Non massive immunization to contain spreading on complex networks

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Optimal strategies for epidemic containment are focused on dismantling the contact network through effective immunization with minimal costs. However, network fragmentation is seldom accessible in practice and may present extreme side effects. In this work, we investigate the epidemic containment immunizing population fractions far below the percolation threshold. We report that moderate and weakly supervised immunizations can lead to finite epidemic thresholds of the susceptible-infected-susceptible model on scale-free networks by changing the nature of the transition from a specific-motif to a collectively driven process. Both pruning of efficient spreaders and increasing of their mutual separation are necessary for a collective activation. Fractions of immunized vertices needed to eradicate the epidemics which are much smaller than the percolation thresholds were observed for a broad spectrum of real networks considering targeted or acquaintance immunization strategies. Our work contributes for the construction of optimal containment, preserving network functionality through non massive and viable immunization strategies.

I. INTRODUCTION

Modern societies are strongly regulated by networked systems such as face-to-face \cite{1–3} and remote social \cite{4} interactions, transportation infrastructures \cite{5,6}, communication networks \cite{7–9}, and so on. These substrates can also be underlying structures through which threats propagate, such as the spreading of contagious diseases \cite{10,11}, computer viruses \cite{12}, and fake news \cite{13–15}. Therefore understanding immunization or knockout (depending on the context) strategies is fundamental \cite{16} either by the necessity to prevent a menace propagation, as in a contagion disease or criminal network \cite{17,18}, or to protect vital components, as in communication \cite{7,9} and transportation \cite{5,6} infrastructures. Epidemic models can be interpreted as generic spreading processes \cite{11} and we hereafter adopt epidemiology jargons without loss of generality.

Containment methods are frequently associated with the percolation analysis \cite{19}, in which the immunization of nodes or edges would lead to the fragmentation of the transmission network into small components, hindering the spreading \cite{20–22}. One remarkable property of scale-free (SF) networks, represented by degree distributions with power-law tails in the form $P(k) \sim k^{-\gamma}$ with degree exponent $2 < \gamma < 3$, is their resilience to random immunization that is ineffective to fragment the network \cite{23,24}. However, targeted immunization of a fraction $f < 1$ of the most central nodes can dismantle SF networks \cite{16,24}. Immunization based on degree and betweenness \cite{25} using global properties of the network are commonly used \cite{23,26,27}; see \cite{16} for a survey of strategies. A drawback of global methods is that knowledge about properties of all nodes is frequently not accessible due to either computational limitations or lack of information of the network topology. An efficient and flexible alternative considers acquaintances of randomly selected nodes based only on local information \cite{20}. This approach, in which a neighbor of a randomly selected vertex is chosen to be immunized, is grounded on the fact that acquaintances are, on average, more central than randomly selected nodes \cite{28}.

Network fragmentation into subextensive components will certainly prevent large-scale epidemic spreading \cite{20,21}. However, if the percolation threshold is high, network fragmentation can be an impracticable attitude due to the costs and harmful side effects. So, how do epidemic processes evolve on moderately immunized (no fragmented) networks? And which fraction of immunization is needed to prevent the epidemic spreading in comparison with the percolation threshold? Despite of explicit analyses of epidemic spreading on immunized networks \cite{26,29–32}, these issues have not been addressed thoroughly to the best of our knowledge.

