Chaotic Population Dynamics and the Evolution of Aging

Proposing a Demographic Theory of Senescence

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Abstract

According to accepted evolutionary theories, aging has evolved as a side-effect of strong selection pressure for early fertility, despite the fact that it has no adaptive value of its own. I have argued elsewhere that recent experimental results make these theories untenable, and that there is now a broad array of evidence indicating that aging has evolved as an adaptation, selected for its own sake. To explain nature's preference for aging is a substantial theoretical challenge. The classical Weismann hypothesis, "making room for the young," fails because the benefit to the population accrues in the form of enhancement to the rate of increase of population average fitness, while the cost affects individual fitness directly and efficiently. In multi-level selection models, the aging genes are lost before their benefit can accumulate. I propose here that aging has evolved based on a different benefit: its contribution to demographic homeostasis. I argue that population dynamics are inherently chaotic, and that the stable ecosystems that we commonly observe in nature are a highly evolved phenomenon. Natural selection for population homeostasis is far more efficient than selection for rate of evolution because chaotic population dynamics can be lethal on a time scale of just a few generations, while enhanced rate of evolution takes far longer to affect population mean fitness. My thesis is that aging can evolve based on its ability to damp population fluctuations. For illustration, I offer an individual-based computational model that reproduces chaotic population dynamics with a delayed-feedback logistic equation. Genes for aging emerge handily.

Introduction

Two classic dilemmas

The problem of altruism is an area where computational modeling has made a decisive contribution to evolutionary theory. Historically, there have been two classic cases of extreme altruism, where fundamental understanding has been elusive, and substantial controversy remains. These are reproductive restraint and the evolution of aging. They constitute "extreme" altruism in the sense that the cost to the individual is high and direct, while the benefit to the population is far too diffuse to be accounted for by kin selection. They are open questions because theory has robustly predicted the impossibility of effects that field biologists claim are commonplace.

Population self-regulation. Do individuals temper their reproductive potential in order to stabilize population swings and avoid exhausting food species on which they depend? Field biologists have collected extensive documentation of this effect (Wynne-Edwards 1962; Nudds 1987; Kolenosky 1972). Yet prevailing theoretical arguments dismiss the possibility. Williams (1966) argued persuasively that altruistic populations would be susceptible to invasion by selfish individuals. This reasoning is widely accepted to this day. But recently, computer modelers and complexity theorists have demonstrated mechanisms by which reproductive restraint might evolve (Pepper & Smuts 2001; Rand *et al* 1995; Haraguchi & Sasaki 2000; Rauch *et al* 2003; Pels *et al* 2002). All these models have confirmed a thesis that had been meticulously demonstrated in an early monograph by Gilpin (1975), which remains widely unappreciated. Because of the communication barrier that insulates the biological community from the complexity community, the prevailing wisdom in the former has not yet adapted.

My own experience (Mitteldorf *et al* 2002) as well as these published accounts has convinced me that it is not difficult to model the evolution of reproductive restraint in an ecological context.

Evolution of Aging is an area where a theoretical consensus has prevailed, but a broad array of experimental data has emerged in contradiction to that theory.

Once again, the theory is founded on the primacy of individual selection. Since aging detracts unambiguously from individual fitness, it must have evolved as a side-effect, via *pleiotropy*, or genetic linkage. The pleiotropic theories come in two flavors, both invoking tradeoffs between longevity and fertility. The original flavor (Williams 1957) is based on direct genetic tradeoffs. The more recent flavor is the Disposable Soma theory (Kirkwood 1977), based on metabolic tradeoffs, apportioning a scarce resource (food energy) between demands of reproduction and the cellular repair and maintenance that is the basis for longevity.

