# Probing Complexity with Epidemics: A New Reactive Immunization Strategy

E. Alfinito[1], M. Beccaria[2], A. Fachechi[3] and G. Macorini[4]
[1]*Dipartimento di Ingegneria dell'Innovazione, Università del Salento, Campus Ecotekne, 73100 Lecce, Italy*
[2,3,4]*Dipartimento di Matematica e Fisica E. De Giorgi, Università del Salento & INFN, Via Arnesano, 73100 Lecce, Italy*

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Abstract: Epidemic evolution on complex networks strongly depends on their topology and the infection dynamical properties, as highly connected nodes and individuals exposed to the contagion have competing roles in the disease spreading. In this spirit, we propose a new immunization strategy exploiting the knowledge of network geometry and dynamical information about the spreading infection. The flexibility and effectiveness of the proposed scheme are successfully tested with numerical simulations on a wide set of complex networks.

## 1 INTRODUCTION

Epidemic processes and the related immunization problem is a very hot topic in statistical mechanics of complex networks (Pastor-Satorras et al., 2015). The central issue in the realization of an effective vaccination program is in fact the identification of the most relevant features in the infection spreading. This is in general a highly non-trivial problem, so that novel immunization programs, looking at some particular aspects of the complex system, have been realized. According to their application and the required knowledge about the system, immunization programs can be roughly classified in two opposite approaches. The first one is the *preventive* case, which aims to make the healthy network stronger against future epidemic events by using (global or local) information about its topology. This is the case of Targeted Immunization (TI) (Pastor-Satorras and Vespignani, 2002), Acquaintance Immunization (AI) (Cohen et al., 2003) as well as their variations and refinements (Stauffer and Barbosa, 2006; Hu and Tang, 2012). On the other hand, the *reactive* approach is designed for dealing with an already spreading infection. In this case, topological knowledge and information about the epidemic state can be combined so that a dynamical reaction can be carried out, see for example (Ruan et al., 2012; Altarelli et al., 2014; Liu et al., 2014; Perra et al., 2012; Yan et al., 2014; Pereira and Young, 2015). A paradigmatic example of this approach is the so-called High-Risk Immunization (HR) (Nian and Wang, 2010), in which only individuals interacting with infected nodes are vaccinated. In some real situations, only the reactive setting is possible, for example to control unexpected disease outbreaks or, in a technological context, to fight malware diffusion. In such situations, a good control of epidemic contagion needs a fast intervention, so it is more convenient to have an immunization program working well on the largest possible set of networks. Unfortunately, known immunization strategies have been usually designed to work in very specific settings. We propose a new immunization strategy which generalizes TI in order to take into account the presence of an already propagating epidemic. Our claim is that the most relevant individuals in the epidemic diffusion have to be identified according to a clever balance between network topological features and the epidemic state at the vaccination time. We test the effectiveness of our protocol with a Monte Carlo implementation of SIR model (Kermack and McKendrick, 1927; May and Anderson, 1979) and extensively compare it with standard immunization strategies on a variety of complex (theoretical and real) networks.

## 2 THEORETICAL BACKGROUND

### 2.1 Implementation of SIR Model

In the SIR model (Kermack and McKendrick, 1927), individuals are classified according to their condition in susceptible ($S$), infected ($I$) and recovered ($R$). Epi-



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demic dynamics is therefore represented by the transitions $S \to I$ and $I \to R$. In our implementation of SIR model, the initial condition is a single infected node (*patient zero*) in the network. At each step, an infected individual is randomly selected and recovers with probability $p_{\mathrm{SIR}}$. If this is not the case, one of its susceptible neighbors is randomly chosen and gets infected. The immunization takes place as a reaction to the epidemic spreading when a given fraction $f$ (the *epidemic threshold*) of the total population is infected. In the procedure, the fraction $g$ of individuals to be vaccinated is selected according to some score assigned to each node, which results in a priority list for the nodes to be vaccinated. The spreading algorithm goes on as long as infected individuals are present. Once the system reaches its final state, we consider the final density $d_R$ of recovered individuals as a measure of total number of individuals involved in the epidemic. Clearly, an immunization strategy is effective if it significantly reduces the density $d_R$ by immunizing a relatively small fraction $d_V$ of the population. Since the evolution of the system is stochastic, we perform a large number of simulations and the effectiveness of a given strategy is measured in terms of the averages $\langle d_R \rangle$ and $\langle d_V \rangle$.

