{u}_{B,k} + o(\epsilon^2)\,, \tag{4.1}$$

where $a_k = (\mathbf{u}_{A,1}\mathbf{P}\mathbf{u}_{B,k})/(\lambda_{A,1} - \lambda_{B,k})$, and $\mathbf{P}$ is a matrix representing the connector links in such a way that $\mathbf{M_T} = \mathbf{M_{AB}} + \epsilon\mathbf{P}$. Note that, since $\mathbf{u}_{A,1}\mathbf{P}\mathbf{u}_{B,1} = \sum (\mathbf{u}_{A,1})_i P_{ij} (\mathbf{u}_{B,1})_j$, only the connector nodes (i.e., those connected by P_{ij}) will contribute to this latter term. See [26] for more details and its application to the case of directed networks (i.e., asymmetric networks, with unidirectional links) and more than two networks.

4.3 Identifying Successful Strategies

The eigenvector $\mathbf{u}_{T,1}$ can be used to determine the outcome of a competition between networks A and B. In this section, we focus on how two networks compete for acquiring the maximum importance inside the interconnected network, while in the next sections we will discuss how to apply these concepts to other dynamical processes. The eigenvector centrality, which is given directly by the eigenvector $\mathbf{u}_{T,1}$ is used as a measure of the topological importance of a node. Subsequently, the centralities of networks A (C_A) and B (C_B) are obtained from the fractions of the total centrality that remain in the nodes of A and B after the connection:

$$C_A = \frac{\sum_{i=1}^{N_A} (\mathbf{u}_{T,1})_i}{\sum_{i=1}^{N_T} (\mathbf{u}_{T,1})_i}\,, \tag{4.2}$$

$$C_B = 1 - C_A\,. \tag{4.3}$$

Suppose that a networks's goal is to accumulate as much C as possible. Regarding Eqs. (4.1) and (4.2), and taking into account that $a_1 > a_{k+1}$, with $k \geq 1$ (since eigenvalues are ranked according to their value), the final outcome of the competition depends mainly on a_1: $\mathbf{u}_{T,1} \to \mathbf{u}_{A,1}$ when $a_1 \to 0$ and therefore, $C_A \to 1$, since the elements of $\mathbf{u}_{A,1}$ are zero for all nodes belonging to network B (see Appendix for details). Otherwise, C_B will grow when a_1 grows.

But, how does a_1 depend on networks A and B, and on the connector links? Inspecting the expression of a_1 (i.e., $a_1 = [\mathbf{u}_{A,1}\mathbf{P}\mathbf{u}_{B,1}]/[\lambda_{A,1} - \lambda_{B,1}]$) we can observe that it relies on two main factors: (i) the difference between the highest eigenvalues associated to both networks, $\lambda_{A,1}$ and $\lambda_{B,1}$, and (ii) $\mathbf{u}_{A,1}\mathbf{P}\mathbf{u}_{B,1}$, a quantity that is proportional to the centralities of the connector nodes when the networks are still disconnected, and to the number of connector links. Importantly, these two factors will control the distribution of centrality between the two competing networks. While (i) is independent of the connection strategy, (ii) depends crucially on the nodes that are chosen to establish connections between A and B.

This way, when the connector nodes are the most central (i.e., $\mathbf{u}_{A,1}\mathbf{P}\mathbf{u}_{B,1}$ is maximum), network B (the weakest) shows its best results in centrality. On the contrary, when the connector links join peripheral nodes of both networks, the value of a_1 reaches its minimum. Consequently, when $a_1 \to 0$, most centrality distributes over network A and therefore $\mathbf{u}_{T,1} \to \mathbf{u}_{A,1}$, leading to $C_A \to 1$. Finally, the larger the number of connector nodes, the higher the term $\mathbf{u}_{A,1}\mathbf{P}\mathbf{u}_{B,1}$, leading to an increase of a_1 and, as a consequence, to a decrease of C_A, indicating that the strong network does not benefit from multiple connections.

It is remarkable that the expression of $\mathbf{u}_{T,1}$ can be approximated, up to first order, to a linear combination of $\mathbf{u}_{A,1}$ and $\mathbf{u}_{B,1}$ (terms $k > 1$ in Eq. (4.1) are less relevant and mainly affect the connector nodes). In spite of the fact that the percentage of centrality captured by both networks is altered by introducing connector links, the distribution of centrality inside each network after the connection is therefore to some extent proportional to what it was before.

