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Self-Organization of Aging in a Population Approaching the Steady State

T. S. Ray[1]

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The nonequilibrium asymptotic dynamics of a model for aging in a population of individuals initially having a random distribution of survival rates is studied. The model drives itself toward a steady state, and the average age tends toward a well-defined value. An analytic derivation shows that the average age of the members of the population decays in a power law fashion with the leading term of order t^{-1}. Monte Carlo simulations agree with the analytic work, and show that the t^{-1} decay is universally observed even when somatic mutations are introduced into the population.

KEY WORDS: Self-organizing; nonequilibrium dynamics; aging; mutations.

1. INTRODUCTION

Systems far from equilibrium exhibit many surprising features in their dynamics. For example, in recent years many systems have been studied which organize themselves into a steady state where both spatial and temporal properties exhibit power law behavior.[1-3] Novel ideas such as self-organized critically have been proposed to describe their general class.[4]

There are also numerous systems which drive themselves toward a steady state but where the final state does not appear to be critical in the usual sense. They nevertheless follow a power law in time as they approach the steady state. Examples of such systems are found in fields ranging from surface roughening and chemical kinetics to models of formation of distributions of galactic clusters.[5-8]

[1] St. Francis Xavier University, Department of Physics, Antigonish, Nova Scotia B2G 1C0, Canada.

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Another system of the latter type arises in the field of population biology. This is a model for aging in a population of a particular species.[9-11] The model leads to a Malthusian (i.e., exponential) growth for the population of the form $\exp(rt)$. Here t is the time and r is a real-valued function of general properties of the genome. The function r is called the "fitness." The significance of r is that it provides a measure of the increase of the number of individuals having a particular genome relative to other members of the populaton. The general properties of the genome which serve as the independent variables for the fitness factor are the probabilities for survival of the individual from one stage of life to the next.

A simple version of the aging model[12] results when only three discrete phases of life are assumed: infancy, juvenile, and adulthood. The fitness r is, in this case, a function of two independent variables: the survival probability J of an individual from babyhood to juvenile age and the survival probability A from juvenile to adulthood. Partridge and Barton[12] recently proposed coupling J and A in the form $J + A^4 = 1$. Physically, this condition is meant to model the hypothesis that a large amount of energy expended as a young individual to reach the juvenile stage of life exhausts its intrinsic resources so as to decrease the probability of reaching adulthood. Mathematically, the number of independent variables is reduced to one, and an optimum nontrivial fitness factor r_c results.

In the above modified version of the aging model, individuals having the maximum fitness factor r_c will gradually become more numerous than those with different r values. The population as a whole tends toward a steady-state growth rate equal to $\exp(r_c t)$.

This paper is concerned with the approach to the steady state. The relevant quantity is the average age (or survival rate) of the population from the juvenile stage to adulthood denoted by $\langle A(t) \rangle$. It will be shown that the system drives itself toward the steady-state value A_c by means of a power law in time. The mathematical framework of the model will be given in Section 2. The analytic derivation of the asymptotic dynamics will be developed in Section 3 for a broad range of physically interesting scenarios. Section 4 contains Monte Carlo studies of the model. Monte Carlo studies of the steady-state properties have been explored in the work of Stauffer and Jan.[13]

2. DESCRIPTION OF THE AGING MODEL

Consider a species where the stage of life for a given individual is classified according to three distinct categories: baby, juvenile, and adult. When an individual is born, it is classified as a "baby" and remains so for one generation. A baby survives it's first generation of life with a probabil-

ity J. If an individual lives through it's first generation, it enters the second stage of life called the "juvenile" stage. If a baby does not survive, it is eliminated from the population.

A juvenile lives through an additional generation with a survival probability A. If an individual survives it's second generation of life, it is classified as an "adult." If it does not survive, it is eliminated from the population. An adult is never allowed to survive a third generation.

Both adults and juveniles are able to produce new offspring. The offspring inherit the values of the survival probabilities J and A directly from their parents. The "fecundity" is defined as the average number of offspring born to a given individual. In general, the fecundity for the juveniles m_j is different than that for the adults m_a. The present work considers the special case $m_j = m_a = 1$. Babies cannot produce new offspring.

Given that they survive childhood, the average age of individuals having an adult survival probability A will be $1 + A$ generations. By convention, only A is used as a measure of age rather than $1 + A$. The interesting quantity which is studied in the present article is the value of A averaged over the entire population, denoted by $\langle A \rangle$.