## 2.2 A New Immunization Strategy

Motivated by the issues discussed in the introduction, we propose a local refinement of TI. Specifically, our score takes into account both static knowledge of the network geometry and dynamical information about the epidemics, and it is given by

$$s_i = d_i + \sum_{j \in N_i} \left[ \beta \frac{\delta_{j,I}}{(d_j)^{1/2}} + \gamma \frac{\delta_{j,S}}{d_i} \frac{d_i - d_j}{d_i + d_j} \right]. \qquad (1)$$

Here, $N_i$ is the $i$-th node neighborhood, $d_i$ its connectivity degree and $\delta_{j,I}$ and $\delta_{j,S}$ are Kronecker deltas selecting respectively infected or susceptible nodes. Finally, $\beta$ and $\gamma$ are tunable free parameters. Clearly, the $\beta$-term favors the vaccination of nodes *near* the epidemic front, where the factor $(d_j)^{-1/2}$ is justified by the fact that less connected neighbors constitute bottlenecks for the epidemic spreading. The $\gamma$-term is a further improvement based on the *leverage centrality* (Joyce et al., 2010).

## 2.3 Standard Immunization Strategies

In our numerical tests, we extensively compare our proposal with the following standard immunization strategies, suitably modified in the reactive setting:

- **Targeted Immunization (TI)**. Originally designed for scale-free networks, this scheme selects nodes to be immunized among the susceptible ones according to their degree.
- **Acquaintance Immunization (AI)**. In this case, random neighbors of randomly selected nodes are vaccinated (if susceptible). This is a degree-based vaccination strategy using only local information about the network.
- **High Risk Immunization (HR)**. Nodes to be immunized are chosen among the susceptible neighbors of infected nodes.

# 3 COMPLEX NETWORKS

In this section, we briefly describe the networks we considered as background for the epidemic spreading. They range from the standard theoretical cases to a collection of real world systems.

## 3.1 Theoretical Networks

In the first class, we consider the Barabàsi-Albert (Albert and Barabási, 2002; Bornholdt and Schuster, 2006) and the Watts-Strogatz (Albert and Barabási, 2002; Bornholdt and Schuster, 2006; Watts and Strogatz, 1998) models, which are respectively the prototypes of scale-free and small-world graphs. We denote with BA[$Q$] the Barabàsi-Albert graph obtained adding $Q$ new links at each step of the growth algorithm. On the other hand, we indicate as WS[$Q$] the Watts-Strogatz graph obtained starting with a regular network with each node connected to $2Q$ adjacent sites. The rewiring probability in constructing WS graphs is denoted by $\theta$. In this paper, we consider BA[2] and WS[2] networks with two different rewiring probabilities $\theta = 0.1, 0.5$ for the WS case. Since some real networks are believed to be approximately or locally scale-free, we also propose two variants of Barabàsi-Albert model. The first one is realized starting with a BA graph and then randomly rewiring $\mathcal{R}$ of its links. This variant can be thought as a model that interpolates, depending on $\mathcal{R}$, between the pure scale-free networks and the completely random graphs. We start with a BA[2] network and consider $\mathcal{R} = 500, 1000, 2000$ rewiring events. The second variant is realized starting with a network with $\mathcal{N}$ nodes equally distributed in $m$ BA centers. The centers are then further randomly connected adding new links between individuals in different clusters. In this case, we start with $m = 5, 10, 20$ BA clusters with $\mathcal{N} = 5000$ total nodes and add $k = 100, 500, 2000$ new edges.

