Journal of Statistical Physics, Vol. 74, Nos. 5/6, 1994

Evolution by Damage Spreading in Kauffman Model

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Received October 23, 1993

In Kauffman's Boolean automata model on the square lattice, the Darwinian fitness of survival can be defined as the fraction of elements which do not change from one iteration to the next. Biological mutations are simulated by flipping one bit in the rule of one site. Selection of the fitter mutant then optimizes the whole lattice completely. This optimization is particularly effective near the critical point of the transition to chaos, but is in itself not a critical phenomenon. Also a two-dimensional spin glass can be optimized in this way.

KEY WORDS: Cellular automata; critical phenomena; optimization; spin glass.

Simplified mathematical models of biology have an old history, e.g., in population dynamics. Discrete mathematics as in cellular automata is particularly useful for genetics, if every gene is assumed to be either on or off, depending on its interactions with other genes and on mutations. In cellular automata, each site of a large lattice carries a variable which is either zero or one (gene off or on) and which physicists like to call a spin. The value of a spin at the next time step t is determined completely by the value of its neighbor spins at time t. Thus both time and space are discrete, allowing high-speed parallel or vector simulation techniques. Kauffman[1] used similar cellular automata to describe cell differentiation in genetics.

The Kauffman model of random Boolean automata is a mixture of all possible cellular automata.[2] Its computer simulation is particularly efficient if the $N = L*L$ sites of a square lattice are influenced by their $K = 4$ nearest neighbors. Then nearly 10^9 spins are treated per second on one processor of a NEC-SX3 supercomputer,[3] and many millions on the DEC

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0022-4715/94/0300-1293$07.00/0

Alpha and HP workstations used in this study. Each site carries a spin which is either up or down; thus each spin is influenced by one of the $2^K = 16$ neighbor configurations. For each of these neighbor configurations, the spin selects at the initialization of the simulation whether it will point up (probability p) or down (probability $1-p$). These 16 bits of choices made in the initialization for one site constitute the rule of this site, which means the later reaction of the gene to whatever the neighbor configuration is.

A mutation then can be a change of one spin, or of one bit in its rule[4] which affects the time development of the lattice. The number of spins differing in the site-by-site comparison of the changed with the unchanged lattice is the Hamming distance; if the initial change is restricted to one site or another localized region (as is the case here), we denote this Hamming distance as the damage, and check if the damage spreads over the whole lattice, remains localized, or eventually dies out. For the nearest-neighbor Kauffman model on the square lattice, $p_c = 0.31$ (or $= 1 - 0.31 = 0.69$) seems to be the critical point[5] for this transition to chaos: For $p < p_c$ damage does not spread, whereas for $p_c < p < 1/2$ damage can spread to infinity. The size distribution of the damage avalanches obeys a power law at the critical point, but not away from it.[8]

Biologically the spins of the Kauffman model may correspond to genes, turned on or off, of one species. If biological evolution is based on random mutation, then the damage should not die out or remain very small, since then the mutation has no effect. On the other hand, damage should not be too large, changing, e.g., one-third of all the genes and trying to transform a rat into a dog. Thus, according to Kauffman, a successfully evolving genome should be poised at the edge of chaos, close to the critical point 0.31, where the damage is large but not infinite. The fitness of the Kauffman model is defined as the fraction of sites which do not change from one iteration to the next; thus, should the system reach a fixed point where nothing moves any longer, the fitness is optimal and equals one. The present note implements this idea, checks if this optimization really works, and looks for the importance of the critical point. (Our "fitness" definition here may agree with the one used by Kauffman,[1] but does not correspond to a biological growth factor and its response to changes in external parameters.[6])

Thus our computer simulates two different Kauffman square lattices simultaneously, one without and the other with mutations. After every mutation, we give the damage ten sweeps through the lattice to spread, check which of the two lattices has the higher fitness, and then copy the one with the higher fitness into the array used before for the lower fitness. Then another mutation is introduced, and the selection process is repeated

again and again. The time t is the number of updates of the whole lattice, requiring Nt updated spins. Biologically, t might at first be identified with the number of cell divisions.