If one assumes a large population so that fluctuations due to its finite size may be neglected, standard population dynamics can be used to solve the model.[12] According to the above prescription for the model, the following coupled equations govern the evolution of a subset of the population having the same J and A values:

$$\begin{aligned} a_{n+1} &= A j_n \\ j_{n+1} &= J b_n \\ b_{n+1} &= j_{n+1} + a_{n+1} \end{aligned} \tag{1}$$

Here, n is a discrete time index measured in generations. The variables b_n, j_n, and a_n denote the number of babies, juveniles, and adults in the nth generation having survival probabilities J and A. The fecundities for both juveniles and adults have been set equal to one.

A solution of the above equations is

$$\begin{aligned} a_n &= a_0 e^{rn} \\ j_n &= j_0 e^{rn} \\ b_n &= b_0 e^{rn} \end{aligned} \tag{2}$$

where the fitness r is given by[12]

$$r = \ln\left[\frac{J}{2} + \frac{J}{2}\left(1 + 4\frac{A}{J}\right)^{1/2}\right] \tag{3}$$

and the constant factors are

$$a_0 = \tfrac{1}{2} A N_0/(e^r + A)$$
$$j_0 = \tfrac{1}{2} e^r N_0/(e^r + A) \tag{4}$$
$$b_0 = N_0(J, A)/2$$

The function $N_0 = N_0(J, A)$ is the initial number of individuals in the population with survival probabilities J and A.

Partridge and Barton[12] have assumed an additional coupling between the survival probabilities: $J + A^4 = 1$. As mentioned in the introduction, this coupling is meant to model the idea that a high survival probability J implies that individuals must use up intrinsic biological resources to ensure a good chance of reaching the juvenile stage of life, and that as a result the probability of reaching adulthood is decreased. The condition reduces the number of independent variables to one, which is chosen as J for the rest of the present work. The fitness factor r then has a nontrivial maximum value r_c for some J_c where $0 < J_c < 1$. The individuals having a survival probability J_c will eventually dominate the population, so that the average adult survival probability will tend toward the steady-state value $A_c = (1 - J_c)^{1/4}$.

In addition, two types of mutations are allowed in the population. The first type is called a "somatic" mutation. This mutation affects the survival of an individual, but it is not passed on to any of the individual's offspring. It is a model for external influences such as background radiation which can lead to early death through the possibility of cancer or other diseases. The resistance of an individual to somatic mutations is given by the magnitude of J and A. The mutations are modeled by effective survival probabilities given by

$$J_{\text{eff}} = J \exp(-\varepsilon q_j)$$
$$A_{\text{eff}} = A \exp(-\varepsilon q_a) \tag{5}$$

Here, ε is an external parameter taken to be small. The variables q_j and q_a are the number of mutations which the individual suffers while trying to survive from babyhood to juvenile and from the juvenile stage to adulthood, respectively.

The second type of mutation is called the "hereditary" mutation. It models the variation in the J values passed on to offspring by the parents. Mathematically, hereditary mutations are detemined by adding a quantity

to the survival probability from the parent so that the J value for a particular offspring is given by

$$J_{\text{offspring}} = J_{\text{parent}} + \varepsilon c \tag{6}$$

where c is a stochastic variable and $-1 < c < +1$.

3. THEORY FOR THE ASYMPTOTIC DYNAMICS

It is possible to derive an analytic expression for the asymptotic dynamics in the case where there are neither somatic nor hereditary mutations in the population. The general features of the dynamics carry over to the full model where mutations are included. Consider a population where the J values are initially distributed according to the function $f_0(J)$. The initial distribution is assumed to be reasonably smooth so that it may be differentiated as many times as necessary. This is mainly done to facilitate the analytic treatment and it is somewhat unrealistic for a finite population where $f_0(J)$ is not even continuous. However, the long-time behavior of the dynamics in simulations of the model (where the population must be finite) is not particularly sensitive to this fact, as will be seen in Section 4.