Table 1: Some common graph metrics for the real networks considered in the MC simulations: in the four columns we report the mean value, variance, maximum and minimum for the degree, vertex eccentricity, closeness centrality and betweenness centrality.

| **CA-HepTh-pruned** | Degree | Eccentricity | Clos. centr. | Bet. centr. |
|---|---|---|---|---|
| mean | 5.74809 | 12.5302 | 0.171894 | 21357.1 |
| variance | 41.7448 | 1.07741 | 0.000597495 | $3.32398\times10^{9}$ |
| max | 65. | 18. | 0.247167 | $1.15586\times10^{6}$ |
| min | 1. | 10. | 0.0813124 | 0. |
| **p2p-Gnutella08** | Degree | Eccentricity | Clos. centr. | Bet. centr. |
| mean | 6.5966 | 7.16003 | 0.218169 | 11471.8 |
| variance | 72.9622 | 0.423737 | 0.000621478 | $4.16777\times10^{8}$ |
| max | 97. | 9. | 0.295973 | 305191. |
| min | 1. | 6. | 0.147952 | 0. |
| **AA** | Degree | Eccentricity | Clos. centr. | Bet. centr. |
| mean | 4.74715 | 8.01616 | 0.227506 | 1836.35 |
| variance | 77.3613 | 0.857017 | 0.00110411 | $7.90946\times10^{7}$ |
| max | 145. | 11. | 0.35615 | 213598. |
| min | 1. | 6. | 0.13556 | 0. |
| **Internet_AS** | Degree | Eccentricity | Clos. centr. | Bet. centr. |
| mean | 4.18991 | 7.15294 | 0.282169 | 14661. |
| variance | 1095.36 | 0.359583 | 0.00163744 | $6.53432\times10^{10}$ |
| max | 2389. | 10. | 0.474276 | $2.03136\times10^{7}$ |
| min | 1. | 5. | 0.141305 | 0. |
| **ProteinYeast** | Degree | Eccentricity | Clos. centr. | Bet. centr. |
| mean | 2.73388 | 13.275 | 0.150965 | 4234.31 |
| variance | 12.2956 | 1.68203 | 0.000581309 | $1.38406\times10^{8}$ |
| max | 56. | 19. | 0.234773 | 225922. |
| min | 1. | 11. | 0.0791633 | 0. |

## 3.2 Real Networks

Finally, the set of real networks we consider in this paper is the following:

1. **Internet_AS**, 11174 nodes, 23408 links. Undirected unweighted Internet Network[1] (Colizza et al., 2006) at the Autonomous System level. Nodes represent Internet service providers and links connections between them. Data were collected by the Oregon Route Views Project (http://www.routeviews.org/) in May 2001.
2. **AA**, 1057 nodes, 2502 links. Interactions between metabolites of E. coli during the metabolic cycle[2] (Jeong et al., 2000). We consider the **AA** case.
3. **CA-HepTh-pruned**, 8638 nodes, 24836 links. Arxiv HEP-TH (High Energy Physics - Theory) collaboration network[3] from the e-print arXiv. A paper is represented as a completely connected subgraph in which nodes are its authors.
4. **p2p-Gnutella08**, 6300 nodes, 20776 links. Sequence of snapshots of the Gnutella file sharing network from August 2002.[4] Nodes are hosts of Gnutella network and links connections between them.
5. **ProteinYeast**, 1870 nodes, 2350 links. Protein Interaction Network[5] (Jeong et al., 2001).

Further information about the collection of real networks can be found in Table 1, in which some standard metrics are reported.

# 4 RESULTS

The discussion of our results is focused on the ability of the various immunization strategies to reduce the epidemic prevalence $\langle d_R \rangle$ as function of the vaccinated fraction of the population. In particular we use the 50% and 25% of the original (*i.e.* without vaccination) prevalence as references (horizontal dotted lines in the plots).

