

Correlation length evolution in cellular automata with long-range stress transfer

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Abstract

In recent years, theoretical research in seismology has been stimulated by the infusion of ideas from statistical physics. Bak and Tang (1989) [1] proposed that the Earth's crust is in a state of Self-Organised Criticality (SOC). This proposal was based upon the statistical similarities between earthquake statistics and those of highly simplified models called cellular automata. A SOC system is one which organises into a state in which events of all sizes may occur at any time. Assuming the Earth's crust is SOC, forecasting large earthquakes is seemingly impossible. More recently, it has been proposed in the literature that regional seismicity may be an example of a Critical Point (CP) system. CP systems progressively approach, and retreat from, a critical state in which large events occur. Cumulative energy release prior to the largest events is predicted to follow a power-law time-to-failure relationship, suggesting that intermediate-term forecasting of large earthquakes may be possible. Cellular automata with differing nearest neighbour energy transfer may display behaviour similar to CP systems or SOC systems (Weatherley *et al.*, 2000 [2]). In these models, energy is only transferred to the nearest neighbours of a failed cell. Such short-range interactions lead to unphysical stress discontinuities at the boundaries of ruptures. For this reason, we have devised a new type of automaton with long-range energy transfer. We find that in a certain regime of parameter space, such long-range automata can display behaviour consistent with the critical point hypothesis of earthquakes. Prior to the largest events, long-range stress correlations progressively form due to the action of external loading and small to moderate sized events. Large events only occur when the stress field is sufficiently correlated at large wavelengths. In a significant number of cases, the largest events are preceded by a period of accelerating cumulative energy release.

A description of the model

The model consists of a rectangular grid of $N = 128 \times 128$ cells. Each cell, $i = (1, 2, \dots, N)$ has two properties: a constant, scalar strength, σ_{ci} and a variable, scalar stress $\sigma_i(t)$. The cell strengths are defined by a statistical fractal distribution of dimension $D = 2.3$, with values in the range $0.1 \leq \sigma_c \leq 1.00$. The dynamical state of a cell is given by the difference between cell strength and stress. This parameter is defined as the stress deficit of a cell:

$$\phi_i(t) = \sigma_{ci} - \sigma_i(t) \quad .$$

The model is driven by periodically increasing the stress of all cells by a small stress increment, $\Delta\sigma$. As a consequence, the stress deficit of cells is driven towards zero. When the stress deficit of a cell $\phi_i \leq 0.0$, the cell is said to fail. The stress of a failed cell is redistributed to other cells according to stress transfer laws described below. Stress redistribution may trigger failure of other cells. In this fashion, ruptures with a range of sizes may cascade through the model.

Since both external loading and stress redistribution changes the stress of cells, model evolution is a two stage process. In the first, loading stage the stresses of all cells are incremented by the minimum stress necessary to fail at least one cell i.e. $\Delta\sigma(t) = \min \phi_i(t)$. The purpose of the second, relaxation stage is to redistribute the stress of failed cells and any cells which fail as a result of stress transfer. Throughout a simulation, the model relaxes to equilibrium prior to the next loading stage. This simulates a slow rate of loading and defines two distinct timescales in the model: a loading timescale and a relaxation timescale. The relaxation timescale is implicitly assumed to be much shorter than the loading timescale.

Unlike traditional cellular automata, stress redistribution in the model is long-range. The stress of a failed cell is redistributed to all cells within a square region surrounding the failed cell. The proportion of stress transferred to a given cell within the transfer region, is governed by an interaction function of the form $f(r) = r^{-p}$ where r is the distance between the failed cell and the cell of interest.

To ensure that all the stress of a failed cell is transferred to cells in the transfer region, we define interaction coefficients I_k for each cell k within the transfer region, with the restriction:

$$\sum_{k=1}^{N_k} I_k = 1 \quad ,$$

where $N_k = s^2$ is the number of cells within a transfer region with a side length, s . Each cell in the model is assigned a grid location (x, y) where $x, y = 1, 2, \dots, 128$. If the failed cell is located at (x_f, y_f) and a cell within the transfer zone is located at (x_k, y_k) , the distance to cell k is $r_k = \sqrt{(x_k - x_f)^2 + (y_k - y_f)^2}$ and the interaction coefficient is:

$$I_k = \frac{r_k^{-p}}{I_o} \quad ,$$

$$I_o = \sum_{k=1}^{N_k} r_k^{-p} \quad .$$

Models with a range of transfer region sizes, s and a variety of interaction exponents, p have been examined. Stress transfer in models with $s = 3$ is nearest neighbour and the interaction function is unimportant; all cells in the transfer region receive equal amounts of stress from the failed cell. As the transfer region size increases, the interaction function becomes progressively more important in defining the fraction of stress transferred to a given cell within the transfer region. For sufficiently large interaction exponents $p \geq 1.00$ and transfer region sizes $s \geq 32$, the interaction function is the dominant factor determining the fraction of stress transferred to a given cell.

