Mean-Field and Anomalous Behavior on a Small-World Network

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> We consider various equilibrium statistical mechanics models with combined short- and long-range interactions and identify the crossover to mean-field behavior, finding anomalous scaling in the width of the mean-field region, as well as in the mean-field amplitudes. We then show that this model enables us, in many cases, to determine the universal critical properties of systems on a small-world network. Finally, we consider nonequilibrium processes.

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of path lengths on a small-world network [10].

ing to compare this model to a related mean-field solution

In some cases, the long-range model may also be more appropriate than the usual small-world model. In the spread of a disease, for example, people tend to spread the disease to those geographically nearby (the regular lattice). There is a chance of a long-range spread of the

The appropriate description for many complex realworld systems is as a network [1], a general connection of nodes and vertices which need not have the structure of a regular lattice. The small-world network model combines both long-range and short-range aspects, and interpolates between regular lattices and random graphs. This model [2], in which a regular lattice is modified by either randomly "re-wiring" links or else by simply randomly adding long-range links, has become a standard model of real-world networks. It incorporates some notion of locality, as most of the links remain the same as that of the original underlying lattice. Yet it also includes the ''smallworld effect'' that the average path length between sites on the network scales only as the logarithm of the network size.

Studies of equilibrium statistical on small-world systems have shown rich behavior [3–6], with mean-field critical exponents, contrasting with the behavior on scalefree networks [7,8]. We seek to explain the mean-field behavior in the small-world systems. However, the presence of quenched randomness makes the small-world model difficult to treat analytically. We thus consider a different model which is easier to handle, lacking quenched randomness. In many cases, depending on inequality (11) below, this model gives the same universal behavior as the small-world model, thus explaining the critical phenomena in the small-world system. We show the existence of mean-field behavior, albeit with anomalous exponents describing the width of the critical region and various mean-field amplitudes. These anomalous exponents can complicate the interpretation of numerical data. Finally, we will consider a nonequilibrium case, describing the relaxation to a stationary state via a branching process.

To define our model, we again start with a regular lattice of *V* sites in *d* dimensions. Rather than adding long-range links with probability *p*, we give each site of the lattice a weak coupling, of order p/V , to every other site in the lattice [9]. We will refer to this as the longrange model. We will find that many results can be obtained on this system by combining mean-field with standard renormalization group techniques. It is interest-

disease. However, this is not necessarily due to fixed longrange links. Rather, it is due to the random probability that a given person travels a long distance, typically by air. Thus, a slight probability of long-range contact between any two people may be a better description than a set of fixed long-range links.

*Equilibrium statistics.—*We consider equilibrium statistical mechanics models with uniform, ferromagnetic couplings, such as Ising models, *XY* models, etc. ... These models can be represented by introducing a field $\vec{\phi}(x)$, where *x* labels lattice sites and where ϕ has $n = 1, 2, \ldots$ components, with a partition function

$$
Z = \sum_{\{\vec{\phi}\}} \exp[-S[\vec{\phi}]],\tag{1}
$$

where $S = E/kT$ is a statistical weight for a configuration of energy *E* at temperature *T*.

For a model on a regular *d*-dimensional lattice, $S[\vec{\phi}]$ = $S_{\text{local}}[\vec{\phi}]$, where $S_{\text{local}}[\vec{\phi}]$ includes only short-range interactions. We refer to this as the local system. We choose instead for the long-range model a statistical weight with additional long-range couplings of strength p/V :

$$
S[\vec{\phi}] = S_{\text{local}}[\vec{\phi}] - \frac{p}{2V} \sum_{x_1, x_2} \vec{\phi}(x_1) \cdot \vec{\phi}(x_2). \tag{2}
$$

We neglect the temperature dependence of the second term on the right-hand side of Eq. (2) in what follows, as it leads to corrections to physical quantities which are higher order in *p*.

Now, decouple the long-range interaction to find

$$
Z = \int_{R^n} d^n \vec{h} \exp\left[-\frac{Vh^2}{2p}\right] Z(\vec{h}),\tag{3}
$$

where \vec{h} has *n* components and where

$$
Z(\vec{h}) = \sum_{\{\vec{\phi}\}} \exp\biggl[-S_{\text{local}}[\vec{\phi}] + \vec{h} \cdot \sum_{x} \vec{\phi}(x)\biggr]. \tag{4}
$$

Here, $Z(\vec{h})$ is equal to the partition function of the local system in the presence of a magnetic field, *h*. Defining $h = |\vec{h}|$, then $Z(\vec{h}) = Z(h)$, as the value of the partition function does not depend on the direction of \vec{h} .