In summary, these results allow developing a general set of strategies that competitors A and B (with $\lambda_{A,1} > \lambda_{B,1}$) should follow in order to obtain as much centrality as possible after the connection. Recalling that the *strong* network is the one with the largest first eigenvalue, and the *weak* network the one with the smallest, the general rules to maximize the outcome of a network that competes for centrality tell us that:

- Connecting the most central nodes of two networks optimizes the centrality of the weak network.
- Connecting the most peripheral nodes of two networks optimizes the centrality of the strong network.
- Increasing the number of links reinforces the centrality of the weak network.

From all above, we stress that the goal of each competitor is not really to overcome the adversary, but to obtain the optimum outcome measured with the eigenvector associated to the largest eigenvalue of the interconnected network. Importantly, the strategy played by each network depends on whether its largest eigenvalue is higher or lower than its competitor, i.e., strong and weak networks must play different strategies to maximize its outcome.

4.4 Applications

As we have seen, the selection of connector nodes between networks strongly influences the eigenvector $\mathbf{u}_{T,1}$ of the interconnected network and how its elements are distributed between the two networks forming it. Since the eigenvector centrality of the nodes is given by the eigenvector $\mathbf{u}_{T,1}$, the competition for $\mathbf{u}_{T,1}$ between two

networks can be interpreted as a struggle for acquiring the highest possible centrality for the nodes inside a network. Interestingly, $\mathbf{u}_{T,1}$ may also contain information about the dynamical processes undergoing inside the network. In this section, we will show two particular examples in population dynamics and disease spreading. We will see how the previous strategies can be interpreted as a way of maximizing the outcome of a dynamical process and that this can be done by just looking at $\mathbf{u}_{T,1}$.

Population Dynamics

A variety of dynamical processes occurring on a network can be mathematically described as

$$\mathbf{n}(t+1) = \mathbf{M}\,\mathbf{n}(t)\,, \tag{4.4}$$

where $\mathbf{n}(t)$ is a vector whose components give the state of each node at time t (for example, the population of individuals at each node), and $\mathbf{M}$, with $M_{ij} \geq 0$, is a connectivity matrix that contains the peculiarities of the dynamical process (usually named as "transition matrix" in this context).

$\mathbf{M}$ is a primitive matrix. For this reason, its largest eigenvalue is positive, it verifies that $\lambda_1 > |\lambda_i|$, $\forall\ i > 1$, and its associated eigenvector is also positive (i.e., all its elements are positive). After t steps, the state of the system is given by

$$\mathbf{n}(t) = \mathbf{M}^t\mathbf{n}(0) = \sum_{i=1}^{m}(\mathbf{n}(0)\cdot\mathbf{u}_i)\lambda_i^t\mathbf{u}_i\,, \tag{4.5}$$

where $\mathbf{n}(0)$ is the initial condition, $\mathbf{u}_i$ the $i-$th eigenvectors of $\mathbf{M}$, and m the size of the network. As we consider $\boldsymbol{M}$ to be a real symmetric matrix, $\mathbf{u}_i$ for $i = 1, 2, \ldots, m$ can be conveniently chosen so as to form an orthonormal basis that permits the spectral decomposition above.

From Eq. (4.5) we obtain that the system evolves towards an asymptotic state independent of the initial condition and proportional to the first eigenvector $\mathbf{u}_1$,

$$\lim_{t\to\infty}\left(\frac{\mathbf{n}(t)}{(\mathbf{n}(0)\cdot\mathbf{u}_1)\lambda_1^t}\right) = \mathbf{u}_1\,, \tag{4.6}$$

while its associated eigenvalue λ_1 yields the growth rate at the asymptotic equilibrium. If $\mathbf{n}(t)$ is normalized such that $|\mathbf{n}(t)| = 1$ after each iteration, $\mathbf{n}(t) \to \mathbf{u}_1$ when $t \to \infty$. Therefore, there is a correspondence between the eigenvector centrality and the asymptotic state of the system at equilibrium: both quantities are proportional to the eigenvector $\mathbf{u}_1$ associated to the largest eigenvalue of the transition matrix $\mathbf{M}$.