The interaction exponent p and the transfer region size s are two important factors determining the statistical behaviour of the model. If stress transfer is near-uniform over a broad region (large s and small p), the size distribution of events is strongly characteristic earthquake with a population of small to moderate events following a Gutenberg-Richter distribution and a separate population of characteristic large events. Conversely, if stress

transfer is relatively short range (small s or large p), the size distribution is Gutenberg-Richter for the entire range of event sizes. Models with parameters intermediate between these two extremes display moderately characteristic event size distributions (see Figure 1).

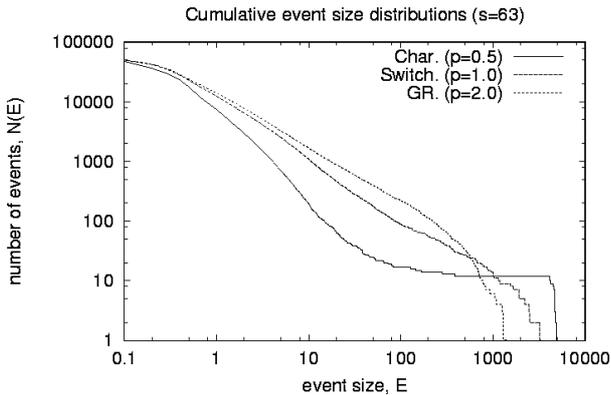


Figure 1: Cumulative event size distributions for three models displaying characteristic earthquake behaviour, Gutenberg-Richter behaviour, and mode-switching behaviour.

Examination of mean stress evolution reveals that characteristic earthquake models display large stress fluctuations, whereas Gutenberg-Richter models display relatively small stress fluctuations. Intermediate models display mode-switching behaviour, switching between time intervals with relatively large fluctuations and intervals with relatively small fluctuations. The results in the following section illustrate the dynamics of a model with $s = 63$ and $p = 1.00$, a mode-switching model. This model serves to highlight the important features of the dynamics of cellular automata with long-range stress transfer.

Although all of the stress from a failed cell is transferred to the cells within the transfer region, some stress is dissipated from the model during ruptures. Stress dissipation arises in two different ways. Firstly, if part of the transfer region for a failed cell lies outside the model boundaries, the stress transferred to that portion of the region is lost from the model. Thus, a failed cell on a boundary transfers half of its stress to cells within the model and half of its stress is lost from the model. Similarly, a failed corner cell will transfer only a quarter of its stress to cells in the model. Stress dissipation also occurs when stress is transferred to a cell which has failed during the current relaxation stage. It is assumed that the healing timescale for cells is larger than the relaxation timescale but smaller than the loading timescale. Failed cells cannot support any stress until the next loading timestep.

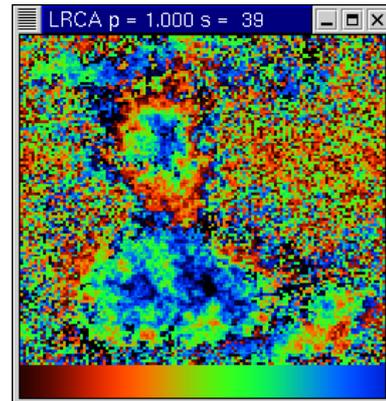
Key results illustrating model dynamics

Stress heterogeneity and rupture dynamics

The stress transfer law in an automaton determines the degree of stress heterogeneity in a system once it has settled into a statistical steady-state (Weatherley, *et al.*, 2000 [2]). In the long-range model described above, all the stress from failed cells is redistributed to neighbours. Subsequent to a rupture, the stress of all failed cells is zero. Based upon this, one might expect the stress field to be relatively smooth in long-range automata; cells which fail concurrently will have similar stresses. As may be seen in the snapshot of stress deficit (Figure 2), some regions of the model have a relatively smooth stress field, while other regions have a heterogeneous stress field. On average, any given cell may receive stress from multiple failed cells during a given interval of time. However, even closely spaced cells may receive different amounts of stress from the failed cells. Consequently, the stress of cells may be quite different even over small distances. The most strongly heterogeneous regions correspond with

the weaker regions of the model. Cells in these regions support relatively little stress so failure of one of these cells may not cause failure of nearby cells which have a low stress. As a result, stress heterogeneity may be quite strong in the weakest regions.

