

# Inherently unstable networks collapse to a critical point

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Nonequilibrium systems that are driven or drive themselves towards a critical point have been studied for almost three decades. Here we present a minimalist example of such a system, motivated by experiments on collapsing active elastic networks. Our model of an unstable elastic network exhibits a collapse towards a critical point from any macroscopically connected initial configuration. Taking into account steric interactions within the network, the model qualitatively and quantitatively reproduces results of the experiments on collapsing active gels.

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## I. INTRODUCTION

Critical phenomena in equilibrium phase transitions have been studied for almost two centuries [1]. The observation of criticality usually requires fine tuning of the system parameters. In contrast, it has been suggested that systems out of equilibrium can be driven, or can even drive themselves, toward a state that exhibits features of criticality [2–4]. Here we present a model of such a system that drives itself toward a critical state. We do this in the context of elastic biopolymer networks that are unstable to internal stress generation by molecular motors. Recent experiments have shown that such active gels collapse into domains that exhibit a critical-like power-law distribution of sizes [5].

Our basic model consists of an elastic, stressed network pinned at its boundaries or constrained to a closed shape. The contractile stress in the network can either be due to internal activity or due to prestress, e.g., for zero rest length of the network elements. If the network is above the connectivity percolation threshold, the boundaries prevent the percolating cluster from collapsing to a point under the contractile stress. The rest of the nonpercolating, isolated clusters do, however, collapse to their centers of mass. If bonds in the network can break, either due to network tension or dynamic cross linking, as many biopolymer networks exhibit [6–9], then the network can become unstable. If the breaks are rare, such that the network relaxes to its mechanical equilibrium between subsequent breaks, then network collapse can be considered quasistatic. An example of such a process is illustrated in Fig. 1 and the animation in the Supplemental Material [10] (see more details below). The collapse of the network results in foci—collapsed points in space containing many network elements. The main result of this paper concerns the distribution of sizes of these foci, which we find to be power-law distributed. Thus, this system possesses features reminiscent of self-organized criticality: starting from any connected configuration the network collapses to a configuration with signatures of criticality.

Below, we present the model and its results in greater detail, followed by a comparison to the experiments of Ref. [5]. Our model is very simple and based on a very general set

of assumptions. Since it does not depend on the microscopic details, we expect it to capture coarse-grained features of other collapsing systems. As we discuss below, it is able to account for results of experiments with a cross-linked actin network, collapsing due to myosin motor activity.

## II. COLLAPSE MODEL

We consider a spring network, initialized on a triangular lattice in  $d = 2$  dimensions, with periodic boundary conditions and total mass (i.e., number of nodes) of  $L \times L = M$ . Each pair of nearest-neighbor nodes is initially connected by an elastic Hookean spring with zero-rest length. In addition, the springs are inherently unstable. This property we implement by breaking the springs one by one in a random fashion. We assume that the probability for each element to break is independent of the tension it experiences. Even a completely relaxed spring with zero length can break with the same probability as any other spring. This is a crucial assumption of our model, which makes it different from, say, the Griffiths-like crack propagation. The mass of the spring is assumed to be equally distributed between the nodes, such that the total mass of the network (i.e., the number of nodes) is conserved during the collapse process.

We assume that the breaks are rare, such that the network relaxes to its mechanical equilibrium in between of any two subsequent breaks. Thus, we quasistatically evolve the network after each break to its mechanical equilibrium, subject to the remaining constraints. An example of the process is shown in Fig. 1 and the animation in the Supplemental Material. The final state of the model consists of disjoint foci, each potentially containing many disjoint network nodes (see Fig. 1). We define a cluster to be the original, connected subnetwork from which the nodes in a focus come.

