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Permeability as a toggle switch in fluid-controlled crustal processes

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Abstract

Fluid transport in the earth's crust is either extremely rapid, or extremely slow. Cracks, dikes and joints represent the former while tight crystalline rocks and impermeable fault gouge/seals represent the latter. In many cases, the local permeability can change instantaneously from one extreme to the other. Instantaneous permeability changes can occur when pore pressures increase to a level sufficient to induce hydro-fracture, or when slip during an earthquake ruptures a high fluid pressure compartment within a fault zone. This 'toggle switch' permeability suggests that modeling approaches that assume homogeneous permeability through the whole system may not capture the real processes occurring. An alternative approach to understanding permeability evolution, and modeling fluid pressure-controlled processes, involves using local permeability rules to govern the fluid pressure evolution of the system. Here we present a model based on the assumption that permeability is zero when a cell is below some failure condition, and very large locally (e.g. nearest neighbors) when the failure condition is met. This toggle switch permeability assumption is incorporated into a cellular automaton model driven by an internal fluid source. Fluid pressure increases (i.e. from porosity reduction, dehydration, partial melt) induce a local hydro-fracture that creates an internally connected network affecting only the regions in the immediate neighborhood. The evolution, growth, and coalescence of this internal network then determines how fluid ultimately flows out of the system when an external (drained) boundary is breached. We show how the fluid pressure state evolves in the system, and how networks of equal pore pressure link on approach to a critical state. We find that the linking of subnetworks marks the percolation threshold and the onset of a correlation length in the model. Statistical distributions of cluster sizes show power law statistics with an exponential tail at the percolation threshold, and power laws when the system is at a critical state. The model provides insights into mechanisms that can establish long-range correlations in flow networks, with applications to earthquake mechanics, dehydration, and melting. © 2000 Elsevier Science B.V. All rights reserved.

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1. Introduction

* Corresponding author. Fax: +41-1-633-1108; E-mail: steve@erdw.ethz.ch Fluids (and their pressure state) play a dominant role in many geological, geophysical, and petrologic processes [1,2]. Substantial evidence suggests that fluid pressures in many parts of

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the crust are close to lithostatic [3–5], and ubiquitous evidence in the form of mineral bearing veins point to hydro-fracture as a primary means of fluid transport [6]. Over-pressured fluid affects the mechanical strength of rock, and was proposed by Hubbert and Rubey [7] as the mechanism responsible for overthrust faulting. Overpressuring in sedimentary basins is widespread below depths of 1-2 km [8], and abrupt vertical and lateral transitions form distinct pore pressure states within sedimentary sequences [9]. Thin faults or hydrothermally altered seals add to the spatial and temporal variations in crustal hydraulics. Rapid changes in pore pressure are suggested as an important precipitation mechanism in gold deposits [10,11], and dilatancy hardening is a wellknown phenomenon associated with crustal faulting [12,13]. Reaction-induced hydro-fracture in high-pressure anatectic melting experiments [14] confirms that this mechanism exists, and may be important in fluid-controlled crustal processes.

A principal problem in modeling fluid-controlled crustal processes is that the nature in which fluids move in the crust is controlled by a parameter that spans about five orders of magnitude for common geologic materials. In general, permeability (k) ranges from about 10^{-14} m² for common sandstone to less than 10^{-19} m² in shales and clays [15,16]. This problem is compounded by evidence that fluid flow in the crust is channeled [17] and episodic [18], resulting in strong spatial and temporal variations in pore pressure and hydraulic properties. Given such an uncontrolled parameter, fluid flow models developed for midcrustal levels can generate a wide range of results, with potentially little or no validity. Here we present a simple model where the internal permeability network develops within the system in response to some physical or chemical process. Specifically, we consider an increasing pore pressure mechanism through porosity reduction or a direct fluid source that increases pore pressure until hydro-fracture. To model rapid changes in hydraulic properties, permeability is treated as a toggle switch (e.g. on or off), being either zero or very large to nearest neighbors. That is, permeability is zero while the fluid pressure state is below some failure condition, and locally very large when the

fluid pressure reaches this condition. Conceptually, this translates to two extreme flow states in the crust. Over short time scales in an impermeable medium, fluid flow is restricted, and pore pressures increase at rates that depend on the fluid source rate and the compressibility of the medium. At the time of failure, fluid flow is rapid to the immediate neighborhood. The reduced fluid pressures that accompany an increase in crack porosity or sudden hydraulic connectivity to a low-pressure region thus limit flow to the immediate neighborhood. Whether the high fluid pressure perturbation propagates depends on the state of the neighbor: (1) If the neighbor is sufficiently far from failure, the crack (or fluid flow) is arrested, or (2) if the state of the neighbor is also near the failure condition, then the instability can propagate.