Consider the SIS model \cite{10} on the top of complex networks \cite{11,12} where nodes can be in one of two states: infected, which become spontaneously susceptible with rate $\mu = 1$, or susceptible, which can be infected by each of their infected contacts with rate $\lambda$. The epidemic threshold $\lambda_c$ determines the infection rate above which the epidemics can thrive indefinitely in an extensive portion of the network. A remarkable feature of the SIS model is its null epidemic threshold for SF networks as the size $N \to \infty$ \cite{12,33,34} meaning that the epidemics always reaches a finite fraction of the network irrespective of the value of $\lambda$. The activation mechanisms of epidemic process and, in particular, of SIS can be quite tricky to analyze \cite{35–38}. We can classify the activation into motif-driven and collective processes \cite{37,39}. In the former, a subextensive fraction is responsible for the triggering the epidemics and spreading it out to the rest of the network infecting an extensive fraction the population and the epidemic threshold vanishes as $N \to \infty$. This is the case of the SIS model on power-law networks for which activation can be triggered by either hubs or a densely connected subgraph given by the maximal index of a $k$-core decomposition, depending on the degree exponent \cite{38}. In the case of collective activation, an extensive part of the network is directly involved \cite{37,39}. This happens, for example, in the Harris contact process for any value of
the degree exponent \[39\] and in the susceptible-infected-recovered-susceptible (SIRS) model for \(\gamma > 3\) \[37\]. In the SIRS model, the infected individual stays for a while in an immunized state before becoming susceptible again (waning immunity) \[10\].

Since the SIS model possesses a fluctuating active steady state, its connection with percolation is not immediate as in the susceptible-infected-recovered (SIR) model \[20, 40\], in which an immunized node becomes recovered and cannot be reinfected. Whereas random immunization is ineffective, targeted strategies \[26, 29\] can lead to a finite epidemic threshold in SF networks through the immunization of the most connected nodes in both SIS and SIR models. Acquaintance immunization can also lead to an infinite epidemic threshold of the SIR model on SF networks \[20\]. Recently, Matamalas et al. \[32\] considered removal of edges with the highest probability to transmit the disease considering a discrete-time version of the pairwise mean-field theory for the SIS model \[41\]. This method successfully promoted epidemic containment preserving a connected giant component. However, this is a global approach prone to aforementioned difficulties of applications in large networks.

In this paper, we push forward this field investigating the evolution of the susceptible-infected-susceptible (SIS) model on synthetic and real networks where a fraction of the nodes far below the percolation threshold is immunized. We consider distinct immunization strategies, including global and local methods. We report that a non massive and weakly supervised immunization can promote containment by altering the nature of the epidemic transition from a specific-motif to a collectively driven activation, permitting that other processes remain functioning after network immunization. We also show that immunized networks are structurally different from their randomized counterparts and a finite epidemic threshold can emerge even when its randomized version still has a vanishing one.

The rest of this paper is organized as follows: In Section II, we discuss the immunization strategies and the structure of synthetic networks resulting from them. We present the investigation of the epidemic threshold of the SIS model on immunized synthetic networks in Sec. III. Effects of immunization on a collection of real networks are presented in Sec. IV. In Section V, we summarize the findings of this paper and draw our concluding remarks. Appendix A presents a summary of the real networks used in the current work while Appendices B and C complement the paper with some computational and analytical methods used throughout the paper.

**II. IMMUNIZED NETWORK ANALYSES**

Consider an initially connected network with \(N\) nodes, in which a fraction \(f\) will be immunized, which means that the vertex and all edges connected to it will be removed. We considered only adaptive methods \[27, 40\], in which the network properties are recalculated every time a node is immunized. In the targeted immunization (TgI), each step corresponds to immunize the most connected vertex of the network. In acquaintance immunization (AcI), a vertex and one of its nearest-neighbors (acquaintances) are sequentially selected at random, being a local strategy \[20, 30\]. The neighbor is immunized with probability

\[
\Phi(k) = \frac{\langle k \rangle^s}{\langle k \rangle^s + k^s},
\]

where the degree \(k\) is the number of nonimmunized nearest-neighbors of the vertex to be immunized, \(\langle k \rangle\) the average degree of the original network, and \(s\) is a parameter. If \(s = 0\), it is the adaptive version of the acquaintance strategy of Ref. \[20\]. If \(s > 0\), hubs are protected having a smaller probability to be immunized while \(s < 0\) implies that hubs are selected preferentially resembling TgI. Here, we present results for \(s = 0\) and \(s = +1/2\), hereafter, called AcI and AcI with hub protection (AcI-HP), respectively. The latter can be considered a weakly supervised strategy due to its limited capacity to determine the most efficient spreaders.