Figure 1 shows how a lattice with five million spins tries to optimize its fitness without the help of any mutations: The fraction of changed sites, i.e., the difference of the fitness from its optimal value of unity, decreases somewhat during the first time steps, but then stays constant. Clearly this optimization strategy is not very successful.

Random mutations change the results drastically: Fig. 2 shows that now after a sufficiently long time the fitness reaches its optimal value, since the number of changing sites decreases exponentially with time, as $\exp(-t/\tau)$. Figure 3 shows for various times the fitness as a function of the parameter p. After about a million time steps, the fitness reaches unity even for $p = 1/2$. We see that for intermediate times the largest improvement in the fitness, i.e., the largest reduction in the fraction of changing spins, is reached for p near the critical point 0.31. In this sense the data justify the hypothesis that life might evolve best near the edge of choas.

However, this optimum is not a critical phenomenon like damage spreading in Kauffman models, the Curie point in ferromagnets, or vapor–liquid equilibria near their critical point. We see in Fig. 2 that the relaxation time τ and the initial fraction of changing sites increase monotonically with the parameter p. Thus, for small p the fitness gain is small, since the

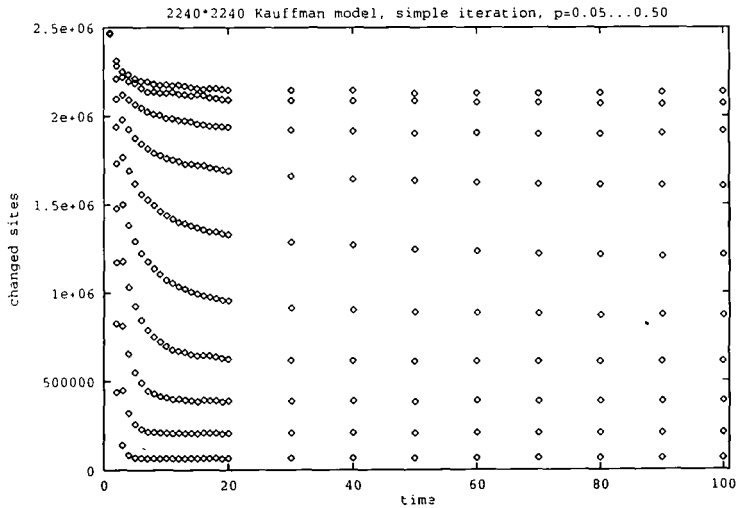


Fig. 1. Number of changing sites versus time t without mutations. This optimization is seen to stop long before it is complete.

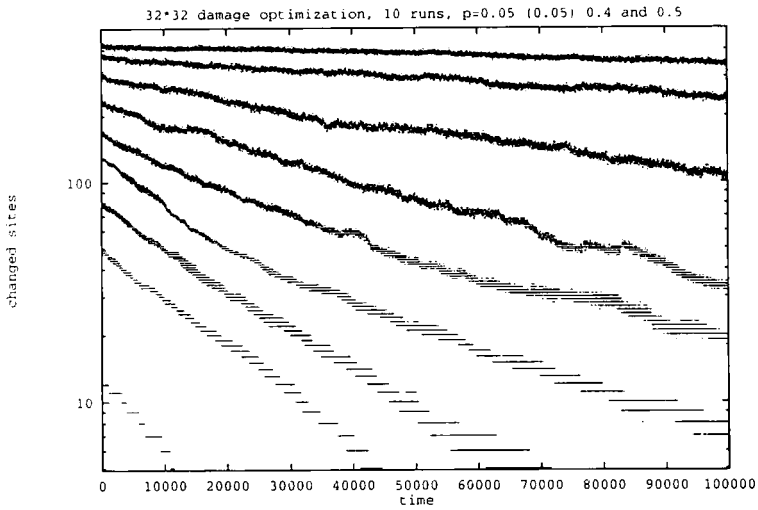


Fig. 2. Number of changing sites, out of 1024 sites, versus time t with mutations happening after every 10 time steps. Now optimization continues to completion: Fitness = 1, no site change any longer. From bottom to top, p varies from 0.05 in steps of 0.05–0.4, followed by $p = 0.5$.

