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Statistical model of earthquake foreshocks

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We propose a statistical model of rupture as a mechanism for the occasionally observed marked increase of seismic activity prior to a great earthquake. The physical ingredients of the model are those of geometrical inhomogeneity and viscoelastic creep. We demonstrate that the observed inverse power law for the rate of increase of seismicity before a large earthquake requires no assumptions beyond those of a random featureless distribution of inhomogeneities and a typical power law of creep with exponent m. On the average, the rate of energy release in earthquakes dE/dt before a large earthquake that will occur at time t_r increases with time as $(t_r-t)^{-\beta}$, with β decreasing from $(t+1)$ for $m=0$ to 1 as m increases to infinity; in two dimensions $t=1.3$ is the percolation-conductance exponent. For large m, which is appropriate for ductile-brittle-fracture laboratory measurements, the value of this exponent is in agreement with observations of the number rate of occurrence of earthquake foreshocks. The exponent β is independent of the amount of initial disorder within a broad interval. The power law is a consequence of the many-body interactions between small cracks formed before an impending large rupture. As a consequence of variations in the initial configuration of inhomogeneities, there are large fluctuations in the rate of energy release dE/dt from system to system, a result also consistent with observations of foreshocks in nature.

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I. INTRODUCTION

Some, but not all, great shallow earthquakes are preceded by foreshock activity whose rate increases as the time of the great event approaches. In some sense, the behavior of foreshocks is thus the inverse of the more commonplace observation that most great earthquakes will trigger aftershocks whose rate decays with time after the great event.

Aftershocks are a universal and abundant consequence of large earthquakes and are observed to occur frequently after intermediate-magnitude events. Their rate of occurrence decays according to the empirical Omori [1] law as

$$\dot{n}(t)=A(M)(t-t_r)^{-\alpha(M)}\,, \tag{1}$$

with an exponent that ranges from 0.9 to 1.8 with a mean around 1.2 [2]; Omori's own value for the exponent was 1.0. The quantity t_r is the time of occurrence of the triggering earthquake. The prefactor $A(M)$ decreases monotonically with magnitude M. In general, the larger aftershocks occur earlier in the series, the rate of energy release varying as

$$\dot{E}\approx(t-t_r)^{-\beta}\,, \tag{2}$$

with an exponent about 2.0 [2].

Foreshocks are significantly less numerous antecedents of large earthquakes. In those cases in which they have been observed, foreshocks occur in increasing numbers and with increasing energies as the time of the main shock approaches. A recent example is the increase in seismicity prior to the Loma Prieta ($M=7.1$) earthquake of November 19, 1989 [3]; in this case the increase in seismicity took place over a time interval of the order of several years; it is more usual to associate foreshock activity with shorter time scales, of the order of days, before a great earthquake. Because of its erratic character, the statistics of an individual foreshock series is difficult to determine. Kagan and Knopoff [4] and Jones and Molnar [5] have superimposed many foreshock series by assigning them a homologous time referred to that of the subsequent great earthquakes. In this case, the rate of occurrence of foreshocks is found to follow an inverse Omori law

$$\dot{n}\approx(t_r-t)^{-\alpha}\,, \tag{3}$$

with an exponent also near 1. Efforts to fit foreshock energy rates in analogy with (2) have not been fruitful because of the erratic character of the series. Laboratory experiments, however, show not only that the rate increases, but also that the energies increase as the time of the large earthquake approaches [6].

With regard to mechanism, the persistence of aftershock series for times of the order of many months

calls for a stress-redistribution process with a time scale intermediate between the very rapid rates of rupture for faults and propagation of elastic waves, corresponding to the observed duration of rapidly running earthquakes, and the tectonic time scale of repetition of the largest earthquakes in a given region. The latter is a time of the order of one hundred to several hundred years in seismically active areas.

Benioff [7] recognized early that some form of generalized viscosity or creep was required to account for the extended time delays between the triggering and the triggered events, without specifying the precise nature of the viscous mechanism. Recently Yamashita and Knopoff [8] and Reuschle [9] proposed that the time delays for aftershocks are due to the agency of stress-corrosion processes that serve to degrade the strengths of barriers between the master crack developed by the strong earthquake and smaller cracks in the neighborhood; the large stress field at the edge of the master crack promotes fusion of nearby satellite cracks with the parent in a scenario of ever accelerating degradation of the barriers between them.

