

Determining whether a class of random graphs is consistent with an observed contact network

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Abstract

We demonstrate a general method to analyze the sensitivity of attack rate in a network model of infectious disease epidemiology to the structure of the network. We use Moore and Shannon’s “network reliability” statistic to measure the epidemic potential of a network. A number of networks are generated using exponential random graph models based on the properties of the contact network structure of one of the Add Health surveys. The expected number of infections on the original Add Health network is significantly different from that on any of the models derived from it. Because individual-level transmissibility and network structure are not separately identifiable parameters given population-level attack rate data it is possible to re-calibrate the transmissibility to fix this difference. However, the temporal behavior of the outbreak remains significantly different. Hence any estimates of the effectiveness of time dependent interventions on one network are unlikely to generalize to the other. Unfortunately, we do not yet have a set of sufficient statistics for specifying a contact network model.

Keywords: Network reliability, Epidemic modeling, Network structure, ERGM, Epidemic potential

1. Introduction

The role of complex networks has become increasingly important in diverse fields of study, ranging from biology to social sciences to engineering. In the field of social sciences, networks often model contacts among a population. The nodes
5 represent the individuals in the population and edges represent the contacts

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or interactions between them. Applied to infectious disease epidemiology, each edge is associated with a probability of transmitting infection. Simulations draw an instance from the joint probability of infecting any set of people, providing insights into the spread of the disease through the population. Historically, the focus of mathematical epidemiology has been on properties such as the period of incubation, the duration of illness and the mortality rate, and less on the structure of the contact network. Hence simple compartmental approaches that assume a more or less homogeneous mixing have been adequate [1, 2, 3, 4, 5, 6]. However, especially in the context of evaluating targeted control efforts for sexually transmitted diseases, there has been increased emphasis on contact networks.

The transmission of an infectious disease through a contact network can be modeled as a diffusive process on a graph [7, 8, 9]. The size and overwhelming complexity of modern epidemiological problems calls for new approaches and tools like stochastic processes, random walks or Markov Chain Monte Carlo methods. With the aid of computers, agent based models on realistic social networks [9, 10, 11] can bridge from the individual level to population-level. Such models have provided useful insights into the implications of interaction patterns for the spread of disease. These simulations provide a platform to test and understand the spread of diseases and the effects of any intervention measures targeted at specific sub-population [10, 12, 13]. Generalizing results from one region to another requires studying variations in the network and the sensitivity of results to those variations. Because it is difficult to measure large contact networks, these studies rely on drawing sample networks from a network model.

It is known that the structure of the contact network significantly affects the spread of diseases. Even though the behavior of real world systems can sometimes be predicted by random graphs with constraints on structural properties such as degree distributions, discrepancies between theory and simulation, suggesting the presence of different social structures which are not captured by these constraints [14]. The effective degree approach [15, 16], edge-based compartmental models [17, 18], modifications to simple compartmental models [19] are a few of the ways researchers have tried to incorporate both the duration of the contacts in the network and the heterogeneities in numbers of partners.

For networks whose structure is tree-like, a large class of epidemic models can be solved exactly to provide analytic expressions for the sizes of both epidemic and non-epidemic outbreaks and for the position of the epidemic threshold [20]. For an arbitrary network, the epidemic threshold condition is closely related to the largest eigenvalue of the adjacency matrix, under reasonable approximations [21]. Further, it is shown that the time taken for the epidemic to die out depends on the difference of the two largest eigenvalues of the adjacency matrix [22]. The fluctuations in the connectivity of the network also influence the overall behavior of epidemic spreading by strongly enhancing the incidence of infection [23]. This work presented here investigates whether a class of networks exhibits similar behavior to an observed network under the *same* dynamical model.

A variety of mathematical models are used in the literature to create net-

works that can imitate the patterns of the links in real networks. Some are random in nature with few parameters fixed whereas others are more structured and take into account more network properties. Methods such as preferential attachment [24] can generate networks with a certain degree distribution while small world models reproduce the clustering in observed networks [25]. Exponential random graphs [26, 27] create a network model with maximum entropy consistent with matching user-specified properties. Networks drawn from this model have values of these statistics that are closely fitted to those of an observed network, but otherwise random [27]. Recent studies have used the exponential random graph models (ERGMs) to model friendship networks [28]. For example, the spread of sexually transmitted disease has been studied extensively [29, 30, 31, 32, 33, 34] using data available from Wave I of the National Longitudinal Study of Adolescent to Adult Health (Add Health). Add Health is a longitudinal study of a nationally representative sample of more than 90,000 adolescents in grades 7 through 12 in the United States, obtained from the data collected between 1994 and 1995 through a stratified sample of 80 schools. This survey data combines the different demographic factors with the social interactions of these school students. They have been analyzed by Resnick et. al. [35] and Udry and Bearman [36], for example, to identify the characteristics associated with health and risky behaviors among the adolescents. ERGMs generate networks efficiently when the constraints are placed only on local statistics like degree distribution (number of people connected to a certain individual), clustering or number of triangles. But the spread of a disease depends on global properties of the network, which are more difficult to match with an ERGM.