Let us replace the discrete measure of time in generations n by a continuous time variable t. The average survival probability $\langle A \rangle$ as a function of t is of the form

$$\langle A(t) \rangle = N(t)\, A(J)\, f_0(J)\, e^{rt}\, dJ \tag{7}$$

where the normalization constant $N(t)$ is given by

$$N(t) = \left[\int_0^1 f_0(J)\, e^{rt}\, dJ \right]^{-1} \tag{8}$$

When t is large, one can use a saddle point approximation to write the exponential factor in a form accurate to the third power of $J - J_c$:

$$\exp(rt) \approx \exp\left\{ \left[r(J_c) + \frac{1}{2!} r''(J_c)(J - J_c)^2 \right] t \right\} \times \left[1 + \frac{1}{3!} r'''(J_c)(J - J_c)^3\, t \right] \tag{9}$$

The terms $r'(J_c)$, $r''(J_c)$, etc., denote the derivatives of the fitness [Eq. (3)] evaluated at the maximum value J_c. The limits of integration in both integrals of Eq. (7) may be replaced by $\pm\infty$ in accordance with the same

approximation. The saddle point approximation used here is slightly different than the usual one in that the cubic term in $(J-J_c)$ is retained. This is necessary so that all terms to leading order in t emerge from the calculation.

Both the initial distribution $f_0(J)$ and the function $A=(1-J)^{1/4}$ may also be expanded around J_c. The original expression for $\langle A(t)\rangle$ then simplifies to a sum of integrals of the form

$$\int_{-\infty}^{+\infty} x^n e^{-ax^2}\,dx \tag{10}$$

where $a=1/2\,|r''(J_c)|$. The fact that r has a maximum value at J_c ensures that $r''(J_c)<0$.

In principle, the asymptotic dynamics can be calculated from any smooth initial distribution. To illustrate the features of the dynamics, let us choose a uniform initial distribution $f_0(J)=c_0$ where c_0 is a constant. The resulting form for $\langle A(t)\rangle$ is then

$$\langle A(t)\rangle \approx A_c - \left(\frac{3}{32\,|r''(J_c)|\,A_c^7} + \frac{r'''(J_c)}{8A_c^3 r''(J_c)^2}\right) t^{-1} + \mathcal{O}(t^{-2}) \tag{11}$$

According to this analysis, the asymptotic dynamics results in a power law decay for $\langle A(t)\rangle$ of order t^{-1} to the steady-state value A_c. The amplitude of the leading t^{-1} term will generally depend upon the initial distribution $f_0(J)$, but the power law itself is quite universal. It is, however, possible to choose $f_0(J)$ carefully so that the t^{-1} term vanishes leaving a t^{-2} dependence.

When somatic mutations are included, the values of J_c and A_c will change, but the power law form of the asymptotics will remain as in Eq. (11). Quite generally, as long as the fitness factor has a well-defined nontrivial maximum, the decay to steady state will go as t^{-1}.

Hereditary mutations, on the other hand, will change the asymptotic dynamics. Mathematically, the power law arises from the narrowing of the distribution of J values as time increases (without hereditary mutations). The distribution tends toward a delta function centered at J_c. If hereditary mutations are present, the distribution cannot become infinitely sharp, due to their "smearing out" effect. This means that the system will exhibit power law behavior until the width of the distribution is of the order of the size of the hereditary mutations. At that time, the power law will be cut off sharply, probably in an exponential fashion. Interestingly, although the steady-state distribution turns out to be symmetric with respect to J_c, the asymmetry of the function $A=(1-J)^{1/4}$ will result in a steady-state value for A not equal to A_c.

4. MONTE CARLO SIMULATIONS

In order to explore the aging dynamics and to check the analytic work in the preceding section, populations governed by this Partridge–Barton model were simulated using the Monte Carlo technique (see, e.g., ref. 14). A population of individuals having a uniform initial distribution of J values was first generated. The simulations proceeded by going through the population and updating it once every new generation. The survival of a baby was determined by selecting a random number and comparing it to the J for that particular individual. If the number was less than J, the baby became a juvenile, otherwise it was discarded. Similarly, a juvenile's survival to adulthood was determined by the comparison of a random number with the A value for that particular juvenile. Any adults of the previous generation were eliminated from the population.

After running through the population and updating the life stage of all individuals, new babies were introduced into the population. Every juvenile and adult produced an average of one baby per individual. The actual number of babies was determined by sampling an exponential distribution. The value of J assigned to each baby was inherited directly from the parent. If hereditary mutations were included in the simulation, the value of J was modified by adding to or subtracting from it a small constant multiplied by a random number between -1 and $+1$ taken from a uniform distribution.