Let us discuss one specific example showing the evolution of a population of genomes (e.g. RNA sequences) that duplicates and mutates inside a genotype

network, where each node represents a different sequence. Two nodes are linked if they differ in only one nucleotide, and therefore one sequence can evolve from one node to the other via point mutations. At each node i of the network, we consider a certain population n_i. At each time step: (i) the population n_i replicates with a growing rate $R > 1$, (ii) its daughter individuals leave the node with probability μ, being $0 < \mu \leq 1$, and (iii) the parameter S controls how probable it is for an individual to remain alive after leaving a node (see Fig. 4.2a for a qualitative description of the process). The transition matrix describing the evolution of the

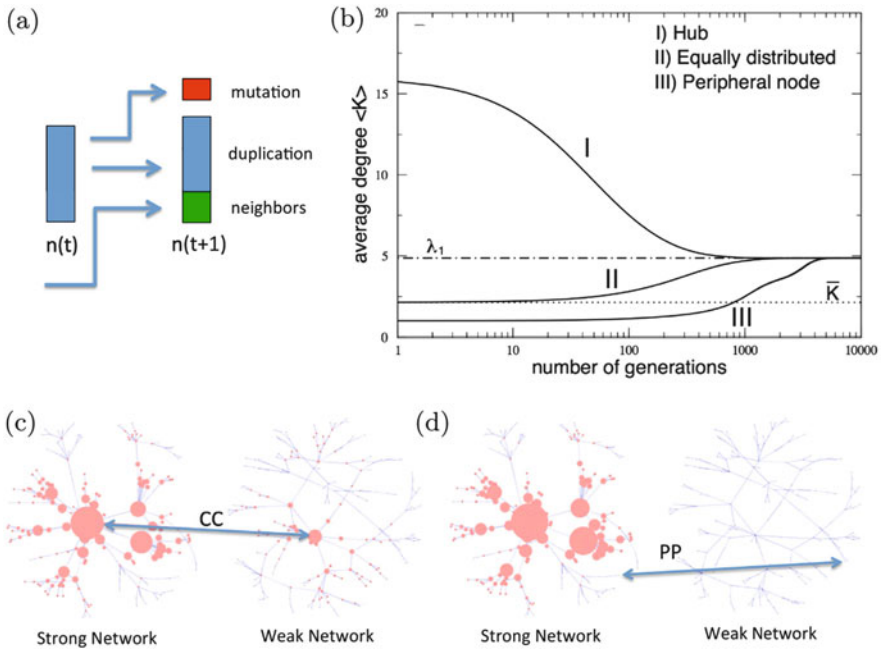

Fig. 4.2 Evolutionary dynamics of a population of genomes. **(a)** Schematic representation of the evolution of the vector state of the system $\mathbf{n}(t)$ when the population spreads on a single network of genomes. The population evolves through a duplication+mutation process, each node sending/receiving population from its neighbors when mutation occurs. **(b)** The evolution of the average degree $\langle K \rangle$ of the population shows that the final distribution is independent from the initial conditions, and higher that the average node degree $\bar{K}$. Three different initial distributions are considered: (*I*) the whole population placed at the most central node ("hub"), (*II*) uniformly distributed over the network, and (*III*) placed at the most peripheral node. **(c)** and **(d)** Evolution of the population when two networks are connected through the most central nodes (CC) and two peripheral nodes (PP) respectively. While in the CC configuration the weak network is able to retain 14.6 % of the population, in the PP case the population is almost completely absorbed by the strong network and only 10^{-4} % stays in the weak one. The parameter values are $\lambda_{A,1} = 1.9135$, $\lambda_{B,1} = 1.9109$, $R = 2$, $\mu = 0.1$, and $S = 36$ (Note that the networks used are artificial examples that verify the basic topological properties of genotype networks but do not represent real cases; see [30, 33] for more details)

system is given by [30]:

$$\mathbf{M} = (R - \mu)\mathcal{I} + \frac{\mu}{S}\mathbf{G}\,, \tag{4.7}$$

where $\mathbf{G}$ is the adjacency matrix (i.e., $G_{ij} = 1$ if nodes i and j are connected and zero otherwise) and $\mathcal{I}$ is the identity matrix (i.e., $\mathcal{I}_{ij} = 1$ if $i = j$ and zero otherwise).