The purpose of this paper is to demonstrate the basic behavior of a model based on the toggle switch permeability assumption, presented as a cellular automaton model, and describe how this model behaves in terms of permeability evolution, self-organization, and critical states. The model has no inherent length scale above the grain scale, so its utility is limited to understanding evolutionary processes to a critical state and a study of model statistics. We have purposely reduced the problem to the simplest possible scenario, excluding for now processes that can be initiated by rapid fluid pressure reductions, such as precipitation, dissolution, dehydration, melting, or dilatancy hardening. Coupling the model to some of these processes is addressed elsewhere [19,20].

2. Conceptual model

The cyclic (or toggle switch) model for permeability is postulated as a possible mechanism operating within the crust or in fault zones. For earthquakes [21,19], fault compaction increases pore pressure in zero permeability cells during quiescence [22,23]. Permeability is transiently very large when dilatant slip accompanies an earthquake [12,13,24,25]. After fluid pressures are redistributed among cells participating in the event, rapid sealing toggles permeability back to





Fig. 1. Conceptual model of toggle switch permeability used for modeling fluid-controlled crustal processes. (a) Earthquakes. Fault zone compaction or other fluid sources in a low permeability fault zone increase fluid pressure in discrete compartments. High fluid pressures induce a dilatant slip event that locally increases permeability and equilibrates fluid pressure in the immediate neighborhood. Healing and sealing then reduce permeability. (b) Dehydration/melting. Isolated nucleation sites from a dehydration or melting reaction increase pore pressure and can induce local hydro-fracture. Hydro-fracture increases the local permeability, and with continued reaction, the crack network grows and coalesces.

zero. Fluid flow within the fault plane is thus episodic and only transiently over-pressured. For dehydration, melting, or other phase transitions with positive Clapeyron slopes (Fig. 1b), scattered nucleation sites within the body generate local over-pressures at time of the reaction [26]. These sites are initially hydraulically disconnected, both to a drained boundary or to other sites $(k \sim 0)$. Increased fluid pressure can buffer the reaction unless it is sufficient to induce hydro-fracture [14]. If hydro-fracture occurs, then the site is hy-

draulically connected to the local environment, thus reducing fluid pressure via increased crack porosity or hydraulic connectivity to low-pressure regions. The reduced fluid pressure has the same thermodynamic effect as an increase in temperature (although much reduced), and the dehydration/melting reaction continues. The resulting positive feedback is an evolving system where the permeability network is created internally from the loop of fluid pressure increase \rightarrow hydro-fracture \rightarrow fluid pressure decrease \rightarrow kinetics. In this case, rapid sealing from precipitation can reduce permeability, or the toggle switch permeability comes into play because permeability is zero between simultaneously evolving crack networks that are hydraulically unconnected. When isolated networks become connected, then k is very large within this subsystem, but is still zero between similar systems developing independently within the body. Scale invariance can be seen conceptually to emerge because the same mechanisms are operating whether one considers the interaction of two individual cells, two interacting networks of many cells, or many isolated networks of unlimited cells. Eventually all subnetworks merge, creating a permeable pathway where the macroscopic value for k becomes applicable. Merging of internal networks results in a percolation-type threshold within the system [27,28].

3. A cellular automaton model of fluid flow

These concepts are well modeled with cellular automata [29–32]. Cellular automata are simply numerical bookkeeping algorithms to distribute a parameter to nearest neighbor cells once some prescribed condition is reached. Typically, a random distribution of a parameter is assumed scattered in a two-dimensional matrix (although it can be extended to three dimensions). The system is then driven by this parameter until a failure criterion is satisfied, where the 'load' is redistributed to nearest neighbors. These extremely simple models produce complex and rich behavior, and are commonly used in studying critical phenomena and phase transitions. The original Ising model for a ferromagnet is an example of a cellular automaton. In this paper a cellular automaton model is proposed for crustal fluid flow, where the driving parameter is pore pressure within the system.