It is imperative to understand how the choice of properties to constrain affects the simulated spread of disease. This paper applies the concept of network reliability, introduced by Moore and Shannon [37] to characterize the effects of the network model. The network reliability $R(x; \alpha)$ takes into account the structural properties (i.e., topology) as well as the dynamics of contagion on the network. It gives the probability of observing an infection attack rate of at least α for an SIR (susceptible-infected-recovered) process with transmission probability x on a particular network. This is a measure of the “epidemic potential” introduced by Hamilton [38]. Since ERGMs are thought to capture the structural features of social networks [26, 28, 39, 40], the network reliability of an ensemble of ERGMs intended to represent a specific network in the Add Health survey is evaluated. It is observed that even though these models are a good representation of local structure in the network, they lead to significantly different dynamics for the propagation of an epidemic. However, because the transmission probability and the network structure are not separately identifiable, there is a simple transformation of the transmission probability that can erase these differences. This suggests supplementing the ERGM model with a description of this transformation to arrive at an ensemble of social network plus the probability of transmission for simulating the spread of disease. Unfortunately, a model calibrated to reproduce the overall attack rate does not necessarily reproduce the full time dependence of the epidemic curve, and thus is not well suited for estimating the effects of time-dependent control measures.

2. Methods

One of the Add Health friendship networks is chosen as the “observed” network for this study. The results on this population-based survey are compared to those obtained from networks created using the ERGM [41] model based on the observed network. One of the ERGM models yields the well-known Faux Magnolia dataset [41, 42, 43]. The Faux Magnolia network matches the Add Health data in degree distribution, clustering, number of triangles and other centrality measures, indicating that the ERGM is a good candidate model for a social network. The population-based data used for this study is obtained from Wave I of the Add Health study (<http://www.cpc.unc.edu/projects/addhealth>). One of the friendship networks, school 86 (based on the schools 086 and 186, a junior and a senior high school) is used as the original dataset. A network containing the mutual friends is considered for this study. The ERGMs are used to model the underlying structure of the friendship network. There are different ERGMs available in the statnet package [41] depending on the property to be constrained. Four distinct ERGMs are used to generate four sets of networks, each containing an ensemble of 100 networks. Each of these four sets match the features of the original network, e.g., the total number of edges, node attributes and different values of the GWESP (geometrically weighted edgewise shared partner) statistic [41], a parameter that combines the clustering and the number of triangles in the networks [26, 40]. The details of these models are mentioned in the Supplementary Notes. One of these models was used to build [41, 42, 43] the Faux Magnolia network from the school 86 data.

The Faux Magnolia network extracted from the statnet package [41] is an ERGM fit based on this Add Health data. Model 1 constrains the total number of edges of the original dataset; model 2 constrains the node attributes, like race, grade and sex in addition to the total number of edges; model 3 constrains the total number of edges and the number of triangles, but it fails to converge in the trials, so it is not reported here. Model 4 constrains the total number of edges, the node attributes and an additional statistic called GWESP [26, 40, 41], which is related to the number of triangles and clustering in a network. The networks in the last two sets are generated using this model, with two different values of the GWESP statistic, i.e., model 4.1 has a GWESP value 0.25 and for model 4.2 it is 0.5. These models are named to be consistent with the statnet naming convention.

2.1. Statistics on Networks

Typical measures of network structure like degree distribution, number of triangles, clustering coefficients and centrality measures - closeness and betweenness centrality are calculated for all the networks generated by the different models as well as the Faux Magnolia and the school 86 networks (Supplementary Notes). Comparison of these measurements demonstrates that the final model, model 4, and the Faux Magnolia network are best calibrated while all the models meet the constraints to the other statistics as well.

2.2. Epidemic Threshold for the Networks

The epidemic threshold condition for networks which have tree-like structure locally given by Newman's formula [20] can be written in terms of the mean $\langle k \rangle$ and the variance $Var[k]$ of the degree distribution [15, 16]. This quantity, called
 145 x_c in this paper, is given by the equation 1.

$$x_c = \frac{1}{\langle k \rangle - 1 + \frac{Var[k]}{\langle k \rangle}} \quad (1)$$

For an arbitrary graph, x_c is inversely related to the largest eigenvalue of the adjacency matrix [21]. The values of the largest eigenvalues for these networks are similar, $\lambda_{86} = 5.05$ and $\lambda_{FM} = 4.98$. The Figures 1a and 1b show that x_c for the school 86 network and the Faux Magnolia are similar compared to the other
 150 network models. (Plots for the largest eigenvalues and the difference of the two leading eigenvalues are in the Supplementary Notes.) The mean epicurves of 10^4 SIR simulations on the Faux Magnolia and the school network for a probability of transmission, $x = 0.85$ are plotted in Figure 2.

