# Scaling of nodal resilience and influence in complex dynamical networks **⊘**

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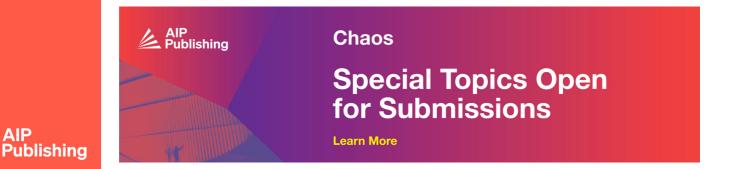




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#### **ABSTRACT**

In complex dynamical networks, the resilience of the individual nodes against perturbation and their influence on the network dynamics are of great interest and have been actively investigated. We consider situations where the coupling dynamics are separable, which arise in certain classes of dynamical processes including epidemic spreading, population dynamics, and regulatory processes, and derive the algebraic scaling relations characterizing the nodal resilience and influence. Utilizing synthetic and empirical networks of different topologies, we numerically verify the scaling associated with the dynamical processes. Our results provide insights into the interplay between network topology and dynamics for the class of processes with separable coupling functions.

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In applications involving complex dynamical networks, it is often of interest to assess the ability of individual nodes to withstand disruptions or perturbations. This defines the nodal resilience. A network with more resilient nodes is naturally more resistant to global catastrophic dynamical events such as cascading failures. It is also useful to quantify an individual node's influence on the behaviors of other nodes in the network, as such influences determine the dynamics on the whole network. Are there general scaling relations characterizing the dependence of nodal resilience and influence on the degree? To answer this question for arbitrary nodal dynamical interactions is difficult. However, if the nodeto-node coupling dynamics are separable, the scaling relations exist and can be derived analytically. Situations where complex networks host separable coupling dynamics can in fact arise in physical and biological contexts such as epidemic spreading, population dynamics, and regulatory processes. This paper presents an analytic theory to show that both nodal resilience and influence scale with the degree algebraically, with extensive numerical support from a large number of synthetic and empirical networks of different topologies. The algebraic scaling relations are robust against variations in network properties such as the clustering coefficient, degree correlation, and heterogeneity. The findings provide insights into the interplay between network topology

and dynamics for tasks such as robustness analysis, critical node identification, and network stability enhancement.

#### I. INTRODUCTION

Extensive research has established that the structures of many complex networks in the real world, to some extent, exhibit general features including random,1 small-world,2 and scale-free3,4 topologies. In addition to these three well-studied topologies, networks possessing a self-similar structure can also arise.<sup>5</sup> Each topology can be generated by a set of elementary governing rules, e.g., the preferential-attachment rule that results in the scale-free topology.<sup>3,4</sup> Complex networks in the real world host dynamical processes, leading to complex dynamical networked systems. Well-studied processes include epidemic transmission,6-9 traffic flows,10 biological competitions,<sup>11–13</sup> cascading failures,<sup>14–16</sup> and cellular signaling.<sup>17</sup>

Considerable efforts were devoted to searching for general dynamical behaviors on complex networks. Earlier, a class of dynamics on weighted complex networks was uncovered,<sup>24</sup> where the topological details of various properly weighted real-world networks tend to have little influence on a variety of dynamical processes on the network, suggesting the possibility of developing

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general strategies for controlling network dynamics. The networks can be biological, physical, technological, or social, and the dynamical processes studied include synchronization, epidemic spreading, and percolation. Later, some common network dynamics were identified<sup>25</sup> and it was also found that a diverse array of flow patterns on complex networks can be mapped into and described by some specific function.<sup>26,27</sup> Subsequently, three generic modes associated with spatiotemporal signal propagation in complex networks were discovered: distance-limited, degree-limited, and composite propagation.<sup>28</sup> Quite recently, discrete stability categories in complex networked dynamics were unveiled: asymptotically unstable, sensitive, and asymptotically stable.<sup>29</sup>

In this paper, we investigate nodal resilience and influence in complex dynamical networks. In particular, an individual node's resilience measures its capability to withstand disruptions or perturbations, which underscores the robustness of the entire system.<sup>30</sup> A node's influence on the network dynamics is also of interest, as certain nodes can have a disproportional effect on the interactions and information flow in the network.<sup>31,32</sup> Studies also offered insights into the related issue of adaptive mechanisms of nodes in dynamical networks<sup>30,33,34</sup> and the evolving landscape of nodal influences.<sup>35,36</sup> In spite of these works, a theoretical framework to explain the resilience and influence of nodes was lacking. To partially address this issue, we consider a special class of dynamical networks whose coupling dynamics are separable and investigate the scaling of the resilience and influence with the nodal degree under short-term or long-term perturbations. Representative dynamical processes with separable coupling functions include certain classes of epidemic spreading, population dynamics, and regulatory processes. We analytically find that the scaling is algebraic and validate them using synthetic and empirical networks. Depending on the specific dynamical process, the nodal resilience and influence can be degree enhancing, degree uniform, or degree suppressing. Our results offer further insights into the interplay between network topology and dynamics for tasks such as robustness analysis, critical node identification, and network stability enhancement.