I have recently amassed evidence against these theories, and made the case that substantial experimental evidence compels us to consider an adaptive theory, in which aging has been selected for its own sake (Mitteldorf 2004). There are four major lines of evidence (as well as several lesser lines):

- Laboratory animals bred for longevity fail to show depressed fertility. (Leroi *et al* 1994) This is direct evidence against the hypothesis of tradeoffs.
- Some mechanisms of aging appear to be conserved over vast stretches of evolutionary time. (Guarente & Kenyon 2000) Maintenance of these mechanisms in the genome implies that the mechanism has served a purpose.
- In caloric restriction experiments, animals evince the

ability simultaneously to forestall aging and increase stress resistance and immune function, even while under dietary stress. (Weindruch & Walford 1986) Why should the body withhold its best efforts to forestall aging when it is unstressed and well-fed?

- Genes have been discovered in wild populations of mice, worms and flies that appear to have no other function than to hasten the progress of senescence. When such genes are knocked out or artificially disabled, experimental animals live longer, and without apparent cost. (Migliaccio *et al* 1999; Dillin *et al* 2002; Lin *et al* 1998) Why has natural selection failed to eliminate such genes?

The need for a new theory of aging

These observations demand an adaptive theory of senescence. But whereas modeling the evolution of population regulation has shown itself to be suprisingly straightforward, no comparable model for the evolution of aging has emerged. What makes modeling the evolution of senescence so difficult?

Since the pioneering work of Weismann (1889), proposed mechanisms for the evolution of aging as an adaptation have been based on a benefit to the group described as "making room for the young." When this effect is fully elaborated (as Weismann never did), it takes the following form:

Mortality rates for young organisms in the wild are elevated by competition with adult conspecifics, which are generally larger and stronger, (and sometimes smarter and more experienced). Aging drains the population of strong adults, so that more of the young can advance to maturity. Therefore, a population that knows aging has a higher turnover rate, and will adapt more nimbly to changing environmental conditions. In addition, aging tempers the advantage of the more fit over the less fit, enhancing population diversity, which also contributes to the adaptability of the population, and enhances the rate of evolutionary change.

The cost of aging is forgone reproductive opportunity. Models can be constructed in which the population-level advantage is pitted against the individual cost, and these models invariably fail. Genes for aging reliably diminish in frequency, and are quickly extinguished from the population. This is my experience, in five years of trying more than a dozen distinct ideas; and it is attested by the fact that there are no published computational models that evolve aging (while there are several that evolve reproductive restraint.)

The reason that aging is not able to get a toehold in the population is that its costs act directly and quickly against individual fitness, while its benefits accrue on a much longer time scale. The costs of aging affect the reproductive success of any individual that carries an aging gene; but the benefits accrue on an evolutionary timescale, and are spread widely over the evolving population. Genes for aging must persist and dominate a population while

- First, the population grows gradually more diverse
- Then the greater diversity leads to better gene combinations
- These gene combinations grow in prevalence and the population as a whole becomes more competitive, relative to neighboring groups
- Finally, this change in fitness must prove decisive in group-wise competition that drives competing populations to extinction.

For all this time, the aging population must be protected from invasion by freeloaders that do not carry the aging gene, and would rapidly take over the population if they had the chance.

I have found it impossible to construct a plausible model with these characteristics, and I now understand why.

How population dynamics changes the picture

The above argument is predicated on the assumption that aging carries a high cost to the individual, because aging must evolve in a context where there is intense competition for individuals reproductive rate. But enforced reproductive restraint can change that context. If Gilpin and his successors are correct, individual reproductive rate is not maximized. It cannot be maximized, because high reproductive rates lead to chaotic population dynamics that are fatal to the group.

In a context where individual reproductive rate is not maximized, it becomes plausible to evolve senescence as an adaptation. At the least, a *weak hypothesis* would be that the cost of aging can be offset 100%, by pairing higher aging with higher fertility. (This hypothesis gives new meaning to fertility/longevity tradeoffs, and stands pleiotropy on its head.) At best (*strong hypothesis*) aging can make its own unique and valuable contribution to taming the dragon of chaos.