During the evolution of the network failure and collapse, the bond-breaking events in nonpercolating subnetworks have no effect on the final state. This is because every finite subnetwork is collapsed to a point after it is detached from the infinite, percolating subnetwork, due to the quasistaticity assumption. Therefore, only the breaking events within the percolating

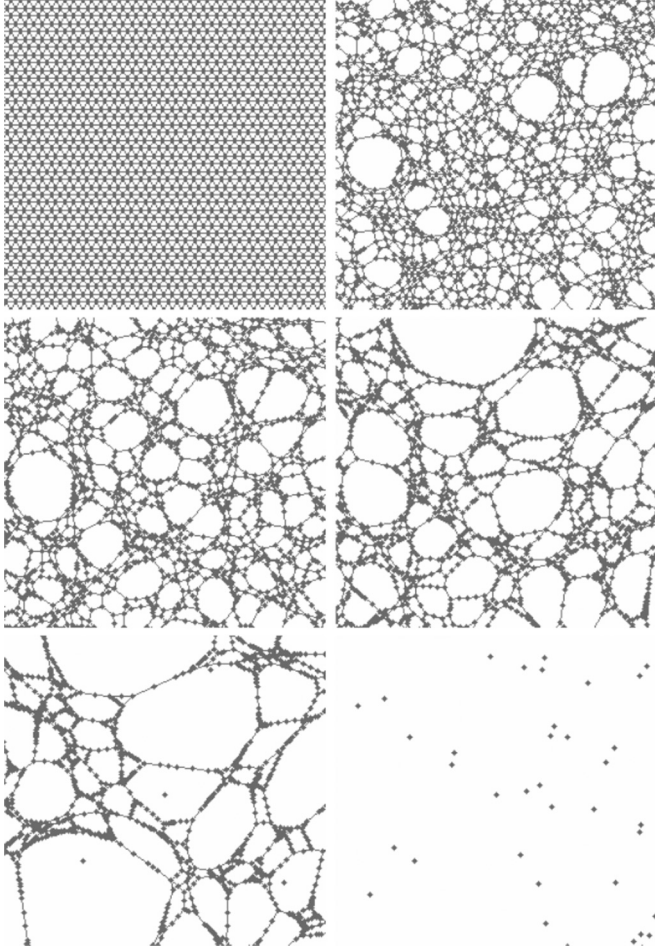


FIG. 1. An example of the collapse process of a triangular network from the initial to final, collapsed state. Only a quarter part of the  $100 \times 112$  nodes network is shown. Note, that during the process the total mass (total number of nodes) is conserved. In the final state of the network all the mass is concentrated in foci. The movie of the collapse of the full network can be seen in the Supplemental Material [10].

subnetwork are relevant for the configuration of the foci in the final state.

In addition to the elastic forces one can also take into account steric interactions within the network, which prevent geometric overlap of different filaments in the network. In two-dimensional (2D) systems these interactions trap enclavic clusters in their surroundings during the collapse (see Fig. 2 for illustration) [11]. As we show below, this qualitatively and quantitatively impacts the collapse process and its final state in the experiment. We first analyze the phantom version of our model of collapse, ignoring steric interactions.

#### A. Phantom version of the collapse model

Without steric interactions, in the final state of the two-dimensional phantom model, the mass  $s$  of foci (i.e., the number of nodes they contain) is found to be distributed in a scale-free fashion,

$$n_s \sim s^{-\tau'} \quad (1)$$

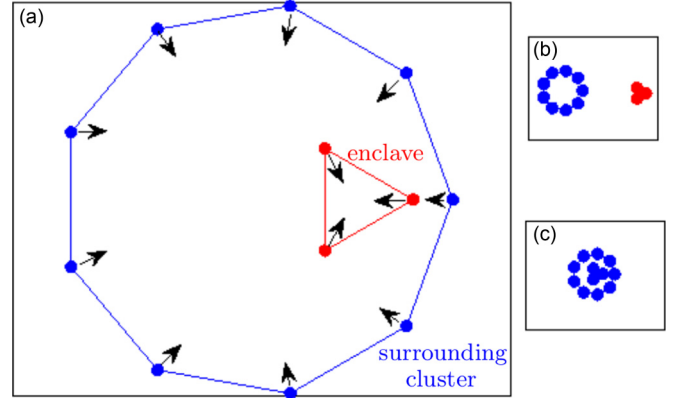


FIG. 2. (Color online) An illustration of the influence of steric interactions on the collapsed state properties. The initial state presented in (a) without steric interactions collapses to two foci with masses 3 and 9, as shown in (b). Due to steric interactions the enclave is trapped by its surroundings, leading to a collapsed state with one focus of mass 12, as shown in (c).