Somatic mutations were incorporated into the simulations in the following manner. First, an exponential distribution was sampled to obtain the number of mutations q that a given individual would suffer. For babies trying to become juveniles, the average number of mutations $u = \langle q_j \rangle$ was set externally, usually to a value 10. The survival probability J for the baby was then reduced by a factor $\exp(-\varepsilon q)$, where ε is a small, positive constant (0.01–0.001). When a juvenile was trying to become an adult, the analogous procedure was followed with the adult survival probability A. The average number of mutations $v = \langle q_a \rangle$ was typically chosen to be several times the average value for the babies ($v = 16u$), as is customary.[12]

The results of a simulation where the initial distribution of J values was chosen to be uniform and neither type of mutation was included are shown in Fig. 1. This is a double-logarithmic plot of the average adult survival rate minus the steady-state value $\langle A(t) \rangle - A_c$ versus time. The dashed line is the theoretical asymptotic behavior as obtained from Eq. (11). The agreement past about the 70th generation appears to be excellent.

Figure 2 shows the simulation results where both somatic and hereditary mutations have been included. The stationary-state value has

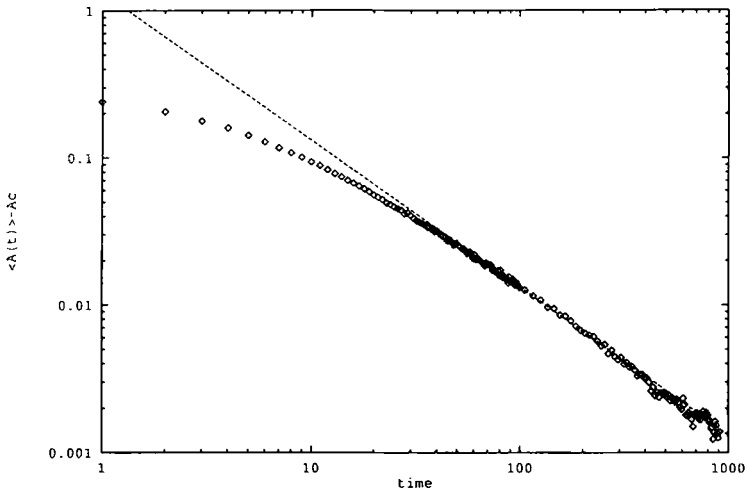

Fig. 1. Average A of members in a population versus time without mutations. The dashed line is the theoretical asymptotic behavior as computed from Eq. (11).

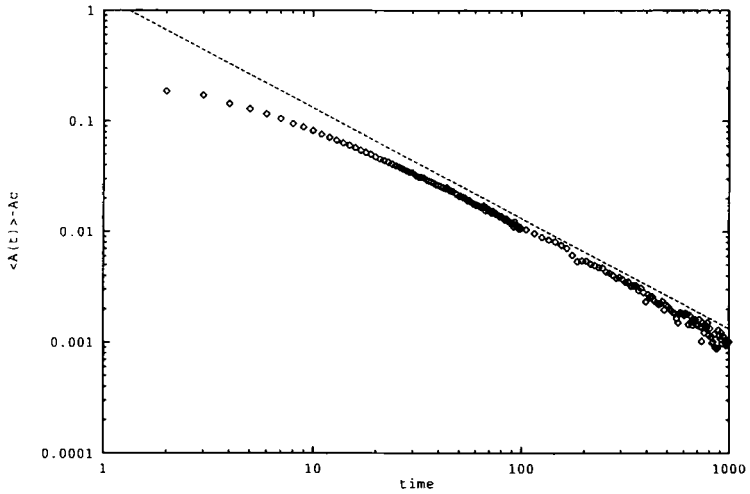

Fig. 2. Average A of members in a population versus time with somatic mutations where $\varepsilon = 0.001$, $u = 10$, and $v = 160$. For the hereditary mutations, $\varepsilon = 0.0001$. The dashed line is the theoretical asymptotic behavior for a population *without* mutations.