The physical basis for this model derives from a formulation of the diffusion equation that includes source terms from time-dependent porosity reduction $\dot{\phi}$, or a direct fluid source $\dot{\Gamma}$. Various derivations are given elsewhere and are not repeated here [33–35]. The equation is:

$$\frac{\partial P_f}{\partial t} = \frac{1}{\phi(\beta_{\phi} + \beta_f)} \left[\frac{k}{v} \nabla^2 P_f - (\dot{\phi}_{\text{plastic}} - \dot{\Gamma}) \right]$$
(1)

where the first part in brackets describes pore pressure reduction through diffusion, and the second part in brackets represents pore pressure increases from a fluid source. In Eq. 1 ϕ is porosity, β_{ϕ} and β_f are the pore and fluid compressibility $(\beta_{\phi} = (1/\phi)(\partial \phi/\partial P))$, v is the viscosity, and k is the intrinsic isotropic permeability of the matrix. For an impermeable matrix $(k \sim 0)$, Eq. 1 reduces to:

$$\frac{\partial P_f}{\partial t}|_{\text{noflow}} = \frac{(\dot{\Gamma} - \dot{\phi})_i}{\phi_i \beta_i} \tag{2}$$

where *i* is the cell matrix index and the compressibility has been lumped into a single parameter $\beta = \beta_{\phi} + \beta_f$ [34]. When porosity is reduced in a system faster than the permeability network can transport it away ($k \approx 0$; $\phi < 0$), pore pressures increase at rates proportional to the storage capacity of the rock ($\phi\beta$). Proposed mechanisms for porosity reduction include healing and sealing of cracks [33], pressure solution [5,36-38], compaction by sedimentation [39,40], and compaction creep of fault gouge [41-43]. In such systems, pore pressures can increase until they overcome the least principal stress and initiate hydro-fracture. In the case of earthquakes, pore pressure increases reduce the frictional resistance to sliding and can induce a dilatant slip event [21,24]. In both cases, fluid pressures are reduced rapidly with local permeability increases $(k \approx \infty)$. The new permeability network may include opening old pathways, or creating new ones. The cycle of reduced porosity, hydro-fracture, and repeated porosity reduction is proposed as the mechanism for the observed episodic crustal fluid flow [13,18]. Field evidence of spatially varying, episodic fluid flow within mature fault zones supports the assumption in Eq. 2 that permeability is sufficiently small as to be approximated as zero [22,44]. Examples of a direct fluid source include a fluid source at depth [45], or devolitization reactions [46].

When the failure condition is reached, fluid pressures instantaneously equilibrate with nearest neighbor cells. This can be viewed as a diffusion time of one time step and fixes a relationship between the diffusion length and the size of the smallest model element [47]. The equilibrium pressure, determined by conserving fluid mass (and ignoring gravity), is:

$$\overline{P} = \frac{\sum_{i=1}^{m} (\phi \beta)_i P_i}{\sum_{i=1}^{m} (\phi \beta)_i}$$
(3)

where \overline{P} is the average pressure of the affected cells, P_i is the pre-failure pore pressure in cells *i*, and *m* is the number of cells involved in the redistribution. We do not currently include increases in crack porosity associated with an event, so any fluid pressure reductions result solely from hydraulic connectivity with lower-pressure regions. Including other important mechanisms such as increased crack porosity, mechanical strength variations, and time-dependent healing would allow larger fluid pressure variations, but are currently not explicity modeled.

The numerical algorithm is as follows: (a) calculate increase in fluid pressure in each cell (Eq. 2), (b) check for cells meeting the failure condition and monitor the size of the connected regions, (c) for failed cells, redistribute fluid pressure among nearest neighbors (Eq. 3), (d) repeat (b) and (c) until all cells are below the failure condition, and (e) advance to next time step. During the redistribution phase, cluster size is determined by counting the distinct number of cells involved in the event. The algorithm keeps track of the state of each cell, and after applying Eq. 3, the matrix is scanned to see if the high pore pressure zone propagates, or stabilizes. Initially, only localized events occur because the system is hydrauli-



Fig. 2. Histograms of initial pore pressure increase rates of four cases considered. The rates were determined by choosing a random distribution of source terms (numerator) or material properties (denominator) in Eq. 2. The distributions of the source term and material properties are shown in the insets.

cally disconnected. As the system evolves, evolving subsystems link to a point where communication between cells can propagate spontaneously through the system, creating a hydraulically connected body.