Logistic Population Dynamics

Dynamics of the time-delayed logistic equation

The logistic equation is the oldest and simplest dynamic model of a population limited by finite resources.

$$\frac{1}{x}\frac{dx}{dt} = b(1 - x/K)$$

where $(1/x)\ dx/dt$ is the logarithmic population growth rate, b is the maximal growth rate in the absence of intraspecific competition, and K is the steady state population level. For x that is small compared to K, the solution exhibits exponential growth; and for $x >> x_{ss}$, the solution declines exponentially. It is well-known that populations governed by the logistic equation are extremely well-behaved: x approaches K asymptotically from either above or below, without overshooting (Abrams 2000).

But the logistic equation is equally prominent in another context entirely: as a difference equation, it is frequently used to study dynamic chaos. The behavior of the logistic equation with finite time increments may be either smooth or chaotic, depending on the size of Δt. For small Δt

(compared to the timescale $1/b$), the behavior is very much like the differential equation; for larger Δt, there are cycles in which x overshoots x_{ss}, and if Δt is increased further, the behavior undergoes a transition to dynamic chaos, such that x jumps wildly about from one time step to the next (Bar-Yam 1997).

Fig 1 shows solutions to the logistic difference equation, starting with very small x, for Δt=1, 2, 2.5 and 2.99. As Δt approaches 3 from below, dynamics become increasingly chaotic. For Δt>3, solutions tend to negative infinity exponentially fast.

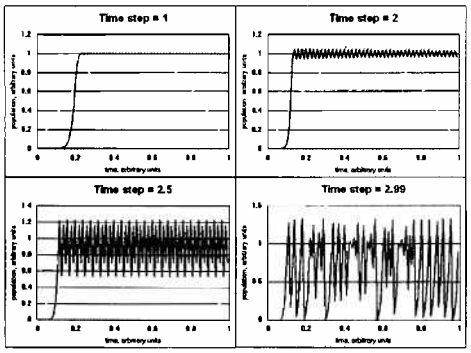

Fig 1. Logistic dynamics, for four values of Δt. The series illustrates the approach to chaos at Δt=3.

Of course, natural population systems cannot afford this kind of dynamic; they would soon fluctuate to extinction. But from a theoretical perspective, the behavior with finite Δt may be a more plausible model for the behavior of real populations than the smooth differential version. Hence it is reasonable to hypothesize that nature has maintained strong selection pressure at the group level to keep population dynamics out of the chaotic regime.

The reason that we find a predominance of stable ecosystems in nature is not that population dynamics are inherently stable, but that demographic homeostasis has been the target of intense selection.

Ways to stabilize population dynamics

If we believe that stable population dynamics is a major target of natural selection, what kinds of adaptations may nature be expected to have found? All such adaptations have a direct, negative impact on individual reproductive value. In this sense, there is a substantial tradeoff between individual and group levels. But while r (reproductive value) must needs be lowered, it is not necessary to lower K (steady-state population level) in order to stabilize dynamics; on the contrary, higher numbers are generally associated with less volatility and less risk of extinction.

(Benton and Grant (2000) make a compelling case that K is a meaningful measure of fitness while r is not; but invocation of r as a proxy for fitness is deeply ingrained in the culture of population genetics. Many of the evolutionary models invoked in the ALife community implicitly reward r.)

We may assume that nature has found the way to population stability that has the least cost, measured by some combination of group and individual fitness. But it is not easy for us to gauge the cost of nature's various options. Birth rates have a direct impact on the effective Δt. In fact, the parameter that determines approach to chaos is the product of the birth rate and the time delay. Lowering the birth rate b offers the fringe benefit of lowered resource requirements, shrinking the environmental footprint.

