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Chaotic population dynamics and the evolution of ageing

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ABSTRACT

Problem: Genetic and demographic studies suggest that ageing is an adaptive genetic program, but population genetic analysis indicates that the benefit of ageing to the group is too slow and too diffuse to offset its individual cost.

Premise: Demographic homeostasis is a major target of natural selection at the group level, with a strength that can compete with the imperative to higher individual reproductive value.

Hypothesis: Ageing has evolved based on its contribution to stabilizing population dynamics, helping prevent population growth overshoot, exhaustion of ecological resources, and local extinction.

Model: Asexual individuals carrying a mutable ageing gene are tracked on a geographic grid with slow migration between neighbouring sites. Birth rate is constant; death probability for individuals is the sum of two terms: (1) a logistic crowding term proportional to the local site population and (2) a Gompertz ageing term, in which mortality increases exponentially with age at a rate that is governed by the individual's gene. The logistic crowding term is computed with a time delay that simulates the momentum of population growth, and causes solutions to undergo chaotic population fluctuations if net growth rates are excessive.

Results: Within each site, individual selection pushes life spans progressively longer. Once life spans have increased at a site, its population may fluctuate to extinction. Shorter-lived individuals re-seed it from neighbouring sites. The result is a dynamic steady state in which ageing is selected without pleiotropy.

Keywords: ageing, altruism, chaos, group selection, population dynamics, senescence.

INTRODUCTION

Standard theories of evolution regard senescence as an epiphenomenon of selection, with no adaptive value of its own. The reasons for this are deeply rooted, not in empirical data, but in the axioms and methods of population genetics. If individual reproductive value is taken as a surrogate for fitness, and selection is assumed to operate incrementally on gene frequencies, then it follows that the contribution of ageing to fitness is wholly negative, and therefore that ageing cannot be selected for its own sake.

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In stark contrast to theoretical expectations, field studies and genetic experiments indicate that ageing *has* been selected for its own sake. The classic theories for the evolution of ageing are untenable in light of experimental findings of the last two decades, and ageing must be regarded as an adaptation (Mitteldorf, 2004).

But what adaptive value can be associated with ageing? By definition, the effect on individual fitness is negative. A century ago, Weismann proposed that ageing exists to 'make room for the young'. This hypothesis is not vacuous and can be translated to specify an explicit mechanism. But it is an unworkable model. The mechanism operates far too slowly and its benefits are too diffuse to compete with the direct cost exacted at the individual level.

I propose that ageing has been selected for its ability to help stabilize population dynamics. In theoretical systems (e.g. Lotka-Volterra), populations are liable to violent swings and chaotic fluctuations, risking extinction. My hypothesis is that demographic homeostasis is an adaptation, an evolved trait of populations, and perhaps of entire ecosystems. This is an attractive candidate for a theory because: (1) population dynamic fluctuations can be swift and lethal, and frequent local extinctions have the potential to help account for strong group selection (e.g. Pels *et al.*, 2002; Pepper and Smuts, 2002); and (2) population dynamics operates outside the framework of population genetic theory, which has been the basis for doubts that ageing can be adaptive.

The mechanism for evolution of ageing that I have modelled operates as follows: Natural selection at the individual level pushes birth rates inexorably higher, until the population's growth rate approaches three times the ecosystem's rate of recovery. Chaotic population dynamics inevitably ensue. (The transition to chaos in mathematical systems tends to be a bright line rather than a gradual progression.) This becomes a collective problem, a tragedy of the commons that can never be redressed with individual selection. The extinctions that follow will be as frequent as necessary to overpower individual selection and enforce growth restraint, bringing the system back from the brink of chaos.

The emergence of ageing as an adaptation fits comfortably within this picture. Ageing would become one of the mechanisms by which a species can take control of its death rate, suppressing violent fluctuations that might otherwise cause extinctions. Ageing (together with reproductive and predatory restraint) would help hold the growth rate down below the chaotic threshold.

As an agent of demographic stability, ageing may offer specific advantages over restrained reproduction. Ageing takes its biggest toll when the population is expanding too rapidly, and adds only marginally to mortality rates when the population is collapsing from starvation. Keeping the birth rate high also helps with genetic diversity and adaptability in an unpredictable environment.