![Fig. 1. Percolation analysis on synthetic SF networks with \(N = 10^7\) nodes considering three immunization methods defined in the main paper. Two values of the degree exponent (a) \(\gamma = 2.3\) and (b) \(2.8\) are presented. The curves correspond to averages over 100 networks with one realization of immunization per network. Abscissas are the same in both plots.](image)

We consider synthetic networks generated with the uncorrelated configuration model (UCM) \[42\] using a minimal degree \(k_{\text{min}} = 3\) and an upper cutoff \(k_c = \sqrt{N}\). We performed percolation analyses to determine whether the fraction of immunized vertices \(f\) fragments or not the network into small components. Figure 1 shows the fraction of nonimmunized nodes \(P_\infty\) which belong to the largest connected component (LCC) \[43\] as a function of \(f\) for synthetic SF networks of size \(N = 10^7\) and two values of the degree exponent \(\gamma < 3\). The percolation thresholds \(f^{\text{per}}\), separating phases with an extensive \((P_\infty > 0)\) and subextensive \((P_\infty = 0)\) LCC, are given in Table I. To deal with finite networks we assume that a relative size...
of the LCC below $10^{-3}$ corresponds to the percolation thresholds, being the results little sensitive to this choice. Figure 1 shows that the LCC for $f = f_{\text{vir}}^{\text{per}}/5$ corresponds to more than 90% of the nonimmunized vertices in all cases, being nearly 100% for AcI and AcI-HP methods.

In the case of AcI-HP, the exponents present smaller values $1/b \approx 0.185$ and 0.053 for $\gamma = 2.3$ and 2.8, respectively. All regressions provided correlation coefficients $r \geq 0.9995$. The degree of the most connected vertex is given by $N\Pi(k_{\text{max}}) \approx 1$ [19] resulting $k_{\text{max}} \sim (\ln N)^b$ for stretched exponentials and $k_{\text{max}} \sim N^{1/(\gamma-1)}$ for power-laws while a finite upper rigid cutoff quickly appears in the case of TgI; see Fig. 2(c).

TABLE I. Percolation thresholds estimated from Fig. 1. Numbers in parenthesis are the uncertainties in the last digit.

| Method   | Threshold |
|----------|-----------|
| AcI-HP   | $\gamma = 2.3$ 0.50(1) 0.46(1) 0.20(1) |
| AcI      | $\gamma = 2.8$ 0.55(1) 0.51(1) 0.24(1) |
| TgI      | $\gamma = 3.0$ 0.56(1) 0.50(1) 0.30(1) |

Last but not least, Figure 3 shows the size dependence of the average shortest paths calculated using breadth first search algorithm [43] for nonimmunized and immunized UCM networks with $f = f_{\text{vir}}^{\text{per}}/5$. To verify if the immunized networks preserve the small-world behavior, in which distances increase logarithmically with size, we fitted the data to the expression

$$\langle l \rangle = l_0 + C_0 w^\alpha,$$  

where $w = \ln N$. The ansatz given by Eq. (3) is not expected to work exactly but to indicate a growth slower than power-laws that is sufficient to characterize the small-world property. All curves are very well fitted (correlation coefficient $r \geq 0.9998$) by Eq. (3) as shown in Fig. 3. The scaling exponents for $\gamma = 2.3$ ( $\gamma = 2.8$ ) were $\alpha = 1.32$, 2.77, and 3.05 ($\alpha = 1.05$, 1.56, and 2.33) for AcI-HP, AcI, and TgI, respectively. The values larger than unity indicate a super-logarithm growth for immunized networks and is larger for more efficient immunization. For the nonimmunized networks, we found $\alpha < 1$ which reflects the sub-logarithm growth expected for random SF networks [44].