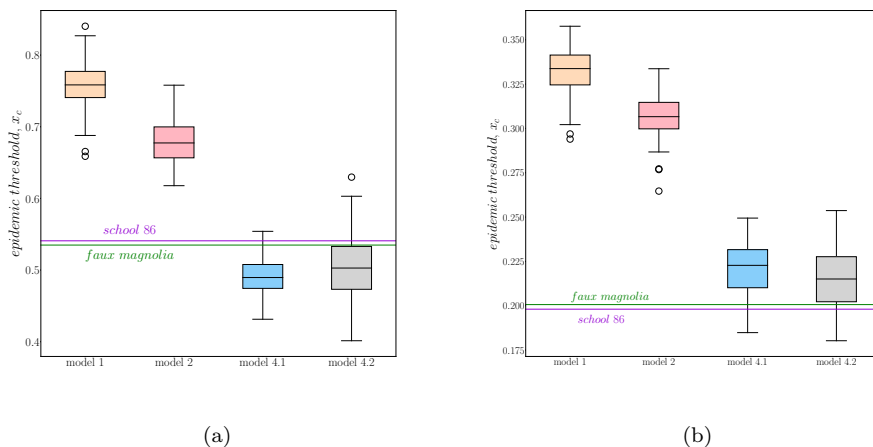


Figure 1: Boxplots showing the values of the epidemic threshold, x_c for the networks. The threshold is calculated using Newman's formula in (a) and using the largest eigenvalue of the adjacency matrix in (b).

The boxplots indicate that the Faux Magnolia matches the x_c values much
 155 better than the others. It is to be noted that the networks obtained using model 1 and model 2 have a higher threshold value in contrast to the models 4.1 and 4.2. Also, despite the similarity in the shape of the epicurves of the two networks in Figure 2, there is a systematic difference between them. The curve corresponding to Faux Magnolia overestimates the length of an outbreak and the height of the peaks for a particular value of the transmission probability.
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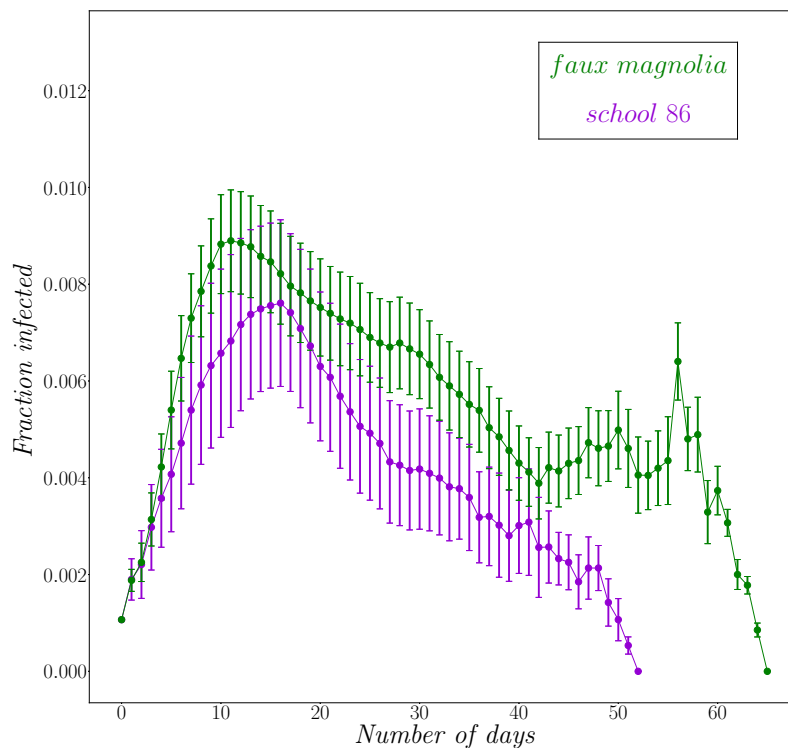


Figure 2: Mean epicurves for the Faux Magnolia and the school 86 networks for a probability of transmission, $x = 0.85$. The error bars are the probable errors for the estimated mean value.

Further, the average attack rate - defined as the average of the total number of the people infected when a randomly chosen individual is infected with the given values of x - is ~ 0.0651 for Faux Magnolia and ~ 0.0402 for school 86 when $x = 0.85$.

