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Percolation on random networks with proliferation

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The literature on growing network models, exemplified by the preferential attachment model and the copying model, has followed an exponential growth in the last few years. In many real-life scenarios, however, the adding of new nodes and edges is not an exogenous process, but involves inheritance and sharing of the local environment of the existing ones. In this paper, we develop a mathematical framework to analytically and numerically study the percolation properties of the random networks with proliferation. We compare random attack and localized attack on benchmark models, including Erdős-Rényi networks, random regular networks, and scale-free networks, with proliferation mechanism. Our results highlight the non-monotonic connections with robustness and growth, and unravel an intriguing opposite effect for random and localized attacks. In particular, it is shown that unbalanced proliferation enhances robustness to random attack while mitigates robustness to localized attack, both independent of the network degree distribution.

Keywords: Complex network; growth model; robustness; attack; inheritance.

1. Introduction

Networks on which many natural and synthetic systems such as the Internet, electric power grids, and brains are built provide efficient and simplistic settings to understand the structure and function of networked systems and to unravel the emergence of complex phenomena ^{1,2}. The research in networks focused initially on static networks with fixed numbers of nodes and edges, while presently it focuses more on evolving and growing ones, approximating the growth of real-world networks. The class of growing network models, exemplified by the seminal preferential attachment or Barabási-Albert model ³, has been made widely popular in statistical physics and computer science. Preferential attachment, meaning that the more connected nodes are more likely to acquire links in the future, is found responsible for the very common scale-free property—typically characterized by the intriguing power-law degree distributions—in diverse growing networks including the Internet, World Wide Web, protein p53 and citation networks ^{4,5,6}. Such networks (called scale-free networks), containing a small number of highly connected nodes (hubs) and a huge number of low-degree nodes, are extremely robust against

random decay but vulnerable to hub-targeted attack⁷. The preferential attachment mechanism has been generalized to incorporate nonlinearity^{8,9}, fitness¹⁰, ranking¹¹, and node similarity (homophily)¹². Another popular growing network model involves the copying rule¹³, where a new node is attached to a randomly chosen node and copies some links of the latter. A variant of duplication-mutation mechanism¹⁴ characterizes the growth of some biological networks including the protein interaction networks and gene regulatory networks.

The main motivation of network growth studies so far has been to link growth mechanism to emergent phenomena of the network such as a power-law degree distribution, giving rise to a common pattern of adding new nodes into the network and connecting them to either random or optimized existing ones. However, there is an entire class of real-life growing networks in which the birth of nodes has an intimate relationship with the old ones and their associated local resources. Cell division is an illustrative example of such growth model with biological relevance^{15,16}. The newly born nodes inherit and share the surroundings of their parent nodes, which are distinct from preferential attachment and duplication mechanism (see Fig. 1). In socioeconomic networks, the property inheritance has much the same flavor¹⁷. Other examples include setting up a new airport in a metropolis with an existing one—they together share the passengers leading to a redistribution of the city’s air traffic—and backup/auxiliary components in diverse technical networks to improve the performance of critical systems by sharing the load^{18,19}. Given the much studied “robust yet fragile” paradigm of scale-free networks that are typically generated through preferential attachment or copying, we still have very limited knowledge on the resilience of networks with the above proliferation mechanism, which, nonetheless, are of important practical relevance and attractive in its own right.

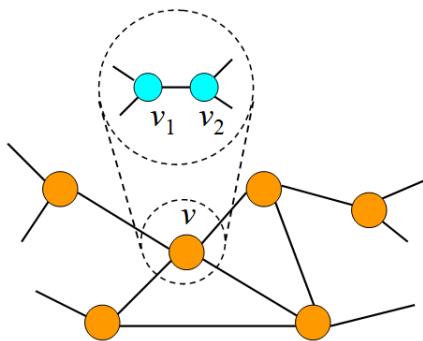


Fig. 1. Schematic of a node v of degree 4 that is divided into two connected nodes v_1 and v_2 , both having degree 3. Here, $a_4 = b_4 = 2$.

2. Main results

To fill this gap, here we introduce a minimal model of percolation on random growing networks, which captures key features of proliferation growth mechanism. We investigate the network robustness under random attack (RA)^{20,21,22} as well as localized attack (LA)^{23,24}. We investigate the critical occupation probability q_c of the network, above which a giant component with a size proportional to that of the entire network first emerges, and the relative size of the giant component P_∞ .