Within this framework, the eigenvector $\mathbf{u}_1$ of the matrix $\mathbf{M}$ yields the final distribution of the population at the stationary state. As expected from the reasoning above, the final distribution does not depend on the initial conditions as illustrated in Fig. 4.2b, where the population is initially distributed in three different ways. Furthermore, the average degree of the stationary population $\langle K \rangle$ is given by the largest eigenvalue λ_1, which fulfills that $\lambda_1 \geq \bar{K}$, being $\bar{K}$ the average degree (number of connections) of the nodes in the network [31].

Now, let us analyze the evolution of the population when two (sub)networks are joined through a connector link. This situation could resemble, for example, the evolution on one very modular RNA neutral network (in [32] the high modularity of such networks was recently analyzed), or two different RNA neutral networks connected via a unique link, representing each neutral network A and B the total set of sequences that fold in two different secondary structures [33]. In Fig. 4.2c, d we observe that the election of the adequate link between those two networks has critical consequences on the population accumulated at each network. Following the rules explained in the previous section, the weak network benefits from the CC connection, acquiring 14.6 % of the total population ($C_B = 0.146$). This is the best outcome that the weak network would be able to achieve when connecting through one link. On the contrary, when the PP strategy is followed, the strong network absorbs the majority of the population and the weak network remains virtually empty ($C_B = 10^{-6}$) .

Spreading Processes: The SI Model

The highly developed mathematical modeling and statistical physics analysis of spreading processes have successfully described the existence of, for example, fixed points, phase transitions or spreading thresholds [34]. Among the different examples of spreading, such as rumor spreading or packet transmission through the WWW, disease spreading has been studied the most [35]. The prediction of disease evolution and the dynamics of contagions have been analyzed with a diversity of models which combine both the state of the system at different scales (from the individual to the whole population) and the structure of connections between individuals [34]. In this section our focus will be on how the structure of the network of contacts between individuals affects the probability of individuals being infected by a disease. Several works have investigated how the network topology constrains the epidemic dynamics and, more specifically, the outbreak of a disease and the

properties of the epidemics in equilibrium [12, 13]. Nevertheless, less attention has been paid to the fact that social networks are typically organized in modules (or subnetworks), which interact between them through certain connector links. What is the effect of the connector links on the spreading of a disease through different subnetworks? As we are going to see, the concepts and tools defined in the previous sections will help us to understand this issue. With this aim, we are going to implement a specific disease model, the Susceptible-Infected (SI) model [36], over two networks that interact by creating interlinks as it is explained in Fig. 4.1. But first of all let us describe in detail the model and its implementation on a single network.

The SI model distinguishes two different states of the individuals: Susceptible (S) of acquiring the disease and Infected (I). When a susceptible individual (i.e., a person prone to be infected) meets an infected one, it will acquire the disease with a certain probability, which is controlled by the spreading rate β:

$$S + I \xrightarrow{\beta} 2I, \tag{4.8}$$

Next, we construct a network where the nodes are individuals and the links account for interactions between them. The connectivity matrix $\mathbf{M}$ of the network contains the connections between individuals (i.e., $M_{ij} = 1$ if two individuals are connected, and zero otherwise). The probability that a node (i.e., a person) k becomes infected is given by $I_k(t)$, while $S_k(t) = 1 - I_k(t)$ is the probability of it being susceptible (i.e., not infected). The network structure strongly influences the probability that node k becomes infected between times t and $t + dt$, as it is proportional to the number of neighbors that are already infected $\beta \sum_j M_{kj} I_j$. Since only susceptible individuals can get infected, the dynamics of $S_k(t)$ and $I_k(t)$ can be described by a set of N differential equations, N being the total number of individuals:

$$\frac{dS_k}{dt} = -\beta S_k \sum_j M_{kj} I_j = -\beta S_k \sum_j M_{kj}(1 - S_j), \tag{4.9}$$

$$\frac{dI_k}{dt} = \beta S_k \sum_j M_{kj} I_j = \beta (1 - I_k) \sum_j M_{kj} I_j, \tag{4.10}$$

with $S_k + I_k = 1$. If the disease starts from a small number of nodes, in the limit of large system size N and ignoring quadratic terms, Eq. (4.10) becomes:

$$\frac{dI_k}{dt} = \beta \sum_j M_{kj} I_j, \tag{4.11}$$

which in matrix form reads

$$\frac{d\mathbf{I}}{dt} = \beta \mathbf{M}\mathbf{I}, \tag{4.12}$$

$\mathbf{I}$ being a vector of components I_k. The temporal evolution of $\mathbf{I}$ can be expressed as a linear combination of the eigenvectors $\mathbf{u}_k$ of the connectivity matrix $\mathbf{M}$:

$$\mathbf{I}(t) = \sum_{k=1}^{N} a_k(t)\mathbf{u}_k, \tag{4.13}$$

where $\mathbf{u}_k$ is the eigenvector associated with the eigenvalue λ_k of $\mathbf{M}$. Then

$$\frac{d\mathbf{I}(t)}{dt} = \sum_{k=1}^{N} \frac{da_k(t)}{dt}\mathbf{u}_k = \beta \mathbf{M} \sum_{k=1}^{N} a_k(t)\mathbf{u}_k = \beta \sum_{k=1}^{N} \lambda_k a_k(t)\mathbf{u}_k. \tag{4.14}$$

Comparing the terms that multiply $\mathbf{u}_k$, we obtain:

$$\frac{da_k}{dt} = \beta \lambda_k a_k, \tag{4.15}$$

which has the solution

$$a_k(t) = a_k(0)e^{\beta \lambda_k t}. \tag{4.16}$$

If we substitute Eq. (4.16) into Eq. (4.13) we obtain the following expression for $\mathbf{I}(t)$:

$$\mathbf{I}(t) = \sum_{k=1}^{N} a_k(0)e^{\beta \lambda_k t}\mathbf{u}_k. \tag{4.17}$$

Since the largest eigenvalue λ_1 dominates over the others, we can approximate the infected population as

$$\mathbf{I}(t) \sim e^{\beta \lambda_k t}\mathbf{u}_1. \tag{4.18}$$

Thus, for $t \to \infty$ the exponential term leads to $\mathbf{I} \to 1$, i.e. the whole population gets infected at the final state. Nevertheless, for low to intermediate time scales ($t \ll \infty$), it is $\mathbf{u}_1$, i.e. precisely the eigenvector centrality of the nodes, that controls the distribution of probabilities of getting infected.

As explained in Sect. 4.2, the properties of the eigenvector $\mathbf{u}_{T,1}$ when two networks A and B are connected depend on the kind of interlink. If we consider two networks of individuals and want to understand how the distribution of the probability of being infected depends on the kind of connection between the two