165 *2.3. $R(x; \alpha)$ - Epidemic Potential (Network Reliability)*

$R(x; \alpha)$, the probability of observing an attack rate of at least α for an SIR process with transmission probability x on a particular network is estimated [44]. For example, $R(x; \alpha = 0.3)$ gives the probability that the overall attack rate for an SIR process is at least 30% when the probability of transmission of infection is x . This measure depends on both the network structure and x . Thus, it specifically reflects the behavior of SIR dynamics on a particular network. It

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is evaluated for all the networks for three different values of α , 0.02, 0.05 and 0.08. This is identical to calculating the probability that at least 2%, 5% or 8% of the population is infected. Simulations are used to verify that the method based on the reliability statistic estimates the correct value of the probability of a disease outbreak. Few values of the transmission probability obtained by the transformation are used as the infection rate to calculate the overall average attack rate in these two networks. The table in the Supplementary Notes shows the results of SIR simulations on the Faux Magnolia network and the school 86 network for different values of x .

It is observed that different networks have the same value of $R(x; \alpha)$ for different values of x . Taking advantage of this confounding, one model can be calibrated to another, so that the epidemic potential [38], $R(x; \alpha)$ is the same for both. A low order polynomial transformation provides a good fit for the calibration curve. For this analysis, the Faux Magnolia network, school 86 network and one of the networks from model 2, named net1 in the paper are used. Further, to validate the results obtained, SIR simulations are run on these two networks for different values of x and the values of the average attack rates are compared.

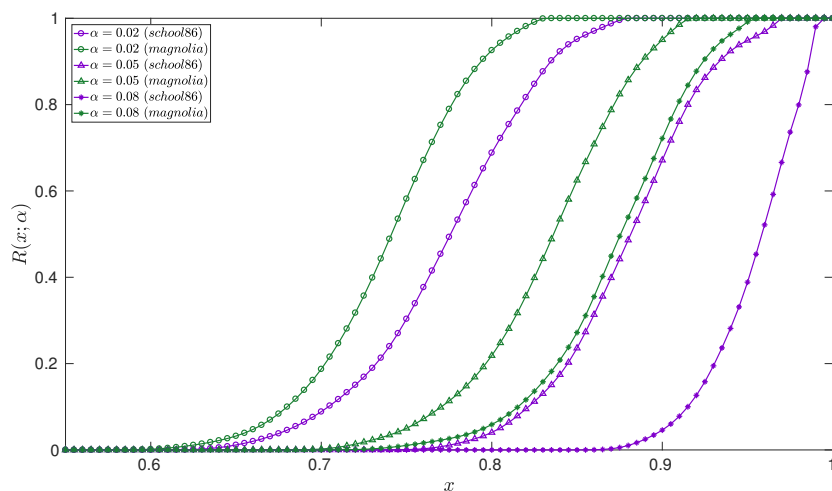


Figure 3: $R(x; \alpha)$ for school 86 and Faux Magnolia networks for three values of the attack rates.

Figure 3 shows the values of $R(x; \alpha)$ for the Faux Magnolia and the school 86 networks for the three values of α mentioned above. Figure 4 shows the values of $R(x; \alpha)$ for the different networks over a range of all the possible values of x for $\alpha = 0.05$. Each shaded region with a curve showing the median value represents each ensemble, and the shaded area lies between the 5th and the 95th quantile curves. (Results for other values of α are in the Supplementary Notes.) From Figure 4, it is evident that different values of the transmission probability, x correspond to the same value of $R(x; \alpha)$.

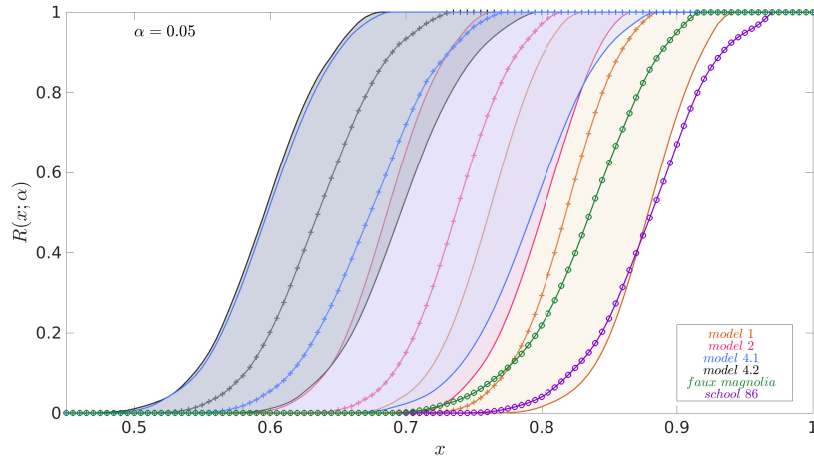


Figure 4: $R(x; \alpha)$ values for all networks for a range of values of x . Each shaded region represents one of the four models with the crossed lines representing the medians for each set and the solid lines denoting the 5th and the 95th quantile curves. The lines with the circles display the values for the Faux Magnolia and the school 86 networks.