We consider the archetypal configuration model that allows for networks with arbitrary degree distribution p_k ²⁵, i.e., the probability that a randomly chosen node has degree k . Configuration model has been a popular choice in percolation studies since it combines the ability to specify the distribution of the number of connections with analytical tractability^{20,26,27}. Let us fix two non-negative integer sequences $\{a_k\}$ and $\{b_k\}$ such that $a_k + b_k = k$ for $k \geq 0$. Let r_k be the reproduction probability that a (parent) node is divided into two connected (child) nodes, which are linked to a_k and b_k different nodes, respectively, in the neighborhood of the parent node given that the parent node has degree k . To fix the notation, we refer to the child node with degree $a_k + 1$ as type-1 and the other one with degree $b_k + 1$ as type-2 (see Fig. 1). Thus, each parent node and its outgoing half-edges are partitioned into two parts, mimicking the microscopic proliferation¹⁵. Further, we define $q_k^{(1)}$ (or resp., $q_k^{(2)}$) as the probability that a type-1 (or resp., type-2) node is occupied given that its parent has degree k . Similarly, q_k^* means the probability that a node is occupied given that it does not reproduce and that it has degree k . Then the probability generating function for occupied node degree distribution is given by

$$F_0(x) = \sum_{k=0}^{\infty} \left[\frac{1}{2} q_k^{(1)} p_k r_k x^{a_k+1} + \frac{1}{2} q_k^{(2)} p_k r_k x^{b_k+1} + q_k^* p_k (1 - r_k) x^k \right], \quad (1)$$

where $\frac{1}{2} q_k^{(i)} p_k r_k$ is the probability of being occupied for a type- i node ($i = 1, 2$), and $q_k^* p_k (1 - r_k)$ is the probability of having degree k and being occupied for a non-reproductive node. Note that the case of $r_k \equiv 0$ reduces to the fundamental non-reproductive node percolation²⁰.

Suppose that we follow a randomly chosen edge, and then the node reached has degree $\frac{k p_k}{\sum_k k p_k}$ if it is non-reproductive, $\frac{(a_k+1)p_k}{\sum_k (k+2)p_k}$ if it is type-1, and $\frac{(b_k+1)p_k}{\sum_k (k+2)p_k}$ if it is type-2. Hence, the excess degree distribution²⁵ for such a node is generated by

$$F_1(x) = \sum_{k=0}^{\infty} \left[\frac{(a_k+1)p_k}{\sum_k (k+2)p_k} q_k^{(1)} r_k x^{a_k} + \frac{(b_k+1)p_k}{\sum_k (k+2)p_k} q_k^{(2)} r_k x^{b_k} + \frac{k p_k}{\sum_k k p_k} q_k^* (1 - r_k) x^{k-1} \right]. \quad (2)$$

Let $H_1(x)$ be the generating function for the distribution of the size of a percolation cluster that is reached by choosing a random edge and following it to its end, say,

v. Therefore, $H_1(x)$ satisfies the self-consistency condition

$$H_1(x) = 1 - F_1(1) + xF_1[H_1(x)], \quad (3)$$

where $1 - F_1(1)$ represents the probability that v is unoccupied, and the term $xF_1[H_1(x)]$ accounts for an occupied v with k other edges leading out of it, distributed according to $F_1(x)$ ^{2,25}. The probability distribution of the size of percolation cluster to which a randomly chosen node belongs is analogously generated by $H_0(x)$, where

$$H_0(x) = 1 - F_0(1) + xF_0[H_1(x)]. \quad (4)$$

In order to gain useful insights on the percolation threshold and its implications on network proliferation, we in the sequel set $q_k^{(1)} = q_k^{(2)} = q_k^* = q$ and $r_k = r$ for all k . Moreover, we set $a_k = \lfloor \alpha k \rfloor$ and $b_k = \lceil (1 - \alpha)k \rceil$ for all k , where $\alpha \in [0, 1/2]$ indicates the ratio between the two types of child nodes. Note that we do not need to consider $\alpha \in [1/2, 1]$, which simply exchanges the two types of nodes and will not affect our results. With these simplifications, Eqs. (1) and (2) reduce to

$$F_0(x) = \frac{1}{2}qr[G^{(1)}(x) + G^{(2)}(x)] + q(1 - r)G^{(0)}(x) \quad (5)$$

and

$$F_1(x) = qr\left[\frac{G^{(1)'}(x) + G^{(2)'}(x)}{G^{(1)'}(1) + G^{(2)'}(1)}\right] + q(1 - r)\frac{G^{(0)'}(x)}{G^{(0)'}(1)}, \quad (6)$$

where $G^{(0)}(x) = \sum_k p_k x^k$ is the generating function for node degree² while $G^{(1)}(x) = \sum_k p_k x^{\alpha k+1}$ and $G^{(2)}(x) = \sum_k p_k x^{(1-\alpha)k+1}$ are responsible for type-1 and type-2 nodes, respectively. It follows from (5) and (6) that $F_0(1) = F_1(1) = q$. In fact, we have $F_0(1) = q$ for arbitrary reproduction probability r_k (c.f. (1)), which can be regarded as the overall fraction of occupied nodes in the entire network.

