Dynamical Response of Networks under External Perturbations: Exact Results

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We introduce and solve a general model of dynamic response under external perturbations. This model captures a wide range of systems out of equilibrium including Ising models of physical systems, social opinions, and population genetics. The distribution of states under perturbation and relaxation process reflects two regimes — one driven by the external perturbation, and one driven by internal ordering. These regimes parallel the disordered and ordered regimes of equilibrium physical systems driven by thermal perturbations but here are shown to be relevant for non-thermal and non-equilibrium external influences on complex biological and social systems. We extend our results to a wide range of network topologies by introducing an effective strength of external perturbation by analytic mean-field approximation. Simulations show this generalization is remarkably accurate for many topologies of current interest in describing real systems.

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Networks have become a standard model for a wealth of complex systems, from physics to social sciences to biology [1, 2]. A large body of work has investigated topological properties [1, 4, 5]. The raison d’être, though, of complex network studies is to understand the relationship between structure and dynamics — from disease spreading and social influence [6, 7, 8, 9, 10] to search [11]. Yet, dynamic response of networks under external perturbations has been less thoroughly investigated [3, 12]. In this paper we consider a simple dynamical process as a general framework for the dynamic response of a network to an external environment. The environment is initially treated as part of the network and then generalized as an external system.

We obtain complete and exact results for the simplest case of fully connected networks and find a nontrivial dynamic behavior that can be divided into two regimes. For large perturbations the environmental influence extends into the system with a distribution which, in the thermodynamic limit, becomes a Gaussian around a value that reflects a balance between the external perturbations driving the system in different directions. For small perturbations the distribution of states has peaks at the two ordered states. Order arises from interactions within the system, and power law tails result from the external perturbation away from these ordered states. The boundary between these regimes is characterized by a uniform distribution where all states are equally likely. The time scale of equilibration is small for large perturbations and diverges inversely as the strength of the perturbation for small perturbations. This characterizes the switching time behavior of the two ordered states. We generalize the exact results to networks of different topologies using a mean field treatment. Simulations show that this generalization, which involves renormalizing the constants in the distributions, is very accurate. Our results reveal and generalize key features of relaxation and dynamic response of models of a wide range of physical systems in the Ising universality class, electoral and contagion models of social systems, and the Wright-Fisher model of evolution in population biology.

Specifically, we consider networks with $N + N_0 + N_1$ nodes. Each node has an internal state which can take only the values 0 or 1. We let the $N_0$ nodes be frozen in state 0, and $N_1$ in state 1, and the remaining nodes change by adopting the state of a connected node. At each time step a random free node is selected; with probability $1 - p$ the node copies the state of one of its connected neighbors, and with probability $p$ the state remains unchanged. The frozen nodes can be interpreted as external perturbations to the subnetwork of free nodes. Analytically extending $N_0$ and $N_1$ to be smaller than 1 enables modeling the case of weak coupling. This model generalizes our previous efforts to derive exact results of network dynamics [13] (see also [14]).

This system is similar to the Ising model, where $N_1 + N_0$ by explicitly representing the impact of thermal perturbations play the roles of the temperature $T$, and $N_1 - N_0$ acts as an external magnetic field $h$. Our dynamics are equivalent to Glauber dynamics [15] for weak fields and high temperatures, where the Ising model parameters are $J/kT \rightarrow 1/(z + N_0 + N_1)$ and $h/J \rightarrow (N_1 - N_0)$, where $z$ is the number of nearest neighbors and $J$ the nearest-neighbor interaction strength. For low temperatures our model is an alternative dynamics that also captures the key kinetic properties of this system. Relevant network structures include crystalline 3-D lattices and random networks for amorphous spin-glasses; fully connected networks correspond to long range interactions or the mean field approximation. Despite the relevance to the extensively studied Ising model, we are not aware of any other exact solution of the response dynamics of a fully connected system or explicit representation of thermal or other perturbation for dynamic response. Specific results are available only for zero temperature dynamics in one-dimensional or mean field systems. [16, 17]
Our system can also model an election with two candidates \([18, 19]\) where some of the voters have a fixed opinion while the rest change their intention according to the opinion of others. Another application is to epidemics that spread upon contact between infected nodes (e.g., individuals or computers). Finally, the model can represent an evolving population of sexually reproducing (haploid) organisms where the internal state represents one of two alleles of a gene \([20]\). Taking \(p = 1/2\), the update of a node mimics the mating of two individuals, with one parent being replaced by the offspring, which can receive the allele of either the mother or the father with 50% probability. Since a free node can also copy the state of a frozen node, the ratios \(N_0/(N + N_0 + N_1 - 1)\) and \(N_1/(N + N_0 + N_1 - 1)\) give the mutation rates.