### III. EPIDEMIC THRESHOLDS FOR IMMUNIZED SYNTHETIC NETWORKS

We ran standard Markovian SIS dynamics [11] on the immunized networks using the algorithm detailed in Ref. [45] and summarized in Appendix B1. We analyzed the steady-state regime with quasistationary (QS) simulations [37, 46] to circumvent the drawbacks of the absorbing states in finite sizes [47]. We determined the epidemic threshold in stochastic simulations using the position $\lambda_\text{c}$ of maximum of the dynamical susceptibility $\chi(\lambda)$ defined as $\chi = N[(\rho^2) - (\rho)^2]/(\rho)$ [48], $\rho$ being the density of infected nodes and the averages are computed in the QS regime [37, 45]. Appendix B2 gives some details of the QS analysis.

For the synthetic SF networks, the epidemic threshold usually depends on the effective size of the LCC of
nonimmunized vertices. So, one cannot investigate the finite-size effects independently of the immunization fraction if we fix the size $N_0$ of the original network. Therefore, larger networks were generated such that the LCC lies in the range $[0.95N_0, 1.05N_0]$. Simulations are run on the LCC. The dependence of the epidemic threshold with size for UCM networks with $\gamma = 2.3$ is shown in Fig. 4 for $f = f^{\text{per}}/5 \approx 0.1$ using both AcI and AcI-HP methods. These thresholds approach finite values as $N \to \infty$ in both cases including the weakly supervised AcI-HP method. The AcI case shows the epidemic thresholds increasing with size after an initial decay since the strategy becomes more efficient as larger hubs appear in the networks. A similar behavior cannot be discarded for AcI-HP for much larger sizes. Simulations for $\gamma = 2.8$ in Fig. 5 are qualitatively similar but subject to different finite-size effects.

In Figs. 4 and 5, simulations are compared with the heterogeneous mean-field (HMF) theory [12], which takes into account only the degree distribution, and the quenched mean-field (QMF) theory [49], which includes the detailed network structure through its adjacency matrix $A_{ij}$ [43]. Details of the theories are presented in Appendix C. The theoretical epidemic thresholds are $\lambda_{c}^\text{HMF} = \langle k \rangle / \langle k^2 \rangle$ [12] and $\lambda_{c}^\text{QMF} = 1/\Lambda_1$ [49], where $\Lambda_1$ is the largest eigenvalue of $A_{ij}$. Notice that HMF outperforms the more detailed QMF theory in the case of immunized networks, conversely to the mean-field performances for the SIS model on nonimmunized networks [50].

The asymptotically finite epidemic thresholds cannot be justified only by the pruning of hub degrees since AcI and, mainly, AcI-HP methods, lead to stretched tail distributions expected to asymptotically produce a null epidemic threshold according to rigorous results [51]. We tackled this point performing a degree-preserving rewiring of the effective immunized network and rerunning the SIS process. Rewired networks for AcI-HP have epidemic thresholds decaying with size, compatibly with QMF and consistent with the conjecture of a vanishing epidemic threshold for a stretched exponential [51] whereas the nonrewired ones present saturation consistent with the HMF theory. In the AcI case, the rewiring changes the tendency of increasing to a very slow decay, still qualitatively compatible the QMF theory but with a much larger prefactor.

A second fundamental ingredient for determining the
This outcome is remarkable since a viable, adaptive, and sive epidemic process (no acquired immunity) as the SIS contain the onset of an endemic state for a very aggres-}<ref>These outcomes act on both properties: pruning the hub’ degrees, reducing their capabilities to stay active, and increasing the distances among them, damping their mutual interactions. The finite epidemic threshold is consistent with a collective [39, 54] rather than specific-motif (hubs, maximum k-core, etc.) driven activation mechanism [36, 38]. This outcome is remarkable since a viable, adaptive, and weakly supervised strategy as the AcI-HP can efficiently contain the onset of an endemic state for a very aggressive epidemic process (no acquired immunity) as the SIS model.