This is a new theory in the sense that the proposed basis on which ageing evolves is unrelated to Weismann's idea. It is an adaptive theory, as demanded by experiment. The proposal entails group selection, as it must, but the mechanism operates on a time scale that can compete with individual selection.

I present a computational model that tracks individual organisms, and invokes no assumptions about the relative strengths of group and individual selection. Individuals compete with each other in a Cartesian network of weakly linked populations, and within each site those individuals that age less rapidly contribute more offspring to each successive generation. Hence life span is always increasing within each site. However, in sites where individual selection has pushed the life span up, population growth rate is higher, population dynamics is more volatile, and extinctions occur more frequently. The system as

a whole evolves towards a dynamic compromise: The population growth rate is kept in check, and ageing helps to dampen population volatility.

EXPERIMENTAL EVIDENCE FOR AGEING AS AN ADAPTATION

I have set out the experimental case for regarding senescence as an adaptation in a companion article (Mitteldorf, 2004). I summarize here the principal arguments:

- The genetic basis of ageing has been conserved over vast spans of evolutionary history (Guarente and Kenyon, 2000).
- Pleiotropic theories demand a direct causal link between fertility trade-offs and the evolution of ageing. The mechanism cannot work if these trade-offs are a subtle or inconsistent effect. But many studies have failed to detect trade-offs (Stearns, 1992), while others have found that longevity can be increased by breeding or by direct genetic manipulation without apparent cost.
- Single 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 disabled (with RNA interference), experimental animals live longer, sometimes without apparent cost (Lin *et al.*, 1998; Migliaccio *et al.*, 1999).
- *Hormetic* phenomena[1] attest to the metabolic ability to extend life span in the face of environmental challenge, suggesting the interpretation that shortened life span in the absence of challenge is a programmed choice (Calabrese and Baldwin, 1998).
- Mortality rates at late ages have low additive genetic variance, which is often regarded as the signature of an adaptation (Curtsinger *et al.*, 1992).
- Late mortality plateaus are difficult to reconcile with theories of ageing based on the declining force of natural selection with age (Pletcher and Curtsinger, 1998).
- There are suggestions that the adaptive mechanisms of *apoptosis*[2] and replicative senescence (via shortened telomeres) may have been co-opted into the ageing programs of higher organisms (Longo *et al.*, 2005).
- There is evidence that the prompt death that characterizes semelparous organisms may not be tightly linked to a burst of reproductive activity, but appears to be an adaptation in its own right (Crespi and Teo, 2002).

The credibility of the present theory, or of any adaptive theory of ageing, depends on this interpretation of experiment. It makes little sense to question long-held methods of evolutionary theory without a compelling empirical impetus. For the remainder of this paper, I shall assume that the experimental case for adaptive ageing is established.

TOWARDS A NEW THEORY OF AGEING

Ideas from the past, and why they are inadequate

Senescence is not a side-effect but a full-blown adaptation. Others have noted as much, and have proposed theoretical frameworks in which ageing might evolve (Bowles, 1998; Skulachev, 1997;

[1] Hormesis is a response of an organism to environmental stress in which, paradoxically, mortality and morbidity are decreased.
[2] Apoptosis is a programmed, orderly process by which a cell destroys itself and recycles its contents as nutrients.

Clark, 1999; Goldsmith, 2004). But whereas possible mechanisms have been described in heuristic terms, no computational model for the affirmative selection of ageing has appeared in the literature. [Exceptions for two specialized circumstances: Travis (2004) demonstrates that altruistic death can be selected in a spatially structured population, only after individuals have already been weakened by senescence. Fabrizio *et al.* (2004) describe a computational model for altruistic apoptosis in a compromised yeast population.] What makes modelling the evolution of senescence so difficult?

Since the pioneering work of Weismann (1889), mechanisms for the evolution of ageing as an adaptation have been proposed based on a benefit to the group described as 'making room for the young'. When this effect is articulated and translated (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. Ageing continually drains the population of strong adults, so that more of the young can advance to maturity. Therefore, a population that knows ageing has a higher turnover rate, and will adapt more nimbly to changing environmental demands. In addition, ageing tempers the advantage of the more fit over the less fit, enhancing diversity, which also contributes to the adaptability of the population and enhances the rate of evolutionary change.

