

# Modeling Epidemic Risk Perception in Networks with Community Structure

Franco Bagnoli<sup>1,2,3(✉)</sup>, Daniel Borkmann<sup>4</sup>, Andrea Guazzini<sup>5,6</sup>,  
Emanuele Massaro<sup>7</sup>, and Stefan Rudolph<sup>8</sup>

<sup>1</sup> Department of Energy, University of Florence, Florence, Italy

<sup>2</sup> Center for the Study of Complex Systems, University of Florence, Florence, Italy

<sup>3</sup> National Institute for Nuclear Physics, Florence Section, Florence, Italy  
`franco.bagnoli@unifi.it`

<sup>4</sup> Communication Systems Group, ETH Zurich, Zurich, Switzerland

<sup>5</sup> National Research Council, Institute for Informatics and Telematics, Pisa, Italy

<sup>6</sup> Department of Psychology, University of Florence, Florence, Italy

<sup>7</sup> Department of Computer Science and Systems, University of Florence,  
Florence, Italy

<sup>8</sup> Organic Computing Group, University of Augsburg, Augsburg, Germany

**Abstract.** We study the influence of global, local and community-level risk perception on the extinction probability of a disease in several models of social networks. In particular, we study the infection progression as a susceptible-infected-susceptible (SIS) model on several modular networks, formed by a certain number of random and scale-free communities. We find that in the scale-free networks the progression is faster than in random ones with the same average connectivity degree. For what concerns the role of perception, we find that the knowledge of the infection level in one’s own neighborhood is the most effective property in stopping the spreading of a disease, but at the same time the more expensive one in terms of the quantity of required information, thus the cost/effectiveness optimum is a tradeoff between several parameters.

**Keywords:** Risk perception · SIS model · Complex networks

## 1 Introduction

Epidemic spreading is one of the most successful and most studied applications in the field of complex networks. The comprehension of the spreading behavior of many diseases, like sexually transmitted diseases (i.e. HIV) or the H1N1 virus, can be studied through computational models in complex networks [4, 20]. In addition to “real” viruses, spreading of information or computer malware in technological networks is of interest as well.

The susceptible-infected-susceptible (SIS) model is often used to study the spreading of an infectious agent on a network. In this model an individual is represented as a node, which can be either be “healthy” or “infected”. Connections between individuals along which the infection can spread are represented

by links. In each time step a healthy node is infected with a certain probability if it is connected to at least one infected node, otherwise it reverts to a healthy node (parallel evolution).

The study of epidemic spreading is a well-known topic in the field of physics and computer science. The dynamics of infectious diseases has been extensively studied in scale-free networks [2, 6, 8, 24], in small-world networks [19] and in several kind of regular and random graphs.

A general finding is that it is hard to stop an epidemic in scale-free networks with slow tails, at least in the absence of correlations in the network among the infections process and the node characteristics [24]. This effect is essential due to the presence of hubs, which act like strong spreaders. However, by using an appropriate policy for hubs, it is possible to stop epidemics also in scale-free networks [2, 9].

This network-aware policy is inspired by the behavior of real human societies, in which selection had lead to the development of strategies used to avoid or reduce infections. However, human societies are not structureless, thus a particular focus must be devoted to the community structures, which are highly important for our social behavior.

Recently, a wave of studies focused the attention on the effect of the community structure in the modelling of epidemic spreading [7, 25, 26]. However, the focus was only set towards the interaction between the viruses' features and the topology, without considering the important relation between cognitive strategies used by subjects and the structure of their (local) community/neighborhood.

Considering this scenario, an important challenge is the comprehension of the structure of real-world networks [14, 15, 21]. Given a graph, a community is a group of vertices that is "more linked" within the group than with the rest of the graph. This is clearly a poor definition, and indeed, in a connected graph, there is not a clear distinction between a community and a rest of the graph. In general, there is a continuum of nested communities whose boundaries are somewhat arbitrary: the structure of communities can be seen as a hierarchical dendrogram [22].

It is generally accepted that the presence of a community structure plays a crucial role in the dynamics of complex networks; for this reason, lots of energy has been invested to develop algorithms for the detection of communities in networks [10, 12, 13]. However, in complex networks, and in particular in social networks, it is very difficult to give a clear definition of a community: nodes often belong to more than just one cluster or module. The problem of overlapping communities was exposed in [23] and recently analyzed in [17]. People usually belong to different communities at the same time, depending on their families, friends, colleagues, etc. For instance, if we want to analyze the spreading of sexual diseases in a social environment, it is important to understand the mechanism that leads people to interact with each other. We can surely detect two distinct groups of people (i.e., communities): heterosexual and homosexual, with bisexual people that act as overlapping vertexes between the two principal communities [1, 7, 18].