[1] https://sites.google.com/site/cxnets/research222
[2] http://www3.nd.edu/ networks/resources/metabolic/
[3] http://snap.stanford.edu/data/ca-HepTh.html

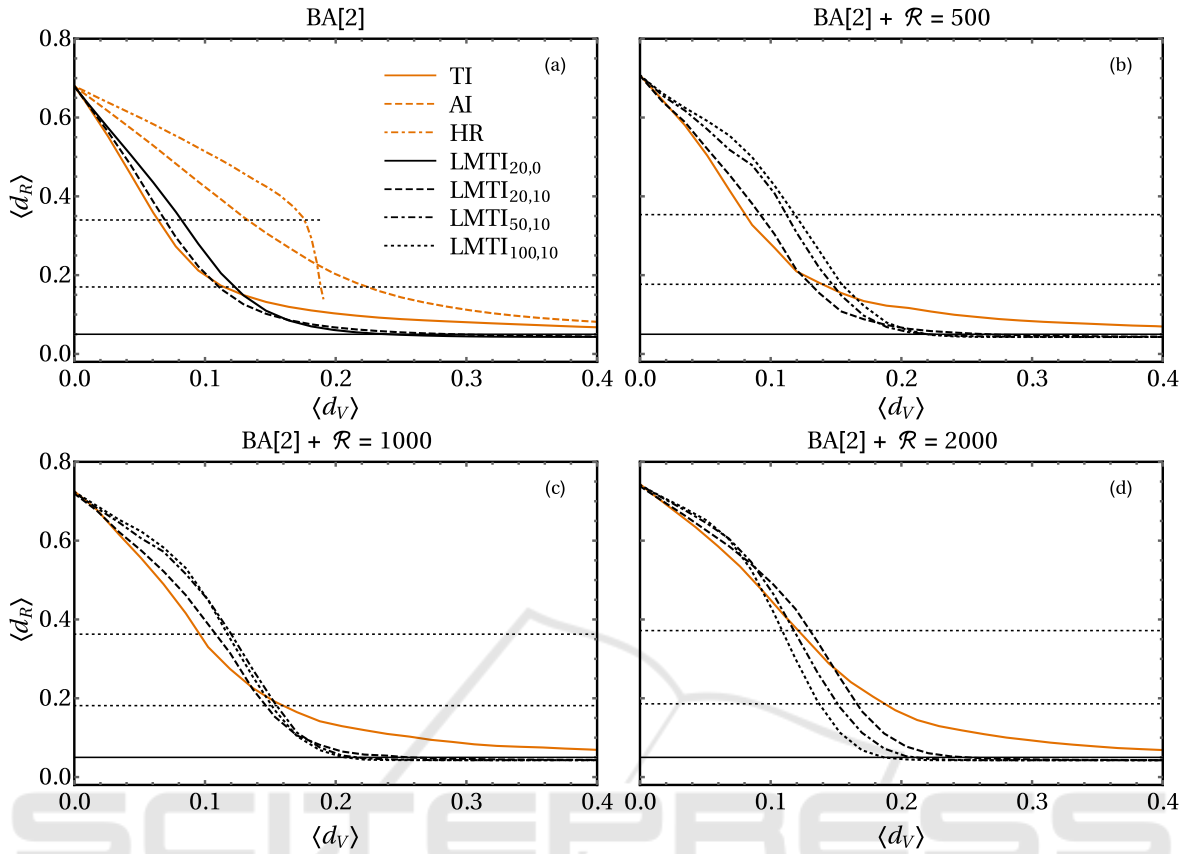


Figure 1: Results for various immunization strategies for randomly rewired BA[2] networks with $\mathcal{N} = 1000$ nodes and $\mathcal{R} = 0$ (pure BA case, a), 500 (b), 1000 (c), 2000 (d) rewiring events. The LMTI scheme is compared to TI, AI and HR strategies for the pure BA case, while various choice of $\beta$ and $\gamma$ are compared with TI only for non zero $\mathcal{R}$. The horizontal solid line is the epidemic threshold $f = 0.05$, while the horizontal dotted lines are 25% and 50% of the mean final density of recovered without any vaccination.