4. Model input

We investigated a matrix of 300×300 cells. The free parameters reduce to assumptions about the fluid sources, initial porosity, and the compressibility of the pore space and fluid. Although many processes are involved in increasing pore pressure with time, we have chosen to simplify the system by investigating a range of pore pressure increase rates (Fig. 2) determined by assumed initial distributions of either the numerator or denominator in Eq. 2. In cases I and IV, normal and uniform distributions of compressibility, respectively, were

assumed in a matrix with a constant source term. In cases II and III, normal and uniform distributions of the source term, respectively, were assumed in a matrix of constant compressibility. Compressibility of the pore space and fluid are relatively well constrained within a range of values [15], and were restricted in cases I and IV to 1×10^{-2} MPa⁻¹ $\leq \beta \leq 1 \times 10^{-3}$ MPa⁻¹ [34]. For cases II and III, compressibility was constant at $\beta = 5 \times 10^{-3}$ MPa⁻¹. For the source term, we assumed porosity reduction rates on the order of geologic strain rates ($\sim 1 \times 10^{-15}$ s⁻¹), constrained in cases II and III to 1×10^{-8} $yr^{-1} \le \dot{\Gamma} - \dot{\phi} \le 1 \times 10^{-6} yr^{-1}$, and held constant for cases I and IV to $\dot{\Gamma} - \dot{\phi} = 1 \times 10^{-6} \text{ yr}^{-1}$. The initial porosity was chosen at 2% for all cases. Fig. 2 shows the input distributions of parameters, and the resulting distributions of pore pressure increase rates. The failure condition was arbitrarily set at the overburden pressure at a depth

of 10 km (270 MPa). This choice of failure condition has no effect on the overall model behavior. The initial cell fluid pressures were evenly distributed between lithostatic and hydrostatic pore pressure to represent the range of possible fluid pressure states in the crust. In the different cases presented, numerous processes that contribute to a non-uniform pore pressure build-up have been grouped into a distribution of pore pressure increase rates. These different rates were determined by assuming a different distribution of sources and compressibilities, ignoring for now random differences in initial porosity, porosity creation with hydro-fracture, changes in the mechanical strength of the rock, and time-dependent healing. Grouping these processes into a single term captured the overall behavior of the model, but specific cases are left to future work.

Eq. 2 provides a model time scale in years, and the time step was chosen at 0.1 yr for all cases. The effects of choosing a range of time steps was not fully explored, but for cases where a time step of 1 yr was chosen showed no effect on the overall evolution of the system. However, some quantitative details are affected by the time step and discussed below.

5. Results

In the absence of a condition for reducing the fluid pressure of the system (e.g. a drained boundary or porosity creation), Eq. 2 requires that the average pressure of the system monotonically increases until the system reaches the failure condition. However, individual cells in the matrix can both increase and decrease in pressure depending on the porosity reduction rate and connectivity with other parts of the matrix at different fluid pressure states. This is shown in Fig. 3 where the pressure history of three arbitrarily chosen cells is shown superposed on the average system pressure. The path of individual cells shows random increases and decreases that depend on the pressure state of the cells to whom they have been connected (Eq. 3), but must necessarily converge to the average pressure state at the end of the simulation. Such pressure reductions were envis-



Fig. 3. Pore pressure profiles of arbitrary cells in the matrix and the average pore pressure of the system. The average pressure of the entire matrix steadily increases toward the failure condition, while the paths of individual cells can include abrupt pore pressure increases or decreases that result from connectivity to separately evolving subnetworks. Ultimately, the average system pressure must reach the failure condition because of the no-flux external boundary condition.