with a Fisher exponent,  $\tau' = 187/91 > 2$ , of the  $d = 2$ -dimensional random percolation model [12,13] [see Figs. 3(a) and 3(b)]. Here  $n_s$  is the number of foci with mass  $s$ . In general, the value of  $\tau'$  is larger than 2 for any  $d$  [14]. This result, Eq. (1), is not expected to depend on the microscopic details of the system, i.e., the lattice and the properties of the initial state of the network, provided that the initial state is macroscopically connected—is above the percolation threshold. The only important features for the critical final state are the randomness and the quasistatic nature of the breaking events. If the breaks are not random, but the probability to break a bond depends on its stress, then one loses the small foci, because the small, isolated subnetworks are not expected to be very stressed and break to even smaller subnetworks. In this case the network's collapse becomes more Griffiths-like [15] with one or few macroscopic cracks. In contrast, if the collapse is not quasistatic, one loses the large foci, because in this case before an isolated, large, massive subnetwork collapses to a focus there is a chance that it will be broken to smaller subnetworks and collapse to a few, less massive foci. Therefore, violation of the assumption of randomness of breaks yields a lower cutoff of the  $n_s$  power-law distribution, while violation of the quasistatic assumption yields an upper cutoff of the distribution. Satisfying the assumptions, however, one gets a scale-free distribution with the lower cutoff equal to the microscopic length-scale and the upper cutoff equal to the system's size. In  $d$  dimensions the distribution of the foci masses is expected to exhibit the Fisher exponent of the  $d$ -dimensional random percolation model.

In order to analyze further the collapsed foci, we evolve them back in time to clusters, similarly to the experimental procedure in Ref. [5] [during this procedure, for instance, panels (b) and (c) evolve back to (a) in Fig. 2]. For comparison we generated the cluster structure of the random percolation model [14] at its critical point [see Fig. 4(a)]. The plot of all clusters is presented in Fig. 4(c). It is similar to the cluster structure of a random percolation model at the critical point [see Fig. 4(a)]. Similarly to the critical point of a random

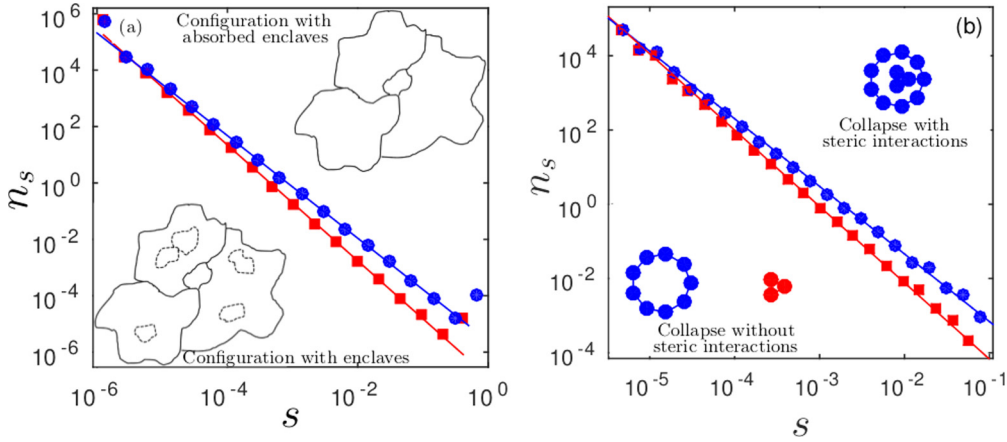


FIG. 3. (Color online) Cluster mass (in nodes) distributions. (a) Critical point of the random percolation model with (circles) and without (squares) absorption of the enclaves into their surroundings (NEP model [11]). Insets illustrate the enclaves—clusters fully surrounded by another cluster—and their absorption in the NEP model. In the bottom inset the enclaves are marked by dashed boundaries and nonenclaves by solid boundaries. In the top inset the result of absorption of enclaves from the configuration of the bottom inset is shown. (b) Collapsed state of the collapse model without (squares) and with (circles) steric interactions. The fits to squares in both panels are with the power law of the random percolation model  $\tau = 187/91 > 2$ , while for the circles the fit is with the NEP model's Fisher exponent from Eq. (3). Insets demonstrate the effective absorption of enclaves during the collapse with steric interactions. The result of collapse of the configuration from Fig. 2(a) is shown for the case of collapse without (bottom inset) and with (top inset) steric interactions.

percolation model, the structure of the clusters, possessing scale-free enclaves, is fractal with a fractal dimension of the percolation model,  $d_f' = d/(\tau' - 1) = 91/48 < 2$ . We conclude that the phantom model of the network collapse

drives itself to the critical point of the random percolation model. This is an important result of the paper.