## 4.1 Barabási-Albert Setting

Let us first consider the pure BA case, see panel (a) in Fig. 1. Degree-based immunizations are clearly the most effective ones in this setting. In particular, the reduction of the epidemic prevalence by 50% is better accomplished with TI. Besides, our strategy (with the best choice $\beta = 20$ and $\gamma = 10$) has similar performances at low $\langle d_V \rangle$. If we want to cut the prevalence down to the 25%, TI and LMTI are almost identical, but the latter is most advisable to further reduce the epidemic strength. The results for partially randomized BAs are collected in panels (b), (c) and (d) of Fig. 1. Here, we compare LMTI for different choices of $\beta$ and $\gamma$ only with TI, as they are the best performers in the original setting. The first visible feature is the gradual worsening of the TI performances for increasing $\mathcal{R}$. This behavior has a simple explanation: the random rewiring procedure works in opposite direction with respect to the preferential attachment of the BA growth algorithm, and the role of big spreaders in the original setting is here gradually downsized. In this setup, the importance of local terms in (1) can be better appreciated and immunization of the individuals close to the epidemic front becomes more and more important in controlling the outbreak.

Another interesting result holds for the randomly connected BAs, see Fig. 2. In this setting, the reduction to the 50% of the epidemic prevalence is generically best suited with TI, while the cutting to the 25% is roughly equivalent for the two strategies (considering the best choice $\beta = 20$ and $\gamma = 10$ for LMTI). However, LMTI always allows to stop the epidemic for a sufficiently large vaccinated fraction (which increase for higher $k$). TI generally appears more effective than LMTI for low values of $\langle d_V \rangle$. This can be explained noticing that for increasing $k$ with fixed $m$ or increasing $m$ with fixed $k$, the connection of each node with individuals belonging to other BA centers

[4] http://snap.stanford.edu/data/p2p-Gnutella08.html
[5] http://www3.nd.edu/ networks/resources/protein/bo.dat.gz