For a fully connected network the nodes are indistinguishable and the state of the network is fully specified by the number of nodes with internal state 1 \([13]\). Therefore, there are only \(N + 1\) global states, which we denote \(\sigma_k\), \(k = 0, 1, ..., N\). The state \(\sigma_k\) has \(k\) free nodes in state 1 and \(N - k\) free nodes in state 0. If \(P_t(m)\) is the probability of finding the network in the state \(\sigma_m\) at the time \(t\), then \(P_{t+1}(m)\) can depend only on \(P_t(m)\), \(P_t(m+1)\) and \(P_t(m-1)\). The probabilities \(P_t(m)\) define a vector of \(N + 1\) components \(P_t\). In terms of \(P_t\) the dynamics is described by the equation

\[
P_{t+1} = UP_t \equiv \left(1 - \frac{(1-p)}{N(N+N_0+N_1-1)}A\right)P_t
\]

where the evolution matrix \(U\) and also the auxiliary matrix \(A\) is tri-diagonal. The non-zero elements of \(A\) are independent of \(p\) and are given by

\[
A_{m,m} = 2m(N-m) + N_1(N-m) + N_0m
\]

\[
A_{m,m+1} = -(m+1)(N + N_0 - m - 1)
\]

\[
A_{m,m-1} = -(N-m+1)(N_1 - N-m).
\]

The transition probability from state \(\sigma_M\) to \(\sigma_L\) after a time \(t\) can be written as

\[
P(L,t;M,0) = \sum_{r=0}^{N} \frac{1}{\Gamma_r} b_{rM} a_{rL} \lambda^t_r.
\]

where \(a_{rL}\) and \(b_{rM}\) are the components of the right and left \(r\)-th eigenvectors of the evolution matrix, \(a_r\) and \(b_r\), with \(\Gamma_r = b_r + a_r\). Thus, the dynamical problem has been reduced to finding the right and left eigenvectors and the eigenvalues of \(A\).

It is easy to check by inspection of small matrices that the eigenvalues \(\mu_r\) of \(A\) are given by

\[
\mu_r = r(r - 1 + N_0 + N_1).
\]

This implies \(0 \leq p \leq \lambda_i \leq 1\), where \(\lambda_i\) are the eigenvalues of \(U\). Because of Eq. (1), the unit eigenvectors completely determine the asymptotic behavior of the system.

The eigensystem \(Aa_r = \mu_r a_r\) leads to the following recursion relation for the coefficients \(a_{rm}\)

\[
\sum_{j=m-1}^{m+1} A_{mj} a_{rj} = \mu_r a_{rm}
\]

with \(a_{r,N+1} = a_{r,-1} = 0\). To solve this equation we multiply the whole expression by \(x^m\), sum over \(m\) and define the generating function \(p_r(x) = \sum_{m=0}^{N} a_{rm} x^m\). The recursion relation then yields the following differential equation for \(p_r\)

\[
x(1-x)p''_r + [(1-N-N_0) - (1+N_1-N)x]p'_r + [N(N_1 - \mu_r/(1-x))p_r = 0.
\]

To understand the asymptotic behavior of the system \((\mu_r = 0)\) we have to consider two cases:

(a) If \(N_0 = N_1 = 0\) then \(\mu_r = 0\) leads to \(r = 0 \text{ or } r = 1\) \([13]\). In this case the differential equation simplifies to \(xp''_r + (1-N)p'_r = 0\), whose two independent solutions are \(p_0(x) = 1\) and \(p_1(x) = x^N\), corresponding to the all–nodes–0 or all–nodes–1 states respectively.