The strategies used to face the infection spreading in a community is itself a complex process (i.e., social problem solving) in which strategies spread (as the epidemics) along the community, and are negotiated and assumed or discarded depending on their social success.

Several factors can affect the social problem solving which is represented by the adoption of a behaviour to reduce the infection risk. Of course, personality factors, previous experiences and the social and economical states of a subject can be considered as influencing variables. Another important variable is represented by the structure of the environment in which the social communities live, because it determines at the same time the speed of the epidemic diffusion and the strategy of the negotiation process; in particular large and more connected communities are often characterized by conservative strategies while small and isolated communities allow more relaxed strategies.

The same strategy can be more or less effective depending on the strategies adopted by the neighbours (community) of the subject. For instance, a subject in a conservative community can adopt a more risky (and presumably profitable) attitude with a certain confidence since he would be protected from the infection because of their neighbours' behaviours. This "parasitic" behavior (like refusing vaccinations) can be tolerated up to a certain level without lowering the community's fitness.

Not only the neighbor's behaviours affect the evolution of the cognitive strategies of a subject, but also the position he has in the network should be a relevant factor. A hub, or a subject with a great social betweenness, is usually more exposed to the infection than a leaf, and as a consequence, the best strategy for him has to be different. In the same way, since the topology of the network (e.g., small world, random) determines variables such as the speed of the spreading, or its pervasiveness, it should also affect the development of the "best strategy".

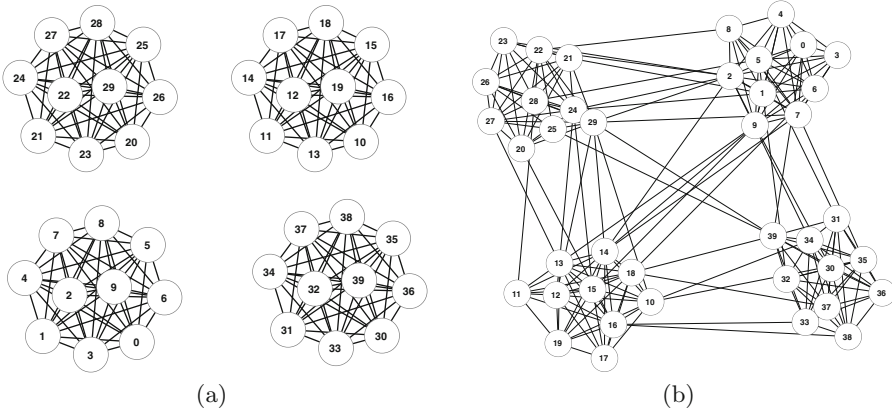
Moreover, while the negotiation process evolves, the cognitive strategies usually develop within the most intimate community of a subject, thus the behaviour adopted by subjects could be an interesting feature for the community detection problem as well.

The understanding of the effects of the community structure on the epidemic spreading in networks is still an open task. In this paper we investigate the role of risk perception in artificial networks, generated in order to reproduce several types of overlapping community structures.

The rest of this paper is organized as follows: we start by describing a mechanism for generating networks with overlapping community structures in Sect. 2. In Sect. 3, we describe the SIS model adopted to model the risk perception of subjects in those networks. Finally, Sect. 4 contains simulation results from our model with a throughout discussion and future work proposals.

## 2 The Networks Model

There are  $n_c$  different communities with  $n_v$  vertices (in this paper we consider only undirected and unweighted graphs); we assume that the probability to



**Fig. 1.** (a) An example network with 4 different communities composed by 10 vertices: in this case, considering  $p_1 = 1$  and  $p_2 = 0$ , we generate 4 non-interconnected fully connected networks. (b) The same 4 communities with parameters  $p_1 = 0.95$  and  $p_2 = 0.05$ .

have a link between the vertices in the same community is  $p_1$ , while  $p_2$  is the probability to have a link between two nodes belonging to different communities. For instance, with  $p_1 = 1$  and  $p_2 = 0$ , we generate  $n_c$  fully connected graphs, with no connections among them as shown in Fig. 1(a). It is possible to use the parameters  $p_1$  and  $p_2$  to control the interaction among different communities, as shown in Fig. 1(b). The algorithm for generating this kind of networks can be summarized as:

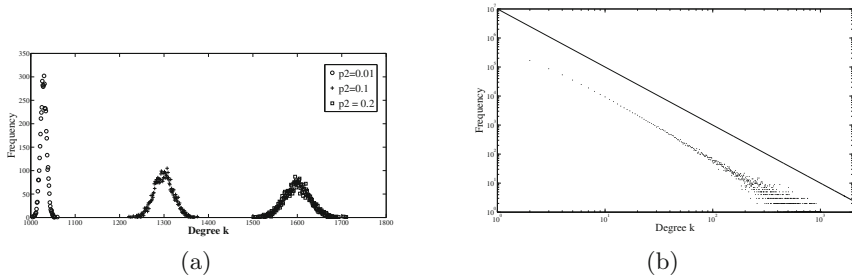
1. Define  $s_1$  as number of vertices in the communities;
2. Define  $n_c$  as number of communities;
3. For all the  $n_c$  communities create a link between the vertices on them with probability  $p_1$ ;
4. For all the vertices  $N = s_1 n_c$  create a link between them and a random vertex of other communities with probability  $p_2$ ;

Constraining the condition  $p_1 = 1 - p_2$ , we can reduce the free parameters to just one. The connectivity degree itself depends on the size of the network and on the probabilities  $p_1$  and  $p_2$ . In particular, the connectivity function  $f(k)$  has a normal distribution from which we could define the mean connectivity  $\langle k \rangle$  as

$$\langle k \rangle = (s_1 - 1)p_1 + (n_c - 1)s_1 p_2 \tag{1}$$

with standard deviation  $\sigma^2(k) = (s_1 - 1)p_1(1 - p_1) + (n_c - 1)s_1 p_2(1 - p_2)$ .

In Fig. 2(a) we show the frequency distribution of the connectivity degree of nodes varying the value of the parameter  $p_2$  for a network composed by  $N = 5000$  nodes and  $n_c = 5$  communities.



**Fig. 2.** (a) Random networks: in this figure, we show the frequency distribution of the connectivity degree changing the value of the parameter  $p_2$ . The circles represent the values for  $p_2 = 0.01$ , crosses for  $p_2 = 0.1$  and eventually squares for  $p_2 = 0.2$ . Here,  $s_1 = 1000$  and  $n_c = 5$ , thus we have generated networks with 5 communities of 1000 nodes for each. (b) Distribution of connectivity degree for the scale-free network generated with the mechanism described above (dots). The straight line is a power law curve with exponent  $\gamma = 2.5$ .

It is widely accepted that real-world networks from social networks to computer networks are scale-free networks, whose degree distribution follows a power law, at least asymptotically. In this network, the probability distribution of contacts often exhibits a power-law behavior:

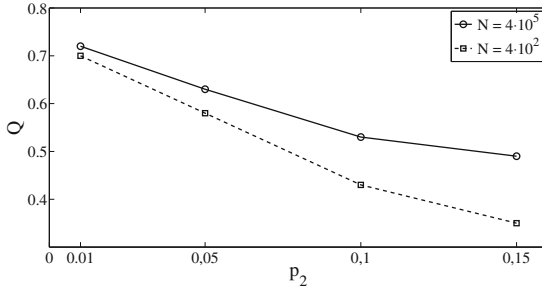
$$P(k) \propto ck^{-\gamma}, \tag{2}$$

with an exponent  $\gamma$  between 2 and 3 [3,11]. For generating networks with this kind of characteristics, we adopt the following mechanism:

1. Start with a fully connected network of  $m$  nodes;
2. Add  $N - m$  nodes;
3. For each new node add  $m$  links;
4. For each of these links choose a node at random from the ones already belonging to the network and attach the link to one of the neighbors of that node, if not already attached.