Models can be constructed in which these advantages are pitted against the direct individual costs of ageing. These models invariably fail. Alleles for ageing reliably diminish in frequency, and are quickly extinguished from the population. This is the prediction of classical population genetic theory. It is also my (unpublished) experience with more than a dozen models; and it is attested by the fact that there are no published computational models of the Weismann mechanism.

The reason that ageing is not able to get a toehold in the population is that its costs act directly and quickly against individual fitness, whereas its benefits are dispersed in time and relatedness. The costs of ageing affect the reproductive success of any individual that carries an ageing allele, but the benefits accrue on an evolutionary time scale (much slower), and are spread widely over the evolving population. Alleles for ageing 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 neighbouring groups; and
- finally, this change in fitness must prove decisive in group-wise competition that drives competing populations to extinction.

For all this time, the ageing population must be protected from invasion by freeloaders that do not carry the ageing allele, and would rapidly take over the population if they had the chance. No plausible model can provide for this. Thus the Weismann hypothesis fails, and so must any theory based on a benefit to the population's *rate of evolution* balanced against a cost in the *life history* of the individual. The benefit is too slow to overtake the cost.

This conclusion will come as no surprise to theorists.

Clues from predator–prey models and population self-regulation

If experimental evidence really demands that ageing has been affirmatively shaped by natural selection, where else can we seek an adaptive value for ageing?

A key insight derives from consideration of a parallel strain in the theory of strong altruism: demographic stability and population self-regulation. Despite a history of scepticism in this field, a number of recent computer models have demonstrated the potency of population dynamics as an evolutionary force (Rand *et al.*, 1995; Haraguchi and Sasaki, 2000; Mitteldorf *et al.*, 2002; Pels *et al.*, 2002; Pepper and Smuts, 2002; Rauch *et al.*, 2003). All these models have confirmed a thesis that had been meticulously demonstrated in an early monograph by Gilpin (1975): If a population of predators increases too rapidly, it can deplete its prey species on a very short time scale, with serious results for the predator population. In fact, population dynamics can be sufficiently swift and lethal as to compete effectively with individual selection, which always favours higher growth rates.

Ageing in the context of population dynamics

Difficulties in accounting for evolution of ageing as an adaptation are connected to the high costs borne by the individual: it has been assumed that ageing must evolve in the midst of an intense competition for individual reproductive potential (r). But reproductive restraint can change that context. If Gilpin and his successors are correct, r of predators is not maximized because mismatch between the reproductive potentials of predator and prey leads to chaotic population dynamics that are fatal to both populations.

In a context where r is not maximized, it becomes plausible for senescence to evolve as an adaptation. Ageing can make its own uniquely valuable contribution to taming the dragon of chaos, since it has the potential to curb the growth rate during population expansion without adding appreciably to the death toll from starvation while the population is in decline. Here is how:

The problem of volatility may be described as a tendency for a population to grow too rapidly when conditions are favourable, and to collapse too precipitously when resources are scarce. The effects of ageing on a population in its growing phase are to limit life span, to diminish the number of offspring per individual lifetime, and thus to suppress the rate of population growth. But when the same population is in collapse, most animals are dying of starvation. Few live to be old enough for old age to contribute to their demise. Thus ageing exacts its greatest toll when the population is freely expanding, but does not add appreciably to the rate of decline when the population is collapsing.

Operation of this mechanism is enhanced by the caloric restriction adaptation. Most animals are able to mitigate the impact of senescence in response to caloric deprivation. This has been observed in the laboratory for a wide range of species, from yeasts to primates (Masoro, 2002; Weindruch and Walford, 1986). The caloric restriction adaptation further enhances the effectiveness of senescence for modulating unstable dynamics, since it pushes the death rate from senescence even lower under the conditions of starvation that attend the contracting phase of the population cycle.