In the crack-fusion model, Yamashita and Knopoff [10] (hereafter called YK) assumed that the stress corrosion process proposed for aftershocks could be extended to foreshocks as well, the principal differences being that fusions among cracks were allowed to take place under the agency of stress corrosion as before, but without the overwhelming influence of the stress field due to the master fracture of the triggering earthquake. In their model, a set of two-dimensional (2D) coplanar cracks is imbedded in a 3D elastic medium; the crack sizes are taken from a power-law distribution, as are the spacings between the cracks. The system is subjected to a plane-strain shear stress σ at infinity. The coordinate x of the tip of each crack is allowed to grow under the generalized creep law

$$\dot{x} \approx K^m , \tag{4}$$

where K is the stress intensity factor at the tip and m is a large exponent, experimentally determined to be of the order of 10 to 170 [11]. The stress intensity factor at the ith crack tip is

$$K_i = \sigma L_i^{1/2} f(\xi_{ij}) , \tag{5}$$

where L_i is the length of the ith crack and $f(\xi_{ij})$ is a dimensionless interaction function of the geometry, i.e., of the lengths and positions of all the cracks in the system relative to the ith crack tip. As the crack tips extend according to (4), the gaps between them become smaller and the stress intensity factor increases. Because of the large value of the exponent m, the rate of growth of the cracks accelerates; when the stress intensity factor reaches a critical preassigned value for the particular gap, a transition from quasistatic growth to an unstable radiative phase takes place, and fusion with the neighbor results. The critical stress intensity factors are randomly selected from a square distribution. This model leads to a simulation of the inverse Omori law for foreshocks with a significant rate of increase of fusion events prior to the last event in which all cracks have fused into one master crack. The exponent $\alpha \sim 1$ from numerical simulations. Other models for foreshocks have appeared in the literature (see YK for references), but none has the quantitative appeal that the crack fusion model has. This model forms the basis for our further discussion.

Model YK has two basic ingredients: (i) a set of preexisting cracks with a given spatial and length distribution and (ii) a creep rheology to provide for time delay and memory effects before ultimate rupture. In this paper, our goal is to demonstrate that the inverse Omori law for foreshocks can emerge naturally from the temporal structure of crack interaction without having to specify the geometry, the spatial distribution, or the crack size distributions. In some sense, a prespecification of a power-law distribution of crack sizes might seem to predispose the result to a power law of foreshock rates, although the precise route is not clear. In this paper, we show that the hyperbolic inverse Omori law is the result of a dynamical evolution of crack growth starting from an initial featureless distribution of inhomogeneities.

In recent years, a vast class of models has been studied with a view toward understanding those features of universality that arise in problems of rupture in random media. In this endeavor, we attempt to recognize a self-organizing principle that has been widely observed in other nonlinear many-body systems with complex interactions. We assume that a complex inhomogeneous system can be decomposed into many simple interactive elements with describable individual responses. The complexity of the global system results from the cooperative behavior of all the elements; the cooperative interactions lead, not to complete randomness on the global scale, but to large-scale features such as fractal structures and, in this case, power-law time dependence. Using this strategy, a partial classification of some of the possible regimes of rupture has emerged [12,13]. Such an approach is attractive in order to be able to attack the important difficulties underlying this field that arise from the presence of many interacting defects, from the long-range nature of the elastic Green's functions and from the irreversible evolution of ruptures, for example. In practice, the execution of this program has taken place on quasistatic lattice models. The evolutionary sequence is obtained by solving the elasticity equations; the first bond that satisfies the rupture criterion is singled out and broken. The process is iterated until a macroscopic fracture appears. In the quasistatic models, dynamics, which yields a time scale, is absent. The process is in the spirit of growth models such as diffusion-limited aggregation which describe the quasistatic, irreversible evolution of complex interfaces [14].

In our model, which is a modification of the thermal fuse model of Sornette and Vanneste [15], we extend previous studies on the statistical physics of rupture by incorporating time dynamics with a scale imposed by the addition of creep processes into the rheology.

II. THE STATISTICAL MODEL

For simplicity, we restrict our study to antiplane deformations of a 2D square lattice, which have the virtue of

having scalar behavior. In this case, the strain field has a scalar potential and the problem is fully equivalent to problems in electrostatics [16,17,15]. We consider a lattice of unit mesh oriented at 45° with respect to the edges; the edges are a distance $L/2^{1/2}$ apart; periodic boundary conditions apply at the edge of the lattice. Each element is characterized by an elastic constant g which is a random variable with a probability distribution $P_g(g)$, chosen in these computations to be uniform in the interval $[1-\Delta g/2, 1+\Delta g/2]$ with Δg varying from 0.1 to 1.6. When a macroscopic antiplane stress S is applied to the network, the antiplane strain field becomes inhomogeneous as a consequence of the inhomogeneity of the elastic constants of the elementary bonds; therefore the small-scale stress field also becomes inhomogeneous.

In the present model we assume that the total strain ϵ can be written as the sum of elastic $\epsilon_{(e)}$ and plastic $\epsilon_{(p)}$ components. The elastic displacement $w_{(e)}$ in the direction normal to the lattice plane is given by the solution to

$$\nabla\cdot[g(x,y)\nabla w_{(e)}(x,y)]=0 \tag{6}$$

together with Hooke's law

$$s=g\epsilon_{(e)} . \tag{7}$$

Then $\epsilon_{(e)x}=\partial w_{(e)}/\partial x$ and $\epsilon_{(e)y}=\partial w_{(e)}/\partial y$ for lattice elements that point in the x or y directions. The elastic shear constant is $g=r^{-1}$. To obtain the plastic or ductile deformation $\epsilon_{(p)}$, we assume that an element of the lattice under a stress s will undergo accelerated creep deformation $\epsilon_{(p)}$ which obeys

$$d\epsilon_{(p)}/dt=rs^m , \tag{8}$$

which is close to the model (4). Here the rate of creep deformation $d\epsilon_{(p)}/dt$ accelerates with applied stress. The coefficient r is a function of temperature through the Boltzmann factor $\exp\{-Q/RT\}$, where Q is the activation energy for ductile flow. From (7) and (8), we see that the two components of the deformation are coupled.