Through this mechanism we are able to generate scale-free networks with an exponent  $\gamma = 2.5$  as shown in Fig. 2(b). There, we show the frequency distribution of the connectivity degree for a network of  $10^6$  nodes. To generate a community structure with a realistic distribution, we first generate  $n_c$  scale-free networks as explained above. Then, for all nodes and all outgoing links, we replace the link pointing inside the community with that connecting a neighbor of a random node in a random community with a probability of  $p_2 = 1 - p_1$ . Thus, the algorithm can be summarized as:

1. Generate  $n_c$  communities as scale-free networks with  $n_v$  vertices;
2. For all the vertices, with a probability  $p_2 = 1 - p_1$ ;
  - Delete a random link;



**Fig. 3.** Different values of modularity ( $Q$ ) after increasing the mixing parameter  $p_2$  for two networks with  $N = 4 \cdot 10^5$  nodes and  $N = 4 \cdot 10^2$  nodes.

- Select a random node of another community and create a link with one of its adjacent vertex;

3. End.

In this way, we are able to generate scale-free networks with a well defined community structure. A good measure for the estimation of the strength of the community structure is the so-called modularity [15]. The modularity  $Q$  is defined to be:

$$Q = \frac{1}{2} \sum_{vw} \left[ A_{vw} - \frac{K_v K_w}{2m} \right] \delta(c_v, c_w), \tag{3}$$

where  $A$  is the adjacency matrix in which  $A_{vw} = 1$  if  $w$  and  $v$  are connected and 0 otherwise.  $m = \frac{1}{2} \sum_{vw} A_{vw}$  is the number of edges in the graph,  $K_i$  is the connectivity degree of node  $i$  and  $(K_v K_w)/(2m)$  represents the probability of an edge existing between vertices  $v$  and  $w$  if connections are made at random but with respecting vertex degrees.  $\delta(c_v, c_w)$  is defined as follows:

$$\delta(c_v, c_w) = \sum_r^{n_c} \hat{c}_{vr} \hat{c}_{wr} \tag{4}$$

where  $\hat{c}_{ir}$  is 1 if vertex  $i$  belongs to group  $r$ , and 0 otherwise.

In Fig. 3 we show the values of modularity for two networks that were generated with the same algorithm, but with different sizes. Here, we consider a network with 4 communities: in the first case  $s_1 = 10^5$ , while in the second case  $s_1 = 10^2$ . What one can observe in Fig. 3 is that the modularity’s behaviour does not change significantly for different network sizes with the same number of communities.

In the case of scale-free networks, the mean connectivity degree  $\langle k \rangle$  is fixed a priori when we choose the number of links the new nodes create. In the case of random networks the mean connectivity is given by Eq. 1.

### 3 The Risk Perception Model

We use the susceptible-infected-susceptible model (SIS) [1, 24] for describing an infectious process. In the SIS model, nodes can be in two distinct states: healthy and ill. Let us denote by  $\tau$  the probability that the infection can spread along a single link. Thus, if node  $i$  is susceptible and it has  $k_i$  neighbors of which  $s_n$  are infected, then, at each time step, node  $i$  will become infected with the probability:

$$p(s, k) = [1 - (1 - \tau)^{s_n}]. \quad (5)$$

We model the effect of risk perception considering the global information of the infection level for the whole network, the information about the infected neighbors and the information about the average state of the community. Thus, the risk perception for the individual  $i$  is given by:

$$I_i = \exp \left\{ -H + J_1 \left( \frac{s_{ni}}{k_i} \right) + J_2 \left( \frac{s_{ci}}{n_{ci}} \right) \right\}, \quad (6)$$

where  $H = J(s/N)$  is the perception about the global network on which  $s$  is the total number of infected agents while  $N$  is the number of agents in the network. The second term of the Eq. 6 represents the perception about the neighborhood, while the third term represents the perception about the local community of the agent  $i$ .

In this model, we assume that people receive information about the network's state through examination of people in the neighborhood. The global information could refer to entities like media while the information about the community could be assumed as *word of mouth*. In this paper, we don't consider the cost that people should pay in order to get these information, but it is clearly an important constraint to consider in future works.

The risk perception  $I_i$ , defined in Eq. 6, is assumed to determine the probability that the agents meet someone in its neighbourhood. The algorithm is given by:

1. For all nodes  $i = 1, 2, \dots, N$ ;
2. For all its neighbors  $j = 1, 2, \dots, k_i$ ;
3. If  $I_i > rand$ ;
- $i$  meets  $j$ ;
- If  $j$  at time  $t - 1$  was infected then  $i$  becomes ill with probability  $\tau$ ;
4. End.