 $\leq 95\%$ Hydrofracture

Fig. 4. Snapshots of pressure state showing the late time evolution of large networks of connectivity approaching the failure condition (e.g. within 5%). Little structure is observed prior to frame a because the cells that fail are isolated in space and hydraulic communication with neighbor cells lowers the pressure and heals it. Once the system is self-organized (a–c) and large regions of connectivity are established, a structure is observed, which grows in scale (d–f). On approach to the critical state (g–h), the scale of interaction has increased to the size of the matrix and the whole system nears failure (i). The corresponding times of these frames are indicated in Figs. 5b and 6b. The color bar ranges from <95% of the failure condition to >99% of the failure condition.

aged in qualitative models of episodic fluid flow in the crust [13]. Convergence to the average system pressure will be discussed shortly in the context of increasing correlation and linking of subnetworks on approach to a critical state (e.g. when the system is at incipient failure). The subtle increase in the overall pore pressure increase rate of the system results from the functional dependence of the rates on porosity, which itself is being reduced (Eq. 2).

For the following discussion, an event is defined as the failure (via hydro-fracture) of at least one cell. If the failure of a cell and subsequent pressure redistribution initiates failure of neighbor cells during the same time step, then the number of connected cells are counted when equilibrium is achieved, and a cluster is defined as the size of the connected cells. That is, if pressure equilibration between neighbor cells is still above the failure condition, then pressure is redistributed to the next nearest neighbors. This continues until pressure equilibration of the subnetwork falls below the failure condition. The number of cells involved in this redistribution sequence is a cluster. Connectivity within the system is monitored in the numerical algorithm, thus ensuring that at least one cell length separates the boundaries between clusters.

The general model behavior (for case II) is shown visually as snapshots in time of zones of incipient failure (Fig. 4a-i). This visual map of the pressure state corresponds to the late time quantitative description in Fig. 3. The full animation of this simulation can be found at http://www. erdw.ethz.ch/~steve/press.htm. At early times, cells approaching failure are isolated in space, so when a cell fails, pressure equilibration with neighbor cells does not propagate and cluster size is limited to one or a few cells. After sufficient time has passed, clusters begin to appear (Fig. 4ac) that converge onto a defined structure of incipient failure within the system (Fig. 4d-f). Once this structure is established (e.g. Fig. 4f), it grows in time (Fig. 4g-h), until a critical state is reached (Fig. 4i) whereby the system as a whole is nearing the failure condition. These snapshots are quanti-



Fig. 5. Time line of the number of events, shown as a histogram binned by year. In all cases, the number of events steadily increases until a drastic reduction occurs and which marks the percolation threshold and the onset of a correlation length. A longer range of interaction reduces the effects of individual cells because the pore pressure grows at the effective rate of clusters. A similar pattern in the time line is observed at later times because of the same effect of linking correlated networks. That is, large subnetworks begin to merge and thus change (again) the scale of interaction. The times of the snapshots shown in Fig. 4 are indicated in frame b.



Fig. 6. Time line of the cumulative sum of the size of all events. All cases show a steady increase until reaching a plateau (marked by a square) that corresponds to the abrupt change in number of events in Fig. 5. The explanation for this feature of the record is the same as described in Fig. 5. The statistics of cluster size distributions (Fig. 7) were determined at times marked by the 'x'.

fied as a time line of the number of events (Fig. 5), the cumulative cluster size (Fig. 6), the statistical distributions of cluster sizes (Fig. 7), and a quantification of the cluster size distribution for case II (Fig. 8). Each of these will be discussed in turn, but the reader should refer to Figs. 3 and 4 for the following discussion of how this system behaves.

6. Discussion

A general model behavior, independent of the input, is the manner in which the evolved structure in Fig. 4 relates to the (self-) organization of the system. Fig. 5 shows the time line of the number of events for each of the four cases, with the time of each snapshot of Fig. 4 indicated on Fig.