Drawing on Eqs. (3)-(6), the mean size of cluster to which a randomly chosen node belongs, in the absence of giant clusters, can be calculated as

$$\langle s \rangle = H'_0(1) = q \left\{ 1 + \frac{\frac{qr}{2}[G^{(1)'}(1) + G^{(2)'}(1)] + q(1 - r)G^{(0)'}(1)}{1 - \frac{qr[G^{(1)''}(1) + G^{(2)''}(1)]}{G^{(1)'}(1) + G^{(2)'}(1)} - \frac{q(1-r)G^{(0)''}(1)}{G^{(0)'}(1)}} \right\}. \quad (7)$$

This quantity diverges at the critical percolation threshold q_c , where a giant component first appears, yielding

$$\begin{aligned} q_c &= \left\{ \frac{r[G^{(1)''}(1) + G^{(2)''}(1)]}{G^{(1)'}(1) + G^{(2)'}(1)} + \frac{(1 - r)G^{(0)''}(1)}{G^{(0)'}(1)} \right\}^{-1} \\ &= \left\{ r \frac{(2\alpha^2 - 2\alpha + 1)\langle k^2 \rangle + \langle k \rangle}{\langle k \rangle + 2} + (1 - r) \left(\frac{\langle k^2 \rangle}{\langle k \rangle} - 1 \right) \right\}^{-1}, \end{aligned} \quad (8)$$

where $\langle k \rangle = G^{(0)'}(1)$ is the average degree of the network. The relative size of the giant component (P_∞) in the network is given by

$$P_\infty = 1 - H_0(1) = F_0(1) - F_0[H_1(1)], \quad (9)$$

where $H_1(1)$ satisfies the transcendental equation $H_1(1) = 1 - F_1(1) + F_1[H_1(1)]$.

It follows from Eq. (8) that q_c is an increasing function with respect to $\alpha \in [0, 1/2]$ for any $r > 0$, meaning that a growing network with unbalanced proliferation (i.e., a smaller α) is more robust to RA regardless of the original degree distribution of the network. This is remarkable in the sense that it adds a new dimension into the picture of network resilience independent of degree distribution, and that it offers a potential reason from the viewpoint of resilience behind the observed unbalanced offspring cell size in Chlamydomonas uncovered by the recent studies in the molecular basis underpinning cell division^{28,16}.

For a scale-free (SF) network with power-law distribution $p_k \sim k^{-\gamma}$, we may calculate the order parameter critical exponent η ²⁹: the probability of belonging to the giant component behaves as $P_\infty \sim (q - q_c)^\eta$ near criticality. In view of (9) and setting $u = H_1(1)$, we obtain

$$\begin{aligned} 1 - \varepsilon = & 1 - q + \frac{qr}{\langle k \rangle + 2} \left[\sum_k (ak + 1)p_k u^{\alpha k} + \sum_k ((1 - \alpha)k + 1)p_k u^{(1-\alpha)k} \right] \\ & + \frac{q(1 - r)}{\langle k \rangle} \sum_k kp_k u^{k-1}, \end{aligned} \quad (10)$$

where $u = 1 - \varepsilon$ and $q = q_c + \delta$. Along the same line of derivation in²⁹, by using Taylor's expansion, we are led to the conclusion that the critical exponents η are not model dependent but only depend on both γ and α when $r > 0$ and only depend on γ when $r = 0$ (which recovers the result for non-proliferation SF networks). For example, when $\alpha = 0$, we obtain $\eta = (\gamma - 3)^{-1}$ when $\gamma \in (3, 4)$, and $\eta = 1$ when $\gamma > 4$. For $\alpha > 0$, the precise calculation of η can be quite tedious. Also note that for SF network with power-law distribution $p_k \sim k^{-\gamma}$ with $1 < \gamma \leq 3$, q_c approaches zero for all α and r by (8) since the ratio $\langle k^2 \rangle / \langle k \rangle$ diverges in this range of γ . It suggests that SF networks with proliferation remain robust, reminiscent of the well-known "robust against random decay" paradigm of Cohen et al.²¹ found in non-proliferated scale-free networks.