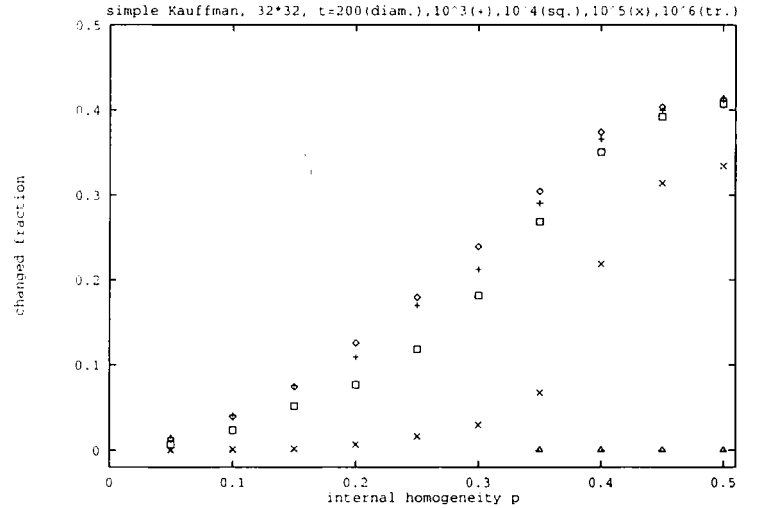
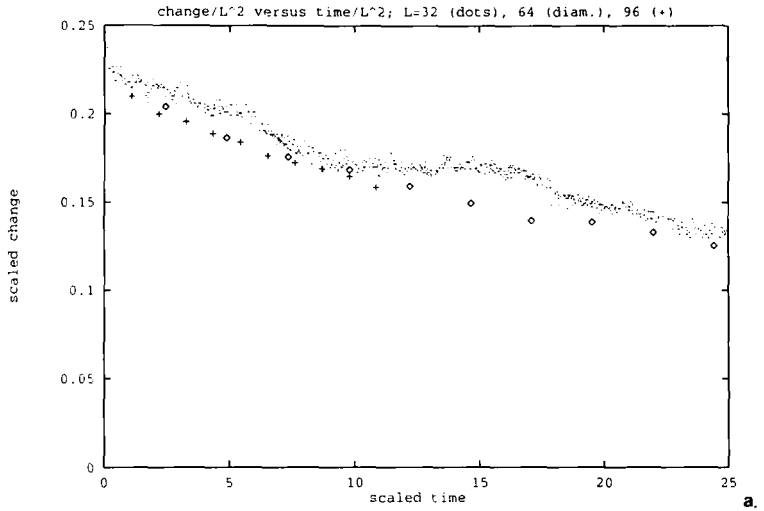


Fig. 3. Fraction of changing sites versus p; this parameter is called the internal homogeneity by Kauffman. Time t increases from diamonds ($t = 200$) over plusses ($t = 1000$), squares ($t = 10{,}000$), crosses ($t = 100{,}000$), to triangles (one million time steps). In most cases, the spins stopped changing already before $t = 10^6$ was reached.



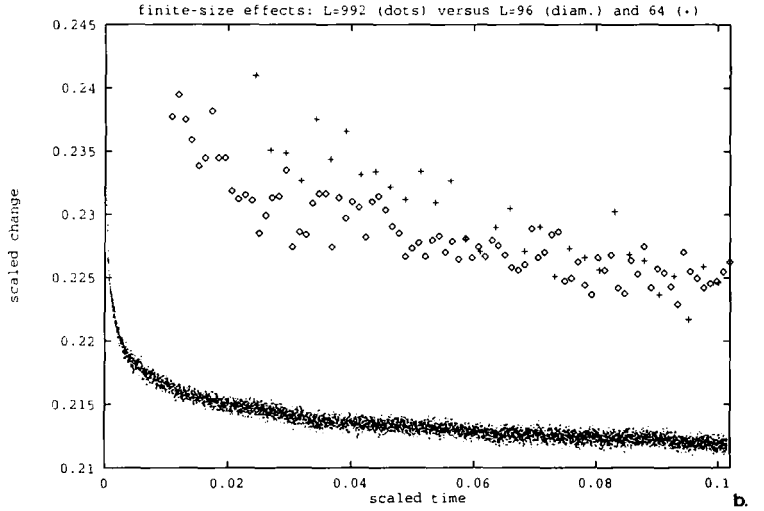


Fig. 4. Comparison of different sizes $N = L * L$ of the square lattice. The scaled change is the number of changing sites, divided by N, and is plotted versus the scaled time t/N. (a) Comparison of $L = 32$ with $L = 64$ and $L = 96$, (b) comparison of the two latter sizes with $L = 992$; for $L = 1472$ at $t/N = 0.046$ the change is 0.217.

fitness starts already close to its optimum. For large p (near 1/2) the fitness gain after 100,000 time steps is small, since the relaxation time is too large. Thus only at intermediate p, around the critical point $p = 0.31$, do we get a fast and large gain in fitness.