5b. The overall shape of the time line of events is the same for all cases. At early times (not shown), the rate is constant and reflects the random failures of cells. As the system evolves, the rate of failure grows steadily as the average system pressure increases and a larger number of cells fail (or fail again). The acceleration in the number of events is followed by an abrupt reduction late in the simulation. Coincident, but not coincidently, the reduction in the number of events occurs at the time when the structure of incipient failure is first established (Fig. 4c-d). As will be shown, this marks the onset of a correlation length, and identifies the percolation threshold of the system. At the percolation threshold, the system is reset at a new scale of interaction. These subsystems then grow in unison, link with other such subsystems evolved elsewhere in the matrix, and evolve toward a critical state. In Fig. 4d-i, this transition is observed as growth of an established structure, and is seen both in the rate at which cells are failing (Fig. 5), and the rate at which failing cells are accumulating (Fig. 6). Fig. 6 is the cumulative sum of all cells that failed during the simulation, and the derivative represents the rate at which cells are failing. The cusp seen in Fig. 6 marks the percolation threshold and the onset of a correlation length in the model. The increasing correlation length (e.g. connected subnetworks) effectively reduces the number of cells that can act independently, and thus reduces the number of individual events that can occur. Following the obvious initial reduction in the number of events, another more subtle drop is observed (Fig. 5) that indicates still another scale of interaction from the merging of correlated subnetworks. Ultimately,

the system reaches a critical state where the scale of interaction is the size of the matrix.

The physical basis for this behavior rests with the self-organization of the system. While zones of incipient failure tend to organize into a correlated structure, they surround (and are surrounded by) zones of low pressure that are also organizing. When the two networks at different pressures meet, the subnetworks merge and equilibrate at a fluid pressure defined by the pressures involved, but below the failure condition. With continued pressure build-up, correlation among cells increases and large portions of the system act together and approximately follow the average pressure of the system. In other words, this healing effect is pronounced at or above the percolation threshold because correlated zones of high pressure tap into low-pressure zones, thus equilibrat-



Fig. 7. Evolution of the cluster size distributions for the four cases, shown as distributions of cluster size (S) and the number of events (N) greater than S. The distributions were determined for the model catalog up to the times indicated by 'x' in Fig. 6. The cluster size is determined by the number of connected cells that fail during one event. In all cases, the final distribution of cluster sizes show power laws over many orders of magnitude, but with slightly different structure at the largest events.

ing a large part of the matrix at a lower pressure. This occurs only when the system is correlated because a large number of cells need to be involved to significantly alter the pressure late in the system evolution. The observed large pressure reductions early in the simulation (e.g. Fig. 3) represent only local equilibration, so only local healing is possible while healing of the overall system is not observed.

The behavior of the system can be quantified by investigating the evolution of cluster size distributions (Fig. 7). The distributions were determined for the entire record up to the times indicated by 'x' in Fig. 6, and span the percolation threshold (indicated by the square). When a cell fails at early times, pressure redistribution with nearest neighbors typically reduces pressure of the affected cells. Therefore, the cluster size is one and the early part of the record is dominated by events of size one (or a few), with no large events. This results in a very steep slope of the cluster size distribution. As correlation among cells is established and grows, larger events are recorded and this results in a wider range of event sizes. As the whole system approaches the failure condition, failure of one cell can initiate failure of neighbor cells, which can then chain-react through the matrix and generate clusters of all sizes up to the size of the matrix.

The cluster size distributions in Fig. 7 are well fit by a simple correlation relationship:

$$D(S) = S^{-\alpha} \exp(-S/L) \tag{4}$$

where S is the cluster size, α is the power law exponent, and L is a correlation length. Eq. 4 is often used in statistical percolation models and critical point phenomena [48,49]. Fig. 8 shows the distribution for case II superposed with a fit of Eq. 4 resulting in estimates of the correlation length in the model. The fitted distributions correspond to the times shown in Fig. 6b, spanning the cusp that we identify as the percolation threshold. Fig. 8 shows that a significant correlation length is established around the cusp of Fig. 6, and grows rapidly following the cusp. In a classic bond percolation model [48], power law statistics are found at the percolation threshold. Power law statistic are also found here, but the power law is appended with an exponential tail controlled by the correlation length. That is, the local correlations created by resetting connected elements at the same fluid pressure introduce similar effects at the smaller scale. As the correlation



Fig. 8. Fit of Eq. 4 to the cluster size distribution in Fig. 7b. The fit shows how the correlation length increases beyond the percolation threshold up to the critical state where the system is at the failure condition and a power law is observed.

length increases (Fig. 8), the power law extends to larger sizes. Ultimately, the system acts as a single unit $(L \rightarrow \infty)$ and shows power law distributions of cluster sizes that span the size of the matrix (approximately five orders of magnitude and 10^5-10^6 individual events). If the system size was extended, the same power laws would result. Some deviations from a power law when the system is at failure are observed (e.g. cases I and III), and show both sub- and supercritical behavior as discussed elsewhere [50,51]. The slope of the power law depends somewhat on the time step. For larger time steps (e.g. 1 yr), slopes of around -1.2 are observed that reflect the coarser merging of networks as the system approaches failure.