MODELLING THE MECHANISM

The model tracks individuals and their progeny in a structured population, which allows for local extinctions. Individuals are associated with a particular location and have limited

mobility. The model described herein is based on explicit groupings with limited between-group migration; a 'viscous' model, based on cellular automata in a continuous geometry is described elsewhere (Mitteldorf, 2002).

Unlike the Weismann hypothesis, the demographic theory does not depend on sexual reproduction. And unlike models based on genetic drift, chaotic population dynamics do not depend on small numbers of individuals per site. [Although the risk of extinction is greater with small numbers per site, the risk rises slowly (logarithmically) as the population per site increases. This is a property of chaotic dynamics: population overshoot collapses exponentially with time.]

Predator–prey interactions would be one way to introduce population fluctuations into the model, but that would add the complication of tracking two (or more) separate species. I have chosen instead to model population dynamics with a logistic equation and a finite time step. The logistic equation links the fates of all individuals within a site, in that their individual mortality rates are proportional to the total site population. Choice of the logistic equation is doubly appropriate because it is a standard model in two fields: population dynamics, with an infinitesimal dt, and chaos theory, substituting a finite Δt. The present model combines features of the two.

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, and the paradigm of *K*-selection.

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

where the left-hand side is the logarithmic population growth rate, r 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 with K, the solution exhibits exponential growth; and for large x, the solution declines towards K. 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: transformed into a difference equation, it is frequently used to study dynamic chaos. The behaviour of the logistic equation with finite time increments may be either smooth or turbulent, depending on the size of Δt. For small Δt (compared with the time scale $1/r$), the behaviour is very much like the differential equation; for larger Δt, x overshoots K, and if Δt is increased further, the behaviour undergoes a transition to dynamic chaos, such that x jumps wildly about from one time step to the next (Bar-Yam, 1997).

Figure 1 shows solutions to the logistic difference equation, starting with very small x, for $\Delta t = 1$, 2, 2.5 and 2.99. As Δt approaches 3 from below, dynamics become increasingly chaotic. For $\Delta t > 3$, solutions tend to negative infinity exponentially fast; but in practice, finite populations risk extinction well before $\Delta t = 3$.

Of course, natural population systems cannot afford to be chaotic; they would soon fluctuate to extinction. But from a theoretical perspective, there is no intrinsic reason why predator populations should be able to avoid instability (Rosenzweig, 1973).