Then, we propose a gain function defined as the number of meetings in time considering different values of  $j = J, J_1, J_2$  and different kind of scale-free and random networks; the gain function  $G(j)$  is given by:

$$G(j) = \frac{\sum_{t=1}^{T_e} M_t}{T_e}, \quad (7)$$

in which  $T_e$  is the time for the extinction, while  $M_t$  is the number of meetings during time. Based on that, we can eventually define a fitness function that

considers the probability to extinct the epidemic in the given time. Thus, the fitness function is given by:

$$F_j^T = G(j)P_e(j) \quad (8)$$

It is possible to make a mean-field approximation of this model. Pastor-Satorras and Vespignani defined the mean-field equation for scale-free networks in [24]. In 2010, Kitchovitch and Liò [16] modeled the mean number of infected neighbors  $g(k)$  for individuals  $i$  with connectivity degree  $k$ . In fact, given the probability of receiving an infection by at least one of the infected neighbors (Eq. 5), it is possible to define the rate of change of the fraction of individuals  $i$  with degree  $k$  at time  $t$  by the following:

$$\frac{di_k}{dt} = -\gamma + (1 - i_k)g(k), \quad (9)$$

on which  $\gamma$  is the rate of recovery (in our simulations we set  $\gamma = 1$ ).

Then, as shown by Boccaletti et al. [5], for any node, the degree distribution of any of its neighbors is,

$$q_k = \frac{kP(k)}{\langle k \rangle}, \quad (10)$$

hence, it is possible to define the number of infected neighbors as:

$$i_n = \sum_{K_{min}}^{k_{max}} q_k i_k, \quad (11)$$

and it allows to give a definition of  $g(k)$  as:

$$g(k) = \sum_{s=0}^k \binom{k}{s} p(s, k) i_n^s (1 - i_n)^{k-s}, \quad (12)$$

where  $s = s_n$  is the number of infected neighbors.

The temporal behavior of the mean fraction  $c$  of infected individuals in the case of a network with fixed connectivity is given by:

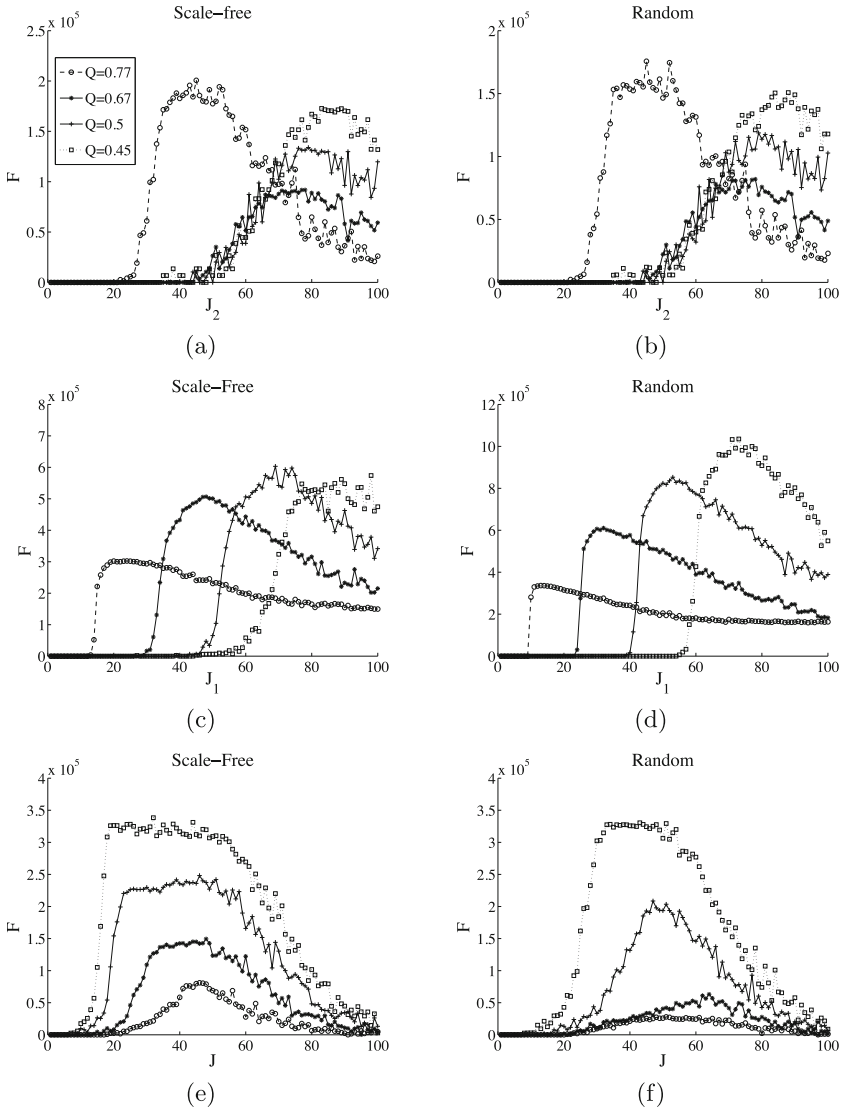
$$c' = \sum_{s_n=1}^k \binom{k}{s_n} c^{s_n} (1 - c)^{k-s_n} [1 - (1 - \tau)^{s_n}], \quad (13)$$