For *p* large, the long-range interaction outweighs the short-range interaction, and the system can be approximately solved by mean-field theory. We will instead consider precisely the opposite case: when *p* is small. This is the only case in which we expect the dependence of physical quantities, such as the specific heat, on *p* to be universal. Then, the critical point of the long-range system, \ddot{T}_c , is close to the critical point of the local system, T_c . Thus, we can use scaling laws for the local system: the magnetization, *m*, obeys $m(T = T_c, h) = A_m h^{1/\delta}$, defining the critical exponent δ . For $T>T_c$, the susceptibility χ obeys $\chi(T, h = 0) = A_{\chi}^+ |T - T_c|^{-\gamma}$. In general, we can write a scaling function: $m =$ $h^{1/\delta} f((T - T_c)h^{-1/(\delta \beta)})$. For $h \gg (T - T_c)^{\delta \beta}$, we use the first result $m(T = T_c, h) = A_m h^{1/\delta}$, while for $h \ll$ $(T - T_c)^{\delta \beta}$, we use the second $m(T = T_c, h) =$ $A_X^+ h |T - T_c|^{-\gamma} - B_X h^3 |T - T_c|^{-\gamma - 2\delta\beta}$, where we have added the h^3 term in the expansion of *m*.

The magnetization is defined by $\partial \ln Z(h)/\partial h = mV$. Thus,

$$
Z(h) = Z(0) \exp\left[\int_0^h dh'm(h')V\right].
$$
 (5)

We first consider the case of *T* near T_c so that $h \gg$ $(T - T_c)^{\delta \beta}$. Then, the dominant contribution to Eq. (5) arises from $h' \gg (T - T_c)^{\delta \beta}$ and thus Eq. (3) gives

$$
Z = Z(0) \int_{R^n} d^n \vec{h} \exp \left[-\frac{V h^2}{2p} + A_m V h^{1+1/\delta} / (1 + 1/\delta) \right].
$$
\n(6)

Since *V* is taken large, we can use a saddle point to arrive at $h = (pA_m)^{\delta/(\delta-1)}$, or

$$
m = A_m (pA_m)^{1/(\delta - 1)}.
$$
 (7)

The correlation length ξ of the local system in the presence of this field is proportional to $m^{-\nu/\beta}$, and hence diverges as $p \rightarrow 0$. The meaning of the correlation length is *not* that spins beyond this length are uncorrelated. Rather, it is that beyond this length the correlations in the long-range system are controlled by the average field *m*, while below this length, the short-range system controls the correlations. Thus, the correlation function $\langle \vec{\phi}(0) \cdot \vec{\phi}(x) \rangle$ decays as a power law up to the correlation length, and then asymptotes to a constant.

We have seen that at $T = T_c$, the system has a net magnetization, and thus $T_c < \tilde{T}_c$. To study the transition itself, we now consider the second case, $h \ll (T - T_c)^{\delta \beta}$. Now, Eqs. (3) and (5) give

$$
Z = Z(0) \int_{R^n} d^n \vec{h} \exp \left[-\frac{V h^2}{2p} + A_X^+ V h^2 |T - T_c|^{-\gamma}/2 - B_X V h^4 |T - T_c|^{-\gamma - 2\delta \beta}/4 \right].
$$
\n(8)

Again using a saddle point, we find a critical point at T_c given by

$$
\tilde{T}_c - T_c = (pA_\chi^+)^{1/\gamma},\tag{9}
$$

with $m = 0$ for $T > \tilde{T}_c$. Slightly below the critical point we find $h = \sqrt{\overline{T}_c - T} \sqrt{\gamma A_{\chi}^+ / B_{\chi}}$ $\sqrt{\gamma A_X^+/B_X}(\tilde{T}_c - T_c)^{\delta \beta - 1/2},$ and a magnetization given by (using the scaling law $\delta \beta - \gamma = \beta$

$$
m = \sqrt{\tilde{T}_c - T} A_X^+ \sqrt{\frac{\gamma A_X^+}{B_X}} (\tilde{T}_c - T_c)^{\beta - 1/2}.
$$
 (10)