If the exponent $m=1$, (8) yields the Newtonian law for liquids; patently, the viscosity of the fluid in this case is thermally activated [18]. In the ductile regime of the earth, the exponent m is about 3 or 4, as a consequence of high-temperature creep in the earth's interior. At the lower temperatures in the seismogenic zone closer to the earth's surface, subcritical crack growth is once again governed by (8), but this time with much larger exponents that range from 10 to 170 as remarked above, and correspond to the brittle regime of deformation regulated by accelerated but unstable crack growth. The nonlinear character of (8) favors the growth of localized strain deformations. In our model, we neglect the temperature dependence of the coefficients m and r and assume that they are fixed and uniform for all elements of the lattice.

A bond element breaks down irreversibly when its total strain ϵ is sufficiently large. In our model, we assume that rupture occurs when the plastic deformation reaches a given threshold $\epsilon_{(r)}$, which is a constant for all elements. Beyond the plane of already formed cracks, which are regions of large elastic deformation, plastic deformation is indeed preferentially localized in the vicinity of crack tips. The extension and deformation of the plastic region, which regulates the growth of the crack, is controlled by the value of the elastic stress according to (8). After the breakdown of a bond, we assume that the stress distribution in the remaining intact elements adjusts itself instantaneously, i.e., the spatiotemporal evolution is solely controlled by the creep deformation; questions of dynamic overshoot are not considered in this approximation.

This model differs from that of YK as follows: here we assume homogeneity of critical strains and inhomogeneity of elastic constants, whereas in YK they are reversed. We do not expect that this feature will change the results [12]. In this paper we solve a problem of antiplane strain while in YK the problem is one of plane strain. In this paper we solve for the evolution of fractures in a 2D fault plane, whereas in YK a 1D problem was solved. Most important, there is no assumption of an initial distribution of broken elements or cracks in our case.

In models such as those of Yamashita and Knopoff [8,10], Reuschle [9] and that of this paper, cracks once formed are not permitted to heal. This is in contrast to the opposite extreme of stick-slip lattice models of Burridge-Knopoff [19] type (see also Carlson and Langer [20], Bak and Tang [21], and others). The absence of healing is appropriate to the description of intermediate-time clustering phenomena that takes place on a time scale that is short compared with the recurrence times of the largest events, and is useful for the description of aftershocks and a variety of precursory clustering phenomena including foreshocks. On the other hand, healing in the stick-slip lattice models is taken to be instantaneous after completion of a rupture event, and these latter models are therefore more appropriate to the long-time scale evolution of seismicity since the recovery of strength is a prerequisite for the restoring of deformational energy on the scale of energies and times of the largest earthquakes.

The computation of the evolution of the sequence of ruptures is carried out as follows. At time $t=0$, a uniform macroscopic stress S is suddenly applied at two edges of the lattice. The stress per bond is S/L in the absence of disordered elastic constants. The elastic stress and strain fields have infinitely short response and are given by (6) and (7). We have used a conjugate gradient technique to solve the equation of elasticity (6), using an error criterion $\epsilon\leq 10^{-20}$. Once the stress in each bond is known, it is substituted in Eq. (8), which gives the time evolution of the creep of the nth element $\epsilon_{(n)}(t)$. The first rupture occurs at $t=t_1$ on that bond which first reaches the plastic deformation threshold $\epsilon_{(r)}$, chosen to be equal to unity for all bonds. After the first fracture, the elastic constant on the fractured element is set equal to zero, and the stresses $s_{(n)}$ in all remaining bonds are calculated again from the equations of elasticity. This new set of stresses $\{s_{(n)}(t_1)\}$ is again substituted into the creep equation (8) with a new set of deformations $\{\epsilon_{(n)}(t_1)\}$. Iterating the procedure after each rupture, noting the time t_i at which the ith rupture event occurs, and resetting the values of $\{s_{(n)}(t_i)\}$ and $\{\epsilon_{(n)}(t_i)\}$, which are the corresponding sets of bond stresses and plastic deformations on all remaining bonds, we obtain the following general

time-dependent plastic deformation expression for the nth bond:

$$\epsilon_{(n)}(t)=\epsilon_{(n)}(t_i)+r_n[s_{(n)}(t_i)]^m(t-t_i)$$
$$\text{for } t_i \le t \le t_{i+1} \text{ if } \epsilon_{(n)}(t) \le 1 . \quad (9)$$

There is a well-defined ordering $0<t_1 \le t_2 \cdots \le t_i \le t_{i+1} < \cdots \le t_r$ of the times at which the successive ruptures take place, ending with the time of the last rupture t_r at which there is a complete disconnection of the network into at least two pieces.