We turn now to consider effects of steric interactions in two dimensions. We show how they change the final state of the collapse from the critical point of the random percolation model to one consistent with both the experiments in Ref. [5] and the no-enclaves percolation (NEP) model of Ref. [11].

### B. Steric interactions

There is an easy way to implement the effects of steric interactions in the two-dimensional version of our collapse model. These interactions prevent distinct but overlapping clusters from collapsing separately, as illustrated in Fig. 2(b). This has the effect of suppressing enclaves—clusters fully surrounded by another cluster. Instead of taking steric interactions into account explicitly during the evolution of our simulation, we identify all enclaves after the collapse. We then absorb these enclaves into their surrounding clusters. Namely, we add the masses of the enclaves to the mass of their surrounding cluster and define a cluster such that it contains all the enclaves and their surrounding cluster. In principle, it would be possible to simulate the full dynamics of the collapse process, taking into account (steric) constraints to suppress configurations with overlapping network elements in two dimensions. This would, however, result in the same configuration in the quasistatic limit we use here, since our network elements are assumed to have zero rest length.

We implement the first, much simpler approach and absorb all enclaves into their surrounding clusters after the collapse. Thus, taking steric interactions into account, the configuration of the clusters has no enclaves, as shown in Fig. 4(d). The configuration is at the critical point of the NEP model [11], shown in Fig. 4(b). The properties of the NEP and a random percolation models' critical points are qualitatively different. In a random percolation model, at the percolation transition,

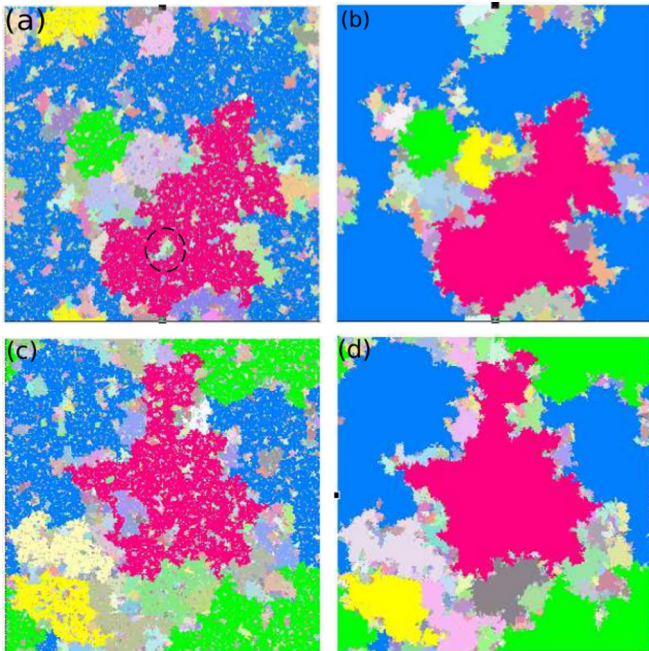


FIG. 4. (Color online) (a), (b) Clusters' structure at the critical point of the random percolation model without (a) and with (b) absorbed enclaves—NEP model. Dashed circle in (a) indicates an enclave, absorbed in (b). (c), (d) Modeling of the motor-driven collapse with and without steric interactions. Configuration of clusters modeled without (c) and with (d) steric interactions. Each cluster is indicated by a different gray level (color in the online version) on each plot.