where  $c \equiv c(t)$ ,  $c' \equiv c(t + 1)$  and the sum runs over the number  $k_{inf}$  of infected individuals.

## 4 Results and Discussion

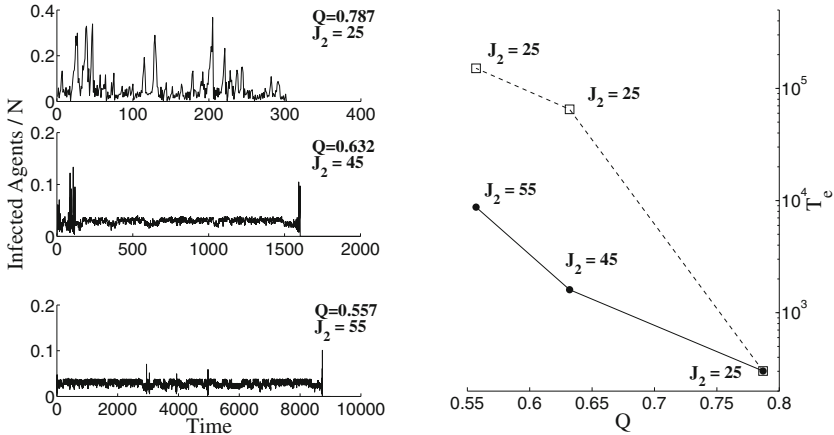
We studied the behavior of our model for different scenarios. In Fig. 5, we show results considering a network of 500 nodes and 5 communities where the initial number of infected agents is  $\approx 10\%$  of all agents in the network. We focus on





**Fig. 4.** Effect of the parameters  $J_2$  (a),  $J_1$  (b) and  $J$  (c) (*x-axis*) on the fitness function  $F$  (*y-axis*) considering different scale-free and random networks with different values of modularity. Results are averaged over 100 simulations for each value of  $J$ ,  $J_1$  and  $J_2$ .

the information about the community (parameter  $J_2$ ), while we kept  $J = J_1 = 1$  fixed. It is very interesting to observe the time necessary for the extinction of the epidemics, with the probability of being infected  $\tau = 0.5$  and changing the community structure of the network.



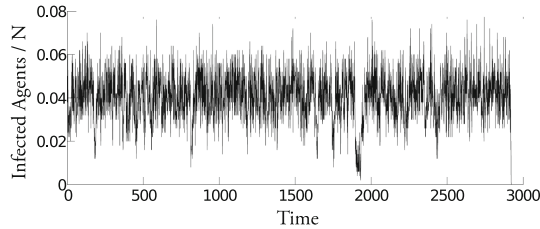
**Fig. 5.** On the left side of the figure we show the temporal evolution of infected individuals by varying the mixing parameter  $p_2$ . The time necessary for the epidemic extinction increases as the modularity  $Q$  decreases. On the right side, we show the effects of the precaution parameter  $J_2$  on the extinction time by varying the modularity  $Q$ . The straight line represents the results for different value of  $J_2$ , while the dashed line represents the results for a constant value of  $J_2$ .

The effects of the parameters  $J_2$ ,  $J_1$  and  $J$  on the fitness function  $F$  considering different scale-free and random networks with different values of modularity are shown in Fig. 4. The results were averaged over 100 simulations for each value of  $J$ ,  $J_1$  and  $J_2$ .