The simplicity of the model stems from the separation of the time evolution of the elastic and plastic deformation fields: the elastic deformation distribution changes instantaneously when the plastic rupture of a new bond occurs and the creep deformation field changes continuously under the fixed elastic stress distribution until the new rupture occurs. This feature simplifies the analysis and the numerical computations. The simplicity of this formulation captures the essential physics of the rupture processes that occur before large impending earthquakes, namely the importance of initial quenched disorder and creep deformations.

III. TIME RATE OF FORESHOCK ENERGIES

The present model contains two well-studied statistical models of rupture as natural limits, namely the quasistatic random fuse model [22] for $m \to +\infty$ and the bond percolation model [23] for $m \to 0$.

A. The quasistatic random-fuse limit ($m \to +\infty$)

When m is very large, the creep deformation rate rs^m of the bond with the largest stress is much larger than those of all other bonds. In this limit, only the bond with the largest stress is significantly deformed compared to the others, and reaches the plastic rupture threshold $\epsilon_{(r)}$ first. Since this is true at all times, the limit $m \to +\infty$ always involves a breakdown of the bond with the largest stress. Thus we recover the *quasistatic* random-fuse model [22] in this limit.

In the limit of large exponent m, we can derive an estimate of the inverse Omori exponent as follows. In this case in 2D, the rupture proceeds by formation of essentially linear cracks of fractal dimension very close to 1 [24]. Consider the growth of a linear macroscopic crack obeying the creep growth law (8) and the corresponding plastic rupture criterion. Except for transients connected with the initial stages of the growth of the crack, one can ignore the influence of the initial disorder on the later stages of the evolution of a very large macroscopic crack, since the rupture dynamics will be controlled by the stress enhancement at its tips. According to Eq. (8), the time needed to break an element subjected to a stress s is of the order of $t=r^{-1}s^{-m}$. When the macroscopic crack length is b, the stress applied to the element at the tip is proportional to $b^{1/2}$ due to stress enhancement. We can reasonably assume that the time to break the element at the crack tip is essentially controlled by the large local stress and is only secondarily sensitive to the plastic deformations which occurred when the bond was more than one lattice interval from the crack tip. This assumption becomes all the more valid in the terminal stage of rupture when the macroscopic crack becomes so large that the rupture process accelerates drastically. In this limit an element far from the tip is subject to relatively small stress and the plastic deformation does not have the time to increase markedly. Thus the approximate time t^* needed to break the element at the tip of a macroscopic crack of length b is proportional to $b^{-m/2}$. During this time, the crack grows by one lattice-mesh interval. Therefore we can write

$$\frac{db}{dt}=\frac{1}{t^*} \approx b^{m/2} . \quad (10)$$

Upon integration for $m>2$, we find $b(t) \approx (t_r-t)^{-2/(m-2)}$ with $t_r=[2/(m-2)]b_{\min}^{-(m-2)/2}$, where $b_{\min}$ is the initial size of the crack. In an infinite system, the elastic energy reduction due to crack formation is proportional to b^2, and the rate of energy release is its time derivative. Thus, finally, $dE/dt \sim (t_r-t)^{-\beta}$, with $\beta=(m+2)/(m-2)$. In the limit $m \to +\infty$, where this computation is valid, the exponent is exactly $\beta=1$. We do not expect this estimate to remain valid for finite values of m due to the fact that the rupture process becomes more complex and our "tip" approximation may be unwarranted. We find below that the value of β for finite values of m is significantly smaller than given by $(m+2)/(m-2)$. If α is independent of magnitude, $\alpha=\beta$.

B. The bond-percolation limit ($m=0$)

In the limit $m \to 0$, the plastic deformation rates are independent of the stress field; the plastic deformation field only depends on the distribution of the elastic constants. Since the elastic constants are independent random variables, the plastic deformation of each bond develops independently. Therefore the successive bond breakdowns are independent random events, solely controlled by the distribution and spatial position of the elastic elements. When a continuous path of ruptured elements appears, the rupture process stops since the elastic modules of the global network becomes zero. At this point, the distribution and position of ruptured bonds is exactly given by the bond percolation model at its critical point $p=p_c$. The rupture dynamics is spontaneously attracted to the critical state of the bond percolation model [23]. The corresponding fraction $q=1-p$ of broken bonds at time t is the fraction of bonds that have reached the rupture threshold $\epsilon_{(r)}=1$, namely $q=\int P_\epsilon(\epsilon,t)d\epsilon$, where $P_\epsilon(\epsilon,t)$ is the distribution of the plastic deformations at time t in the network. $P_\epsilon(\epsilon,t)$ is obtained by performing the change of variable $g \to \epsilon(g,t)$, where $\epsilon(g,t)$ is given by Eq. (6) with $g=r^{-1}$, in the distribution of elastic constants $P_r(r)$, namely $P_\epsilon(\epsilon,t)=t^{-1}P_r(\epsilon/t)$. Global rupture is reached when q attains the bond percolation threshold $q_c=1-p_c$ ($=\frac{1}{2}$ for a square lattice in 2D).