#### II. NODAL RESILIENCE AND INFLUENCE

We consider a complex dynamical network comprising N interconnected nodes represented by a weighted, undirected adjacency matrix  $A_{ii}$ . The system dynamics are governed by

$$\frac{dx_i}{dt} = M_0(x_i) + \sum_{i=1}^{N} A_{ij} M_1(x_i) M_2(x_j), \tag{1}$$

where  $x_i(t)$  is the vector of the dynamical variables of node i,  $M_0(x_i(t))$  characterizes the self-evolution of node i, the product of the functions  $M_1(x_i(t))$  and  $M_2(x_j(t))$  describes the pairwise interaction between nodes i and j, so the coupling is separable. Although modeling the coupling relationship between nodes as the product of two functions—each dependent on the state of an individual node—is a specific choice. However, this assumption is applicable to a variety of real-world networks and dynamic processes, particularly in complex networks where dynamics can naturally be described as state-dependent interactions. For example:

- (1) Epidemic Dynamics (denoted as E) on Social Networks:<sup>6-9</sup> The transmission rate of a disease can often be modeled as the product of an infected individual's infectivity and a susceptible individual's susceptibility, as seen in classical models like SIS (Susceptible-Infected-Susceptible).
- (2) Population Dynamics (denoted as **P**) in Ecological Networks:<sup>37</sup> The interaction strength between species often depends on their population sizes and follows a multiplicative relationship.
- (3) Regulatory Dynamics (denoted as R) in Protein–Protein Interaction Networks: <sup>17,19,20,22,23</sup> In gene regulatory networks or protein interaction networks, the influence of one node (gene or protein) on another is typically state-dependent. For instance, transcription factors regulate gene expression based on the state of the nodes involved.

Consider the situation where the system has attained a steady state when a disturbance occurs, with nodal values stabilized at  $x_i$ . Introducing a small temporary perturbation  $\delta x_m$  at node m, we reset the time to 0. The initial conditions for the nodes in the network are, thus, given by

$$x_i(0) = \begin{cases} x_i, & i \neq m, \\ x_m + \delta x_m, & i = m. \end{cases}$$
 (2)

## A. Steady-state solution

The steady state of the networked dynamical system equation (1) can be obtained by solving

$$\frac{dx_i}{dt} = M_0(x_i) + \sum_{j=1}^{N} A_{ij} M_1(x_i) M_2(x_j) = 0.$$
 (3)

The average of  $M_2(x_i)$  among neighboring nodes is

$$\langle M_2(x) \rangle_i = \frac{1}{k_i} \sum_{i=1}^N A_{ij} M_2(x_j),$$
 (4)

where  $k_i$  denotes the degree of node *i*. Substituting Eq. (4) into Eq. (3), we have

$$M_0(x_i) + k_i M_1(x_i) \langle M_2(x) \rangle_i = 0.$$
 (5)

Letting

$$R(x_i) \equiv -\frac{M_1(x_i)}{M_0(x_i)},\tag{6}$$

we have, from Eq. (5),

$$R(x_i) = \frac{1}{k_i \langle M_2(x) \rangle_i}. (7)$$

For  $\lambda_i \equiv R(x_i)$ , we have  $\lambda_i \sim k_i^{-1}$ , so the steady state  $x_i$  can be obtained through the inverse of  $R(x_i)$ ,

$$x_i = R^{-1}(\lambda_i). (8)$$

# B. Definition of nodal resilience and influence

After a temporary perturbation, the system undergoes a transient phase before returning to its steady state. The instantaneous

response of node m is

$$\Delta x_m(t) = x_m(t) - x_m, \tag{9}$$

where  $\Delta x_m(t)$  measures the deviation of node m from its steady state. To quantify the resilience of node m, we use the concept of resilience triangle.<sup>38</sup> The loss of resilience of node m is defined as

$$LR_m = \int_0^\infty \frac{\Delta x_m(t)}{\delta x_m} dt,$$
 (10)

where the integrand  $\Delta x_m(t)/\delta x_m$  is positive, <sup>38</sup> so  $LR_m > 0$ . A larger value of  $LR_m$  means that it takes longer for the node to recover to its steady state, signifying a weaker resilience. The resilience  $R_m$  of node m can then be defined as the reciprocal of  $LR_m$ , i.e.,

$$R_m = \frac{1}{LR_m}. (11)$$

A smaller resilience loss  $LR_m$  corresponds to a higher resilience  $R_m$ , meaning that the node is more capable of returning to its steady state.

The influence  $I_m$  of node m on the rest of the network is defined as the cumulative resilience losses of other nodes in the system

$$I_m = \sum_{i=1, i \neq m}^{N} LR_i = \sum_{i=1, i \neq m}^{N} \int_0^{\infty} \frac{\Delta x_i(t)}{\delta x_m} dt,$$
 (12)

where larger resilience losses of the other nodes indicate that the influence of node m is greater.

#### **III. SCALING OF NODAL RESILIENCE**

A perturbation  $\delta x_m$  applied to the steady state of node m gives the initial condition:  $x_m(0) = x_m + \delta x_m$ , e.g.,  $\delta x_m = \alpha x_m$  with  $\alpha = 0.1$ . The dynamical evolution of m is governed by

$$\frac{d(x_m + \Delta x_m(t))}{dt} = M_0(x_m + \Delta x_m(t)) + \sum_{j=1}^N A_{mj} M_1(x_m + \Delta x_m(t)) M_2(x_j + \Delta x_j(t)),$$
(13)

where  $\Delta x_m(t)$  is the instantaneous response of node m, as defined in Eq. (9). Linearizing the dynamics about the steady state, we obtain

$$\frac{d\Delta x_m(t)}{dt} = \left(M_0'(x_m) + M_1'(x_m) \sum_{j=1}^N A_{mj} M_2(x_j)\right) \Delta x_m(t)$$

+ 
$$M_1(x_m) \sum_{i=1}^{N} A_{mj} M'_2(x_j) \Delta x_j(t) + o(\Delta x^2).$$
 (14)