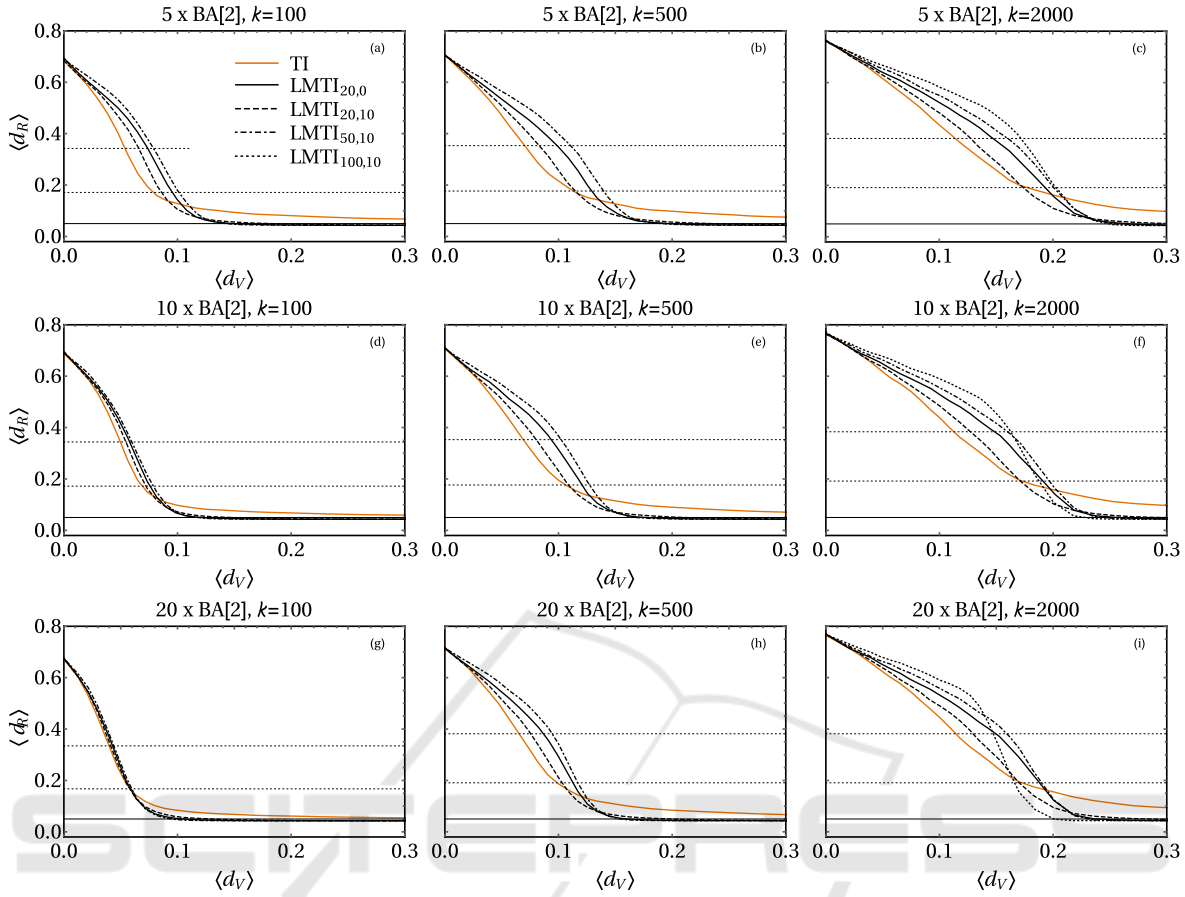


Figure 2: Results for various immunization strategies for randomly connected BA[2] clusters with $\mathcal{N} = 5000$ total nodes. The number of BA centers is $m = 5$ (a,b,c), 10 (d,e,f), 20 (g,h,i), each with $k = 100, 500, 2000$ new links. and $\mathcal{R} = 0$ (pure BA case, a), 500 (b), 1000 (c), 2000 (d) rewiring events. The LMTI scheme is compared to TI only. The horizontal solid line is the epidemic threshold $f = 0.05$, while the horizontal dotted lines are 25% and 50% of the mean final density of recovered without any vaccination.

acquires more and more importance. Thus, it is more likely that the epidemic spreads on the whole network rather than to be localized in the initial cluster, therefore acquiring an extended front. In this case it is convenient to adopt a non-local immunization strategy, unless there is a sufficient vaccinations availability to surround the infection. Finally, an interesting aspect is that choices with higher $\beta$ are more effective in stopping the epidemic as the network becomes highly connected and clustered. This is clear from Fig. 2 for $k = 2000$, where LMTI with the choice $\beta = 100$ and $\gamma = 10$ allows to stop the epidemic for $\langle d_V \rangle = 0.25$ in networks with $m = 5$ centers and for $\langle d_V \rangle = 0.20$ for $m = 20$ initial clusters.

## 4.2 Watts-Strogatz Setting

Fig. 3 collects the results for Watts-Strogatz networks. They are radically different than the BA setting. As a consequence of absence of hubs, a pure TI is indeed a poor strategy in WS graphs. On the other hand, our score shows a much better adaptability. The presence of the local terms is fundamental in reducing the epidemic prevalence, giving to the LMTI curves a profile with a rapid fall-off. The only strategy giving comparable results for small $\theta$ is HR, which was the worst performer for BA. Moreover, HR performances are highly sensitive to the $\theta$ value and dramatically worsen for $\theta = 0.5$. An interesting aspect of our strategy is the role of the leverage term. In fact, for WS network keeping a residual regular structure ($\theta = 0.1$), the $\gamma$-term is totally ineffective. Moreover, it becomes even deleterious for highly randomized WS ($\theta = 0.5$). The choice $\beta = 20$ and $\gamma = 0$ is by far the best performer in all WS settings, always allowing to stop the epidemic with a relatively low vaccinations (note that all the other immunization strategies fail in doing this).