Thus, the magnetization behaves as $m = \tilde{A}\sqrt{\tilde{T}_c - T}$, with $\tilde{A} \propto p^{(\beta - 1/2)/\gamma}$. If the local system is described by mean-field theory, then $\beta - 1/2 = 0$. In other cases, β < $1/2$, and the mean-field amplitude \vec{A} diverges for small \vec{p} . The specific heat of the system can be obtained by differentiating the partition function (8) twice with respect to temperature. The partition function $Z(0)$ is analytic in *T* at $T = \tilde{T}_c$, while the integral over *h* is not, leading to a specific heat jump at $T = T_c$ equal to $(\gamma A_\chi^+ | \tilde{T}_c - T_c | ^{-\gamma - 1})^2 / (2B_\chi | \tilde{T}_c - T_c | ^{-\gamma - 2\delta \beta}) \propto$ $|\tilde{T}_c - T_c|^{2\delta\beta - \gamma - 2} = |\tilde{T}_c - T_c|^{-\alpha}$. This jump in specific heat is comparable to the specific heat of the local system at temperature \tilde{T}_c .

We now consider the width of the mean-field critical region. For $T < T_c$, the average magnetization of the local system in the absence of a field behaves as $|T_c - T|^{\beta}$, and the susceptibility is given by $\chi =$ A_X^{-} $|T_c - T|^{-\gamma}$. In the long-range system, this magnetization produces a field $h \propto p|T_c - T|^{\beta}$, which in turns feeds back and increases the magnetization an amount of order $p|T_c - T|^{\beta}|T_c - T|^{-\gamma}$. For $|T_c - T|^{\gamma} \gg p$, this effect is negligible compared to the averaged field itself, $|T_c - T|^{\beta}$. Thus, at such temperatures the long-range interactions have a negligible effect on the magnetization, and so the mean-field critical behavior extends only to $|T_c - T| \propto p^{1/\gamma} \propto |\tilde{T}_c - T_c|$. Therefore, for small *p*, the width of the mean-field critical region is small. The scaling arguments above all rely on this width becoming narrower than the width of the anomalous critical region in the local system, in which case both mean-field and anomalous scaling will be seen in the same system.

*Effect of randomness.—*In the small-world model one adds a quenched set of strong links, while the long-range model lacks randomness and has links of *strength* p/V . We now identify a criterion, Eq. (11), for when it is justified to ignore the quenched nature of the links in the small-world model, at least for determining the universal scaling of quantities with p , as in Eqs. (9) and

(10). The strategy is to consider the long-range model without randomness, and then to add the effects of randomness in the small-world model as a perturbation, determining when it is self-consistent to ignore randomness for small *p*.

At \tilde{T}_c , the correlation length of the local system is $\xi \propto$ $(\tilde{T}_c - \tilde{T})^{-\nu} \propto p^{-\nu/\gamma}$. Thus, within a correlation volume, there are $p^{-\nu d/\gamma}$ sites. In the small-world model, each site coupled with a long-range link feels an average field proportional to *m*, and also feels statistical fluctuations about this field. These effects lead to perturbations in the two relevant variables, the magnetic field and temperature, which must be treated carefully.

Consider first the average field acting on a correlation volume due to the long-range links. If sites in the correlation volume are chosen instead with probability *p* to have long-range links, then an average of $p^{1-\nu d/\gamma}$ sites are chosen. Since $1 - \nu d / \gamma < 0$, this number diverges as $p \rightarrow 0$. Then, there are a large number of sites with longrange links within each correlation volume and so the sample-to-sample fluctuation in the number of such sites within each correlation volume is negligible in the smallworld model. Thus, the sample-to-sample fluctuations in the average field are negligible.

Next consider the statistical fluctuations in the field, which *reduce* the correlation of the given site with its neighbors. This effectively raises the temperature of a site with a long-range link. Consider the number of sites with long-range links. The root mean square sample-tosample fluctuation in the number of such sites scales as $(p\xi^d)^{1/2}$, and thus the sample-to-sample fluctuation in the temperature averaged over a correlation volume scales as $(p/\xi^d)^{1/2} \propto p^{1/2 + \nu d/(2\gamma)} \propto |\tilde{T}_c - T_c|^{\gamma/2 + \nu d/2}.$ Compare this to the difference in temperatures, $\tilde{T}_c - T_c$. As long as

$$
\gamma/2 + \nu d/2 > 1,\tag{11}
$$

the sample-to-sample fluctuation in temperature is negligible as $p \rightarrow 0$. Equation (11) resembles the Harris criterion [11] for the relevance of disorder, with an additional term $\gamma/2$ on the left-hand side. For any model where Eq. (11) holds (this includes most unfrustrated models), fluctuations in field and temperature are both negligible, and the scaling of both $\tilde{T}_c - T$ and \tilde{A} with *p* will be the same in the small-world and long-range models.