(b) If \(N_0, N_1 \neq 0\) then \(\mu_r = 0\) implies \(r = 0\). In this case equation \((3)\) is that of a hypergeometric function \(F\) and we find \(p_0(x) = F(-N,N_1,1-N-N_0,x)\), which is a finite polynomial with known coefficients \(a_{rm}\). Normalizing this eigenvector, we obtain the probability of finding the network in state \(\sigma_m\) at large times:

\[
\rho(m) = A \frac{(N_1 + m - 1)! (N + N_0 - m - 1)!}{(N-m)! m!}
\]

where \(A = (N,N_0,N_1)\) is a normalization. Because of the frozen nodes, the dynamics will never stabilize in any state, but will always move from one state to another, with mean occupation number \(m = N N_1/(N_0 + N_1)\). The surprising feature of this solution is that for \(N_0 = N_1 = 1\) we obtain \(\rho(m) = 1/(N+1)\), for all values of \(N\). Thus all macroscopic states are equally likely and the system executes a random walk through the state space.

The dynamics at long times is dominated by the second largest eigenvector, with eigenvalue \(\lambda_1\). For large networks \(\lambda_1^t \approx e^{-\tau}/\tau\) where

\[
\tau = \frac{N(N + N_0 + N_1 - 1)}{(1-p)(N_0 + N_1)}.
\]

We obtain a complete description of the dynamics by deriving all eigenvectors with \(\mu_r \neq 0\). The differential equation for \(p_r(x)\) yields

\[
p_r(x) = F(1-r-N_0,1-r-N-N_0-N_1,1-N-N_0,x) \frac{1}{(1-x)^{N_1}}.
\]

Expanding the numerator and denominator in Taylor series gives the coefficients \(a_{rm}\). Although they can easily be written down explicitly, we do not do so here. Similarly, defining the generating function \(q_r(x) = \sum_{m=1-N_1}^{N+N_0-1} b_{rm} x^m\) we obtain a differential equation for \(q_r\) whose solution is

\[
q_r(x) = x^{1-N_1} F(1-r-N_1,1-r-N-N_0-N_1,1-N-N_1,x) \frac{1}{(1-x)^{N_1+1}}.
\]

If \(N_0 = N_1 = 0\) this solution is not valid for \(r = 0\) or \(r = 1\), since the matrix \(A^T\) becomes singular. In this ...
case the two left eigenvectors are given by \( b_{0,m} = 1 \) and \( b_{1,m} = N - 2m \). For other cases the solution is obtained from the power series expansion of \( q_r(x) \). Equations (6) and (7) complete the solution of the problem.

In the thermodynamic limit \( N \to \infty \) we can define continuous variables \( x = m/N \), \( n_0 = N_0/N \) and \( n_1 = N_1/N \) and approximate the asymptotic distribution by a Gaussian \( \rho(x) = \rho_0 \exp\left[-\left(x - x_0\right)^2/2\delta^2\right] \), with \( x_0 = n_1/(n_0 + n_1) \), \( \rho_0 = 1/\sqrt{2\pi\delta^2} \) and

\[
\delta = \left[ \frac{n_0 n_1 (1 + n_0 + n_1)}{N(n_0 + n_1)^3} \right]^{1/2}. \tag{8}
\]

In the limit where \( n_0, n_1 \gg 1 \) the width depends only on the ratio \( \alpha = n_0/n_1 \) and is given by \( \sqrt{\alpha/N}/(1 + \alpha) \).

The problem we just solved can be generalized to treat an external reservoir weakly coupled to the network of \( N \) nodes. We note that the differential equations for the generating functions \( p_i(x) \) and \( q_r(x) \) remain well defined for real \( N_0 \) and \( N_1 \). The solutions for the generating functions remain the same, except that factorials must be replaced by gamma functions. Since \( N_0/(N + N_0 + N_1 - 1) \) and \( N_1/(N + N_0 + N_1 - 1) \) represent the probabilities that a free node copies one of the frozen nodes, small values of \( N_0 \) and \( N_1 \) can be interpreted as representing a weak connection between the free nodes and an external system containing the frozen nodes. The external system can be thought of as a reservoir that affects the network but is not affected by it. Alternatively, we can suppose that there is a single node fixed at 0 that is on for only a fraction \( N_0 \) of the time and off for the fraction \( 1 - N_0 \), and similarly for a single node fixed at 1.