IV. PERCOLATION VERSUS IMMUNIZATION THRESHOLDS ON REAL NETWORKS

The aforementioned impact of non massive immunization on synthetic networks naturally calls for applications on real networks. So, we determined the immunization fraction capable to eradicate a highly endemic steady state in the nonimmunized network [26, 32]. We calculated the infection rate for which the stationary density of infected vertices in the nonimmunized networks is \( \rho = \rho_0 \) using standard simulations (without QS sampling). Starting with \( f = 0 \), we increase \( f \) with small increments \( \Delta f \ll 1 \) at infection rate \( \lambda = \lambda_0 \), until the stationary density drops to zero, determining the immunization threshold \( f^{\text{imm}} \). For our simulations, we used \( \rho_0 = 0.1 \). Results are qualitatively similar for other values of \( \rho_0 \). Figure 6 shows the stationary density and relative size of the LCC as functions of \( f \) for UCM networks using AcI. The stationary density falls to zero far below the percolation threshold, \( f^{\text{per}} \), confirming the high efficacy of non massive immunization for epidemic containment in synthetic networks.

We now turn our attention to a set of 42 real networks of wide spectrum of structural properties previously investigated in Ref. [56]; see Appendix A for basic network information. As in the percolation analysis, we tackle the finite size of the networks assuming that either a stationary density or a relative size of the LCC below \( 10^{-3} \) correspond to the immunization or percolation thresholds, respectively. Again, the results depend little on this choice. Visualizations of the LCC of the adjacency vocabulary network for Japanese and its immunized version using AcI at \( f = f^{\text{imm}} = 0.024 \) are shown in Fig. 7. The immunized network presents a sparser but well connected LCC having more than 70% of the vertices and concentrated in the innermost regions of the network. Considering only the LCC, the average degree decays from \( \langle k \rangle = 5.92 \) to 3.47 while the average distance increases from \( \langle \ell \rangle = 3.07 \) to 4.26 when AcI is applied. The percolation and immunization thresholds are \( f^{\text{per}} = 0.32 \) and \( f^{\text{imm}} = 0.024 \), respectively.

Figure 7(a) compares the immunization and percolation thresholds calculated using AcI for the set of 42 real networks. The data is grouped according to the Pearson coefficient \( p \) [28] defined as [43]

[4] \[ p = \frac{\sum_{i,j} (A_{ij} - \frac{k_i k_j}{N(k)}) k_i k_j}{\sum_{i,j} (k_i \delta_{ij} - \frac{k_i k_j}{N(k)}) k_i k_j} \]

which lays in the interval \(-1 < p < 1\) and ranks the level of degree correlations of the network, being disassortative for \( p < 0 \), uncorrelated for \( p = 0 \) and assortative for \( p > 0 \); see Appendix A for the Pearson coefficients and values of \( \lambda_0 \) for \( \rho_0 = 0.1 \) of the real networks. The efficiency of AcI is negatively correlated with the Pearson coefficient with a immunization threshold much lower than the percolation one for both highly disassortative (\( p < -0.1 \)) and slightly correlated (\( |p| < 0.1 \)) cases and a worse performance for the highly assortative cases (\( p > 0.1 \)). Such a dependence reflects the loss of efficiency of acquaintance-based methods for finding hubs on assortative networks. Our results are in agreement with Ref. [31] where it was observed that immunization efficiency depends on the level of correlation.

Much better performances are attainable if cleverer immunization strategies are adopted. We investigated TgI in Fig. 7(b) which is much more efficient than AcI. The condition \( f^{\text{imm}} \ll f^{\text{per}} \) holds for the whole set of real networks.
networks. One can improve further with more specific centralities rather than degree [36] or use process-targeted strategies [32]. However, whatever the used approach one must rely on the dynamic processes rather than only topological structures.