Intuitively, we expect that the transition temperature in the long-range model will be higher than that in the small-world model: this is definitely true if we ignore the local couplings, and consider only the long-range links. Therefore, in cases when Eq. (11) does not hold, $\tilde{T}_c - T_c$ should scale as at least as large a power of *p* in the small-world model as it does in the long-range model.

*Variational approach.—*We can also show that the long-range model without randomness provides an upper bound to the free energy of a small-world network model, using an argument inspired by the Migdal-Kadanoff bond-moving procedure [12]. Equation (2) defines the

statistical weight *S* without randomness. If instead of connecting every pair of sites with strength p/V , we add connections between pairs of sites with probability p/V and unit strength, we obtain a new statistical weight $\tilde{S}[\vec{\phi}]=S_{\rm local}[\vec{\phi}]-\frac{1}{2}$ $\sum_{x_1, x_2} \vec{\phi}(x_1) \cdot \vec{\phi}(x_2)$, where the sum extends over sites x_1, x_2 which are connected by a longrange link. Then, $\langle S - \tilde{S} \rangle = \sum_{x_1, x_2} w(x_1, x_2) \langle \vec{\phi}(x_1) \cdot \vec{\phi}(x_2) \rangle$, where the brackets $\langle \rangle$ denote an expectation value computed in the long-range system with statistical weight e^{-S} , and where $w(x_1, x_2) = -p/(2V)$ if x_1, x_2 are not connected in the small-world network and $w(x_1, x_2)$ = $1 - p/(2V)$ if x_1, x_2 are connected. The partition function of a small-world network, Z_{sw} , can be expressed in terms of that of the long-range system, *Z*, via Z_{sw} = Z (exp*S* – *S*). The convex inequality states that $\langle \exp[S - \tilde{S}] \rangle \ge \exp[\langle S - \tilde{S} \rangle]$. The quantity $\langle S - \tilde{S} \rangle$ is a random function of disorder. For a large system, this quantity is self-averaging and its average over disorder is equal to zero, so for typical networks, $\langle S - \tilde{S} \rangle = 0$, up to small fluctuations. Inserting this result in the convex inequality gives $Z_{sw} \geq Z$.

*Comparison to numerics.—*An important work was a numerical calculation of some of these quantities, looking for the shift in the transition temperature [6]. In that paper, a different scaling argument was made for the shift, $\tilde{T}_c - T_c \sim p^{1/(vd)}$. This is the temperature at which a correlation volume includes roughly one long-range link. However, we have argued that the shift in transition temperature actually scales as $p^{1/\gamma}$, which is less than $p^{1/(vd)}$ as $p \rightarrow 0$. The difference arises since one longrange link is not sufficient to magnetize an entire correlation volume; several such links are required.

The numerical results in two dimensions are consistent with a shift in transition temperature scaling as $p^{1/\gamma} =$ $p^{0.57...}$. The numerical results in three dimensions indicate a shift scaling as $p^{0.96}$, while taking $\gamma = 1.2396$ from ϵ expansion [13] gives $1/\gamma \approx 0.81$. This indicates some discrepancy with the numerical results. However, in the numerical study [6], it was argued that their results do not yet involve sufficiently large lattices to obtain accurate scaling; certainly, $p^{0.81}$ is closer to the observed scaling than $p^{1/(vd)} \approx p^{0.53}$ is.

*Nonequilibrium dynamics.—*We now consider the generalization to a nonequilibrium process, the contact process [14], in which each site is marked either infected or susceptible. An infected site becomes susceptible at unit rate, while an infected site can turn a neighboring susceptible site infected at a rate λ/q , with *q* the lattice coordination number. The state with all sites susceptible is absorbing. However, above a critical λ_c , if a single infected site is placed in an infinite lattice of susceptible sites, there is a nonzero probability of the epidemic persisting for all time. We modify the model as follows: each susceptible site can be infected by any other infected site, not necessarily a neighbor, at a rate equal to p/V . Although we will not decouple this interaction, the general development will be very similar to the equilibrium case.

We start by recalling some exponents in the local case. For $\lambda > \lambda_c$, there is an average density, $\rho \propto |\lambda - \lambda_c|^{\beta}$. In the presence of a source, where susceptible sites become infected at a rate *h*, the density $\rho(\lambda = \lambda_c, h) = A_\rho h^{1/\delta_h}$.