In this case, the inverse Omori law can be derived analytically as follows. For $m=0$, Eq. (9) simplifies into

$\epsilon_{(n)}=r_n t$. The time needed for the nth element to reach the rupture threshold $\epsilon_{(r)}=1$ is exactly r_n^{-1}. The number of rupture events $n(t)dt$ during the infinitesimal time interval dt is thus simply $n(t)=P_r(r)|dr/dt|$ with $t=r^{-1}$. Thus $n(t)=t^{-2}P_r(t^{-1})$. The fraction $p(t)$ of unbroken bonds at time t is simply $p(t)=1-\int n(t)dt$. The global elastic constant G of the network is proportional to the global conductance of an equivalent network of resistances. From percolation theory, G goes to zero as p goes to p_c according to the power law $G\approx(p-p_c)^t$ with a critical exponent $t=1.300$ in 2D and 2.0 in 3D. The elastic energy dE/dt released per unit time during the rupture process is given by the time derivative of the elastic energy stored within the elastic network. Since the system is subject to a constant stress, the global elastic energy is simply proportional to the inverse G^{-1} of the global elastic constant. Therefore the rate of release of elastic energy dE/dt scales as $[p(t)-p_c]^{-t-1}$ with $p(t)=1-\int t^{-2}P_r(t^{-1})dt$. For concreteness, consider the case of a uniform distribution of elastic constants $P_g(g)=1/\Delta g$ in the interval $[1-\Delta g/2, 1+\Delta g/2]$. Then $P_r(r)=r^{-2}/\Delta g$ and $n(t)=t^{-2}P_r(t^{-1})=\Delta g$ is a constant. Thus $p(t)=1-t\Delta g$ and $dE/dt\sim d(G^{-1})/dt \sim(t_r-t)^{-t-1}$ with $t_r=1/2\Delta g$. We thus obtain a power