We conclude our results comparing the simulation values of immunization thresholds with two mean-field theories. Within a HMF theory, $f^{\text{imm}}_c$ is given by the condition

$$\langle k^2 \rangle_{f^{\text{imm}}_c} / \langle k \rangle_{f^{\text{imm}}_c} = \lambda_0,$$

where the $\langle k^n \rangle_f$ are moments of the degree distribution of the LCC after immunization of $fN$ vertices. Similarly, the QMF immunization threshold is given by

$$\frac{1}{\Lambda_1(f^{\text{imm}})} = \lambda_0,$$

where $\Lambda_1(f)$ is the largest eigenvalue of the adjacency matrix corresponding to the LCC after immunization of $fN$ vertices. The ratio $f^{\text{MF}}_c / f^{\text{imm}}_c$ between theory and simulation for immunization thresholds on real networks are compared in Fig. 8 for AcI and TgI strategies. The closer the ratio is to 1 (solid lines) the better is the mean-field theory. The HMF theory tends to underestimate while QMF to overestimate the immunization thresholds in opposition to the performance of the epidemic thresholds where QMF tends to underestimate and HMF to overestimate the simulation results [50]. For efficient immunizations, namely TgI and AcI at disassortative or weakly correlated networks, the HMF theory performs better than QMF. The cases with highly assortative correlations, the performances are similar. This can be seen with the aid of average value of the ratios computed over each plot and indicated by dashed (QMF) and dotted (HMF) horizontal lines, respectively.

V. CONCLUSIONS

Containment methods for controlling propagation of dynamical processes on the top of networks is crucial for
setting up protection protocols against threatenings that can be disseminated throughout networked substrates. A considerable part of the containment methods are based on percolation analysis while the spreading on partially and weakly damaged networks has received little attention. In the present work, we tackle this problem investigating the epidemic spreading of the SIS model on complex networks using different immunization strategies. We report that a non massive immunization with the removal of a fraction far below the percolation threshold can alter the originally motiff-driven (hubs, maximum k-core, etc...) [36] mechanisms for activation of endemic phases to a collective activation involving extensive parts of the network. Even in the case of a weakly supervised immunization strategy, the absence of an epidemic threshold at originally SF networks is replaced by finite thresholds caused by the concomitant pruning of hubs and increasing of their mutual distances. Backed up by the analysis of a collection of real networks, we also show that immunization can efficiently contain epidemic spreading using non massive levels.

To the best of our knowledge, the information that immunization thresholds are much smaller than the percolation ones has passed unnoticed or underestimated in the vast physics literature concerned with immunization of complex networks. So, we hope that our work will ignite new research activity towards elaboration of optimal and viable immunization strategies. We conclude highlighting the importance of running accurate stochastic simulations of the actual dynamical processes since the long-range interactions cannot be completely reckoned by mean-field methods [50, 51].

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Appendix A: Structural properties of real networks

Tables II, III, and IV show some structural properties of the real networks studied on this work such as the number of vertices $N$, mean degree $\langle k \rangle$, heterogeneity coefficient $\eta = \langle k^2 \rangle / \langle k \rangle$, and Pearson coefficient $p$. Different tables correspond to different ranges of Pearson coefficient used in Fig. 7(a). The infection rate $\lambda_0$ necessary to sustain a stationary state with 10% of infected vertices in the original network are shown in the last columns.

![Figure 8](image-url)

**FIG. 8.** Comparison of the ratio between immunization thresholds obtained in numerical simulations and mean-field theories for a set of 42 real networks immunized using either (a)-(c) AcI and (d) TgI strategies with $p_0 = 0.1$. Data for AcI are grouped according ranges of Pearson coefficient: (a) $p < -0.1$, (b) $|p| < 0.1$, and (c) $p > 0.1$. Dashed and dotted lines represent the ratios averaged over all networks for QMF and HMF theories, respectively. Averages as in Fig. 7.