Empirically, it turns out that the two values of x are related to each other by a quadratic (details in the Supplementary Notes). The transformed x values of the Faux Magnolia network relative to those for the school 86 network are estimated using a quadratic fit, i.e.,

$$x_{FM} = a_0 + a_1 x_{86} + a_2 x_{86}^2 \quad (2)$$

The x values re-calibrated according to equation 2 reproduce the epidemic potential for the networks. The plot in Figure 5 shows these estimated values of $R(x; \alpha)$ obtained using the quadratic polynomial fit from the transformed values of x for the case when $\alpha = 0.05$. (The plots for $\alpha = 0.02$ and 0.08 are in the Supplementary Notes.) It is to be noted that the estimated values calculated using this technique are as good as those calculated numerically for these two networks.

Figure 6 shows the time evolution of the overall fraction of infected people for three different networks - Faux Magnolia, school 86 and net1 (one of the networks from model 2) - for two values of the probability of transmission, $x = 0.85$ and 0.92 . These are the mean epicurves obtained from 10^4 SIR simulations on these networks. The error bars represent the probable error for the estimate of the mean value from the simulations. The epicurves for each run of the simulations and their mean curve are presented in the Supplementary Notes. The plot 6c shows the mean epicurves for different values of x chosen so that the average attack rate is similar for the three networks. The overall attack rate of ~ 0.06 is obtained in the school 86 network for $x = 0.92$ whereas in the Faux Magnolia network it is obtained when $x = 0.85$ (also shown in the table in the

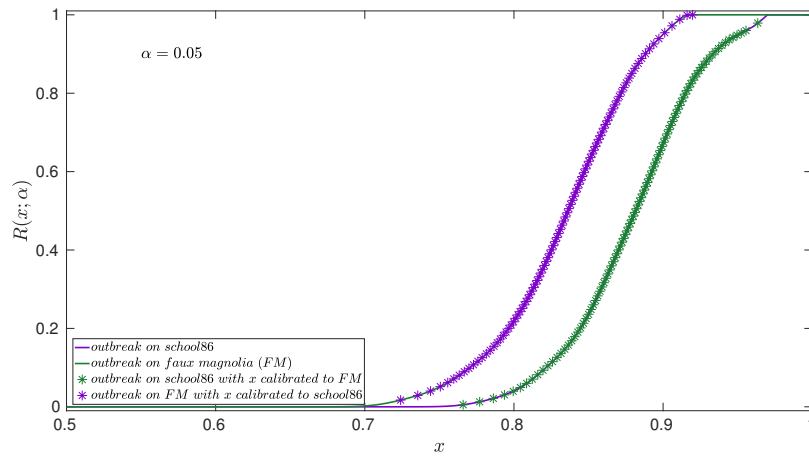


Figure 5: Plot showing that the estimated values of $R(x; \alpha)$ for the Faux Magnolia network calculated using the quadratic polynomial fit from the values obtained from the school 86 network agree with those obtained from the numerical analysis on the Faux Magnolia network.

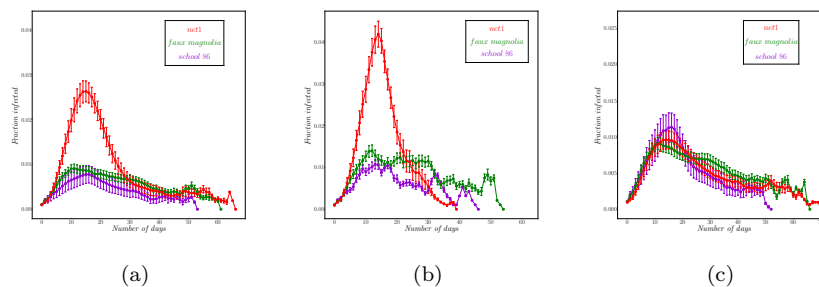


Figure 6: The mean epicurves for the three networks for two values of the probability of transmission, x . (a) $x = 0.85$ and (b) $x = 0.92$. (c) The mean epicurves for the networks for three values of transmission, $x = 0.73$ for net1, $x = 0.85$ for Faux Magnolia and $x = 0.92$ for school 86, to obtain the same average attack rate (~ 0.06). The error bars are the probable errors for the estimated mean value.