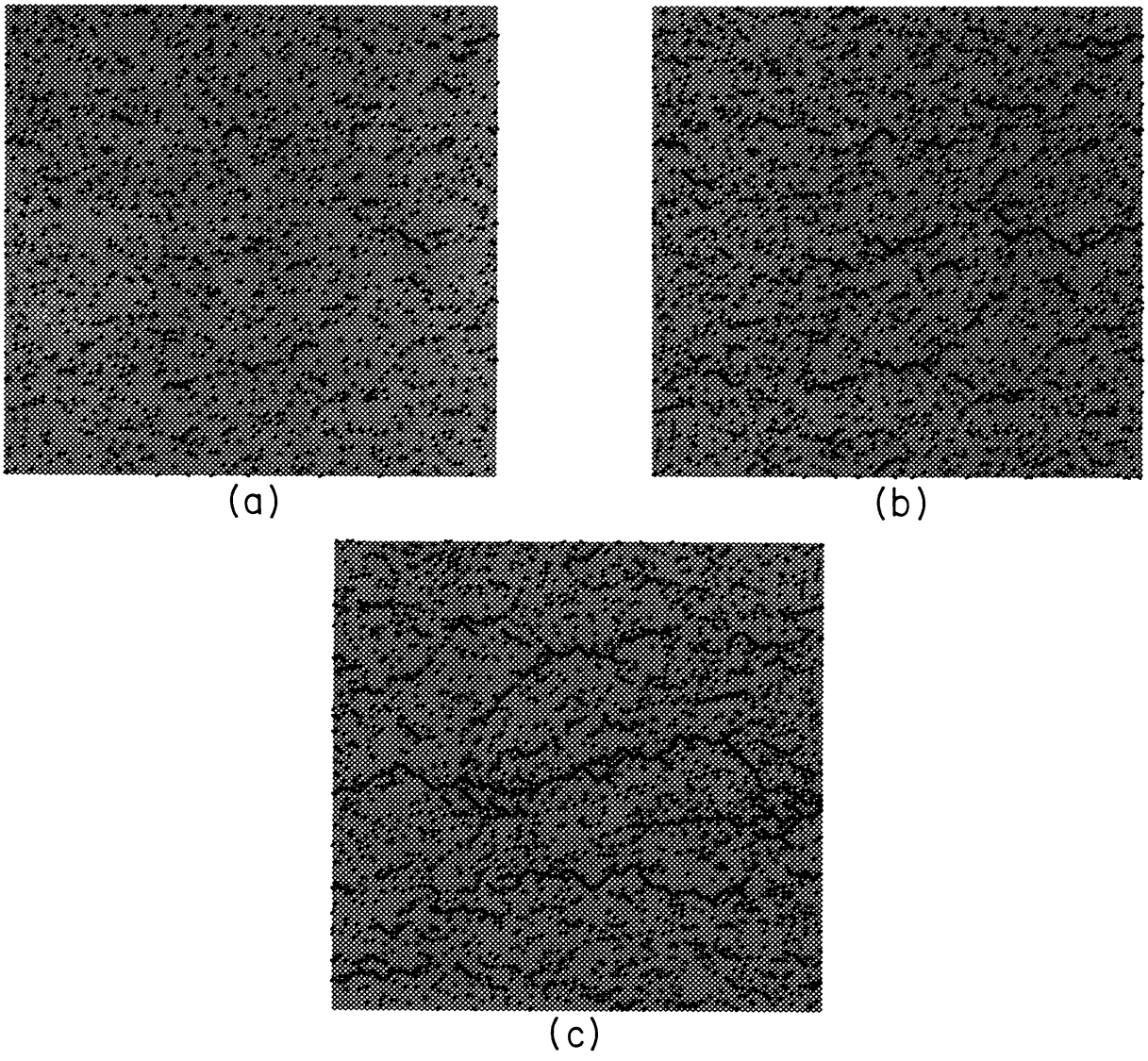

FIG. 1. Crack patterns at three times of the rupture history of the same system for $m=2$, $\Delta g=0.2$, on a square lattice of size 180×180 tilted at 45° to the applied stress. (a) Fracture pattern at $t/t_r=0.9912$; 50% of the elements needed for global fracture have ruptured. One observes mainly isolated, independent breakdown events plus the existence of a few relatively large clusters of broken elements. The former contribute to the increasing damage of the system and the latter tend to dominate the further revolution of the process. The damage occurs late in the process: the first element breaks down at $t/t_r=0.886$, the second at $t/t_r=0.895$. (b) Fracture pattern at $t/t_r=0.9982$; 80% of the elements have ruptured. Many large cracks are competing. It is difficult to predict the path of the ultimate global rupture. (c) System at global failure; $t/t_r=1$. A small change in the initial disorder may change the final rupture pattern drastically.

law of the form of the inverse Omori law with an exponent $\beta = t+1 \approx 2.3$ in 2D. We have verified that the scaling law with the exponent $\beta = t+1$ will hold for a very broad class of distributions of elastic constants. Note that the limit $m=0$ shows that $n(t)$ can be constant, i.e., dn/dt is zero, whereas dE/dt need not be zero. We remind the reader that it is the distribution of $dn(t)/dt$ that is usually observed, while that for dE/dt is not.

C. The general case ($0 \leq m < +\infty$)

The exponent β for the rate of energy increase in foreshocks depends on the value of the creep exponent m, and should decrease from $\beta_0 = 2.3$ in the percolation limit $m=0$ to $\beta_\infty = 1$ in the quasistatic "random fuse" limit $m \to +\infty$. We present results from computer simulations on more general cases $0 \leq m < \infty$ that show that β does indeed decrease continuously as m increases. For $m=2$ for instance, we find $\beta \approx 1.3$. For each value of m (0.1,0.5,1.2,4,8) typically 25 different configurations have been computed for each value of the disorder parameter Δg in networks of size 80×80, a procedure similar to that used in YK.

Figure 1 exhibits a typical example for the time evolution of the rupture process in a random 180×180 network with a uniform distribution of bond elastic constants, with $\Delta g = 0.2$ and $m=2$. Rupture occurs in two main steps. At the beginning of the process, the progressive deterioration of bonds is similar to a random bond dilution in which the initial quenched disorder of the elementary elastic constants dominates the dynamics. At large regimes, the dilution process is followed by a regime characterized by correlated cluster growth and fusion events between extended cracks. In this regime, stress enhancement and screening effects become important. The relative importance of these two steps depends upon the disorder Δg and on the creep exponent. Increasing the disorder and decreasing m favors the uncorrelated random dilution regime. For a given configuration of disorder of elastic constants, it can be proved [25] that the ordered sequence of bond rupture and therefore the final crack pattern at the end of rupture remain invariant as the global applied stress is changed. The dynamical sequence, such as the time ordering of the breakdown of different elements is invariant. Only a global rescaling of the time scale by the factor S^{-m} takes place. In other words, the time needed to observe a macroscopic rupture scales with the externally applied stress S as S^{-m}.

Sornette and Vanneste [15] have studied in some detail the self-similar structure of the cracks that are formed spontaneously during the dynamical rupture process. For systems of size 80×80, the "capacity" fractal dimension has been estimated to be $D = 1.11 \pm 0.02$ for $m=2$ and does not seem to depend on this disorder Δg. Preliminary calculations for $m<2$ indicate an increase of the fractal dimension with decreasing m as follows: $D = 1.3 \pm 0.1$ for $m=1$ and $D = 1.5 \pm 0.1$ for $m=0.5$; at $m=0$, we recover the value of the percolation model $D = 2-\beta/\nu = 129/144 \approx 1.9$. For $m>2$, the fractal dimension decreases down to $D \approx 1$ for $m=8$. This result is to be compared with the value of the quasistatic random fuse model of $D = 1.1 \pm 0.1$ [24], which according to Sec. III A is equivalent to the limit $m \to +\infty$.