For  $\Delta x_j(t) \ll \Delta x_m(t)$ , Eq. (14) can be simplified as

$$\frac{d\Delta x_m(t)}{dt} = P_m \Delta x_m(t),\tag{15}$$

where

$$P_m = M'_0(x_m) + M'_1(x_m) \sum_{j=1}^N A_{mj} M_2(x_j).$$

Solving Eq. (15), we get

$$\Delta x_m(t) = \delta x_m e^{P_m t}. (16)$$

Consequently, the resilience of node m can be obtained as

$$R_m = \frac{1}{\int_0^\infty \frac{\Delta x_m(t)}{\delta x_m} dt} = -P_m. \tag{17}$$

To ensure the mathematical consistency of the framework with its stability requirements, we impose specific conditions on the terms that contribute to  $P_m$ . The sign of  $P_m$  depends on the forms of  $M_0'(x_m)$ ,  $M_1'(x_m)$ , and  $M_2(x_j)$ . Therefore, to guarantee stability and prevent potential instabilities in the system, we assume  $M_0'(x_m) \leq 0$  and  $M_1'(x_m) \leq 0$  to ensure the system stabilizes after perturbations, while requiring  $M_2(x_j) \geq 0$  to prevent destabilizing feedback interactions. Additionally, we assume that  $P_m$  remains negative to ensure  $\Delta x_m(\infty) \to 0$ , which holds when the combined effects of  $M_0'(x_m)$ ,  $M_1'(x_m)$ , and  $M_2(x_j)$  do not induce positive feedback loops capable of causing divergence.

Employing Eq. (6), we have

$$M_0'(x_m) = -\frac{M_1'(x_m)}{R(x_m)} + \frac{M_1(x_m)R'(x_m)}{R^2(x_m)}.$$
 (18)

Utilizing Eqs. (4) and (7), we get

$$M'_1(x_m) \sum_{i=1}^N A_{mi} M_2(x_i) = \frac{M'_1(x_m)}{R(x_m)}.$$
 (19)

Combining these expressions, we finally obtain

$$P_{m} = \frac{M_{1}(x_{m})R'(x_{m})}{\lambda_{m}^{2}}.$$
 (20)

Using the Hahn series,  $M_1(x_m)$  and  $R'(x_i)$  can be expressed as

$$M_1(x_m) = \sum_{n=0}^{\infty} B_n \lambda_m^{\beta_n}$$
 and  $R'(x_m) = \sum_{n=0}^{\infty} C_n \lambda_m^{\psi_n}$ . (21)

In the limit  $\lambda_m \to 0$ , the leading terms  $\lambda_m^{\beta_0}$  and  $\lambda_m^{\psi_0}$  dominate the dynamical evolution. We have

$$R_m \sim \lambda_m^{-2+\beta_0+\psi_0}. (22)$$

Utilizing  $\lambda_m \sim k_m^{-1}$ , we also have

$$R_m \sim k_m^{2-\beta_0-\psi_0},\tag{23}$$

where

$$\zeta = 2 - \beta_0 - \psi_0. \tag{24}$$

It can be seen from Eqs. (23) and (24) that the nodal resilience scales with the nodal degree algebraically with the exponent  $\zeta$ .

To provide numerical support for the scaling relations (23) and (24), we implement epidemic spreading (E), population dynamics (P), and regulatory dynamics (R) on ER (Erdös–Rényi) random and scale-free networks. The details of the network structures and dynamical processes are listed in Table I. These models are grounded in well-established literature and have been adapted to satisfy the separability condition specified in Eq. (1).

TABLE I. Network dynamics.

Dynamics	Equation	Notation
Epidemic	$\frac{dx_i}{dt} = -x_i(t) + \sum_{j=1}^{N} A_{ij} (1 - x_i(t)) x_j(t)$	E
Population	$\frac{dx_i}{dt} = -x_i^{0.8}(t) + \sum_{j=1}^{N} A_{ij} x_j^{0.2}(t)$	P
Regulatory	$\frac{dx_i}{dt} = -x_i(t) + \sum_{j=1}^{N} A_{ij} \frac{x_j(t)}{1 + x_j(t)}$	R

- (1) Epidemic Spreading (E): The equation  $\frac{dx_i}{dt} = -x_i(t) + \sum_{j=1}^{N} A_{ij}(1-x_i(t))x_j(t)$  follows the classic SIS (Susceptible-Infected-Susceptible) framework.<sup>8</sup> In this equation,  $x_i(t)$  represents the infection probability of node i, while the coupling terms  $(1-x_i)x_j$  model the transmission of infection between nodes. No additional parameters are introduced beyond the adjacency matrix  $A_{ij}$ , ensuring that the formulation aligns with standard models.
- 2. Population Dynamics (P): The equation  $\frac{dx_i}{dt} = -x_i^{0.8}(t) + \sum_{j=1}^N A_{ij}x_j^{0.2}(t)$  models birth-and-death processes.<sup>28,37</sup> The exponents 0.8 and 0.2 were chosen to introduce nonlinear effects while maintaining analytical tractability.
- 3. Regulatory Dynamics (R): The equation  $\frac{dx_i}{dt} = -x_i^a(t) + \sum_{j=1}^N A_{ij} \frac{x_j(t)}{1+x_j(t)}$  models regulatory interactions. The first term describes the self-dynamics of a protein, which could include processes such as degradation (where a=1), dimerization (where a=2), or more complex chain reactions (represented by fractional a). The second term represents the regulation of

protein *i* by its interacting partners and is often modeled using a Hill function of the form  $\frac{x_j(t)}{1+x_i(t)}$ .