220 Supplementary Notes). For the random network (net1), a lower value, $x = 0.73$
 225 gives the same attack rate.

3. Discussion

For epidemiological purposes, it is essential to explore the effects of the network structure on the dynamical process of an infectious disease epidemic. The
 225 boxplots in Figure 1 indicate that although the ERGM succeeds in generating random graphs with similar degree distribution, the small amounts of variation

correspond to quite a significant change in the value of the epidemic threshold, x_c . The difference in the threshold values in the plots 1a and 1b could be because of the approximation of the tree-like structure in the Newman's formula [20].
230 Even though this is an important measure to study the spread of an infectious disease, it does not capture much of the dynamics. The epicurves (Figure 2) provide a better understanding of the process. The similarity of epicurves obtained from the SIR simulations implies that the additional constraints used to generate the Faux Magnolia helps to predict the time evolution of a disease on
235 the school 86 network.

Instead of determining how the system behaves for a particular value of the probability of transmission, a measure that estimates the size of an epidemic outbreak for all its possible values, $R(x; \alpha)$ is suggested [44]. Although the Faux Magnolia network model matches the desired statistics of the school 86
240 network better than the other networks generated using ERGMs, the behavior of the disease outbreak is significantly different. Figure 3 shows that the original network is significantly more resistant to disease spread than Faux Magnolia, even though they have similar local statistics. Figure 4 suggests that the school network is more resistant to an epidemic than ERGMs derived from it, i.e., a
245 higher transmission probability is required for widespread disease in the school 86 compared to other networks. In particular, the probability that an outbreak seeded in a single randomly selected individual will spread to at least 5% of the population is biased in this model.

There is a consistency in the values of the epidemic threshold (Figure 1a) and the $R(x; \alpha)$ values for the ensemble of networks generated using the ERGMs
250 (Figure 4). The model 1 and model 2 networks have higher threshold values than those of model 4.1 and model 4.2. The $R(x; \alpha)$ plots also point to the same result. The disagreement in the results for the Faux Magnolia and the school 86 networks may arise from the fact that these two are neither tree-like
255 nor random graphs. The $R(x; \alpha)$ curves demonstrate that the probability of an outbreak of a certain size on the Faux Magnolia and the school 86 networks are not the same, which corresponds to the conclusion from the epicurves. It can be seen that the propagation of infection on most networks is significantly different from that on the original network.

From Figure 6, it can be concluded that the number of people infected in the three networks, Faux Magnolia, school 86 and net1, are mismatched as
260 a function of time. Figures 6a and 6b show that the epicurves for net1 are very different from the other two. Therefore, for a fixed value of x , the Faux Magnolia performs much better than net1 in predicting an outbreak on the original school network. The two epicurves are similar in many respects, but there are systematic differences in the duration of the epidemic and the height of the peak values. The epicurves in Figure 6c lead to a surprising result. For
265 a re-calibrated value of x , the less constrained random graph (net1) performs similar to, sometimes better than the Faux Magnolia network in estimating the outbreak.
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4. Conclusions

The transmission of infectious diseases can be investigated as a diffusive process on networks. It is essential to explore the topology of the network since it affects the course of the propagation of the infection through the population. Even with the knowledge of statistics like degree distribution, number of triangles, clustering coefficients or the centrality measures, it is difficult to predict the propagation of a disease through a population. Here, the epidemic potential measured by $R(x; \alpha)$ and the mean epidemic curve for different networks are compared. These measures depend on both the global structural aspects of the contact network and the dynamics on the network.

Exponential random graph models are used to generate a number of different networks that match local statistics of one of the friendship networks from the first wave of the Add Health study. The Faux Magnolia network is one such network well known in the literature. Network measures like the epidemic threshold for these two networks are similar, suggesting that Faux Magnolia is a better model for the high school friendship data than others. However, it is observed that there are significant systematic differences in spite of the similarity of the epidemic curves. These differences will extend to the effects of time-dependent intervention measures as well.

To obtain a better insight to the behavior of epidemic spreading, the epidemic potential $R(x; \alpha)$ is evaluated. $R(x; \alpha)$ for all these networks show that the school 86 network is more resistant to large outbreaks than any of the others. Treating the transmission probability, x as a free parameter, these models can be calibrated so that they all have the same epidemic potential. But the resulting epidemic curves exhibit systematic biases in the peak height and the outbreak duration. Indeed, it turns out that a calibrated, but less constrained system performs better than one which has more constraints, even when it is calibrated. Moreover, it is suspected that different networks will exhibit different responses to interventions.