clusters possess many scale-free enclaves. The presence of enclaves makes the clusters noncompact. In fact, the clusters are so porous, that their mass scales sublinearly with their area. Such structures are random fractals, with the fractal dimension  $d'_f = 91/48 < 2$ . Since the density of the largest cluster vanishes in the thermodynamic limit, the percolation transition is continuous [14]. However, the properties of the NEP model are different [11]. The configuration of the clusters possesses no enclaves, as shown in Fig. 4(b). The absence of enclaves indicates nonfractal, compact, Euclidean clusters. Moreover, the cluster mass distribution in the NEP model is given by

$$n_s \sim s^{-\tau}, \quad (2)$$

with a Fisher exponent,  $\tau < 2$  [see Fig. 3(a)]. This is inconsistent with a random percolation model, where the order parameter is continuous at the percolation transition, implying  $\tau' = 187/91 > 2$  [14]. In fact, the Fisher exponent and fractal dimension are related via a hyperscaling relation  $d/d'_f = \tau' - 1$  for a random percolation model [14]. Therefore, it is expected that violation of  $d'_f < 2$  [as shown for the NEP model in Fig. 4(b)] also implies violation of  $\tau > 2$  [as shown for the NEP model in Fig. 3(a)].

We find that the critical point signatures of the NEP model naturally emerge from our collapse model, taking into account steric interactions within the elastic network during the collapse process. Specifically, the configuration of the clusters possesses no enclaves and the Fisher exponent is given by the one of the NEP model [see Fig. 3(b)],

$$\tau = 1.82 \pm 0.01 < 2. \quad (3)$$

Thus, we conclude that our model for the collapse with steric interactions is driven to the critical point of the NEP model. This is analogous to how the phantom model of collapse, ignoring steric interactions, is driven to the critical point of a random percolation model. We turn now to discuss how the model presented here, with steric interactions, captures the experimental findings of Ref. [5].

### III. EXPERIMENTAL RESULTS

Our model of inherently unstable networks can be directly applied in the context of internally driven active gels. Motivated by living matter, such networks have been intensively studied during the last decade [16,17]. One example of such an active network is the actin cortex of living cells, which is composed of cytoskeletal actin filaments, cross links, and myosin motors. Taken together, these cellular proteins form an elastic network with contractility induced by nonequilibrium motor stresses [16,18]. The motors generate forces that propagate through the network, changing the viscoelastic properties, structure, and fluctuations in living systems [16,17,19–30].

Recent experiments on reconstituted acto-myosin networks *in vitro* have demonstrated restructuring of the network by the motors. Motor activity can lead to network failure and its subsequent collapse to isolated foci [7,31,32] and, as reported in Refs. [5,11], can robustly lead to scale-free structures.

In order to apply our model of collapse to such active networks we need to make the following assumptions. Within our coarse-grained approach we assume that the actin filaments