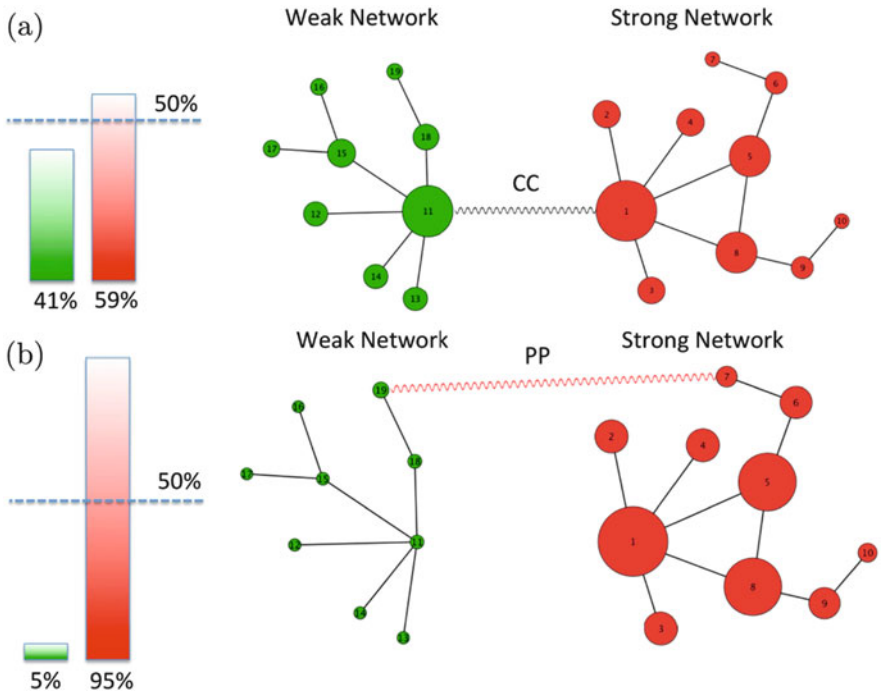

Fig. 4.3 Probability of being infected by a disease (SI model), at the beginning of the spreading process, for two interconnected networks. Two social networks based on romantic connections between young students [37] are connected through a **(a)** CC and **(b)** PP connection. Node size is proportional to the probability of being infected (obtained from $\mathbf{u}_{T,1}$) and *bars* indicate the percentage of infection risk accumulated by each network. The PP strategy leads the strong network to increase its risk of infection as compared to the weak network

networks, all strategies defined in Sect. 4.3 apply. The only difference is that, since the terms C_A and C_B are related to the probability of being infected at low to moderate times after the beginning of the epidemics, the aim of the networks will be to reduce this probability instead of increasing it. Therefore, the strategies are exactly the same as in the case of network centrality or population dynamics, but they must be applied in the opposite way.

In Fig. 4.3 we show an example of how spreading processes on interconnected networks are strongly dependent on the way the networks are linked between them. We consider two social networks based on the romantic relationships between students in an American high school [37]. Specifically, we select two subnetworks that are isolated and evaluate how a connection between one student of each subnetwork affects the probability of being infected by a disease throughout the network ensemble. With this aim, we introduce a SI spreading process with an infection rate $\beta = 0.1$ in the networks and calculate the fraction of the first eigenvector that lies within each subnetwork for a CC and a PP connection. In

this particular example, the largest eigenvalues of the strong and the weak network are, respectively, $\lambda_{A,1} = 2.69$ and $\lambda_{B,1} = 2.39$. Figure 4.3a, b shows how the probability of being infected at short to moderate times is always higher in the strong network. Nevertheless, when the two most peripheral nodes of both networks are connected (Fig. 4.3b) the probability that the strong network gets infected increases dramatically. This is a situation that the strong network has to avoid, since it gets much more vulnerable to the disease than the weak network. Therefore, the PP (CC) connection is now the most harmful strategy for the strong (weak) network, while the CC (PP) connection is the safest one.

4.5 Conclusions

In this chapter we have shown that the way networks interact to form interconnected networks and, more specifically, how they choose the connector links, can have important consequences on the structural and dynamical properties of the networks [26]. A series of dynamical processes occurring on interacting networks, such as population dynamics or disease spreading, can be explained from the analysis of the spectral properties of the transition matrix, which in its turn depends on the way the networks are coupled. We have seen that it is possible to define strategies that maximize the outcome (defined in terms of the dynamical process under consideration) acquired by a certain network. As a general strategy, strong networks (i.e. those with the highest largest eigenvalue) will benefit from establishing connections between peripheral nodes. Weak networks, instead, obtain a higher benefit when the central nodes of both networks are elected as connectors.

Throughout this chapter we considered that the network's goal is to accumulate as much percentage of $\mathbf{u}_1$ as possible. However, in some cases, networks may want to minimize it as in the case of disease spreading.

It is important to stress that the proposed methodology applies for processes where the final state of the system is given by the eigenvector associated to the largest eigenvalue of the transition matrix. For example, this is not the case for diffusion processes where the system dynamics is described by the (weighted) Laplacian matrix $\mathbf{L}$, obtained as $\mathbf{L} = \mathbf{W} - \mathbf{M}$, where $\mathbf{W}$ is a diagonal matrix with W_{ii} containing the total weight of node i [38].

The spectral properties of the Laplacian matrix also determine the stability of the synchronization manifold in the complete synchronization of networked systems [39]. Nevertheless, the influence of the connection strategies in the spectral properties of the Laplacian are much more difficult to interpret than in the case of the transition matrix, as there is no straightforward relation between the spectral properties of both matrices. The reader is referred to Ref. [40] for a detailed theoretical, numerical and experimental study of the effect of different connection strategies on the synchronization of an ensemble of networks.