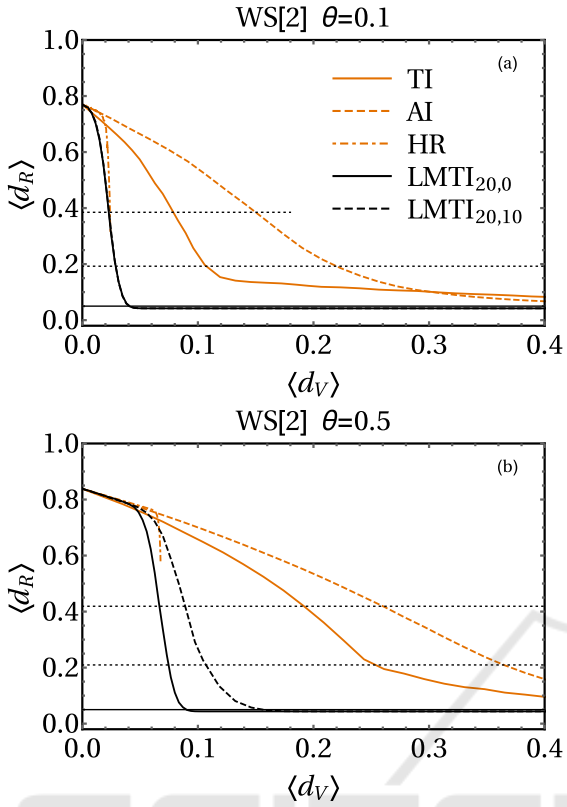


Figure 3: Results for various immunization strategies for WS[2] networks with $\mathcal{N} = 1000$ nodes and $\theta = 0.1$ (a), 0.5 (b). The LMTI scheme is compared to TI, AI and HR strategies. The horizontal solid line is the epidemic threshold $f = 0.05$, while the horizontal dotted lines are 25% and 50% of the mean final density of recovered without any vaccination.

### 4.3 Real Networks

Fig. 4 collects the results for real networks. As a general result, TI and LMTI are the most indicated strategies for reducing the epidemic prevalence by 50% (LMTI can cut it to the 25% with a slightly larger vaccinated fraction). Moreover, HR generally works worse than AI in all real networks we considered.

It is interesting to note that **Internet_AS** and **ProteinYeast** networks show a great structural resistance to epidemic outbreaks. Even without any vaccination, the average size of an epidemic remains relatively small (less than the 30% of the total population). According to the $k$-shell decomposition (Seidman, 1983), **Internet_AS** and **ProteinYeast** networks have a relatively small center and a large periphery, as the first two shells $k = 1, 2$ account for a very large fraction of the nodes (respectively the 78% and the 86% of the entire population). **ProteinYeast** is also the network with smallest mean degree and highest vertex eccentricity. In these networks, the eradication of the epidemic is particularly simple (with LMTI, this can be done respectively with $\langle d_V \rangle \sim 0.15$ and $\langle d_V \rangle \sim 0.05$). The other networks are much more exposed to the risk of a pandemic outbreak. In fact, without vaccination the typical size of the epidemic involves more than the 60% of the population. In **p2p-Gnutella08** and **CA-HepTh-pruned** networks, the results for the TI and LMTI with $\beta = 20$ are almost the same, while in both cases the HR strategy fails even in significantly reducing the epidemic prevalence. It is interesting to note that in the **p2p-Gnutella08** network the AI is particularly efficient, with performances similar to TI or LMTI for small values of $\langle d_V \rangle$. With reference to Table 1, this network presents the higher mean degree and the lowest mean vertex eccentricity, suggesting that it is highly and uniformly connected. As a consequence, in this network it is very difficult to control the epidemic spreading. Finally, results for **AA** network show again that TI and LMTI are almost equivalent for small values of $\langle d_V \rangle$, with the second one doing better for slightly larger values of the vaccinated fraction.

## 5 CONCLUSION AND DISCUSSION

In this work, we proposed a novel reactive immunization strategy. It is based on a local modification of TI protocol which aims to actively take into account the presence of a propagating epidemic and use it as a source of information to trigger an optimized vaccination response. We numerically compared our proposal with other standard immunization strategies and proved that it is a very efficient choice in every case we considered. Moreover, it always allows to stop the epidemic with a relatively small vaccinated fraction. Our proposal naturally fits in the class of techniques using local knowledge about complex systems (see for example the Hebbian learning rule (Hebb, 1949) in the case of neural networks).