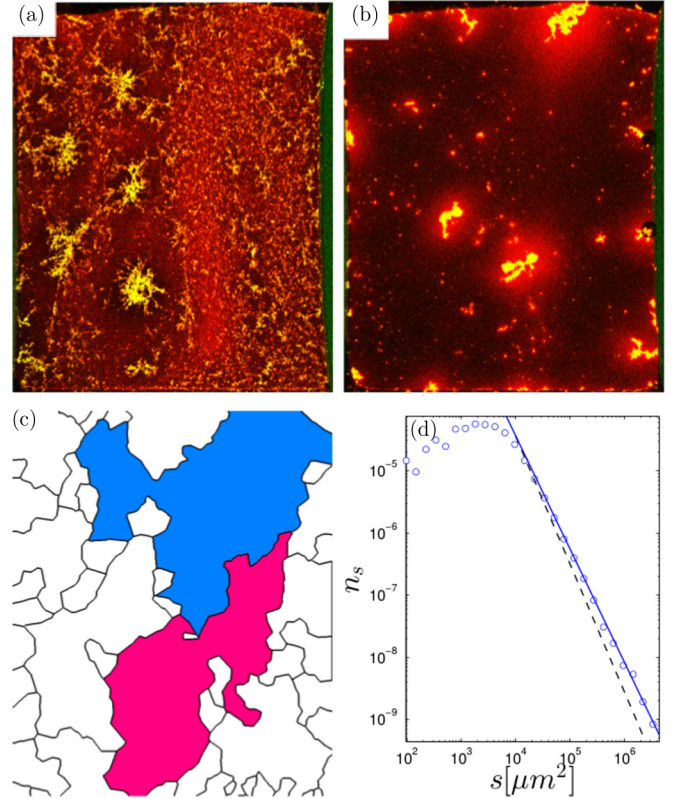


FIG. 5. (Color online) Experimental results of a fascin-crosslinked actin network, collapsed by myosin motors [11]. The concentrations of crosslinks, actin and molecular motors are given by 0.24, 12, and 0.12  $\mu\text{M}$ , respectively. The dimensions are 3.18 mm  $\times$  2 mm, while the sample thickness is 80  $\mu\text{m}$ . (a) The network after 2 min. (b) The network after 104 min. (c) Configuration of the clusters. Colors indicate the largest (blue) and the second-largest (pink) clusters. (d) Histogram (circles) of cluster areas, averaged over 26 samples. For the critically connected regime, the data is statistically more consistent (1.4 standard errors from the Hill estimator of  $\tau = 1.91 \pm 0.06$  [36]) with a power-law distribution with a NEP model's (or the collapse model with steric interactions in two dimensions) Fisher exponent from Eq. (3) (solid line). The agreement of the data with the random percolation model Fisher exponent  $\tau = 187/91$ , indicated by the dashed line, is significantly worse (2.4 standard errors from the Hill estimator).

can be modeled as springs, while the nodes of the network represent the cross-link proteins.

The contractile forces in the network are applied by the myosin motors [20,22,33–35]. This is implemented in the model by setting the rest length of each spring to zero. Thus, the initial state, when the springs' lengths are equal to the lattice constant, is under stress, such that an instability of the network elements leads to network collapse.

The inherent instability of the network elements can follow from several reasons. First, the cross links of the network possess a finite residence time [8,9]. When they unbind, the previously cross-linked actin filaments irreversibly detach from each other, due to contractile forces. Second, motors reduce local connectivity by severing the actin filaments [7] or inducing unbinding of a cross link [6]. This inherent instability of network elements is the key feature, which makes the

network unstable. Within our simple model we implement this reduction of local connectivity by breaking the springs one by one in a random fashion.

In the experiments in Refs. [5,11], motor-driven collapse of a model cytoskeletal system, composed of actin filaments, fascin cross links, and myosin motors was studied in chambers with dimensions  $3.18\text{ mm} \times 2\text{ mm}$ . The sample thickness is  $80\text{ }\mu\text{m}$  (not much larger than a typical length of an actin filament,  $20\text{ }\mu\text{m}$ ), such that the chambers can be considered as quasi-2D. In these experiments, the motors collapse the network to multiple small foci [see Figs. 5(a) and 5(b)]. Surprisingly, scale-free foci mass distribution is obtained over a wide range of experimental parameters and is manifested in a power-law distribution of the foci masses, as shown in Fig. 5(d).

The cluster configuration observed in the experiment shows no enclaves, as illustrated in Fig. 5(c). The absence of enclaves in the experiment suggests nonfractal clusters. Moreover, the Fisher exponent,  $\tau$ , of the cluster mass distribution in Eq. (2) in the experiment is consistent with the one of the NEP model [Eq. (3)]. The robustness of the criticality and its properties is captured by our model of collapse with steric interactions, in which the network collapses to the critical configuration of the NEP model from any initial connected configuration.

#### IV. SUMMARY

Here, we present a minimal model for collapse of inherently unstable networks. The model drives itself to a special, critical state. Ignoring steric interactions of the network elements,

this state corresponds to the critical point of a random percolation model. The same model with steric interactions in two dimensions leads to a collapse to the state, which corresponds to the critical point of a modified version of the percolation model—percolation model with enclaves absorbed in their surroundings, i.e., the NEP model [11]. The structural properties of the NEP model at the transition are in quantitative agreement with the experimentally observed cluster distribution. Thus, the present model with steric interactions accounts for the robustness and the properties of the criticality, observed in the experiment.

The most crucial assumption of the model is that there is no coupling between the stress on a network element and its failure rate. In the opposite regime of a strong coupling between the stress and the failure one gets Griffiths-like behavior with a single crack propagation in the quasistatic case. In contrast, in our case the system drives itself to many disjoint foci with scale-free mass distribution. We expect that, due to general assumptions of the model, it can be applied to other systems.

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