Figure 1 shows examples of the distribution \( \rho(m) \) for a network with \( N = 100 \) and various values of \( N_0 \) and \( N_1 \). Numerical simulations displaying similar results are described in [21].

Figure 2 shows an example of the time evolution of the probability density for a fully connected network compared to numerical simulations. The evolution from the initial to the asymptotic time-independent distribution is the analog of an equilibration process promoted by the external system.

For small values of \( N_0 \) and \( N_1 \) (\( \ll 1/\ln N \)), we can obtain a simplified expression for \( \rho(m) \):

\[
\rho(m) \approx \frac{N_1 N_0}{N_0 + N_1} \left[ \frac{1 - \ln N}{m^{1-N_1}} - \frac{\ln N}{(N - m)^{1-N_0}} \right]. \tag{9}
\]

Thus \( \rho(m) \) displays a power law behavior on both ends of the curve: \( 1/m \) for \( m \) close to 0 and \( 1/(N - m) \) for \( m \) close to \( N \) (see, for instance, the curve with \( N_0 = N_1 = 0.5 \) in Fig. 1). Since the relaxation time \( \tau \) is proportional to \( 1/(N_0 + N_1) \), the equilibration process becomes very slow in this limit.

For networks with different topologies the effect of the frozen nodes is amplified. To see this we note that the probability that a free node copies a frozen node is \( P_i = (N_0 + N_1)/(N_0 + N_1 + k_i) \) where \( k_i \) is the degree of the node. For fully connected networks \( k_i = N - 1 \) and \( P_i \equiv P_{FC} \). For general networks an average value \( P_{av} \) can be calculated by replacing \( k_i \) by the average degree \( k_{av} \).

We can then define effective numbers of frozen nodes, \( N_{0ef} \) and \( N_{1ef} \), as being the values of \( N_0 \) and \( N_1 \) in \( P_{FC} \) for which \( P_{av} \equiv P_{FC} \). This leads to

\[
N_{0ef} = f N_0, \quad N_{1ef} = f N_1 \tag{10}
\]

where \( f = (N - 1)/k_{av} \). Corrections involving higher moments can be obtained by integrating \( P_i \) with the degree distribution and expanding around \( k_{av} \).

Figure 3 shows examples of the equilibrium distribution for four different networks with \( N = 100 \) and \( N_0 = N_1 = 5 \). Panel (a) shows a random network with connection probability 0.3 \( (N_{av} = 30, f = 3.3) \). The theoretical result was obtained with Eq. (6) with \( N_{0ef} = N_{1ef} = 17 \). For a scale-free network (panel (b))
grown from an initial cluster of 6 nodes adding nodes with 3 connections each following the preferential attachment rule \[ f = 99/6 \] and the effective values of \( N_0 \) and \( N_1 \) are approximately 82. Panel (c) shows the probability distribution for a 2-D regular lattice with 10 \( \times \) 10 nodes for which \( f = 99/36 \approx 28 \). Finally, panel (d) shows a small world version of the regular lattice \[ \frac{1}{3} \] where 30 connections were randomly re-connected, creating short-cuts between otherwise distant nodes. These results show that the mean field generalization is accurate for many network topologies. Still, extreme cases such as a star network should be different and this is confirmed by simulations and preliminary analytic results. The relaxation time \( \frac{\tau}{N} = \frac{1}{k_{av} + N_0 + N_1}/(N_0 + N_1) \). It increases linearly with \( N \) for fully connected or random networks, but is independent of \( N \) for regular and scale-free topologies.

Our results have important implications for real systems. In the social sciences they show the importance of opinion makers in stabilizing the outcome of elections: weak external influences result in an arbitrary but seemingly strong opinion that can switch at random (see also \[ \frac{22}{[22]} \]), due to the arbitrary choice of the ordered state in the weak perturbation regime. Thus, an elected candidate winning a landslide election may have no solid support. The slow dynamics can play a crucial role, since the time to switching might occur only after the election day, especially if the number of voters is large. Stronger external influences, counter intuitively, reduce the relation time giving rise to improved internal equilibration.