Figure 2: A snapshot of stress deficit in a long-range automaton. Hot colours represent low stress deficit (cells which are close to failure) and cooler colours represent higher stress deficit (cells which are further from failure). Note that some regions of the model have relatively smooth stress deficit while other regions are quite heterogeneous. Stress heterogeneity is responsible for the two types of ruptures in the model: distributed and consolidated ruptures.



The degree of stress heterogeneity affects the character of ruptures in the model. In nearest neighbour models, only the cells immediately surrounding a failed cell can fail in response to stress transfer from the failed cell. Ruptures tend to be localised and disjoint regions of the system fail almost independantly. With long-range interactions, stress transfer from a failed cell may trigger failure of cells which are spatially separated from the failed cell. In such a case, the rupture is spatially distributed. The small to moderate events are commonly distributed, branching through regions with highly heterogeneous stress and failing relatively few of the cells. Larger events consist of a mixture between localised and distributed rupture. These events occur when a broad strong region has reached a state of high stress. Failure of a cell near such an asperity triggers a rupture cascade which fails the entire asperity. Often failure of a strong asperity cascades into weaker regions, becoming more distributed in character as the rupture front encounters increasingly heterogeneous stress. The boundaries of ruptures are not clearly evident in the long-range automata as there are no high stress concentrations on rupture fronts, as is the case for nearest neighbour automata.

Evolution of stress deficit correlations

Events in the model may be broadly classified into smaller, distributed ruptures and larger, consolidated ruptures. The distributed ruptures occur predominantly in the weaker regions of the model and occur quite frequently compared with the consolidated ruptures. Occuring mainly in the stronger regions, consolidated ruptures require that a sufficiently broad region is close to failure. The approach to failure is driven by external loading and stress transfer from ruptures in surrounding weaker regions. The focus of this investigation is to determine whether the occurrence of consolidated events is preceded by systematic changes in stress deficit correlations within the system, and whether such changes are associated with accelerating cumulative energy release.

In the model, the energy released during an event is defined as the sum of the squared stresses of all cells which fail during a single loading timestep. If $f = 1, 2, \dots, N_f$ is the index of a failed cell and N_f is the number of failed cells for a given timestep t , then the energy release $E(t)$ is given by

$$E(t) = \sum_{f=1}^{N_f} \sigma_f^2(t) \quad .$$

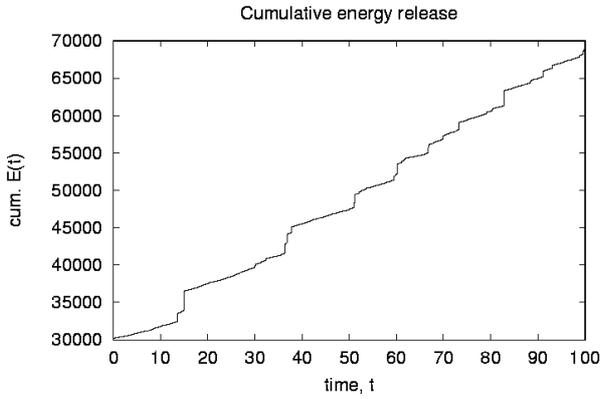


Figure 3: Cumulative energy release for 20000 timesteps in a mode-switching model ($s = 63$, $p = 1.00$). Note that a number of the moderate to large events are preceded by an interval of accelerating cumulative energy release.

Figure 3 is a plot of cumulative energy release for 20000 loading timesteps in a mode-switching model with a transfer region size, $s = 63$ and an interaction exponent, $p = 1.00$. A significant fraction of moderate to large events in this model are preceded by an interval of accelerating energy release. Such accelerating sequences may be fit to a good approximation, by a power-law time-to-failure relationship of the form $\sum E(t) = A + B(t_c - t)^m$.