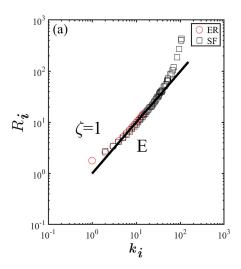
The expressions of the scaling exponents for the E, R, and P dynamics are provided in Appendix A.

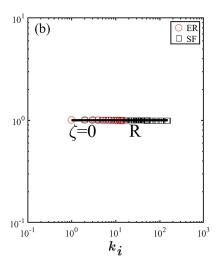
Under an impulsive perturbation, the scaling of the nodal resilience with the degree exhibits three distinct behaviors, as shown in Figs. 1(a)–1(c), respectively, for **E**, **R**, and **P** types of dynamics. For  $\zeta > 0$ , the nodal resilience increases with the degree, indicating that the hub nodes are more resilient. For  $\zeta \approx 0$ , the nodes in the network are uniformly resilient against the perturbation. For  $\zeta < 0$ , nodes of smaller degrees tend to be more resilient.

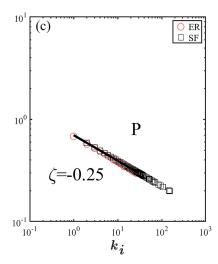
While nodal resilience  $R_m \sim k_m^n$  quantifies the recovery capabilities of individual nodes, the overall resilience of a network depends on both the properties of its nodes and its structural organization. A network made up of highly resilient nodes is not automatically robust at the global level. Network resilience is influenced by several structural factors, including topology, average path length, coupling strength, redundancy, clustering coefficient, and assortativity. For example, in a star network topology, all peripheral nodes may show high resilience, but the network as a whole remains structurally fragile. If the central hub fails, the entire network collapses. This situation illustrates that individual node resilience does not guarantee overall network robustness, emphasizing the crucial role of network structures in determining resilience beyond the characteristics of the nodes themselves.

# IV. SCALING OF NODAL INFLUENCE $I_m$

We first consider the case where the perturbation is impulsive and applies to node m at t = 0. The dynamics of the nearest neighbor







**FIG. 1.** Patterns of nodal resilience under temporary perturbation. (a)–(c)  $R_i$  vs  $k_i$  for **E**, **R**, and **P**, respectively, for all networks. The red circles represent the results from an Erdős–Rényi random network with size N=6000 and average degree  $\langle k \rangle=4$ , and the black diamonds give the results from a scale-free network of size N=6000 and average degree  $\langle k \rangle=4$ .

of node *m*, denoted as *i*, is described by the following equation:

$$\frac{d(x_i + \Delta x_i(t))}{dt} = M_0(x_i + \Delta x_i(t)) + \sum_{j=1}^N A_{ij} M_1(x_i + \Delta x_i(t)) M_2(x_j + \Delta x_j(t)), \quad (25)$$

which can be linearized about the steady state as

$$\frac{d\Delta x_i(t)}{dt} = \left(M_0'(x_i) + M_1'(x_i) \sum_{j=1}^N A_{ij} M_2(x_j)\right) \Delta x_i(t) 
+ M_1(x_i) \sum_{j=1, j \neq m}^N A_{ij} M_2'(x_j) \Delta x_j(t) 
+ M_1(x_i) M_2'(x_m) \Delta x_m(t) + o(\Delta x^2).$$
(26)

Assuming that node *j* is far from the disturbance source *m*, we have  $\Delta x_i(t) \ll \Delta x_m(t)$ . Equation (26) can then be written as

$$\frac{d\Delta x_i(t)}{dt} = P_i \Delta x_i(t) + Q_i, \tag{27}$$

where

$$P_i = M'_0(x_i) + M'_1(x_i) \sum_{j=1}^N A_{ij} M_2(x_j)$$
 and 
$$Q_i = M_1(x_i) M'_2(x_m) \Delta x_m(t).$$

Solving Eq. (27) gives

$$\Delta x_i(t) = e^{P_i t} \int_0^t Q_i e^{-P_i s} ds.$$
 (28)

Similar to  $P_m$ , we get  $P_i < 0$ . Substituting Eq. (16) into Eq. (28), we get

$$\Delta x_i(t) = \frac{M_1(x_i)M_2'(x_m)\delta x_m}{P_m - P_i} \left( e^{P_m t} - e^{P_i t} \right). \tag{29}$$

Consequently, the loss of resilience of node i can be obtained as

$$LR_i = \int_0^\infty \frac{\Delta x_i(t)}{\delta x_m} dt = \frac{M_1(x_i)M_2'(x_m)}{P_i P_m}.$$
 (30)

Similarly,  $M'_2(x_m)$  can be expressed as a Hahn series,

$$M'_{2}(x_{m}) = \sum_{n=0}^{\infty} C_{n} \lambda_{m}^{\varphi_{n}},$$
 (31)

which can be simplified as

$$M_2'(x_m) \sim k_m^{-\varphi_0}.$$
 (32)

Combining Eqs. (17), (23), and (30), we get

$$LR_i \sim k_m^{-2+\beta_0+\psi_0-\varphi_0}$$
. (33)

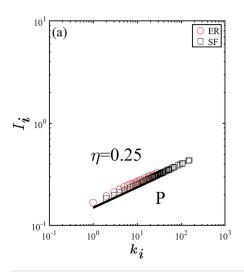
Collecting all neighboring nodes of the perturbed node m, we obtain the scaling relation

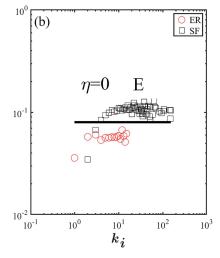
$$I_m \sim k_m^{\eta},$$
 (34)