In theoretical biology our results are equivalent to the exact dynamical solution of the Wright-Fisher model \[ \frac{20}{[20]} \] for arbitrary population sizes and mutation rates. Our equations give not only the asymptotic equilibrium distribution of alleles (see \[ \frac{20}{[20]} \] for approximate expressions), but also its time evolution in two regimes, one where mutations have difficulty overcoming an existing dominant allele (the low perturbation regime) and one where random mutations dominate (the high perturbation regime). Again this is crucial information, since the equilibration time can be extremely long for the typically small mutation rates observed in nature.

Finally, we emphasize that exact dynamical solutions, describing systems out of equilibrium, are rare and important for the study of many statistical properties that can be described in future publications.

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\[ \frac{1}{[1]} \] R. Albert and A.-L. Barabási, Rev. Mod. Phys. \textbf{74}, 47 (2002).
\[ \frac{2}{[2]} \] S. Boccaletti, V. Latora, Y. Moreno, M. Chavez and D.-U. Hwang, Phys. Rep. \textbf{424}, 175 (2006).
\[ \frac{3}{[3]} \] Y. Bar-Yam and I. Epstein, PNAS \textbf{101}, 4341 (2004).
\[ \frac{4}{[4]} \] R. Albert, H. Jeong, and A.-L. Barabási, Nature London \textbf{406}, 378 (2000).
\[ \frac{5}{[5]} \] R. Cohen, K. Erez, D. ben-Avraham, and S. Havlin, Phys. Rev. Lett. \textbf{85}, 4626 (2000).
\[ \frac{6}{[6]} \] R. Pastor-Satorras and A. Vespignani, Phys. Rev. Lett. \textbf{86}, 3200 (2001).
\[ \frac{7}{[7]} \] M. Barahona and L.M. Pecora, Phys. Rev. Lett. \textbf{89}, 054101 (2002).
\[ \frac{8}{[8]} \] T. Nishikawa, A.E. Motter, Y.-C. Lai, and F.C. Hoppensteadt, Phys. Rev. Lett. \textbf{91}, 014101 (2003).
\[ \frac{9}{[9]} \] Y. Moreno, M. Nekovee and A.F. Pacheco, Phys. Rev. E \textbf{69}, 066130 (2004).
\[ \frac{10}{[10]} \] M.F. Laguna, A. Guillermo, and H. Zanette Damin, Physica A \textbf{329}, 459 (2003).
\[ \frac{11}{[11]} \] R. Guimera et al., Phys. Rev. Lett. \textbf{89}, 248701 (2002).
\[ \frac{12}{[12]} \] X. Wang, Y.-C. Lai, and C. H. Lai, Phys. Rev. E \textbf{74}, 066104 (2006).
\[ \frac{13}{[13]} \] M.A.M. de Aguiar, I.R. Epstein and Y. Bar-Yam, Phys. Rev. E \textbf{72}, 067102 (2005).
\[ \frac{14}{[14]} \] H. Zhou and R. Lipowsky, J. Stat. Mech., P01009 (2007).
\[ \frac{15}{[15]} \] R.J. Glauber, J. Math. Phys. \textbf{4}, 294 (1963).
\[ \frac{16}{[16]} \] A. Prados and J. J. Brey, J. Phys. A: Math. Gen. \textbf{34}, L453 (2001).
\[ \frac{17}{[17]} \] Dj. Spasojevic, S. Janicevic and M. Knezevi, Europhys. Lett., \textbf{76} 912 (2006).
\[ \frac{18}{[18]} \] D. Vilone and C. Castellano, Phys. Rev. E \textbf{69}, 016109 (2004).
\[ \frac{19}{[19]} \] V. Sood and S. Redner, Phys. Rev. Lett. \textbf{94},178701 (2005).
\[ \frac{20}{[20]} \] W.J. Ewens, \\emph{Mathematical Population Genetics}, Springer - NY (2004).
[21] N. Boccara, eprint, [arXiv:0704.1790] [nlin.AO] 2007.

[22] M. Mobilia, A. Petersen and S Redner, J. Stat. Mech.