We turn to the time dependence of the rupture process, since this is relevant to the determination of the inverse Omori law. For each system, we have calculated the time dependence of the global elastic constant $G(t)$ of the network. Since the system is subjected to a constant stress S, the global elastic energy is simply proportional to the inverse G^{-1} for the global elastic constant. The rate of energy release is thus simply proportional to dG^{-1}/dt. In Fig. 2 we display the time dependence of G^{-1} on a log-log scale, i.e., the cumulative value of the energy release rate for the case $m=2$, as a function of $(t_r - t)/t_r$, where t_r is the time of occurrence of a macroscopic rupture in the network. Each curve corresponds to a different value of the disorder Δg, with the lowest corresponding to the smallest disorder we have used $\Delta g = 0.1$, and the upper to the largest disorder $\Delta g = 1.6$ Each curve has been obtained by averaging over typically 25 different network configurations ("ensemble" average) with the same average disorder Δg. We observe that G^{-1} indeed follows a power law as a function of $(t_r - t)/t_r$ with an exponent $(\beta - 1) \approx 0.3$ for $m=2$. The value of β does not seem to depend on the strength of the disorder Δg since all curves are approximately parallel. However, β is sensitive to the value of the creep exponent m: for $m=0.1$, we find $\beta \approx 2.2$, which can be compared with the theoretical prediction $\beta = 2.3$ for the percolation limit $m=0$; for $m=1$, $\beta \approx 1.7$; for $m=4$, $\beta \approx 1.15$; for $m=8$, $\beta \approx 1.05$, which again can be compared with the prediction $\beta = 1$ for the linear crack growth limit $m \to \infty$.

Figure 3 presents the evolution of G^{-1} as a function of $(t_r - t)/t_r$ for 30 different realizations with the same average disorder $\Delta g = 0.2$. The curve with index $\Delta g = 0.2$ in Fig. 2 has been obtained by averaging the 30 curves shown in Fig. 3. Figure 3 underlines the importance of fluctuations from system to system. It is clear that the in-

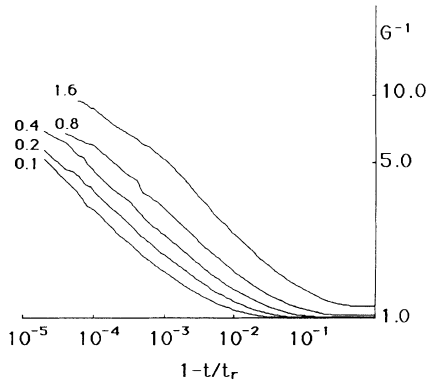

FIG. 2. Time dependence of G^{-1}, which is the cumulative energy release rate as a function of $(t_r - t)/t_r$, where t_r is the time of macroscopic rupture of the network (log-log plot). Each curve corresponds to a different value of the range of disorder Δg, with the lowest for $\Delta g = 0.1$ and the uppermost for $\Delta g = 1.6$. Each curve is the ensemble average over (usually) 25 realizations with same Δg. The average slope of the curves in their linear portion is $(\alpha - 1) \sim 0.3$.

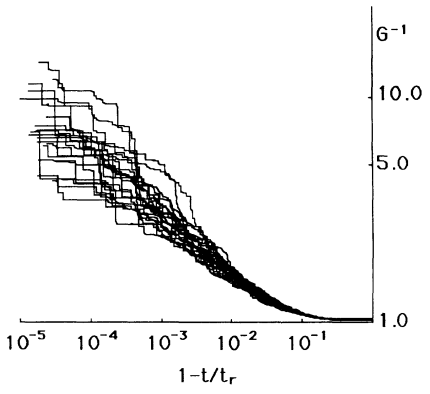

FIG. 3. Evolution of G^{-1} as a function of $(t_r-t)/t_r$ for 30 different network configurations with the same average disorder $\Delta g=0.8$. Each curve corresponds to an individual realization for which no averaging has been carried out.

verse Omori law holds with good precision only by averaging over many realizations; for a specific realization, there may be very large deviations from the average behavior. Thus, if an inverse Omori law is valid in general, large deviations from the mathematical expression can be expected to be the rule rather than the exception, due to the role of initial inhomogeneities which are amplified by the rupture dynamics. The size of the system-to-system fluctuations should decrease as the lattice size increases, as is well known in the two limits $m=0$ and $m\to\infty$. Therefore the quality of the observation of the foreshock rate law depends on the amount of initial disorder, its specific configuration and the size of the great rupture event; the exponent does not depend on these features.

IV. CONCLUSIONS

Assuming random featureless inhomogeneities of the elastic properties and a power law of creep with exponent m, we have demonstrated the existence of a rate law for the increase of seismic activity before an impending great-rupture even at time t_r. The exponent β is independent of the amount of initial disorder within a broad interval but varies with the exponent m of the creep law. Our model provides a simple explanation for the existence of a hyperbolic law of elastic energy release before rupture. The relation is a consequence of the many-body nonlinear interactions among the elements of the network. The complex correlated behavior underlined by the power law does not result from any built-in complexity either into the spatial structure or into the rheological laws, but rather arises from the cooperative behavior of all the elements whose successive correlated fractures eventually lead to the great event. The observations of the power-law rate behavior in nature with $\alpha\approx 1$, first made by Kagan and Knopoff [4] and Jones and Molnar [5], suggest an exponent that is wholly consistent with a high value of the exponent in the creep law which, in turn, is consistent with laboratory observations of crack-growth phenomena in the brittle-fracture regime of deformation. As noted, $\beta=\alpha$ if α is independent of M. Finally, our model might provide a physical basis for understanding other empirical relations of hyperbolic type which give evolution rates of measurable quantities in the terminal stages of failure of many different systems [26].