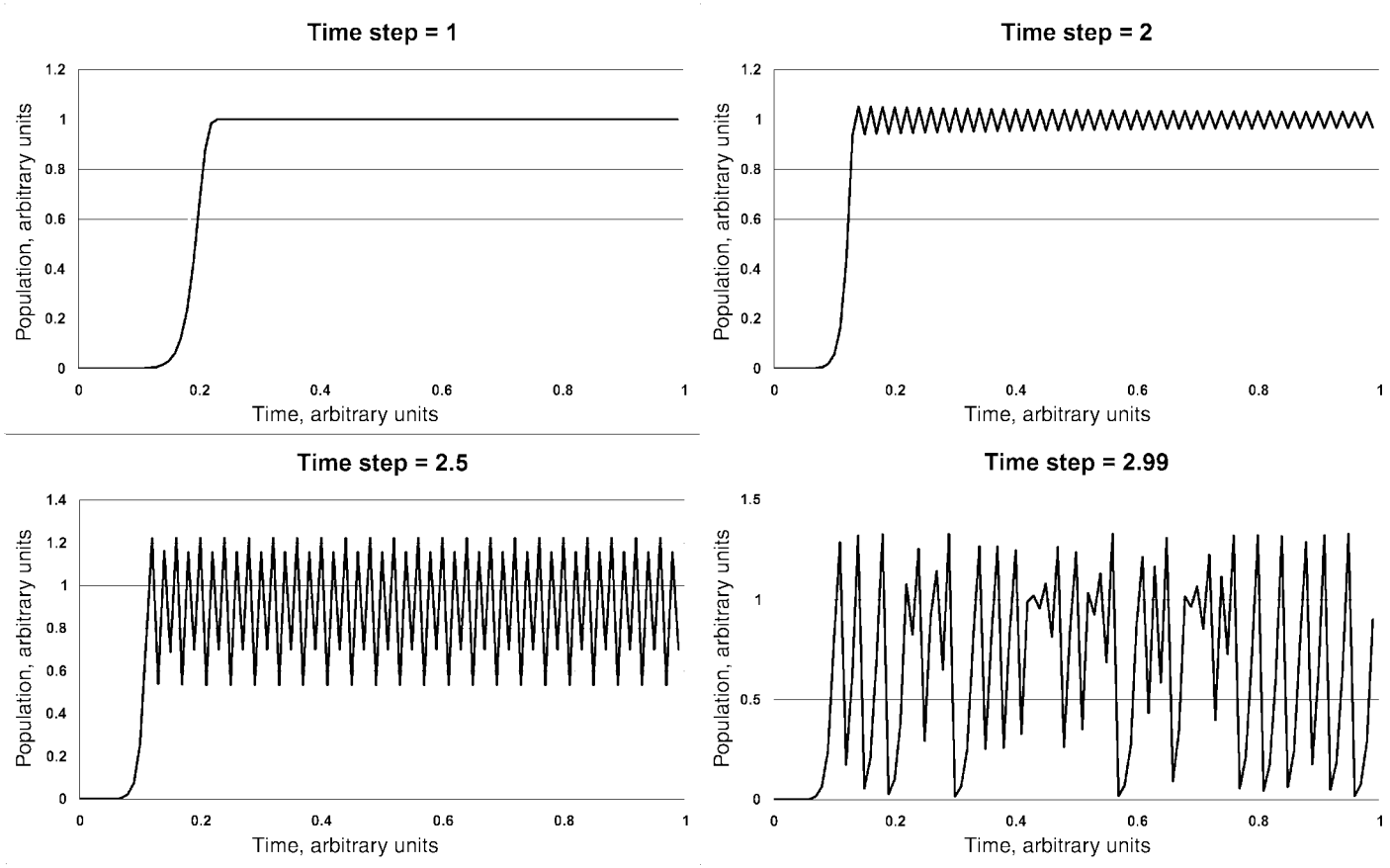

Fig. 1. Logistic dynamics, for four values of Δt. The series illustrates the approach to chaos at $\Delta t = 3$. Note the rapid onset of chaotic behaviour when Δt exceeds 2.5. (For $\Delta t > 3$, population drops to zero, with no chance of recovery.)

If individual selection pressure causes birth rates in a population to rise while the recovery time of its environment remains fixed, then feedback about the maximum sustainable population level is effectively delayed. This is tantamount to increasing Δt in the logistic equation.

We assume that nature has maintained strong selection pressure at the group level to keep population dynamics out of the chaotic regime. If so, the reason that we find persistent ecosystems in nature is not that population dynamics is 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, how might nature have achieved this end? Suppressing chaos entails a substantial negative impact on individual reproductive value. The stage is thus set for a trade-off between individual and group selection levels. But while r (reproductive value) must be lowered, it is not necessary to lower K (steady-state population level) to stabilize dynamics; on the contrary, higher values of K are generally associated with less volatility and less risk of extinction.

Birth rates have a direct impact on the effective Δt. In fact, the parameter that determines the approach to chaos is the product of the birth rate and the time delay. Lowering the birth rate r offers the fringe benefit of lowered resource requirements, shrinking the environmental footprint. There are two reasons, however, why 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 other is the insurance against accidents that larger offspring numbers provide.

Indiscriminately 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. Decreasing K leads to the same population dynamics in a smaller population – not at all a winning proposition. Adding an 'accidental death' term that is independent of population density is equivalent to lowering r; in fact, the r that appears in the logistic equation is really the net population growth rate, or difference between birth and accidental death rates.

Senescence operates differently from accidental death, and has distinct advantages as a regulator of population, described above ('Ageing in the context of population dynamics'). If nature were to seek a path to demographic homeostasis with relatively low cost to individual fitness, she could plausibly recruit senescence as a useful tool.