Lower birth rate decreases r proportionately. The impact on K is less clear. If you think of K as the carrying capacity of a niche, it may seem to be independent of b; but if you imagine the death rate to be a function determined by the set of all environmental and crowding conditions, you may equally well conclude that K is directly proportional to b. It is probably safe to say that reality is somewhere in between. There are two lesser reasons, however, that nature may choose to keep birth rates high. One is population diversity, and the impact on population adaptability in the face of a changing environment. The second is the insurance against accidents that larger numbers provides.

Raising the death rate is *not* a path to demographic stability. In the logistic equation, the only death term is proportional to crowding, and raising the death rate is equivalent to lowering K. Thus decreased K leads to the same population dynamics in a smaller population – not at all a winning proposition. You can, of course, add an "accidental death" term that is independent of population density. This is equivalent to lowering b; in fact, the b that appears in the logistic equation is really the net population growth rate, or difference between birth and accidental death rates. Increasing the accidental death rate rather than decreasing the birth rate avoids the two "lesser reasons" but also misses the resource savings described above.

Senescence operates differently from accidental death, and has a special advantage. When population density is low and expanding freely, the death rate from competitive forces is low, so many individuals live to an age where senescence matters. So in times of population expansion, aging effectively tempers the growth rate. But in tightly competitive times, when the population is high and contracting, starvation and resource scarcity will prevent most individuals from attaining an age at which senescence becomes a life-limiting condition. So as a force for demographic stability, aging has the potential to damp population growth when it is too fast, but then to get out of the way when populations are shrinking and its action would be counterproductive.

In the real world, we find an additional adaptation associated with senescence that further improves its utility as a stabilizer of population dynamics. Most animals are able to lessen the effects of senescence in response to caloric deprivation. This is the "Caloric Restriction effect" (CR), that has been observed in the laboratory for a wide range of species, from yeasts to primates (Weindruch &

Walford 1986; Masoro 2002). The CR adaptation further enhances the effectiveness of senescence for avoiding unstable dynamics. The death rate from senescence is at its lowest under the conditions of starvation that attend the contracting phase of the population cycle. Aging does not reduce ***r***, and arguably has minimal impact on ***K***.

Thus, if nature has sought a least-cost path to demographic homeostasis, it is understandable that she has recruited senescence as a useful device.

Description of the Model

To illustrate the operation of chaotic population dynamics, and the opportunity to evolve aging, I have implemented a multi-level selection model. An asexual population is arrayed on an n*n grid of sites, (opposite edges identified to avoid boundary effects). A small rate of migration links each site with its four von Neumann neighbors. Within each site is a variable population of individuals (I call them "orgs") that reproduce clonally, with a birth rate that determines the probability of reproduction in each time step. The probability per time step of individual death is modeled as the sum of two terms: One term derives from aging, and increases exponentially with the age of the org after maturity (a Gompertz (1825) function). The other term derives from crowding, and is proportional to the number of orgs sharing the site.

The crowding variable that determines the death rate at each site is measured with a time delay. In other words, the death term in the logistic equation is proportional to the site population a number of timesteps in the past. The time delay is crucial to the model, because it makes the difference between a logistic equation that approaches steady state smoothly and a population dynamic that is powerfully unstable.

The delay feature mimics an attribute of real ecosystems. Population growth can have a momentum that continues even after resources on which the population depends have been depleted.

Principal parameters of the model

grid size: length and width of the population grid (typically 16*16)

maturity: the age before which an org is not yet able to reproduce, and senescence has not yet become effective. (typically the reciprocal birth rate)

delay: population levels feed back to death rates, after this delay (typically 50 timesteps, corresponding to a "chaos parameter" of 2.25)

K=steady-state population: the denominator in the term that invokes death from crowding; the number of orgs at each site to which the system would relax with stable dynamics. (typically 100)

migration: the rate at which orgs from one site diffuse to a random neighbor site. (typically 3E-5)

mutation rate: the probability that an aging gene will randomly change its value during reproduction. (typically 3E-2)

The model supports an option for **smart aging**, emulating the CR adaptation. When this option is invoked, aging is muted in response to (delayed) crowding.