We now test our theory numerically for Erdős-Rényi (ER) networks with a Poisson degree distribution $p_k = e^{-\lambda} \lambda^k / k!$ and $\lambda = \langle k \rangle$, random regular (RR) networks following a degenerated degree distribution $p_k = \delta_{k,k_0}$ concentrated on k_0 , and SF networks characterized by $p_k \sim k^{-\gamma}$ with lower and upper cut-offs, k_{\min} and k_{\max} . Percolation transitions are shown in the left columns of Fig. 2 and Fig. 3 for ER, RR, and SF networks, respectively. As can be seen, the theory agrees very well with the simulation results.

A couple of interesting comments can be drawn from the results. First, as expected from the above analysis, an increase in α systematically yields an increase in percolation threshold q_c for all values of $r > 0$ and all network topologies. Moreover, for all networks and any given value of $r > 0$, q_c is found to increase in a linear form when $\alpha \leq 0.3$ and gradually saturate at a plateau when α goes from 0.3 to 0.5. This is better appreciated in the inset of Fig. 2(a) for ER networks, which implies that relatively mild unbalance in reproduction could not gain prominent resilience

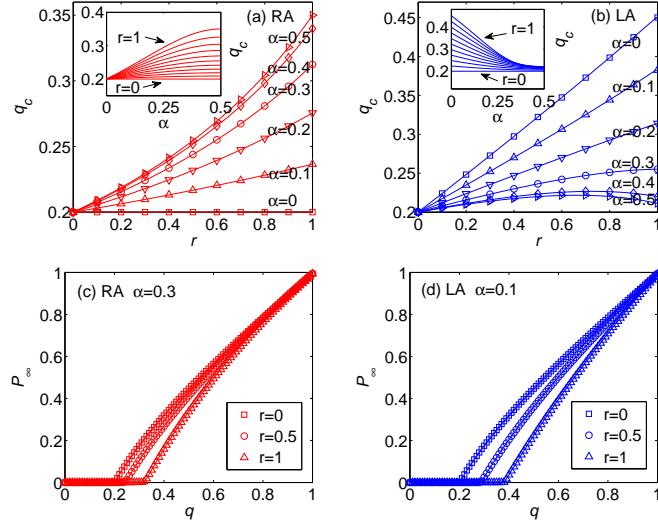


Fig. 2. First row: percolation thresholds q_c for ER networks of $N = 10^6$ nodes and average degree $\langle k \rangle = 5$ under (a) random attack (RA) and (b) localized attack (LA) from simulations with $\alpha = 0$ (squares), $\alpha = 0.1$ (upper triangles), $\alpha = 0.2$ (lower triangles), $\alpha = 0.3$ (circles), $\alpha = 0.4$ (diamonds), and $\alpha = 0.5$ (right triangles). The insets show q_c versus α for increasing r from 0 to 1 with an increment of 0.1. Second row: fractions of giant components P_∞ as functions of q for (c) $\alpha = 0.3$ under RA and (d) $\alpha = 0.1$ under LA from simulations with $r = 0$ (squares), $r = 0.5$ (circles), and $r = 1$ (triangles). Simulation results are averaged over 50 random graphs with 20 independent runs for each data point; solid lines are the theoretical results.

against RA, going some way to pushing the unbalance to an observed-effect level in some real systems²⁸. Second, for ER networks, q_c increases as r grows for any given $\alpha > 0$ but remains unchanged for $\alpha = 0$. For SF networks, q_c is increasing for all $\alpha \geq 0$, while for RR networks, there exists a clear demarcation of monotonicity signified by $\alpha = 0.0417$; see Fig. 3(c). Intuitively, for the network with a heterogeneous degree distribution, breaking nodes into two child nodes generally attenuates the heterogeneity for all α (esp. large α), which makes the network less robust to RA⁷. However, for homogeneous networks, such reproduction may instead increase its heterogeneity in degree (esp. when α is small) providing extra robustness against RA. In fact, by defining the indicator

$$\rho(\alpha) = \alpha(\alpha - 1)\langle k \rangle + \frac{\langle k \rangle^2 + \langle k \rangle}{\langle k^2 \rangle}, \quad (11)$$

which combines the influences of density and heterogeneity of the network, we derive that $q_c(r)$ is increasing, decreasing, and constant when $\rho(\alpha) < 1$, > 1 , and $= 1$, respectively, employing Eq. (8). The results for ER, SF, and RR networks are summarized in Table I, which agrees with the simulations in Figs. 2 and 3.