We seek to determine whether there are systematic variations in stress deficit correlations preceding the occurrence of larger events in the model. The correlation function for stress deficit is defined as

$$C(r) = \frac{\langle (\phi_i - \bar{\phi})(\phi_j - \bar{\phi}) \rangle}{\langle (\phi_i - \bar{\phi})^2 \rangle}$$

where $r = \sqrt{(x_j - x_i)^2 + (y_j - y_i)^2}$, $\bar{\phi}$ is the mean stress deficit at time t , and $\langle \dots \rangle$ represents the global average. The correlation function is computed for $0.0 \leq r \leq 128.0$ every 200 timesteps during the time interval shown in Figure 3. The computed correlation function is shown in Figure 4.

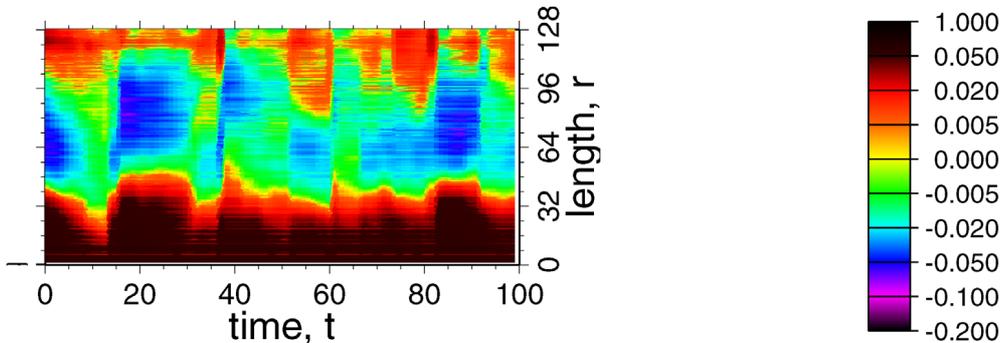


Figure 4: Correlation function of stress deficit field for 20000 timesteps of a mode-switching model with $s = 63$ and $p = 1.00$. The colours represent the value of the correlation function for a given length, r at a given timestep t .

Both positive and negative correlations may occur in the model. Positive correlations for a given length, r implies that cells separated by a distance r have very similar stress deficits. In such a case we would expect a rupture to grow to at least a scale $l \sim r$. Negative correlations imply that, for cells separated by r , on average one cell has a stress deficit greater than the mean deficit ($\bar{\phi}$) and one cell has a stress deficit lower than the mean deficit. In other words, negative correlations for a length r imply that regions of the model separated by a distance r have quite different values of stress deficit. In such a case, we would not expect a rupture to grow to a scale $l \sim r$. Values of the correlation function very close to zero implies that the numbers of cells with high stress deficit is approximately equal to the

number of cells with low stress deficit. Thus, it is possible for a rupture to grow to a scale corresponding to near-zero correlation.

Compare the cumulative energy release (Figure 3) with the correlation function evolution (Figure 4). At small distances ($r < 32.0$), stress deficit correlations are positive all of the time. Thus, small to moderate events may occur at any time. During intervals in which relatively few moderate to large events occur, there are significant negative correlations for lengths in the range $32.0 < r < 96.0$. A large event with $l > 32$ is unlikely to occur during these intervals. As the time of a moderate to large event approaches, correlations at intermediate distances approach zero and positive correlations increase for the largest distances, $r > 96.0$ (for example $t = 12$, $t = 34$, and $t = 50$). In such a case, a broad region has accumulated sufficient stress to allow a large event to occur. Subsequent to this large event, correlations are more negative for lengths associated with the scale of the rupture.

Discussion and conclusions

At intermediate to large distances, the correlation function evolution clearly indicates the preparation of the model for a large event. Cumulative energy release accelerates prior to the largest events and may be fitted by a power-law time-to-failure relationship in a significant number of cases. These two features suggest that the long-range automata display a type of critical point behaviour. The system progressively approaches a state in which a large event may occur, falling away from this critical state after a large event has occurred.

Evolution of stress deficit correlations associated with approach to, and retreat from, a critical state are important features of the long-range automata described here. The dynamics of these models appears to be consistent with the critical point hypothesis of earthquakes. Unlike nearest neighbour automata, small to moderate events play a significant role in the preparation of the system for a large event. For these reasons, it may be beneficial to continue to investigate the dynamics of these models. The simplicity of the models and their computational efficiency, make them prime candidates for research into the physical mechanism underlying critical point phenomena and the applicability of the critical point hypothesis as a theory for regional seismicity.

Acknowledgments

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