Finally, there are other dynamical aspects that may not be explained by the analysis of either the transition or the Laplacian matrix. For example, it is expected

that the complexity of the global dynamics in networks of dynamical systems that are coupled through different connection strategies will be affected by the strategy adopted in creating connections, but it is not clear at this point whether this may be related to the spectral properties of any matrix representing the coupling topology of the system. These and other problems related to networks of networks are still open and will have to be addressed in the future, showing that network interconnection is a promising subfield of network theory with potential applications in several branches of science.

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Appendix

Networks A and B, of N_A and N_B nodes and L_A and L_B links respectively, form the initially disconnected network AB of $N_A + N_B$ nodes and $L_A + L_B$ links. We connect them through L connector links to create a new interconnected network T of $N_T = N_A + N_B$ nodes and $L_T = L_A + L_B + L$ links. For convenience, the nodes of network A are numbered from $i = 1$ to N_A and the nodes of network B from $i = N_A + 1$ to $N_T = N_A + N_B$. The adjacency matrix $\mathbf{G_{AB}}$ of the disconnected network consists of two diagonal blocks corresponding to $\mathbf{G_A}$ and $\mathbf{G_B}$. The relation between the transition matrix $\mathbf{M_{AB}}$, also formed by two blocks, and $\mathbf{G_{AB}}$, depends on the peculiarities of the process. Note that the eigenvectors of $\mathbf{M_A}$ and $\mathbf{M_B}$ are related to those of $\mathbf{M_{AB}}$ as follows: Let us call $\mathbf{x}_{A,i}$ ($i = 1, \ldots, N_A$) and $\mathbf{x}_{B,j}$ ($j = 1, \ldots, N_B$) the eigenvectors associated to the eigenvalues $\lambda_{A,i}$ and $\lambda_{B,j}$ of matrices $\mathbf{M_A}$ and $\mathbf{M_B}$ respectively. Note that the N_A eigenvectors $\mathbf{x}_{A,i}$ are of length N_A, the N_B eigenvectors $\mathbf{x}_{B,j}$ are of length N_B, and the eigenvectors of $\mathbf{M_{AB}}$ are of length N_T. The first $i = 1, \ldots, N_A$ eigenvectors of $\mathbf{M_{AB}}$ verify $(\mathbf{u}_{AB,i})_k = (\mathbf{x}_{A,i})_k$ for $k \leq N_A$ and $(\mathbf{u}_{AB,i})_k = 0$ for $k > N_A$. Therefore, $\lambda_{AB,i} = \lambda_{A,i}$ for $i = 1, \ldots, N_A$. The eigenvectors $i = N_A + 1, \ldots, N_T$ of $\mathbf{M_{AB}}$ verify $(\mathbf{u}_{AB,i})_k = 0$ for $k \leq N_A$ and $(\mathbf{u}_{AB,i})_k = (\mathbf{x}_{B,i})_{k-N_A}$ for $k > N_A$. Therefore, $\lambda_{AB,i} = \lambda_{B,i-N_A}$ for $i = N_A + 1, \ldots, N_T$. For simplicity in the following calculations, due to their evident relation with the eigenvectors of $\mathbf{M_A}$, we denote eigenvectors $\mathbf{u}_{AB,i}$ for $i = 1, \ldots, N_A$ as $\mathbf{u}_{A,i}$. Analogously, we denote $\mathbf{u}_{AB,i+N_A}$ for $i = 1, \ldots, N_B$ as $\mathbf{u}_{B,i}$.

Considering the addition of interlinks as represented by the symmetric matrix $\mathbf{P}$ (with non-zero entries in the off-diagonal blocks of elements (i, j) with $i \leq N_A$ and $j > N_A$ and $i > N_A$ and $j \leq N_A$) to be a small perturbation of parameter ϵ, and Taylor-expanding the largest eigenvalue of M_T and its associated eigenvector around those of M_{AB}, we obtain

$$\mathbf{M_T}\mathbf{u}_{T,1} = \lambda_{T,1}\mathbf{u}_{T,1} \tag{4.19}$$

where

$$\mathbf{M_T} = \mathbf{M_{AB}} + \epsilon \mathbf{P}, \tag{4.20}$$

$$\mathbf{u}_{T_1} = \mathbf{u}_{A,1} + \epsilon \mathbf{v}_1 + \epsilon^2 \mathbf{v}_2 + o(\epsilon^3), \tag{4.21}$$

$$\lambda_{T,1} = \lambda_{A,1} + \epsilon t_1 + \epsilon^2 t_2 + o(\epsilon^3). \tag{4.22}$$