On the left side of Fig. 5 we show the temporal evolution of the percentage of infectious agents for different kind of networks and different values of  $J_2$ . We can observe that the extinction time increases when the modularity of network decreases, even if we use higher values of  $J_2$ . On the right side of Fig. 5, we show the effect of the precaution on the extinction time. The straight line corresponds to different values of  $J_2$ , while the dotted line corresponds to the same value of  $J_2$  in different kind of networks. It is also possible to observe that when a network becomes *less clustered*, the information about the community becomes less important.

In the case of scale-free networks, the mean connectivity degree  $\langle k \rangle$  is related to the number  $m$  of links the new nodes create. In the above example, considering  $m = 5$ , we obtained a mean connectivity degree  $\langle k \rangle = 7.8$ .

For comparisons, we generated random networks with a mean connectivity degree  $\langle k \rangle \in (7, 8)$ . The first result that we obtained is that the extinction time is larger than in the scale-free case. In Fig. 6, we show the temporal evolution of the infected agents for a random network with modularity  $Q = 0.78$  considering  $J_2 = 25$  as in the upper plot on the left side of Fig. 5. For the scale-free network the time necessary for the extinction is  $T_e \simeq 3 \cdot 10^2$  while for the random one it is  $T_e \simeq 3 \cdot 10^3$ .



**Fig. 6.** Percentage of infected agents for a random network of 100 nodes and 5 communities with modularity  $Q = 0.78$ . Adopting the same parameter used for the simulation reported in Fig. 5, we show how the time for the extinction (approx. 2900 units) of the epidemic is greater than for the scale-free case (i.e., upper plot on the left side of Fig. 5).

**Table 1.** Critical values for the extinction of the epidemic on case of scale-free networks of 500 nodes and 5 communities considering a maximum threshold time  $T_{max} = 1000$ , necessary for the extinction of the epidemics.

$Q$ (modularity)	Critical Values		
	$J$	$J_1$	$J_2$
0.78	45	15	25
0.64	40	15	45
0.35	40	20	55

Regarding the effects of the global and local (neighborhood) information, we investigated scale-free networks composed by 500 nodes and 5 communities with a fixed maximum threshold time  $T_{max}$ , necessary for the extinction of the epidemics. We assume  $T_{max} = 1000$  and separately measure critical values of  $J, J_1$  and  $J_2$ . In the Table 1, we show the critical values of the three parameters by changing the modularity  $Q$ . As we can observe, the most variable parameter is  $J_2$  while the other two parameters do not appear to change. From this figure, we observe that the information about the fraction of infected neighbors is the most effective for stopping the disease. However, in order to get this piece of information, each node needs to check the status of all its neighbours, a task that can be quite hard and possibly conflicting with privacy. On the other hand, the information on the average infectious level in the community or in the whole population is more easily obtained. Therefore, one needs to add the cost of information into the model in order to decide what the most effective solution for risk perception is.

Summarizing, we have studied the progression and extinction of a disease in a SIS model over modular networks, formed by a certain number of random and scale-free communities. The infection probability is modulated by a risk perception term (modeling the probability of an encounter). This term depends on the global, local and community infection level. We found that in scale-free networks the progression is slower than in random ones with the same average

connectivity. For what concerns the role of perception, we found that the local one (information about infected neighbours) is the most effective for stopping the spreading of the disease. However, it is also the piece of information that requires most efforts to be gathered, and therefore it may result a high cost/efficacy ratio.

The main element of originality of this paper is that we introduced a network model based on communities, which still retains the scale-free structure with the possibility of changing the modularity, and we think that this structure (albeit being quite theoretical) is more realistic than standard scale-free networks. The fact the knowledge about own community is more effective than other indicators is surely trivial (and we expected to get this result), but it is also the information that is more expensive to get, at least for the standard data gathering existing today. We would like to quantify the advantage in using this indicator in order to compare its efficiency with respect to its cost (and for doing it we need to include a cost model, that will be done in a future work) and also point to the necessity of gathering this kind of local information, that in a real case may also present problems related to the privacy, but might be of great importance in the case of a pandemic.

In regard to extending the model by inserting a cost model, it should also be taken into account what the best strategies are to avoid the spreading of epidemics in different environments considering agents as intelligent entities capable to change or select the best strategies dynamically in order to minimize the risk and to maximize the economy of the system. In combination to this, we plan to add a more complex model such as the SIR eventually with vaccinations, for which there are important factors like the penetration and the possibility that the modular structure may be exploited to “shield” a community that may remain not exposed – similarly like people that refuse vaccinations, but are “shielded” by a surrounding community that vaccinates.