There are two important aspects about our novel immunization strategy we would like to stress. First, our scheme, so as many other standard immunization strategies, is strongly information-demanding. Of course, a full knowledge about the complex system is rather unlikely in real situations, then sampling and interpolation (Ferguson et al., 2014), so as data assimilative strategies (Rhodes and Hollingsworth, 2009) have been recently developed. The second aspect concerns the role of the tunable parameters in our strategy. In fact, we compared our LMTI with other benchmark strategies for an *optimal* choice of $\beta$ and $\gamma$. Such a choice consists in the evaluation of the com-

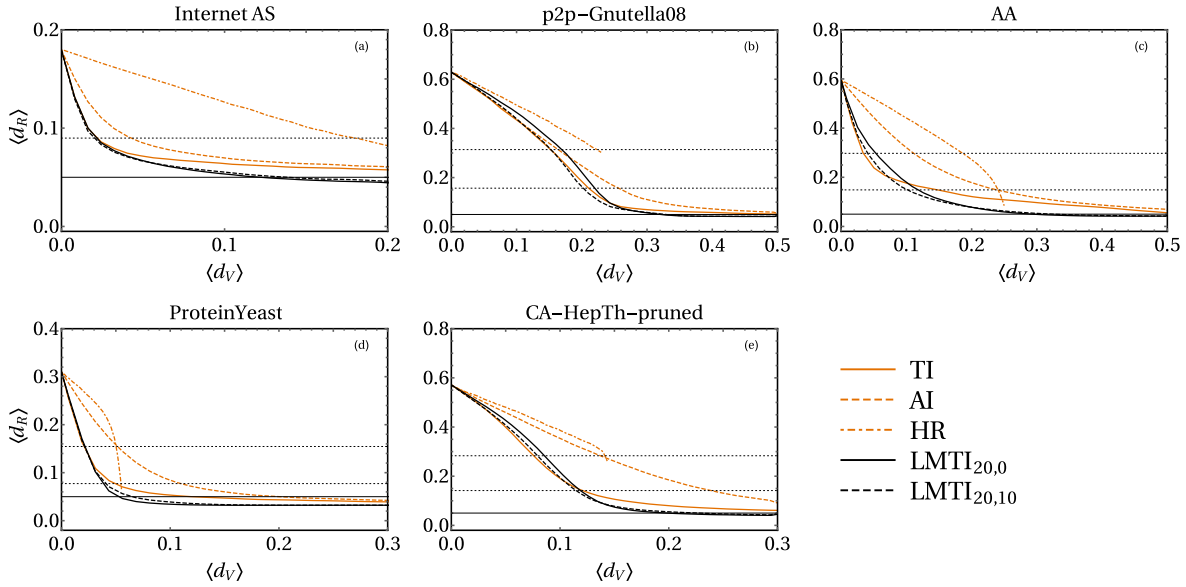


Figure 4: Results for various immunization strategies on real networks (a-e). The LMTI scheme is compared to TI, AI and HR strategies. The horizontal solid line is the epidemic threshold $f = 0.05$, while the horizontal dotted lines are 25% and 50% of the mean final density of recovered without any vaccination. For Internet_AS (a), the horizontal dotted line is the 50% of the mean final density of recovered without any vaccination.

peting roles of global and local features in the epidemic spreading and, in general, it depends on the network model and its size, as well as on the epidemic threshold. The study of such a dependence is generally a non-trivial theoretical task and goes beyond the goal of the present paper. Instead, in our purely phenomenological approach, we tuned the local parameters choosing the best pair $(\beta, \gamma)$ by looking at the performances of LMTI in reducing the epidemics. We therefore did not identify a method to *a priori* fix the best values of these parameters. Hopefully, this issue can be addressed and theoretically better understood in future works. In doing so, a more detailed experimental analysis of the dependence of LMTI performances on the free parameters is necessary.

In conclusion, several extensions of our work are possible. For example, it can be generalized to other classes of ideal networks with good theoretical control, like weighted or directed graphs. It can also be applied to actual specific diseases, *e.g.* TBC, Xylella fastidiosa (Alfinito et al., 2016) or Ebola infections. In doing this, more realistic propagation models, like the delayed SIR considered in (Agliari et al., 2013) and a detailed cost benefit analysis are needed.

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