Taking into account that (i) $|\mathbf{u}_{T,1}| = 1 \Rightarrow \mathbf{u}_{A,1} \cdot \mathbf{v}_1 = 0$ and $\mathbf{u}_{A,1} \cdot \mathbf{v}_2 = 0$, and (ii) $\mathbf{u}_{A,1}\mathbf{P}\mathbf{u}_{A,1} = 0$ because $(\mathbf{u}_{A,1})_i = 0$ for $i > N_A$, we include Eqs. (4.20–4.22) in Eq. (4.19), premultiply by $\mathbf{u}_{A,1}$ and equate the terms of the same order in ϵ. Considering that point (i) above, in its turn, implies that $\mathbf{v}_1$ and $\mathbf{v}_2$ can be expressed as linear combinations of the other eigenvectors of M_T, which are orthogonal to $\mathbf{u}_{A,1}$, and therefore $\mathbf{u}_{A,1} \cdot \mathbf{M_{AB}}\mathbf{v}_1 = 0$ and $\mathbf{u}_{A,1} \cdot \mathbf{M_{AB}}\mathbf{v}_2 = 0$, we obtain to first order in ϵ

$$\mathbf{u}_{A,1} \cdot (\mathbf{M_{AB}}\mathbf{v}_1 + \mathbf{P}\mathbf{u}_{A,1}) = \mathbf{u}_{A,1} \cdot (\lambda_{A,1}\mathbf{v}_1 + t_1\mathbf{u}_{A,1}) \tag{4.23}$$

$$\Rightarrow t_1 = 0 \tag{4.24}$$

$$\Rightarrow (\mathbf{M_{AB}} - \lambda_{A,1})\mathbf{v}_1 = -\mathbf{P}\mathbf{u}_{A,1}\,, \tag{4.25}$$

and for order ϵ^2

$$\mathbf{u}_{A,1} \cdot (\mathbf{M_{AB}}\mathbf{v}_2 + \mathbf{P}\mathbf{v}_1) = \mathbf{u}_{A,1} \cdot (\lambda_{A,1}\mathbf{v}_2 + t_2\mathbf{u}_{A,1}) \Rightarrow t_2 = \mathbf{u}_{A,1}\mathbf{P}\mathbf{v}_1. \tag{4.26}$$

The vector $\mathbf{v}_1$ can be numerically obtained solving Eq. (4.25). However, it can also be analytically expressed as

$$\mathbf{v}_1 = \sum_{k=1}^{N_T} c_k \mathbf{u}_{AB,k} = \sum_{k=1}^{N_A} c_k \mathbf{u}_{A,k} + \sum_{k=N_A+1}^{N_T} c_k \mathbf{u}_{B,k-N_A}. \tag{4.27}$$

We know $c_1 = 0$ because $\mathbf{u}_{A,1} \cdot \mathbf{v}_1 = 0$. Including Eq. (4.27) in Eq. (4.25), and multiplying both sides by $\mathbf{u}_{AB,k}$ from the left, we obtain $c_k = 0$ for $1 < k \leq N_A$ (because $\mathbf{u}_{A,k}\mathbf{P}\mathbf{u}_{A,1} = 0\ \forall k$) and $c_k = \frac{\mathbf{u}_{A,1}\mathbf{P}\mathbf{u}_{B,k-N_A}}{\lambda_{A,1}-\lambda_{B,k-N_A}}$ for $k > N_A$. All this yields

$$\mathbf{v}_1 = \sum_{k=1}^{N_B} \frac{\mathbf{u}_{A,1}\mathbf{P}\mathbf{u}_{B,k}}{\lambda_{A,1} - \lambda_{B,k}} \mathbf{u}_{B,k}\,, \tag{4.28}$$

and including Eqs. (4.28) and (4.26) in Eq. (4.22), we finally obtain

$$\mathbf{u}_{T,1} = \mathbf{u}_{A,1} + \epsilon \sum_{k=1}^{N_B} \frac{\mathbf{u}_{A,1}\mathbf{P}\mathbf{u}_{B,k}}{\lambda_{A,1} - \lambda_{B,k}} \mathbf{u}_{B,k} + o(\epsilon^2)\,, \tag{4.29}$$

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