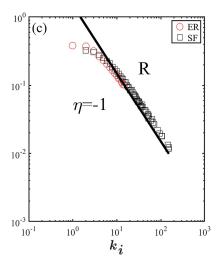
with the algebraic scaling exponent  $\eta$  given by

$$\eta = -1 + \beta_0 + \psi_0 - \varphi_0. \tag{35}$$

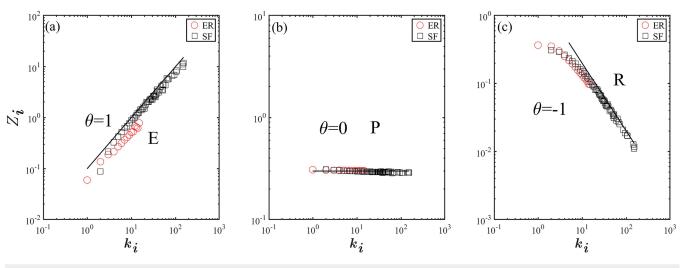
We next consider a constant perturbation:  $\Delta x_m(t) = \delta x_m$ . In this case, the nodal resilience cannot be defined, but the influence







**FIG. 2.** Patterns of nodal influence under temporary perturbation. (a)–(c)  $l_i$  vs  $k_i$  for **P**, **E**, and **R**, respectively, for all networks. The red circles represent the results from an Erdős–Rényi random network with size N=6000 and average degree  $\langle k \rangle=4$ , and the black diamonds give the results from a scale-free network of size N=6000 and average degree  $\langle k \rangle=4$ .



**FIG. 3.** Patterns of nodal influence under a constant perturbation. (a)–(c)  $Z_i$  vs  $k_i$  for **E**, **P**, and **R**, respectively, for all networks. The red circles represent the results from an Erdős–Rényi random network with size N=6000 and average degree  $\langle k \rangle=4$ , and the black diamonds give the results from a scale-free network of size N=6000 and average degree  $\langle k \rangle=4$ .

 $Z_m$  of node m can be defined as

$$Z_m = \sum_{i=1, i \neq m}^{N} \frac{\Delta x_i(t \to \infty)}{\delta x_m},$$
 (36)

where  $\Delta x_i(t \to \infty)$  is the *i*th node's response to the disturbance at the source *m*. From Eq. (27), we get  $Q_i = M_1(x_i)M'_2(x_m)\delta x_m$ . From Eq. (28), we obtain

$$\Delta x_i(t) = \frac{M_1(x_i)M_2'(x_m)\delta x_m}{P_i} \left( e^{P_i t} - 1 \right).$$
 (37)

In the limit  $t \to \infty$ , we get

$$\frac{\Delta x_i(t \to \infty)}{\delta x_m} = -\frac{M_1(x_i)M_2'(x_m)}{P_i} \sim k_m^{-\varphi_0},$$
 (38)

and the scaling relation

$$Z_m \sim k_m^{\theta},$$
 (39)

where

$$\theta = 1 - \varphi_0. \tag{40}$$

To verify the scaling relations (34) and (35) for the case of an impulsive perturbation, we use the same numerical setting as in Fig. 1. The results are shown in Fig. 2. It can be seen that the nodal influence also exhibits three distinct scaling behaviors, as shown in Figs. 2(a)–2(c) for P, E, and R types of dynamics, respectively. For the case of a constant perturbation, the simulation results to support the scaling relations as given by (39) and (40) are shown in Fig. 3. There are again three distinct patterns: (1) for E type of dynamics, we have  $\theta = 1$ , indicating that nodes with larger degrees have greater influence on other nodes, (2) for P type of dynamics, we have  $\theta \approx 0$ , so the nodal influence is independent of the degree, and (3) for R

type of dynamics, we have  $\theta=-1.$  In this case, nodes with smaller degrees have greater influence.

# V. VERIFYING RESILIENCE AND INFLUENCE SCALING WITH EMPIRICAL NETWORKS

We study the following empirical networks to test the scaling:

- (1) **PPI1**. The yeast scale-free protein–protein interaction network, consisting of 1647 nodes (proteins) and 5036 undirected links. The network describes the chemical interactions among proteins.<sup>39</sup>
- (2) **PPI2.** The human protein–protein interaction network, a scale-free network, consisting of N = 2035 nodes (protein) and  $L = 13\,806$  protein–protein interaction links.<sup>40</sup>

**TABLE II.** Structural characteristics of the empirical networks, including the number N of nodes, the average degree  $\langle k \rangle$ , assortativity coefficient r, and clustering coefficient C. The rightmost column indicates the dynamical processes implemented in the simulations.

Network	N	$\langle k \rangle$	r	С	Dynamics
PPI1	1647	3.05	-0.1059	0.1908	R
PPI2	2035	6.78	-0.2192	0.0473	R
PPI3	2938	5.25	-0.1929	0.1256	R
PPI4	2350	2.96	-0.1856	0.0853	R
URIV	1133	9.62	0.0782	0.1662	E
UCIonline	1893	14.61	-0.1880	0.1097	E
ECO1	1044	14.17	-0.1740	0.0376	P

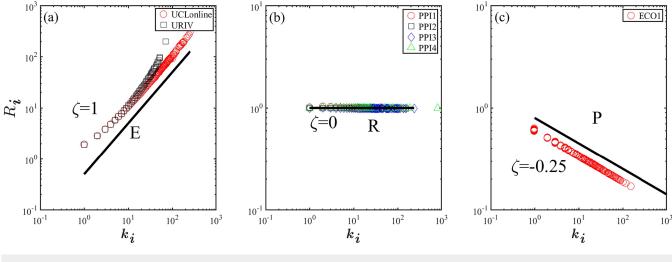


FIG. 4. Scaling of nodal resilience for empirical networks. Shown is  $R_i$  vs  $k_i$  for (a) E dynamics, (b) R dynamics, and (c) P dynamics.