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320 References

- [1] W. O. Kermack, A. G. McKendrick, A contribution to the mathematical theory of epidemics, in: Proceedings of the Royal Society of London A: Mathematical, Physical and Engineering sciences, Vol. 115, The Royal Society, 1927, pp. 700–721.
- 325 [2] N. T. Bailey, et al., The Mathematical Theory of Infectious Diseases and its Applications, Charles Griffin & Company Ltd, 5a Crendon Street, High Wycombe, Bucks HP13 6LE., 1975.
- [3] R. M. Anderson, R. M. May, et al., Population biology of infectious diseases - Report of the Dahlem Workshop, Berlin, 14-19 March 1982., Berlin, German Federal Republic; Springer-Verlag, 1982.
- 330 [4] R. M. Anderson, R. M. May, Helminth infections of humans: Mathematical models, population dynamics, and control, *Advances in Parasitology* 24 (1985) 1–101.
- [5] R. M. Anderson, R. M. May, B. Anderson, *Infectious Diseases of Humans: Dynamics and Control*, Vol. 28, Wiley Online Library, 1992.
- 335 [6] H. W. Hethcote, The mathematics of infectious diseases, *SIAM Review* 42 (4) (2000) 599–653.
- [7] M. Morris, *Epidemiology and social networks: Modeling structured diffusion*, *Sociological Methods & Research* 22 (1) (1993) 99–126.
- 340 [8] M. Barthélemy, A. Barrat, R. Pastor-Satorras, A. Vespignani, Velocity and hierarchical spread of epidemic outbreaks in scale-free networks, *Physical Review Letters* 92 (17) (2004) 178701.
- [9] M. Barthélemy, A. Barrat, R. Pastor-Satorras, A. Vespignani, Dynamical patterns of epidemic outbreaks in complex heterogeneous networks, *Journal of Theoretical Biology* 235 (2) (2005) 275–288.
- 345 [10] S. Eubank, H. Guclu, V. A. Kumar, M. V. Marathe, A. Srinivasan, Z. Toroczkai, N. Wang, Modelling disease outbreaks in realistic urban social networks, *Nature* 429 (6988) (2004) 180–184.

- [11] K. R. Bisset, J. Chen, X. Feng, V. Kumar, M. V. Marathe, Epifast: A
350 fast algorithm for large scale realistic epidemic simulations on distributed
memory systems, in: Proceedings of the 23rd International Conference on
Supercomputing, ACM, 2009, pp. 430–439.
- [12] L. A. Meyers, B. Pourbohloul, M. E. Newman, D. M. Skowronski, R. C.
355 Brunham, Network theory and SARS: Predicting outbreak diversity, *Journal of Theoretical Biology* 232 (1) (2005) 71–81.
- [13] V. Colizza, A. Barrat, M. Barthélemy, A.-J. Valleron, A. Vespignani, Mod-
eling the worldwide spread of pandemic influenza: baseline case and con-
tainment interventions, *PLoS Med* 4 (1) (2007) e13.
- [14] M. E. Newman, S. H. Strogatz, D. J. Watts, Random graphs with arbitrary
360 degree distributions and their applications, *Physical Review E* 64 (2) (2001)
026118.
- [15] A. L. Lloyd, S. Valeika, Network models in epidemiology: An overview,
*Complex Population Dynamics: Nonlinear Modeling in Ecology, Epidemiol-
ogy and Genetics* (2007) 189–214.
- [16] J. Lindquist, J. Ma, P. Van den Driessche, F. H. Willeboordse, Effective
365 degree network disease models, *Journal of Mathematical Biology* 62 (2)
(2011) 143–164.
- [17] J. C. Miller, A. C. Slim, E. M. Volz, Edge-based compartmental modelling
for infectious disease spread, *Journal of the Royal Society Interface* 9 (70)
370 (2012) 890–906.
- [18] J. C. Miller, E. M. Volz, Incorporating disease and population structure
into models of SIR disease in contact networks, *PloS One* 8 (8) (2013)
e69162.
- [19] S. Bansal, B. T. Grenfell, L. A. Meyers, When individual behaviour mat-
375 ters: homogeneous and network models in epidemiology, *Journal of the
Royal Society Interface* 4 (16) (2007) 879–891.
- [20] M. E. Newman, Spread of epidemic disease on networks, *Physical Review
E* 66 (1) (2002) 016128.
- [21] Y. Wang, D. Chakrabarti, C. Wang, C. Faloutsos, Epidemic spreading in
380 real networks: An eigenvalue viewpoint, in: Proceedings 22nd International
Symposium on Reliable Distributed Systems, IEEE, 2003, pp. 25–34.
- [22] A. Ganesh, L. Massoulié, D. Towsley, The effect of network topology on the
spread of epidemics, in: Proceedings IEEE 24th Annual Joint Conference
of the IEEE Computer and Communications Societies., Vol. 2, IEEE, 2005,
385 pp. 1455–1466.