DETAILED SPECIFICATION OF THE MODEL

An asexual population is arrayed on an $n \times n$ grid of sites (opposite edges identified to avoid boundary effects). A small rate of migration links each site with its four nearest neighbours. Within each site is a variable population of individuals with a probability of reproducing clonally in each time step. Birth rate in some versions is a constant, and in others is governed by a gene. The probability per time step of individual death is modelled as a combination of two terms: one term derives from ageing, and increases exponentially with age of the individual after maturity [a Gompertz (1825) function]; the other term derives from crowding, and is proportional to the total population occupying each site. In the present model, it is

assumed that the probabilities of death from senescence and crowding (starvation) simply add. This is a conservative assumption. More realistically, the combined probability of death from two causes is less than the sum of the two separate probabilities, and this fact reduces the individual cost of ageing, thus permitting adaptive ageing to evolve more readily. I hope to relax this assumption in future versions of the model.

The crowding variable 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 time steps in the past. The time delay is crucial to the model, closely analogous to the parameter Δt. For zero time delay, the logistic dynamic approaches steady-state smoothly, but for $\Delta t \times$ [population growth rate] $\gtrsim 2$, population dynamics become too volatile to be sustainable.

Time delay mimics an attribute of real ecosystems. Population growth can continue even after the resources on which the population depends have been depleted. The availability of ecological resources that enable the population to grow and expand depends on the population's history of exploiting these resources in the past.

Principal parameters of the model

- *Grid size*: length and width of the population grid (typically 16×16).
- *Birth rate* (r): the probability of an individual spawning an offspring in each time step. In some runs, this was specified, and in others it was controlled individually by an evolving locus.
- *Maturity*: the age before which an individual is not yet able to reproduce, set to the reciprocal of birth rate. This same parameter appears in the Gompertz function as the age at which the mortality rate begins to increase.
- *Delay*: population levels feed back to death rates, delayed this number of time steps into the future (typically 50 time steps, corresponding to a 'chaos parameter' of 2.25).
- *Steady-state population per site* (K): the denominator in the term that invokes death from crowding; the number of individuals at each site to which the system would relax with stable dynamics (typically 100).
- *Migration*: the rate at which individuals from one site diffuse to a random neighbour site (10^{-5}).
- *Mutation rate*: the probability that an ageing gene will randomly change its value during reproduction (3×10^{-2}).

Note that as in most evolutionary simulations, we track a small population with a large mutation rate to keep the model computationally tractable (Krzanowski, 2001).

Heuristic dynamics of the model

Within each site, selection rewards the individuals that leave the most offspring – that is, those with high birth rates and low rates of ageing. But when the population's overall growth rate exceeds the logistic time lag by a factor of about 2, chaotic population dynamics quickly lead 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 driven the growth too high are constantly blinking out of existence,

then being re-seeded by migrating individuals from sites where the growth rate is lower (on average).

MODEL RESULTS

Calibration and validation

Population dynamics in the model were calibrated with non-evolving individuals in a single grid site, all individuals contributing equally to a common crowding measure. At a given value of *delay*, there is a maximum birth rate that can be sustained without causing the population to fluctuate to extinction. The *delay* parameter was fixed and different (constant) values of birth rate r were specified, with no ageing and no evolution. Values of r that were too high led to global extinction. The maximum viable r was found to be inversely proportional to *delay* for a given K parameter. The constant product $r \times delay$ is the model's representation of the parameter Δt, the 'chaos parameter'. This product is the slope of the lines in the plot, and the fact that the lines are straight is an indication that the chaos parameter is the operational determinant of the limits of growth (Fig. 2).

For large values of K, the critical chaos parameter is about 2.5 in the model, close to the maximum value 3 in the (deterministic) difference equation. For smaller K, there is additional stochastic behaviour resulting from the uncertainty of small numbers, and the critical chaos parameter is only 1.8 for $K = 30$.

Unlike the drift-and-dominate effects described in Wright's (1931) shifting balance theory, the effect of chaotic population dynamics does not depend on small sub-population sizes. Small K per site increases the risk of extinction, leading to lower limits on r; but as K increases, the maximum viable r saturates at a finite value.