- (3) PPI3. Binary protein–protein interaction network of Arabidopsis thaliana, whose giant connected component comprises 2938 nodes and 7720 links.<sup>41</sup>
- (4) PPI4. Multiplex genetic and protein interactions network of *Rattus norvegicus*, composed of 2350 nodes and 3484 links.<sup>42</sup>
- (5) URIV. The email communication network at the University Rovira i Virgili in Tarragona in the south of Catalonia in Spain, composed of 1133 nodes and 5451 links.<sup>43</sup>
- (6) UCIonline. An instant messaging network from the University of California Irvine, 4d capturing 61 040 transactions between 1893 users during a 218-day period. Connecting all individuals who exchanged messages throughout the period leads to a
- network of 1893 nodes with 27 670 links, exhibiting a fat-tailed degree distribution.
- (7) ECO1. A mutualistic ecological network constructed using data on symbiotic interactions of plants and pollinators in Carlinville, Illinois.<sup>28</sup> The resulting network is a bipartite graph linking 456 plants with 1429 pollinators. When a pair of plants is visited by the same pollinator, they mutually benefit each other indirectly by increasing the pollinator populations. Similarly, pollinators sharing the same plants possess an indirect mutualistic interaction.

Table II lists the structural characteristics of the empirical networks and the corresponding dynamical processes. Simulations

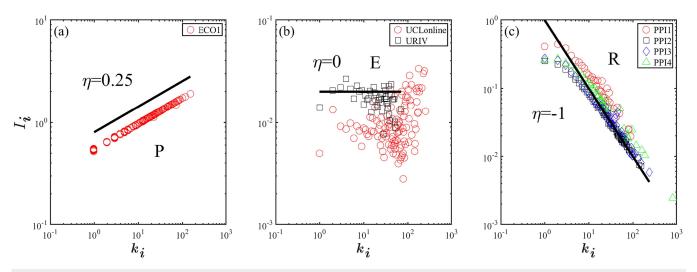


FIG. 5. Scaling of nodal influence under an impulsive perturbation. Shown is  $l_i$  vs  $k_i$  for (a) P dynamics, (b) E dynamics, and (c) R dynamics.

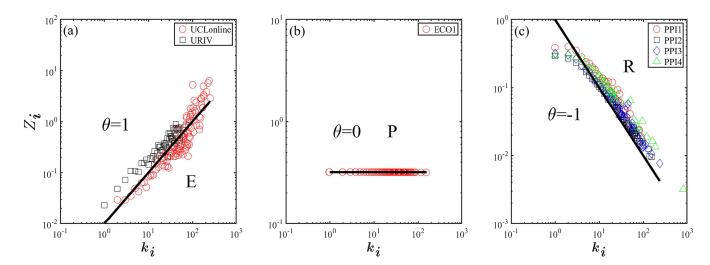


FIG. 6. Scaling of nodal influence under a constant perturbation. Shown is  $Z_i$  vs  $k_i$  for (a) E dynamics, (b) P dynamics, and (c) R dynamics.

reveal the three distinct scaling behaviors governing the nodal resilience and influence as predicted by theory, as shown in Figs. 4–6.

#### VI. DISCUSSION

We have studied the degree scaling of nodal resilience and influence for a class of complex dynamical networks that satisfy the separability condition: the coupling between a pair of nodes can be written as the product of two functions, each depending solely on the dynamical variable of the respective node. While this assumption may be strong so as to make an analytic derivation of the scaling relations possible, certain types of dynamical processes on networks do satisfy this assumption, which include epidemic spreading, population dynamics, and regulatory processes. Given a specific type of dynamical process on the network, e.g., epidemic spreading, the roles played by the individual nodes in the process, which depend on the nodal degree, are of interest, especially from the standpoint of control and mitigation. For example, if it is determined that a small set of nodes contribute disproportionally to the spreading, some optimal control strategy at the nodal level can be devised to suppress (or promote) the dynamics. Our theoretical analysis and extensive numerical computations using a large number of synthetic and empirical networks revealed that, regardless of the network structure, for a given type of dynamical process, the scaling relations of the nodal resilience and influence with the degree is algebraic, which holds with respect to variations in network properties such as the clustering coefficient, degree correlation, and degree heterogeneity, even when the perturbation is large (Appendix B).

It is worth emphasizing that our theoretical derivations of the algebraic scaling of the nodal resilience and influence rely on the network dynamics settling into a stable state in the absence of any perturbation. Whether similar scaling would arise in networks with oscillatory dynamics is an open question. Another direction to extend our study is search for nodal resilience and influence scaling in multilayer and multiplex complex networks that model real-world systems with interdependencies.

#### **ACKNOWLEDGMENTS**

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#### **AUTHOR DECLARATIONS**

#### **Conflict of Interest**

The authors have no conflicts to disclose.

#### **Author Contributions**

Li-Lei Han: Conceptualization (equal); Formal analysis (equal); Investigation (equal); Methodology (lead); Validation (equal); Writing – original draft (equal). Lang Zeng: Data curation (equal); Formal analysis (equal); Investigation (equal); Methodology (equal); Writing – original draft (equal). Hayoung Choi: Conceptualization (equal); Formal analysis (equal); Methodology (equal); Writing – original draft (equal). Ying-Cheng Lai: Conceptualization (equal); Investigation (equal); Supervision (equal); Writing – original draft (equal). Younghae Do: Conceptualization (lead); Funding acquisition (lead); Project administration (lead); Supervision (lead); Writing – original draft (lead); Writing – review & editing (equal).