**Acknowledgments.** We acknowledge funding from the 7th Framework Programme of the European Union under grant agreement n° 257756 and n° 257906.

## References

1. Anderson, R.M., May, R.M.: *Infectious Diseases of Humans Dynamics and Control*. Oxford University Press, Oxford (1992)
2. Bagnoli, F., Liò, P., Sguanci, L.: Risk perception in epidemic modeling. *Phys. Rev. E* **76**, 061904 (2007)
3. Barabási, A.L., Albert, R.: Emergence of scaling in random networks. *Science* **286**, 509–512 (1999)
4. Barrat, A., Barthlemy, M., Vespignani, A.: *Dynamical Processes on Complex Networks*. Cambridge University Press, New York (2008)
5. Boccaletti, S., Latora, V., Moreno, Y., Chavez, M., Hwang, D.-U.: Complex networks: structure and dynamics. *Phys. Rep. Fervier* **424**(4–5), 175–308 (2006)
6. Boguñá, M., Pastor-Satorras, R., Vespignani, A.: Absence of epidemic threshold in scale-free networks with degree correlations. *Phys. Rev. Lett.* **90**, 028701 (2003)

7. Chen, J., Zhang, H., Guan, Z.-H., Li, T.: Epidemic spreading on networks with overlapping community structure. *Phys. A: Stat. Mech. Appl.* **391**(4), 1848–1854 (2012)
8. Cohen, R., ben Avraham, D., Havlin, S.: Percolation critical exponents in scale-free networks. *Phys. Rev. E* **66**, 036113 (2002)
9. Dezső, Z., Barabási, A.-L.: Halting viruses in scale-free networks. *Phys. Rev. E* **65**(5), 055103+ (2002)
10. Van Dongen, S.: Graph clustering via a discrete uncoupling process. *SIAM. J. Matrix Anal. Appl.* **30**, 121–141 (2009)
11. Dorogovtsev, S.N., Mendes, J.F.F., Samukhin, A.N.: Structure of growing networks with preferential linking. *Phys. Rev. Lett.* **85**, 4633–4636 (2000)
12. Dorso, C., Medus, A.D.: Community detection in networks. *Int. J. Bifurcat. Chaos* **20**(2), 361–367 (2010)
13. Fortunato, S.: Community detection in graphs. *Phys. Rep.* **486**(3–5), 75–174 (2010)
14. Fortunato, S., Castellano, C.: *Community Structure in Graphs*, December 2007
15. Girvan, M., Newman, M.E.J.: Community structure in social and biological networks. *Proc. Natl. Acad. Sci., USA* **99**, 7821–7826 (2002)
16. Stephan, K., Pietro, L.: Risk perception and disease spread on social networks. *Procedia Comput. Sci.* **1**(1), 2339–2348 (2010)
17. Lancichinetti, A., Fortunato, S., Kertész, J.: Detecting the overlapping and hierarchical community structure of complex networks. *New J. Phys.* **11**, 033015 (2009)
18. Liljeros, F., Edling, C.R., Amaral, L.A., Stanley, E.H., Åberg, Y.: The web of human sexual contacts. *Nature* **411**(6840), 907–908 (2001)
19. Moore, C., Newman, M.E.J.: Epidemics and percolation in small-world networks. *Phys. Rev. E* **61**, 5678–5682 (2000)
20. Newman, M.: *Networks: An Introduction*. Oxford University Press Inc., New York (2010)
21. Newman, M.E.J.: Detecting community structure in networks. *Europ. Phys. J. B.* **38**, 321–330 (2004)
22. Newman, M.E.J., Girvan, M.: Finding and evaluating community structure in networks. *Phys. Rev. E* **69**, 026113 (2004)
23. Palla, G., Derényi, I., Vicsek, T.: *Nature* **435**, 814 (2005)
24. Pastor-Satorras, R., Vespignani, A.: Epidemic dynamics and endemic states in complex networks. *Phys. Rev. E* **63**, 066117 (2001)
25. Salathé, M., Jones, J.H.: Dynamics and control of diseases in networks with community structure. *PLoS Comput. Biol.* **6**(4), e1000736 (2010)
26. Saumell-Mendiola, A., Serrano, M.A., Bogu ná, M.: Epidemic spreading on inter-connected networks. [arXiv:1202.4087](https://arxiv.org/abs/1202.4087) (2012)