Next, we perform a comparative study on LA, a promising alternative for RA, where nodes surrounding a seed node are removed layer by layer, causing aggregated damage of adjacent components limited to a specific area, referred to as an “attacked

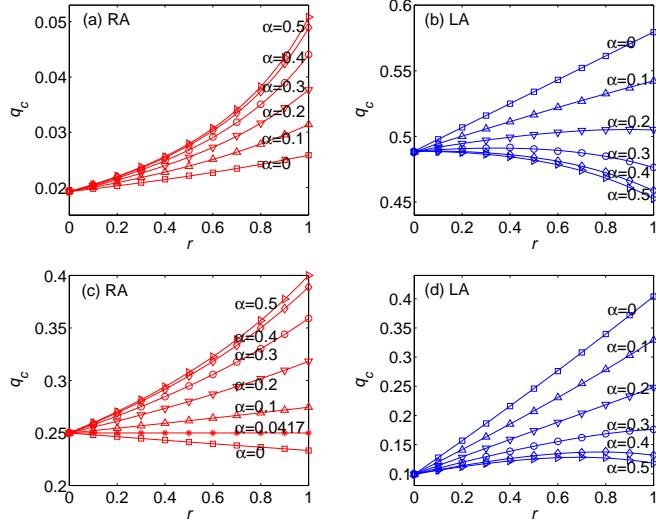


Fig. 3. First row: percolation thresholds q_c for SF networks of $N = 10^6$ nodes, $\gamma = 2.4$, $k_{\min} = 2$, and $\langle k \rangle = 5$ under (a) RA and (b) LA from simulations with $\alpha = 0$ (squares), $\alpha = 0.1$ (upper triangles), $\alpha = 0.2$ (lower triangles), $\alpha = 0.3$ (circles), $\alpha = 0.4$ (diamonds), and $\alpha = 0.5$ (right triangles). Second row: percolation thresholds q_c for RR networks of $N = 10^6$ nodes and $k_0 = 5$ under (a) RA and (b) LA from simulations with $\alpha = 0$ (squares), $\alpha = 0.0417$ (stars), $\alpha = 0.1$ (upper triangles), $\alpha = 0.2$ (lower triangles), $\alpha = 0.3$ (circles), $\alpha = 0.4$ (diamonds), and $\alpha = 0.5$ (right triangles). Simulation results are averaged over 50 random graphs with 20 independent runs for each data point; solid lines are the theoretical results.

	ER	SF ($1 < \gamma \leq 3$)	RR ($k_0 \geq 2$)
$\rho(\alpha) = 1$	$\alpha = 0$	—	$\alpha = \alpha^*$
$\rho(\alpha) < 1$	$\alpha \in (0, 0.5]$	$\alpha \in [0, 0.5]$	$\alpha \in (\alpha^*, 0.5]$
$\rho(\alpha) > 1$	—	—	$\alpha \in [0, \alpha^*)$

Table I Given $\alpha \in [0, 0.5]$, the indicator $\rho(\alpha)$ determines the monotonicity of percolation threshold $q_c(r)$ under RA. Results for ER, SF, RR networks are characterized, where $\alpha^* = \frac{1}{2} \left(1 - \sqrt{1 - \frac{4}{k_0^2}} \right)$.

hole”^{23,30,31,32}. We begin with the initially intact network, namely, $q_k^{(1)} = q_k^{(2)} = q_k^* \equiv 1$, and hence the equivalent of Eq. (1) for a randomly chosen node is

$$G_0(x) = \sum_{k=0}^{\infty} \left[\frac{1}{2} p_k r_k x^{a_k+1} + \frac{1}{2} p_k r_k x^{b_k+1} + p_k (1 - r_k) x^k \right]. \quad (12)$$