Consider also the infection spreading from a single source. At $\lambda = \lambda_c$, the survival probability of the infection after time *t*, $P(t)$ obeys $P(t) = A_p t^{-\delta}$. The number of infected sites is a random variable, $n(t)$; the average number of such sites obeys $\overline{n}(t) = A_n t^n$. The radius of the infection scales as $t^{z/2}$. For $\lambda < \lambda_c$, the infection dies out exponentially, with an asymptotic survival probability $P(t) \propto e^{-t/\tau}$, with $\tau \propto |\lambda_c - \lambda|^{-\nu_{\parallel}}$. This gives rise to a divergent susceptibility: in the presence of a source *h*, the susceptibility, $\chi \equiv \partial_h \rho$, obeys $\chi(\lambda, h = 0) = A_{\chi} |\lambda_c - \lambda|^{-\gamma}.$

Now, consider the dynamics in the long-range model, with $\lambda = \lambda_c$, with a single source for an infection. This source grows as described, with the given $P(t)$, $\overline{n}(t)$. However, the local outbreak starting from that source can produce other local outbreaks elsewhere, via the long-range links, at a rate equal to *p* times the number of infected sites. For *p* small, the number of infected sites $n(t)$ will be large before such an event, and thus the fluctuations in the $n(t)$ are described by a random process with a universal distribution. In the large *V* limit, at fixed *t*, each new local outbreak produced via a longrange link is well separated in space from the other local outbreaks. Thus, we can describe the dynamics of the spread from a single source simply: there is initially one local outbreak, created at time 0, which survives at time *t* with probability $P(t)$, and which produces additional local outbreaks at a rate equal to $pn(t)$. Each local outbreak, created at time t' , evolves independently, surviving with probability $P(t - t')$, and producing additional local outbreaks with rate $pn(t - t')$. This fully describes the dynamics via a branching process. For $\lambda \neq \lambda_c$, this description of the dynamics remains valid with a changed $P(t)$ and distribution of $n(t)$.

At short times, the average number of infected sites in this dynamics is equal to $\overline{n}(t)$. At long times, the average number of infected sites grows exponentially [15]. To describe this exponential growth, realize that at long times the number of local outbreaks becomes large. If $s(t')$ describes the number of local outbreaks started at time t' , then the average number of particles at time t is equal to $\int_0^t dt' s(t')n(t-t')$, and thus on average $s(t)$ = $p \int_0^t dt' s(t')n(t-t')$. The ansatz $s(t) = e^{\alpha t}$ gives

$$
\alpha = [A_{\eta}p\Gamma(1+\eta)]^{1/(1+\eta)}.
$$
 (12)

Each local outbreak takes a volume of order $t^{dz/2}$. Eventually, at sufficiently large time, such that $e^{\alpha t}$ ~ $(V/t^{dz/2})$, the individual local outbreaks start to merge, and the dynamics of different local outbreaks become coupled. This time t is of order $ln(V)$.

Beyond this time *t*, one approaches a stationary state with density ρ . At $\lambda = \lambda_c$, the dynamics is equivalent to the local system with a source of particles $h = p\rho$. Thus, we find that the density obeys $h = pA_0h^{1/\delta_h}$, or $h = (pA_\rho)^{\delta_h/(\delta_h-1)}$, and thus

$$
\rho = A_{\rho}(pA_{\rho})^{1/(\delta_h - 1)}.
$$
 (13)

Equation (13) should be compared to Eq. (7). It implies that the transition to a spreading epidemic happens at $\lambda =$ $\lambda_c < \lambda_c$. For $\lambda \approx \lambda_c$, following the same steps as in the equilibrium case leads to the same result as Eq. (10), except that the mean-field transition has $\rho \propto \lambda - \lambda_c$, rather than $m \propto \sqrt{\bar{T}_c - T}$. Thus, the stationary results in this nonequilibrium model are described by the same scaling theory as in the equilibrium models, while the spread of infection starting from a single source is described by an interesting branching dynamics.

We have developed a general scaling theory for describing equilibrium and nonequilibrium systems with both short- and long-range interactions. We find that the long-range interactions lead to mean-field behavior, but with a scaling region whose width vanishes as $p \rightarrow 0$. We have also developed a branching process description of the spread of infection from a single source in the contact process with long-range interactions.

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