7. Conclusions

We have presented here a simple model that follows through on the common observation that hydro-fracturing and other sudden changes in hydraulic properties probably play an important role in crustal hydraulics. The model is based on a basic assumption that crustal permeability can be approximated as very small (i.e. effectively zero), or very large (i.e. effectively infinite), with the transition between the two extremes activated by processes such as dilatant slip during earthquakes or hydro-fracture. The actual limits of the toggle switch are not well known, but can be argued to be in the range of 10^{-18} $m^2 \leftrightarrow 10^{-16} m^2$. The toggle switch permeability is supported by numerous field observations, and provides a strong non-linearity necessary to produce the resulting complex statistics. The model results present a plausible scenario for crustal fluid flow, with implications for most fluidcontrolled crustal processes. We show that the average behavior of the system pressure is indistinguishable from a continuum approach, but also show that sudden, large amplitude fluctuations occur in response to the linking of independently evolving subnetworks. These highfrequency perturbations govern the internal connectivity structure. The evolution of the connectivity structure shows the onset of a correlation length at the percolation threshold that can

be described as a power law with an exponential tail. After the percolation threshold, the correlation length increases until the entire system is at the verge of failure where we observe power law statistics of cluster size distributions, indicating scale invariance. Physically, scale invariance means that the snapshots in Fig. 3 could be viewed either as an entire system itself, or as merely a minor subsystem operating within a larger network. That is, the snapshots could be both a representation of the processes occurring at the level of pore structure, or at the level of large scale fluid pressure development and expulsion within the crust. When the fluid pressure state of the system is about to reach the failure condition (Fig. 4i), then it could equally represent incipient failure of one computational cell, or a large system that is on the verge of meeting its nearest neighbor. The no-flux model boundaries require that the fluid pressure within the system ultimately reaches the failure condition. In the earth, drainage occurs over long time scales, and an eventual balance is reached between sources and sinks, thus resulting in a state of criticality over geologic time scales.

This model shows critical behavior in the same sense as other cellular automata models [29] that produce power law statistics at a critical state, and can be described in terms of critical state phase transitions, percolation theory, and renormalization group theory [52]. However, we arrive at these results from an approach that uses physical processes to explicity drive the system and show quantitatively and visually what these theoretical results show mathematically. The rich behavior of the model presented here results from the simplest possible scenario of instantaneous large changes in local permeability. We investigated a wide range of inputs for probable crustal conditions, and show that the general behavior of the model is independent of that input. The model shows that the system self-organizes to a state where the merging of isolated networks resets the scale of interaction. At this new scale of interaction, larger events become possible, until at the critical state all event sizes are possible because the scale of interaction is on the scale of the model.

The utility of this model is that the rules that

drive the system are based on simple concepts with substantial natural observations to back up the initial assumptions; namely that permeability can take on extreme values in response to a sudden event. A unique aspect of the model is that a natural large-scale correlation mechanism is inherent in the system self-organization, and is based on the physically probable mechanism of connecting isolated networks at different fluid pressures. The model can be further developed by explicity modeling other processes that are currently ignored. This includes the simultaneously evolving stress field in response to the evolving crack network, and poroelastic effects [53]. Numerous feedbacks and couplings can be included in future models, with applications to fluid pressure-induced mineral precipitation or melting and dehydration reactions. As already pointed out, there exists no length scale in this model above the grain scale. Although this limits its immediate ability to compare with geologic observations, it does serve to point out a physical basis for scale invariance of permeability and crack networks often observed in nature. The model has many potential applications in modeling fluid-controlled crustal processes. For example, coupling this simple model to elastic dislocation theory provided a length scale necessary to develop a deterministic fluid-controlled three-dimensional earthquake model [19], and coupling this model to dehydration kinetics has produced a dynamical system in P-T space for understanding permeability evolution in dehydrating systems [20]. A further coupling to melt kinetics and elastic dislocation theory [54] provides the necessary length scale for an envisaged forward and deterministic model of volcanic seismicity.

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