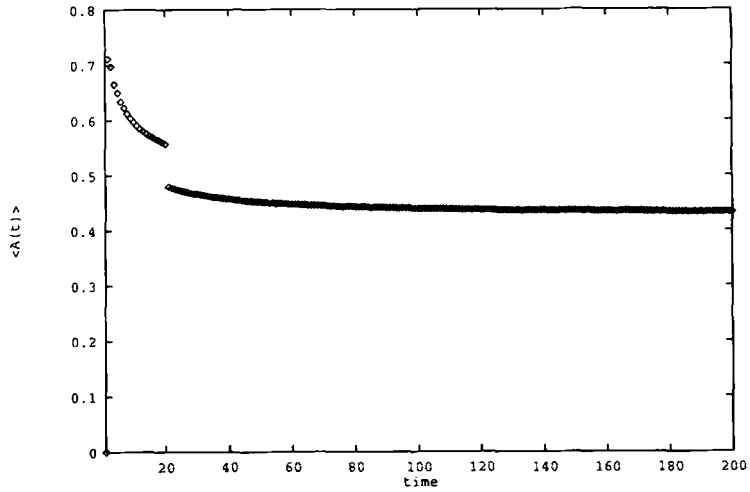

Fig. 3. Average A of members in a population versus time. Up to $t = 20$, there are no mutations. For $t > 20$, mutations are turned on with $\varepsilon = 0.001$, $u = 10$, and $v = 160$.

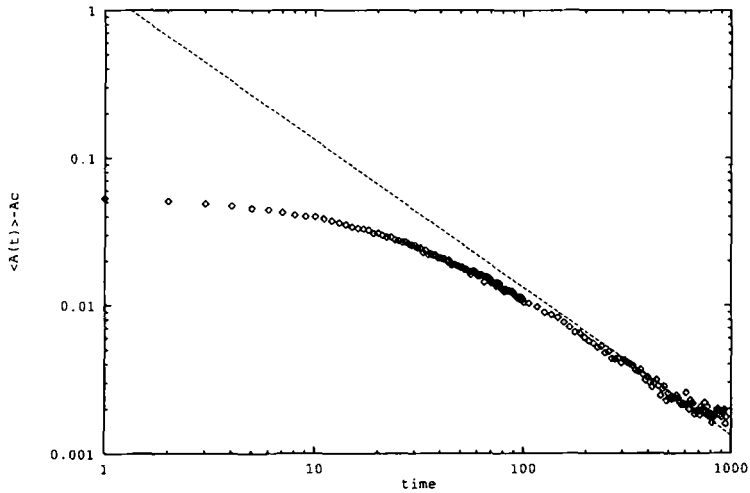

Fig. 4. Data from Fig. 3 with the time origin shifted to the 20th generation. The dashed line is proportional to t^{-1}.

changed noticeably. The asymptotics still give a t^{-1} decay to steady state, although the amplitude is slightly different (15–20%). The simulation was not run along enough to see the cutoff in the power law due to the hereditary mutations.

Figure 3 shows how the average adult survival rate behaves when somatic mutations are suddenly introduced into the simulations at a given time. This could model, say, the sudden increase in mutation rates due to the partial degradation of the ozone layer in the upper atmosphere. The resulting increase in the level of ultraviolet radiation causes an increased incidence of somatic mutations possibly leading to malignant skin cancer. In Fig. 4, the same data are plotted on a double-logarithmic plot after subtracting the final steady-state value and shifting the time origin to the point at which the somatic mutations were introduced. The same asymptotic plot as in Figs. 1 and 2 is included to show a t^{-1} behavior. Again, the asymptotics show a t^{-1} power law decay. At large times, the data seem to indicate premature saturation, which is probably caused by the finite size of the system.

5. DISCUSSION

The universality of the t^{-1} decay law in the asymptotic dynamics is evident in all of the simulation data. The analytic theory presented in Section 3 seems to capture most of the observed behavior of the model, even though it is an idealization of the true discrete model. The generality of the analytic arguments suggests that virtually any coupling between J and A which results in a nontrivial optimum fitness factor for $0 < J < 1$ will produce the t^{-1} decay.

In a more realistic model, the population will have additional environmental pressures so as to contain the exponential growth. For example, a limit in the amount of food available for consumption or a limited living space may cause the population to saturate to a given size. As long as the external pressures act in a manner which is independent of the J distribution, this should not affect the asymptotic dynamics. The important aspects of the model lie in the relative differences of the survival rates of individuals dictated by their particular J values. Anything which causes a global nondiscriminating reduction of the population will not affect the overall domination by individuals having J values close to J_c. In fact, the simulations are run in precisely this manner: when the population contains too many members for the computer memory to handle, the population is reduced by removing individuals at random.

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NOTE ADDED IN PROOF

For different biological experiments see E. Nieschlag, S. Nieschlag, and H. M. Behre, *Nature* **366**:215 (1993) and C. Kenyon, J. Chang, E. Gensch, A. Rudner, and R. Tabtiang, *Nature* **366**:461 (1993).

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