Heuristic dynamics of the model

Within each site, selection rewards the orgs with genes to create the most offspring, i.e., high birth rates and low rates of aging. But high individual birth rates and low aging rates are a recipe for chaotic population dynamics, leading to extinction. When the system is allowed to evolve globally, a steady-state is established in which individual growth parameters skirt the edge of chaos. Sites in which individual selection has taken the growth too high are constantly blinking out of existence, then re-seeded by migrating orgs from sites where the growth rate is lower (on average).

Experiments with the model

Three kinds of experiments were programmed:

1. Population dynamics were calibrated with non-evolving orgs. In the absence of aging, the maximum sustainable birth rates were recorded.
2. The grid was populated with orgs that carry a gene that determines their individual rate of aging. The aging gene is permitted to mutate, and its evolved distribution was subsequently measured. Evolved rate of aging was plotted as a function of programmed birth rate.
3. Orgs were given two evolving genes, one of which determines individual birth rate and the other individual rate of aging. Birth rate and aging rate were permitted to evolve independently.

Since population dynamics can be stabilized either by moderating the birth rate or by increasing the rate of aging, this latter option allowed for direct competition between these two solutions to the chaos problem.

Model Results

1. Demonstration of population dynamics

In this calibration experiment, **delay** was fixed and different (constant) values of **b** were initialized on the grid, with no aging and no evolution. Values of **b** that were too high led to global extinction. The maximum viable **b** was found to be inversely proportional to **delay** for a given **K** parameter. Small **K** per site increased the risk of extinction, leading to lower values of maximum viable **b**. But this effect saturated for large values of **K**. Unlike the drift-and-dominate effects described in Wright's (1931) shifting balance theory, the effect of chaotic population dynamics is not limited to small sub-population sizes.

The straight lines in this plot are an indication that the chaos parameter, (**time delay** * **birth rate**), is the operational determinant of the limits of growth. In deterministic

models, dynamics becomes chaotic as this parameter approaches 3. In the results below, (from a stochastic model) the slopes of the lines range from 1.8 for the smallest population per site to 2.5 for the largest.

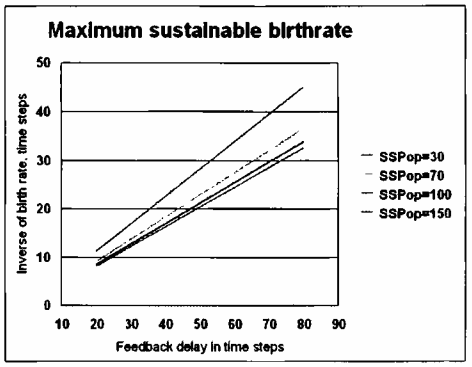

Fig 2. The chaos parameter (time delay * birth rate) determines stability of population dynamics. Higher **K** (steady-state population per site) permits a slightly higher chaos parameter.

2. Evolution of aging when birth rate is fixed

Rate of aging was programmed by an individual gene that was permitted to evolve through 10^6 time steps, attaining a steady state. The terminal value depends on the fixed value of ***b***: the higher the birth rate, the more aging is required to stabilize population dynamics.

In Fig 3, a family of three curves is plotted for different values of the migration parameter. The migration parameter is important because a very high value of migration is equivalent to a single panmictic population, while a very low value corresponds to n^2 independent panmictic populations.

The migration parameter programs the relative importance of group selection vs. individual selection. This is the "rescue effect": if migration is high, then extinct sites are quickly repopulated from neighboring sites, while low values of migration impose a higher cost for extinction. In the present results, lower values of migration are associated with slightly higher evolved rates of aging. (For the lowest value of migration and highest values of ***b***, extreme volatility extinguished the global population before a stable level of aging could be established.)