# DATA AVAILABILITY

The data that support the findings of this study are available from the corresponding author upon reasonable request.

## APPENDIX A: THREE TYPES OF NETWORK DYNAMICS

#### 1. Epidemic spreading dynamics

From the epidemics model in Table I, the three functions in Eq. (1) are  $M_0(x) = -Bx$ ,  $M_1(x) = 1 - x$ , and  $M_2(x) = x$ . We have

$$R(x) = \frac{1-x}{Bx}$$
,  $R'(x) = -\frac{1}{B}x^{-2}$ , and  $R^{-1}(x) = \frac{1}{1+Bx}$ .

Their Hahn expansions are

$$M_1(R^{-1}(x)) = \frac{Bx}{1+Bx} = Bx - B^2x^2 + B^3x^3 - \cdots,$$

$$R'(R^{-1}(x)) = -\frac{1}{B} - 2x - Bx^2,$$

$$M'_2(R^{-1}(x)) = 1.$$

From Eqs. (21), (23), (31), (34), and (39), we get

$$\beta_0 = 1, \quad \psi_0 = 0, \quad \text{and} \quad \varphi_0 = 0,$$
 (A1)

$$\zeta = 1, \quad \eta = 0, \quad \text{and} \quad \theta = 1.$$
 (A2)

#### 2. Regulatory dynamics

From the regulatory dynamics model shown in Table I, we have  $M_0(x)=-Bx^a, M_1(x)=1,$  and  $M_2(x)=\frac{x^h}{1+x^h},$  and

$$R(x) = \frac{1}{Bx^a}, \ R'(x) = -\frac{a}{B}x^{-(a+1)}, \ R^{-1}(x) = B^{-\frac{1}{a}}x^{-\frac{1}{a}}.$$

Their Hahn expansions are

$$M_1(R^{-1}(x)) = 1,$$

$$R'(R^{-1}(x)) = -aB^{\frac{1}{a}}x^{\frac{a+1}{a}},$$

$$M'_2(R^{-1}(x)) = \frac{hB^{-\frac{h-1}{a}}x^{-\frac{h-1}{a}}}{\left(1 + B^{-\frac{h}{a}}x^{-\frac{h}{a}}\right)^2} = hB^{\frac{h+1}{a}}x^{\frac{h+1}{a}} + \cdots.$$

Substituting these expansions into Eqs. (21), (23), (31), (34), and (39), we get

$$\beta_0 = 0$$
,  $\psi_0 = \frac{a+1}{a}$ , and  $\varphi_0 = \frac{h+1}{a}$ , (A3)

$$\zeta = \frac{a-1}{a}$$
,  $\eta = -\frac{h}{a}$ , and  $\theta = 1 - \frac{h+1}{a}$ . (A4)

As an example, for a=1 and h=1, we have  $\zeta=0, \ \eta=-1$  and  $\theta=-1.$ 

# 3. Population dynamics

From the population dynamics model in Table I, we have  $M_0(x) = -Bx^a$ ,  $M_1(x) = 1$ , and  $M_2(x) = x^h$ , leading to

$$R(x) = \frac{1}{Bx^a}, R'(x) = -\frac{a}{B}x^{-(a+1)}, R^{-1}(x) = B^{-\frac{1}{a}}x^{-\frac{1}{a}}.$$

Their Hahn expansions are

$$M_1(R^{-1}(x)) = 1,$$

$$R'(R^{-1}(x)) = -aB^{\frac{1}{a}}x^{\frac{a+1}{a}},$$

$$M'_2(R^{-1}(x)) = h(B^{-\frac{1}{a}}x^{-\frac{1}{a}})^{h-1} = hB^{-\frac{h-1}{a}}x^{-\frac{h-1}{a}}.$$

Substituting the expansions into Eqs. (21), (23), (31), (34), and (39), we get

$$\beta_0 = 0$$
,  $\psi_0 = \frac{a+1}{a}$ , and  $\varphi_0 = -\frac{h-1}{a}$ , (A5)

$$\zeta = \frac{a-1}{a}, \quad \eta = \frac{h}{a}, \text{ and } \theta = 1 + \frac{h-1}{a}.$$
 (A6)

As an example, for a=0.8 and h=0.2, we have  $\zeta=-0.25$ ,  $\eta=0.25$ , and  $\theta=0$ .

# APPENDIX B: EFFECTS OF PERTURBATION MAGNITUDE, NETWORK CLUSTERING COEFFICIENT, DEGREE CORRELATION, AND DEGREE HETEROGENEITY ON SCALING

#### 1. Effect of perturbation magnitude

In our theoretical derivation of the nodal resilience and influence scaling, small perturbations are assumed:  $\alpha = \Delta x_m/x_m \ll 1$ , so that the approximation of linearized dynamics about the stable steady state is applicable. The simulation results in the main text are obtained with  $\alpha = 0.1$ . To find out if the three algebraic scaling relations hold for large perturbations, we set  $\alpha = 0.4$ ,  $\alpha = 0.7$ , and  $\alpha = 1$ . The simulation results are shown in Fig. 7. It can be seen that, in spite of the large perturbation, the nodal resilience and influence still follow the three classes of algebraic scaling.

#### 2. Effect of network clustering coefficient

The configuration model for complex networks stipulates that the clustering coefficient tends to zero if the network is sparse and large. However, empirical networks tend to have a nonzero clustering coefficient  $^{45}$  (e.g., 0.1). To address the effects of the clustering coefficient C on the nodal resilience and influence scaling, we generate networks with C=0.15 and calculate the scaling relations for different types of dynamical processes. The simulation results are shown in Fig. 8, revealing the emergence of the three classes of algebraic scaling.