Evolving ageing when birth rate is fixed

Sites on a Cartesian grid were populated with individuals carrying a gene that determines their individual rate of ageing. The ageing gene was permitted to mutate, and its evolved

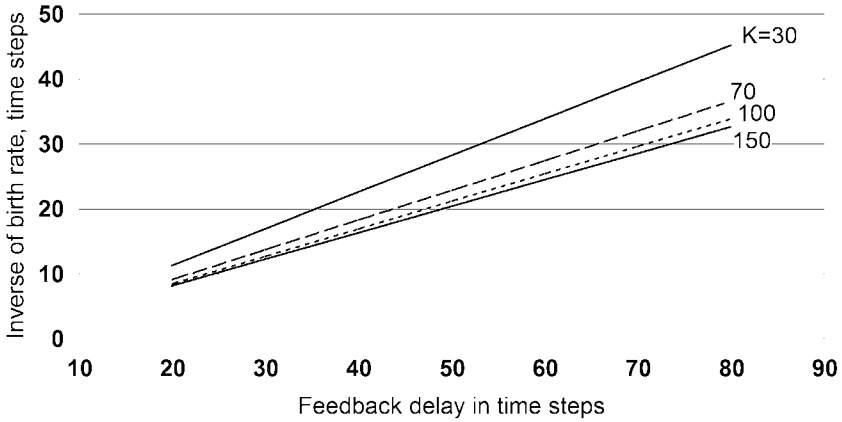

Fig. 2. Birth rates that are too high lead to global extinction. The maximum permissible birth rate is inversely proportional to the time delay Δt in the model. Hence the graph of $(1/r)$ versus *delay* is a straight line. The slope of each line is the constant product $r \times delay$, which is the 'parameter' of the model.

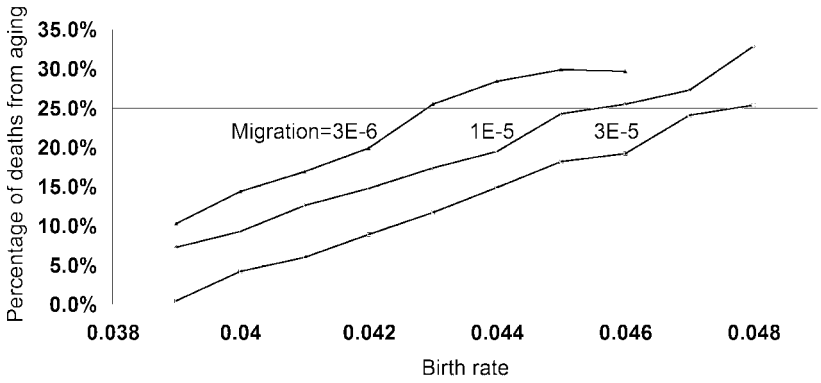

Fig. 3. Birth rate is fixed, and rate of ageing is allowed to evolve for 10^6 time steps. Larger fixed birth rates necessitate higher rates of ageing for stability. The three series correspond to different values of the migration parameter.

distribution was subsequently measured. Rate of ageing 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 r: the higher the birth rate, the more ageing is required to stabilize population dynamics. Average evolved rate of ageing was charted as a function of programmed birth rate. A family of curves was generated for different values of migration.

In Fig. 3, a family of three curves is plotted for different values of the migration parameter. The migration parameter programs the relative importance of group selection versus individual selection. If migration is high, then extinct sites are quickly repopulated from neighbouring 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 ageing. (For the lowest value of migration and highest values of r, extreme volatility extinguished the global population before a stable level of ageing could be established.)

This experiment demonstrates the principle that, with birth rate fixed, ageing can be selected as an adaptation to moderate population fluctuations. The caveat is that the birth rate must be fixed 'too high' to see this result. The model result suggests that the predator's problem of chaotic population dynamics can be solved by moderating birth rate, by limiting the life span with senescence, or a combination of the two. Above, I have offered qualitative arguments for the particular advantages of ageing as a population regulation device. In future versions of this model, I hope to simulate direct evolutionary competition between organisms that curb their ecological footprint by limiting birth rate to organisms that accomplish the same end by means of senescence.