All deaths in the model are either caused by crowding or by aging. The y axis in Fig 3 corresponds to the proportion of all deaths attributable to aging, a dimensionless quantity that might be crudely compared to the impact of aging in nature. The proportion of aging deaths ranges up to 35% in the model results. Measuring the corresponding parameter for real ecosystems is a field biologist's nightmare, and data are scarce. Bonduriansky and Brassil (2002) estimate that the fitness cost to antler flies in the wild is about 20%.

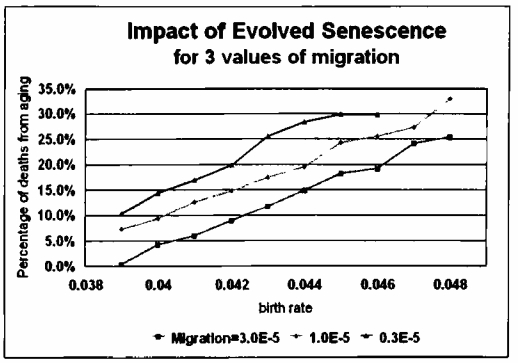

Fig 3. Birth rate is fixed, and rate of aging is allowed to evolve for 10^6 time steps. Larger fixed birth rates necessitate higher rates of aging for stability. The three series correspond to different values of the migration parameter.

3. Simultaneous evolution of aging and birth rate

In these runs, each individual carried two genes, determining its rate of aging and its birth rate, respectively. In principle, the chaos problem could be solved either with a high birth rate and a high aging rate, or a low birth rate and low aging rate.

Fig 4 below is a scatter plot placing each of ~10^4 individuals on a map according to their evolved birth rate (x axis) and aging rate (y axis). Always a compromise was reached, where both moderated birth rate and substantial aging contribute to demographic stability.

Two separate runs are superimposed in the scatterplot. In one of the runs, aging was programmed to be "smart" in the sense of the CR response. In the smart aging run, the effect of aging was moderated during times of high (delayed) population density when, presumably, animals would be stressed by tight food supplies. The figure shows how smart aging shifts the center of the distribution toward higher values of aging, and correspondingly higher values of ***b***.

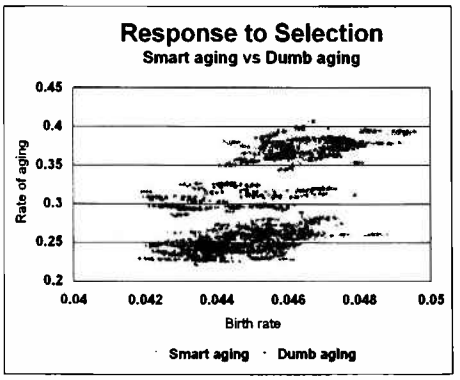

Fig 4. Scatter plot of evolved individuals by birth rate (x) and aging rate (y). With "smart aging" (CR response) turned on, aging is preferred over low birth rate as a solution to the chaos problem.

Summary and conclusions

Experimental results demand an adaptive theory of aging. There has been a century of ideas on the subject, but no quantitative models in which senescence evolves as an adaptation have emerged. Considerations of demographic homeostasis and chaotic population dynamics may be able to bridge this gap, and provide a plausible mechanism by which senescence may be selected.

It would be dishonest to minimize the radical revision in thinking that I am proposing. The idea that natural selection may not be optimizing individual reproductive value casts a shadow on a great body of evolutionary theory. This theory enjoys wide acceptance, although much of its foundation is untested and therefore vulnerable, because complex real-world ecosystems so seldom provide opportunities for clean tests of simple hypotheses.

Perhaps 95% of evolutionary biologists think that individual selection is all the selection that there is. 5% have argued bravely that group selection can sometimes overcome the more direct effect of individual selection.

In this landscape of opinion, I am staking out a position on the far left wing, claiming that evolution not just of populations but of entire ecosystems is the rule rather than the exception. The imperative for demographic homeostasis is a primary agent of natural selection, rivaling reproductive potential in its import.

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