TABLE II. Some properties of real networks with Pearson coefficients $p < -0.1$. Size $N$, average degree $\langle k \rangle$, heterogeneity coefficient $\eta$, and infection rate $\lambda_0$ able to produce a steady state with 10% of infected vertices are shown.

| Network      | $N$  | $\langle k \rangle$ | $\eta$ | $p$      | $\lambda_0$ |
|--------------|------|---------------------|--------|----------|-------------|
| spanish      | 11,558 | 7.45              | 457    | -0.28    | 0.0424     |
| japanese     | 2,698  | 5.92              | 108    | -0.26    | 0.0624     |
| english      | 7,377  | 12.0              | 320    | -0.24    | 0.0287     |
| french       | 8,308  | 5.73              | 218    | -0.23    | 0.0599     |
| Jung         | 6,120  | 16.4              | 991    | -0.23    | 0.0225     |
| JDK          | 6,434  | 16.7              | 982    | -0.22    | 0.0224     |
| politicalblogs | 1,222 | 27.5              | 81.2   | -0.22    | 0.0203     |
| Internet     | 22,963 | 4.21              | 261    | -0.20    | 0.0899     |
| ASCaida      | 26,475 | 4.03              | 280    | -0.19    | 0.0924     |
| EMail        | 224,832 | 3.02             | 567    | -0.19    | 0.0949     |
| UC Irvine    | 1,893  | 14.6              | 55.6   | -0.19    | 0.0349     |
| UCMailingList | 24,567 | 12.9              | 341    | -0.19    | 0.0349     |
| AS Oregon    | 6,474  | 3.89              | 165    | -0.18    | 0.103      |
| Linux Soft   | 30,817 | 13.8              | 853    | -0.18    | 0.0274     |
| Google       | 15,763 | 18.9              | 902    | -0.12    | 0.0224     |
| Euron        | 33,696 | 10.7              | 142    | -0.12    | 0.0412     |

TABLE II. Some properties of real networks with Pearson coefficients $p < -0.1$. Size $N$, average degree $\langle k \rangle$, heterogeneity coefficient $\eta$, and infection rate $\lambda_0$ able to produce a steady state with 10% of infected vertices are shown.

Appendix B: Numerical methods

1. Computer implementation of SIS

To simulate the SIS model on graphs, we used the optimized Gillespie algorithm (OGA), described in Ref. [45]. We determine the number of infected vertices $N_i$ and their total number of edges $N_e$. At each time step, one of the events healing or infection attempt is chosen with probabilities $p = N_i / (N_i + \lambda N_e)$ and $1 - p$, respectively. In the former, one infected vertex is selected at random...
and become susceptible. In the latter, one infected vertex is selected with probability proportional to its degree and one of its nearest-neighbors is chosen with equal chance. If the selected neighbor is susceptible, it becomes infected. Otherwise, the simulation proceeds to the next step without change of configuration. At the end of this infection/healing process, the time is incremented by \( \Delta t = 1/(N_t + \lambda N_e) \) while \( N_t \) and \( N_e \) are updated accordingly. This process is iterated until the predetermined simulation time is reached.

### 2. Quasistationary analysis

The QS analysis is a method to investigate dynamical processes with absorbing states as the SIS model. It consists of evaluating averages only over samples that did not visit the absorbing states [47]. For subcritical and critical simulations the dynamics falls very often into the absorbing state resulting in short and noisy intervals of stationary data. Dickman and de Oliveira [46] proposed a method to overcome this problem where the dynamics jumps to an active configuration previously visited along the evolution of the process every time the system falls into the absorbing state. Computationally, configurations visited during the simulation are stored and constantly updated. One of them is randomly selected to restart the simulation every time the absorbing state is visited.