SUMMARY AND DISCUSSION

I have proceeded from the assumption that there is a robust and diverse array of experimental results, which, taken together, demonstrates that ageing is an adaptation. Yet, adaptive ageing is implausible in the context of classical population genetic models, where allele frequencies are tracked incrementally.

To resolve this dilemma, I propose a population dynamic theory, which I call the 'demographic theory of ageing'. Individual selection pushes birth rates inexorably higher, until population growth outpaces the ability of the underlying ecosystem to recover. The onset of chaotic population dynamics is sudden and powerful. A small increase in birth rate can push the system over the edge into a chaotic regime where a population may fluctuate to extinction in just a dozen generations.

Classical literature examines isoclines to determine the stability of predator–prey interactions (e.g. Rosenzweig and MacArthur, 1963). Because this approach uses differential equations, it relies on an implicit assumption that populations change at a rate slow compared with a single life span. The chaotic regime explored here is characterized by more rapidly changing populations – comparable to or faster than the individual's life span. This regime is entered when individual selection pushes the predator to sufficiently high reproductive efficiency. Then group selection grows as strong as it needs to be to oppose further increases in the population growth rate.

We speculate on this basis that demographic homeostasis has been a major target of natural selection. The persistent ecosystems that we commonly observe are the result of a group selection process. This hypothesis can resolve the paradox of adaptive ageing. Any combination of decreased birth rate and increased senescence may suffice to restrain r, but ageing offers its own particular benefit towards stabilization of population dynamics.

The hypothesis that demographic homeostasis is part of nature's definition of 'fitness' may also offer an answer to a broader paradox: Why is it so easy to breed (or engineer) animals that seem to have higher individual reproductive value than those found in the wild? For example, Reznick *et al.* (2000) ask this question about *Daphnia*; Kenyon has found a similar issue in *C. elegans* (Guarante and Kenyon, 2000); and Rose's breeding experiments with *Drosophila* have produced a fly with both higher fertility and greater longevity than the wild type (Leroi *et al.*, 1994). Reznick *et al.* (2004) report that guppies collected from high-predation river pools appear to be fitter in every aspect of their life history than those from low-predation sites. It is fair to ask why there appears to be so much additive genetic variance in characteristics that contribute so directly to reproductive potential. My answer is that nature is not in the business of maximizing reproductive potential, but rather of creating robust ecosystems.

Senescence is a ubiquitous feature of the biosphere, and any explanation of its evolution must be comparably universal in scope. Is predator–prey population dynamics too specialized a principle to explain the general prevalence of ageing? Perhaps not. Abrams (2000) emphasizes the universality of predator–prey interactions. All living things are part of a food chain, and we need not think of a predator as a carnivore, or even an animal: host–parasite interactions lead to similar population dynamics as predator and prey.

A second essential feature of the present model is division of the population into competing demes, with the possibility of local extinctions and competition to re-seed sites that thus become vacant. The model seems to require particular combinations of migration rate and population growth rate to generate the results of interest. But I would argue that geography, locality of interactions, and the possibility of local extinctions are broad features of the biosphere that operate, for any given species, on a multitude of scales. If 'rescue' is too rapid at a small scale, then we are free to regard populations on that scale as a single deme. As population growth rates evolve ever higher, there will always be some geographic scale, dependent on motility, for which local resources are exhausted, the population suffers extinction, and rescue is sufficiently slow to set up an inter-demic

competition for resource management. Thus it may be that the model's requirement of multi-level selection is also a general feature of the biosphere.

Ageing may be best understood in the context of other ubiquitous traits that benefit adaptability and the pace of evolution: obligate sex, intraspecific diversity, programmable mutation rates, genomes structured to promote evolvability. All of these traits have important but very long-term benefits that accrue to a lineage or a population; none of them can evolve in the context of a fierce competition for short-term reproductive potential.

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, based on experiments *in the laboratory*. Laboratory evolution has successfully demonstrated the primary principles of population genetics within a controlled setting. It is less clear that population genetics is a good model for natural selection in the wild; complex real-world ecosystems seldom provide opportunities for clean tests of simple hypotheses. If radical new thinking can be justified, it must be because the old models fail to account for some prominent features of the biosphere. Adaptive ageing may be an example of such.

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