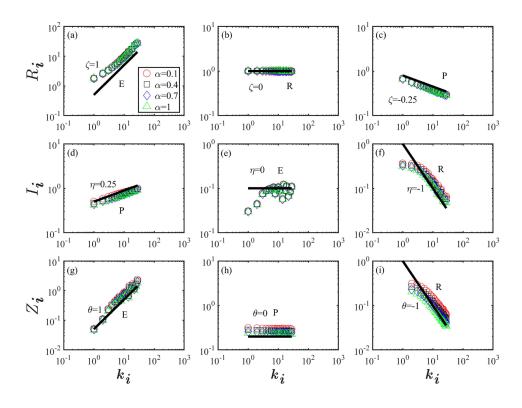
## 3. Effect of degree correlation

We generate networks with three different values of the degree–degree correlation: r = -0.2, 0, and 0.2. The resulting scaling relations are shown in Fig. 9. It can be seen that the correlation has little effect on the emergence of the three classes of algebraic scaling for the nodal resilience and influence.

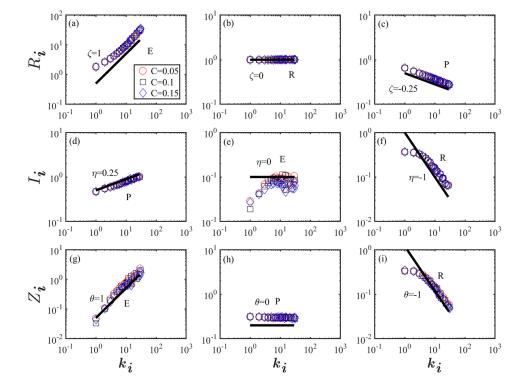
# 4. Effect of degree heterogeneity

We generate networks with three different values of the degree heterogeneity:  $^{46-49}$   $\nu=2.1$ , 3, and 4. The resulting scaling relations

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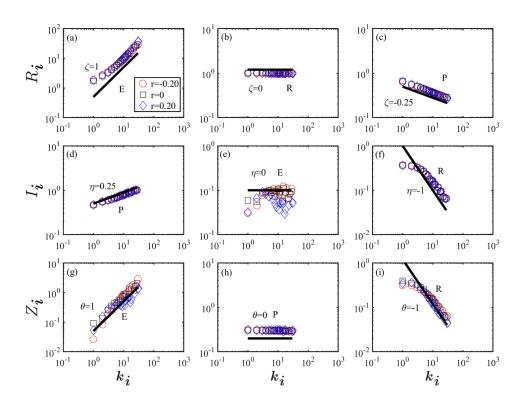


**FIG. 7.** Scaling of nodal resilience and influence with the degree under a large impulsive perturbation. The values of the relative perturbation magnitude are  $\alpha=0.4$ ,  $\alpha=0.7$ , and  $\alpha=1$ . (a)–(c)  $R_i$  vs  $k_i$  for E, R, and P types of dynamics, respectively. (d)–(f)  $I_i$  vs  $k_i$  for P, E, and R types of dynamics, respectively. (g)–(i)  $Z_i$  vs  $k_i$  for E, P, and R types of dynamics, respectively. The scaling is algebraic.

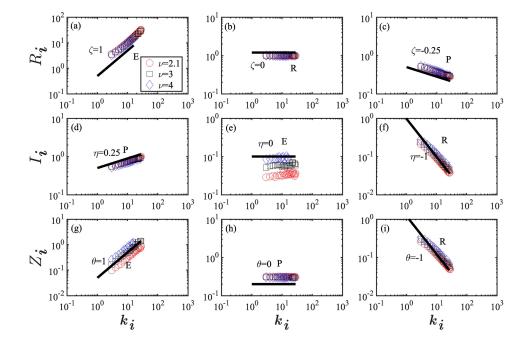


**FIG. 8.** Impact of network clustering coefficient on nodal resilience and influence scaling. The cluster coefficient is set to be C=0.15. (a)–(c)  $R_i$  vs  $k_i$  for **E**, **R**, and **P** types of dynamics. (d)–(f)  $I_i$  vs  $k_i$  for **P**, **E**, and **R** types of dynamics. (g)–(i)  $Z_i$  vs  $k_i$  for **E**, **P**, and **R** types of dynamics. The scaling remains algebraic.

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**FIG. 9.** Impact of degree–degree correlation on nodal resilience and influence scaling. Three values of the correlation are used: r = -0.2, 0, and 0.2. (a)–(c)  $R_i$  vs  $k_i$  for **E**, **R**, and **P** types of dynamics. (d)–(f)  $l_i$  vs  $k_i$  for **P**, **E**, and **R** types of dynamics. (g)–(i)  $Z_i$  vs  $k_i$  for **E**, **P**, and **R** types of dynamics. The scaling remains algebraic.



**FIG. 10.** Impact of degree heterogeneity on nodal resilience and influence scaling. Three values of the heterogeneity are used:  $\nu=2.1,3,$  and 4. (a)–(c)  $R_i$  vs  $k_i$  for **E**, **R**, and **P** types of dynamics. (d)–(f)  $I_i$  vs  $k_i$  for **P**, **E**, and **R** types of dynamics. (g)–(i)  $Z_i$  vs  $k_i$  for **E**, **P**, and **R** types of dynamics. The scaling remains to be algebraic.

are shown in Fig. 10. It can be seen that the degree heterogeneity has little effect on the algebraic scaling for the nodal resilience and influence.

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