- [23] Y. Moreno, R. Pastor-Satorras, A. Vespignani, Epidemic outbreaks in complex heterogeneous networks, *The European Physical Journal B-Condensed Matter and Complex Systems* 26 (4) (2002) 521–529.
- [24] A.-L. Barabási, R. Albert, Emergence of scaling in random networks, *Science* 286 (5439) (1999) 509–512.
- [25] D. J. Watts, S. H. Strogatz, Collective dynamics of ‘small-world’ networks, *Nature* 393 (6684) (1998) 440–442.
- [26] G. Robins, P. Pattison, Y. Kalish, D. Lusher, An introduction to exponential random graph (p^*) models for social networks, *Social Networks* 29 (2) (2007) 173 – 191, special Section: Advances in Exponential Random Graph (p^*) Models. doi:<http://dx.doi.org/10.1016/j.socnet.2006.08.002>. URL <http://www.sciencedirect.com/science/article/pii/S0378873306000372>
- [27] J. Park, M. E. Newman, Statistical mechanics of networks, *Physical Review E* 70 (6) (2004) 066117.
- [28] S. M. Goodreau, J. A. Kitts, M. Morris, Birds of a feather, or friend of a friend? Using exponential random graph models to investigate adolescent social networks*, *Demography* 46 (1) (2009) 103–125.
- [29] E. O. Laumann, Y. Youm, Racial/ethnic group differences in the prevalence of sexually transmitted diseases in the United States: A network explanation, *Sexually Transmitted Diseases* 26 (5) (1999) 250–261.
- [30] A. Jolly, S. Muth, J. Wylie, J. Potterat, Sexual networks and sexually transmitted infections: A tale of two cities, *Journal of Urban Health* 78 (3) (2001) 433–445.
- [31] F. Liljeros, C. R. Edling, L. A. N. Amaral, Sexual networks: Implications for the transmission of sexually transmitted infections, *Microbes and Infection* 5 (2) (2003) 189–196.
- [32] W. C. Miller, C. A. Ford, M. Morris, M. S. Handcock, J. L. Schmitz, M. M. Hobbs, M. S. Cohen, K. M. Harris, J. R. Udry, Prevalence of Chlamydial and Gonococcal infections among young adults in the United States, *JAMA* 291 (18) (2004) 2229–2236.
- [33] M. Morris, M. S. Handcock, W. C. Miller, C. A. Ford, J. L. Schmitz, M. M. Hobbs, M. S. Cohen, K. M. Harris, J. R. Udry, Prevalence of HIV infection among young adults in the United States: Results from the Add Health Study, *American Journal of Public Health* 96 (6) (2006) 1091–1097.
- [34] M. Morris, A. E. Kurth, D. T. Hamilton, J. Moody, S. Wakefield, Concurrent partnerships and HIV prevalence disparities by race: Linking science and public health practice, *American Journal of Public Health* 99 (6) (2009) 1023–1031.

- 425 [35] M. D. Resnick, P. S. Bearman, R. W. Blum, K. E. Bauman, K. M. Harris,
J. Jones, J. Tabor, T. Beuhring, R. E. Sieving, M. Shew, et al., Protecting
adolescents from harm: Findings from the National Longitudinal Study on
Adolescent Health, *JAMA* 278 (10) (1997) 823–832.
- [36] J. R. Udry, P. S. Bearman, New methods for new research on adolescent
430 sexual behavior, Cambridge University Press, 1998.
- [37] E. Moore, C. Shannon, Reliable circuits using less reliable relays, *Journal
of the Franklin Institute* 262 (3) (1956) 191–208.
- [38] D. T. Hamilton, M. S. Handcock, M. Morris, Degree distributions in sexual
435 networks: A framework for evaluating evidence, *Sexually Transmitted
Diseases* 35 (1) (2008) 30.
- [39] G. Robins, T. Snijders, P. Wang, M. Handcock, P. Pattison, Recent de-
velopments in exponential random graph (p^*) models for social networks,
Social Networks 29 (2) (2007) 192–215.
- [40] T. A. Snijders, P. E. Pattison, G. L. Robins, M. S. Handcock, New speci-
440 fications for exponential random graph models, *Sociological Methodology*
36 (1) (2006) 99–153.
- [41] M. S. Handcock, D. R. Hunter, C. T. Butts, S. M. Goodreau, M. Morris,
statnet: Software tools for the Statistical Modeling of Network Data, Seat-
tle, WA (2003).
445 URL <http://statnetproject.org>
- [42] M. S. Handcock, D. R. Hunter, C. T. Butts, S. M. Goodreau, P. N. Kriv-
itsky, M. Morris, ergm: Fit, Simulate and Diagnose Exponential-Family
Models for Networks, The Statnet Project (<http://www.statnet.org>), r
package version 3.6.0 (2016).
450 URL <http://CRAN.R-project.org/package=ergm>
- [43] D. R. Hunter, M. S. Handcock, C. T. Butts, S. M. Goodreau, Morris,
Martina, ergm: A package to fit, simulate and diagnose exponential-family
models for networks, *Journal of Statistical Software* 24 (3) (2008) 1–29.
- [44] M. Youssef, Y. Khorramzadeh, S. Eubank, Network reliability: The effect
455 of local network structure on diffusive processes, *Physical Review E* 88 (5)
(2013) 052810.