![Figure 9: Susceptibility curves of the SIS model for immunized (solid lines) and nonimmunized (dashed lines) UCM networks with (a) \( \gamma = 2.3 \) and (b) \( \gamma = 2.8 \) and different sizes indicated in the legends. The immunization strategy is AcI-HP with \( f = 0.1 \) in both cases.](image)

In our simulations, we started with all nodes infected and relax the system for a time interval \( t_{th} = 10^7 \). The QS probability \( Q(n) \) that the system has \( n \) active (infected) vertices are computed during the time interval \( t_{av} = 3 \times 10^7 \). A list with \( M = 100 \) active configurations is built. With probability \( p_t = 0.01 \) per time unit, the list is updated replacing one of the configurations by the current state. The QS density and dynamical susceptibility are defined in terms of moments

\[
\langle \rho^s \rangle = \frac{1}{N_s} \sum_{n \geq 1} n^s Q(n) \quad (B1)
\]

as \( \rho_{qs} = \langle \rho \rangle \) and \( \chi = N(\langle \rho^2 \rangle - \langle \rho \rangle^2) / \langle \rho \rangle \), respectively. Figure 9 shows typical susceptibility curves for immunized and nonimmunized UCM networks. The epidemic threshold is estimated as the position of maximum susceptibility value.
Appendix C: Mean-field theories for the SIS model

1. HMF theory

The probability $p_k$ that a vertex of degree $k$ is infected in the HMF theory evolves as

$$\frac{dp_k}{dt} = -p_k + \lambda k(1 - p_k) \sum_{k'} P(k'|k)p_{k'},$$

(C1)

where $P(k'|k)$ is the conditional probability that a vertex of degree $k$ is connected to a vertex of degree $k'$. Using linear stability analysis around the absorbing state $p_k = 0$, the following Jacobian is found

$$J = \lambda k \sum_{k'} \frac{P(k'|k)}{k'}.$$

Considering uncorrelated networks where $P(k'|k) = \langle k' \rangle / \langle k \rangle$ the epidemic threshold of the SIS model becomes $\lambda_c^{\text{HMF}} = \langle k \rangle / \langle k^2 \rangle$ [12]. Taking the asymptotic limit of the moments $\langle k^n \rangle$ we obtain

$$\lambda_c^{\text{HMF}} = \frac{\langle k \rangle}{\langle k^2 \rangle} \simeq \frac{k^{-3+\gamma}}{k_{\text{max}}} \quad \text{if } \gamma < 3,$$

$$\text{const.} \quad \text{if } \gamma > 3,$$

(C2)

which goes to zero for $\gamma < 3$ and becomes larger than zero for $\gamma > 3$.

2. QMF theory

The QMF theory includes the network structure by explicitly using the adjacency matrix $A_{ij}$. The probability that a given vertex $i$ is infected evolves as

$$\frac{dp_i}{dt} = -p_i + \lambda(1 - p_i) \sum_{j} A_{ij}p_j,$$

(C3)

The linear stability analysis around $p_i = 0$ leads to a Jacobian matrix $J_{ij} = -\delta_{ij} + \lambda A_{ij}$, such that the epidemic threshold is given by $\lambda_c^{\text{QMF}} = 1/\Lambda_1$, where $\Lambda_1$ is the largest eigenvalue of $A_{ij}$. Plugging the expression for $\Lambda_1$ of uncorrelated networks derived in Ref. [57], one obtains the following behavior for the epidemic threshold

$$\lambda_c^{\text{QMF}} = \frac{1}{\Lambda_1} \simeq \begin{cases} \langle k \rangle / \langle k^2 \rangle & \text{if } 2 < \gamma < 5/2, \\ \frac{1}{\sqrt{k_{\text{max}}}} & \text{if } 5/2 < \gamma \end{cases},$$

(C4)

which goes to zero for any power-law degree distribution irrespective of the value of $\gamma$.

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