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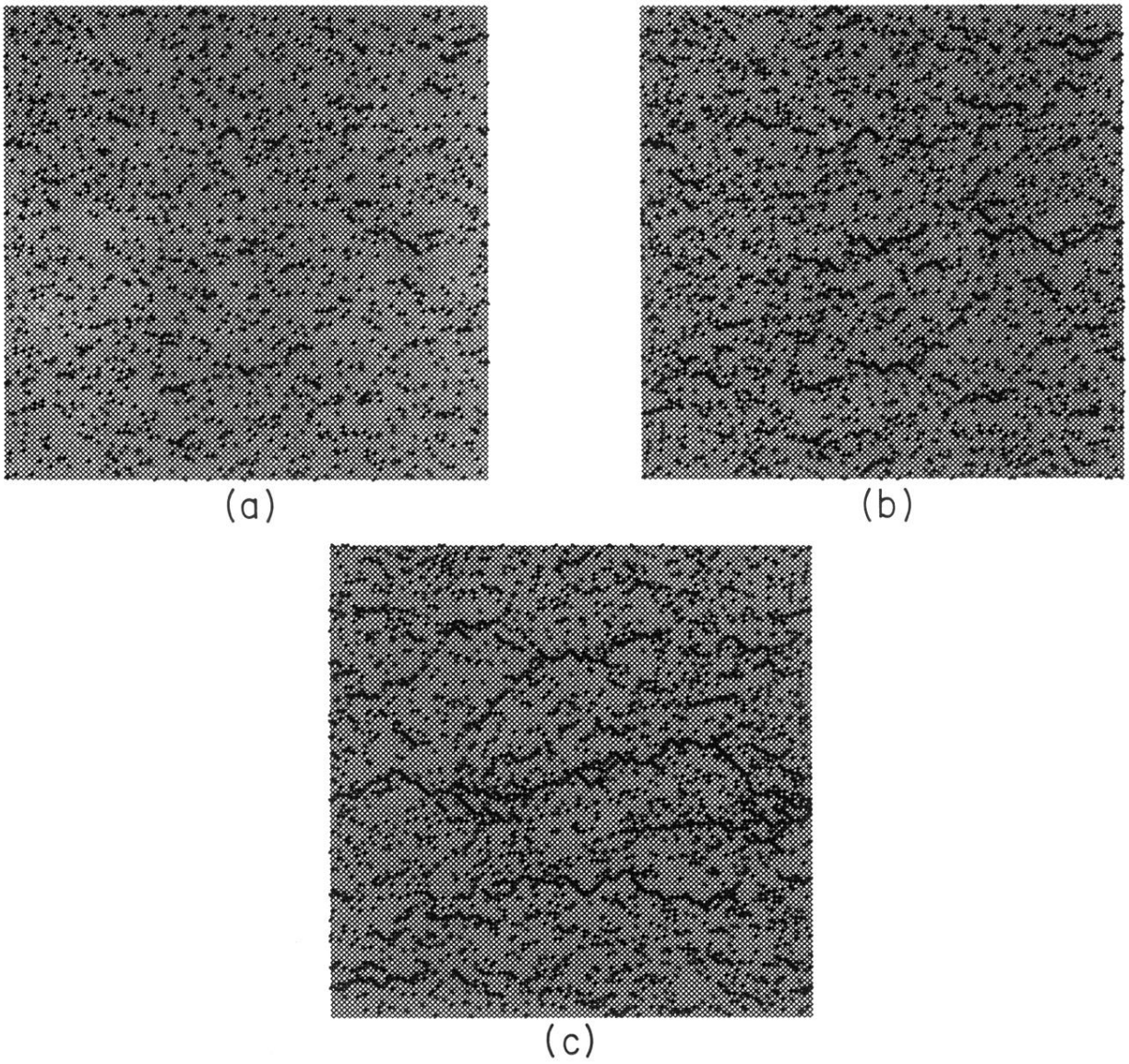

FIG. 1. Crack patterns at three times of the rupture history of the same system for $m=2$, $\Delta g=0.2$, on a square lattice of size 180×180 tilted at 45° to the applied stress. (a) Fracture pattern at $t/t_r=0.9912$; 50% of the elements needed for global fracture have ruptured. One observes mainly isolated, independent breakdown events plus the existence of a few relatively large clusters of broken elements. The former contribute to the increasing damage of the system and the latter tend to dominate the further revolution of the process. The damage occurs late in the process: the first element breaks down at $t/t_r=0.886$, the second at $t/t_r=0.895$. (b) Fracture pattern at $t/t_r=0.9982$; 80% of the elements have ruptured. Many large cracks are competing. It is difficult to predict the path of the ultimate global rupture. (c) System at global failure; $t/t_r=1$. A small change in the initial disorder may change the final rupture pattern drastically.