With the same simplification leading to Eqs. (5) and (6), we see that $G_0(x) = F_0(x)/q$, where $F_0(x)$ is given by Eq. (5). By performing the LA process until a

fraction $1 - q$ of nodes in the network are removed, the generating function of the degree distribution of the remaining network is²³

$$\tilde{G}_0(x) = \frac{1}{G_0(f)} G_0 \left[f + \frac{G'_0(f)}{G'_0(1)} (x-1) \right], \quad (13)$$

where $f \equiv G_0^{-1}(q)$. Let $\tilde{G}_1(x) = \tilde{G}'_0(x)/\tilde{G}'_0(1)$. Using the criterion for the network to collapse, $\tilde{G}'_1(1) = 1$, the critical percolation threshold q_c satisfies²⁵

$$G''_0[G_0^{-1}(q_c)] = G'_0(1). \quad (14)$$

The fraction P_∞ of the giant component in the network is given by

$$P_\infty = q \{1 - \tilde{G}_0[\tilde{H}_1(1)]\}, \quad (15)$$

where $\tilde{H}_1(1)$ satisfies the transcendental equation $\tilde{H}_1(1) = \tilde{G}_1[\tilde{H}_1(1)]$.

The right columns of Figs. 2 and 3 show the corresponding percolation transitions for ER, RR, and SF networks, respectively, under LA. Theoretical results and simulations agree well with each other. Surprisingly, the results for LA display distinctive behaviors as compared to RA. First, at any give reproduction level of $r > 0$, the percolation threshold q_c decreases with respect to α for all networks. Furthermore, the results show a linear decay in $q_c(\alpha)$ for $\alpha \in [0, 0.3]$ gradually becoming flat when α goes beyond about 0.3. One intuitive explanation is that the higher degree nodes are more likely within the attacked hole accelerating the network fragmentation, and that the smaller α is the more heterogeneous the resulting degree distribution becomes regardless of the initial degree distribution of the network. Second, for a given α , $q_c(r)$ is no longer monotonic in general (especially for large α). In particular, proliferation could improve the robustness for SF networks against LA (see Fig. 3(b)) but not for ER or RR networks, in contrast to the RA situation, where only RR networks could be made more robust (see Fig. 3(c)). We contend that this intriguing phenomenon may find its origin in the delicate balance between the two competitive factors behind LA²³, namely, the heterogeneity factor accelerating the fragmentation as discussed above and the localization factor that only nodes on the surface of the attacked hole contribute to the breakdown mitigating the fragmentation. When α is large, the heterogeneity factor (esp. for SF networks) declines, and the localization factor becomes more dominant making the network more robust with the growth of r . For ER and RR networks with proliferation, it seems that the first effect stays dominant and the robustness will be weakened as compared to the non-proliferation case (i.e., $r = 0$).

Finally, we test robustness of real-world networks with proliferation under LA using a road network of California³³ where intersections and endpoints are represented as nodes, and a movie actor collaboration network³ where two actors are connected if they were cast together in the same movie. The road network is a low-dimensional grid network with average degree around 3 while the movie actor network has a power-law exponent approximately 2.1. Fig. 4(a) shows that when $\alpha = 0.5$ the critical threshold $q_c(r)$ increases attaining a maximum at around $r = 0.6$

and then decreases in line with the above analysis. We also observe from Fig. 4(b) that the network robustness is improved when $\alpha = 0.5$ for any $r > 0$ supporting our theoretical results for SF networks under large α .

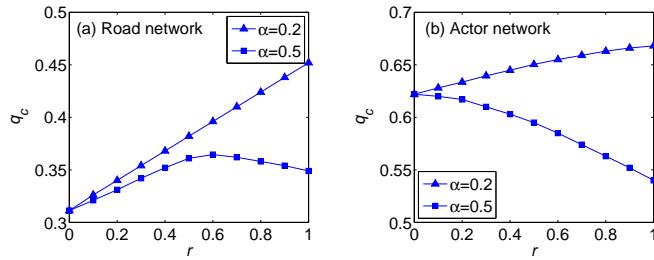


Fig. 4. Percolation thresholds q_c as a function of r under LA for (a) Road network of $N = 1965206$ nodes and (b) Actor network of $N = 382219$ nodes with $\alpha = 0.2$ (triangles) and $\alpha = 0.5$ (squares). Simulation results are averaged over 100 independent runs for each data point.

3. Conclusion

To conclude, we have studied the percolation properties of a random network model with proliferation under RA and LA. We identified the critical ratio α^* between the offsprings that the percolation transition remains unchanged for all level r of reproduction under RA for networks with an arbitrary degree distribution. Among other results, we find that proliferation has a non-monotonic effect on network robustness for LA, which seems opposite, at least in terms of the percolation thresholds for ER, RR, and SF networks, to that for RA. The percolation critical exponents can also be explored analytically under our framework. The proposed model could form the basis for future investigations of many interesting properties, including epidemic processes and dynamical systems on endogenous growing networks.

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10 *Yilun Shang*

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