One may question if 100,000 time steps should be described as a "fast" fitness evolution. However, nature does not operate like a workstation, but treats genes in parallel. Mutations may occur independently of each other in different parts of the genome. Then our model does not decribe the whole set of genes, but only a small part within which the probability of two simultaneous mutations is negligibly small: With 10^5 genes active in the human genome, the 10^5 time steps $= 10^4$ mutations for our 10^3 genes in Fig. 2 correspond to only ten mutations per gene, which is no longer a huge requirement for the number of steps needed to find an optimal fitness.

In other words, biological time should be proportional to t/N and not just to the number t of iterations of the lattice of $N = L*L$ spins, in this mutation algorithm. Indeed, Fig. 4 plots the fraction of changing sites s versus t/N, and now we see that different lattice sizes give nearly the same results.

Similar results are obtained for the two-dimensional Edwards–Anderson spin glass with $\pm J$ interactions, recently optimized[7] by another Darwinian procedure. Now the "fitness" is merely the negative interaction energy per

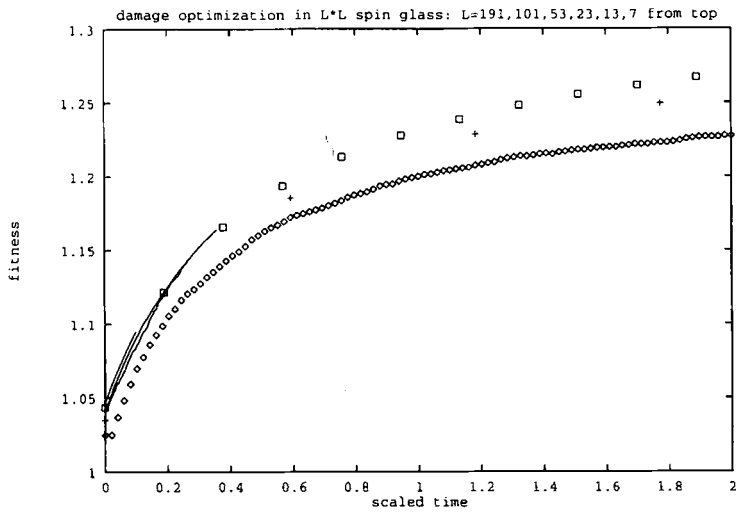


Fig. 5. Comparison of different sizes for the square-lattice spin glass. The fitness now is the negative interaction energy and is plotted versus the scaled time t/(number of spins).

site in units of J and could vary between -1 and 1. First we flip a spin only if this flip lowers the energy and soon arrive at a fixed point (local fitness optimum near 1.047) (Fig. 5). The mutations through damaging one spin increase this fitness toward 1.35 (for $t = 50{,}000$ Monte Carlo steps per spin in a $53 * 53$ lattice), about the same as the fitness reached in ref. 7 for this ground-state energy. In contrast to the Kauffman model, this zero-temperature spin glass always reaches a fixed point after a few iterations.

Thus this computer simulation found what nature found billions of years ago, that damage spreading due to mutations is a possible way in which evolution and selection of the fitter mutant can work. Surprisingly, optimization by damage spreading leads to *ideal* fitness = 1 in the Kauffman model, if we wait long enough. But for our model the edge of chaos is not a sharply defined critical phenomenon in the optimization results.

ACKNOWLEDGMENTS

I thank N. Jan and T. S. Ray for encouraging discussions and the Canada Council for support.

NOTE ADDED IN PROOF

Results similar to the two-dimensional Edwards–Anderson spin glass were obtained for the three-dimensional system (M. Cleary, private communication).

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Communicated by W. Klein