

Post-reproductive life span and demographic stability

Josh Mitteldorf and Charles Goodnight

J. Mitteldorf (josh@mathforum.org), Dept of Ecology and Evolutionary Biology, Univ. of Arizona, Tucson, AZ 85720, USA.

– C. Goodnight, Dept of Biology, Univ. of Vermont, Burlington, VT 05405, USA.

Recent field studies suggest that it is common in nature for animals to outlive their reproductive viability. Post-reproductive life span has been observed in a broad range of vertebrate and invertebrate species. But post-reproductive life span poses a paradox for traditional theories of life history evolution. The only commonly-cited explanation is the ‘grandmother hypothesis’, which is limited to higher, social mammals. We propose that post-reproductive life span evolves to stabilize population dynamics, avoiding local extinctions. Predator–prey and other ecosystem interactions tend to produce volatility that can create population crashes and local extinctions. Total fertility rates that exceed the ecosystem’s recovery rate contribute to population overshoot, followed by collapse. These local extinctions may constitute a potent group selection mechanism, driving evolution toward controlled rates of population growth, even when there is a significant individual cost. In this paper, we consider the question: what life history characteristics support demographic homeostasis at the least cost to individual fitness? In individual-based evolutionary simulations, we find that reduction in fertility is sufficient to avoid population instabilities leading to extinction, but that life histories that include senescence can accomplish the same thing at a lower cost to individual fitness. Furthermore, life histories that include the potential for a post-reproductive period are yet more efficient at stabilizing population dynamics, while minimizing the impact on individual fitness.

Post-reproductive life span (PRLS) is common. It is not only human females that survive beyond menopause, but also whales (McAuliffe and Whitehead 2005), elephants (Packer et al. 1998), opossums (Austad 1993), quail (Ottinger and Balthazart 1986), parakeets (Holmes and Ottinger 2003), domestic hens (Brody et al. 1923), guppies (Reznick et al. 2006), lab mice (Gosden et al. 1983), the roundworm *C. elegans* (Goranson et al. 2005), and even yeast (Minoia et al. 2005). There is evidence PRLS exists in the wild and not just in the lab (Holmes and Ottinger 2003, Cohen 2004, Goranson et al. 2005, McAuliffe and Whitehead 2005). It is likely that this list is limited by the number of species that have been studied, and that the phenomenon of PRLS is actually ubiquitous in nature (Packer et al. 1998, Cohen 2004).

The only established theories for PRLS are the ‘mother hypothesis’ (Gaulin 1980, Pechei 2001), and the ‘grandmother hypotheses’ (Blurton Jones et al. 1989, Hawkes and Blurton Jones 1997, O’Connell et al. 1999, Pechei 2001, Hawkes 2003). The mother hypothesis posits that menopause is adaptively advantageous because it limits the number of offspring a female can produce, and as a consequence increases offspring survivorship. The grandmother hypotheses posit that post-reproductive females assist their reproductive age offspring to raise their grand offspring. Different published versions vary as to whether menopause is an adaptation in and of itself, or a side effect of selection for

increased life span, and whether the individuals helped are daughters or sons; however, all share in the explanation that post reproductive life span evolves as a result of the increase in inclusive fitness that arises from helping to raise grandchildren (Pechei 2001). These ideas were originally proposed for humans only, and later were tentatively extended to other social mammals, with mixed success (Packer et al. 1998, Pavelka and Fedigan 1999, Ward et al. 2009, Herndon and Walker 2010). Since PRLS is not restricted to social vertebrates and organisms with extended parental care, these hypotheses are not applicable for many organisms with PRLS. Even for humans, with our extraordinary duration of juvenile dependence, there has been debate about whether the grandmother hypothesis alone is sufficient to explain PRLS. Kachel et al. (2011a) have published a model which suggests that the costs of adding non-fertile years are not commensurate with the fitness benefits, while Hawkes et al. (2011) finds faults with their model, and Kachel et al. (2011b) have defended the generality of their results.

Regardless of whether the explanations from inclusive fitness are adequate for humans, there remains a good deal of PRLS in non-social animals that remains unexplained. PRLS presents a challenge for any of the established theories of aging. Based on the assumption of life history tradeoffs that have come to define the field, Williams (1957) once made the prediction that ‘There should be little or no post-reproductive period in the normal life-cycle of any

species'. His reasoning was that PRLS represents a genetic cost with no offsetting fitness benefit. If, indeed, PRLS carries a significant cost in individual fitness, then attributing PRLS to an accident of history or development is not adequate; a general explanation for its existence and evolution is requisite. This benefit can only accrue to a larger selective unit than the individual. Though the hypothesis of group selection continues to suffer from a perception that it is likely to be weaker and slower than individual selection (Maynard Smith 1976), theoretical and experimental work in the intervening decades indicates that group selection may be much more common (Stevens et al. 1995), and powerful (Muir 1996, Goodnight and Stevens 1997, Swenson et al. 2000, Bijma et al. 2007) than previously considered. Although always controversial, group selection has a long history in biological thought, tracing back at least to Darwin (Borrello 2010). Current disagreements over the efficacy of group selection trace to the rejection of Wynne-Edwards's (1962) view of group selection by Maynard Smith (1976), Williams (1966) and others (Borrello 2010). Sober and Wilson (1998) cite numerous examples of group selection, and describe the reluctance of the evolutionary community to extend its theoretical paradigm to accommodate diverse evidence for the ubiquity of group selection in nature. With this in mind it makes sense to consider whether aging in general, and PRLS in particular may be an adaptation to selection acting above the level of the individual organism.

It was May (1973), who first analyzed natural populations as complex dynamical systems, and Gilpin (1975) who developed an early computational model of a simple predator/prey system, suggesting that population dynamics might lead to a swift and effective form of group selection. Thomas et al. (1980) proposed that the reason chaotic population dynamics are not frequently observed in nature is that population chaos leads rapidly to extinction, resulting in a powerful group-selective force. Ferriere and Fox (1995) emphasized that long-term evolution probably could not be understood without incorporating complex dynamics of populations. It is now widely agreed that natural population dynamics are generally more stable than naïve theory or laboratory models might suggest. But the field is dominated by a suspicion of group selection rooted in classical population genetic theory, which is premised on slowly-changing gene frequencies in constant populations. Hence there has been a good deal of attention to the question whether population stabilization could evolve as an incidental by-product when life histories are individually optimized to respond to population density (Heckel and Roughgarden 1980, Turelli and Petry 1980). This field is reviewed by Mueller and Joshi (2000).

Mitteldorf (2004b, 2010) has argued that the broad phenomenology and genetic character of senescence could only have evolved as an adaptation, and that an adaptive theory of aging is required. He proposed a picture in which senescence rescues the population from chaotic dynamics leading to extinction (Mitteldorf 2006), and demographic homeostasis becomes a major target of natural selection at the population level. In this context, Mitteldorf (2004a) suggested that aging could be favored by selection at the population level due to its leveling effect on the death rate. Populations subject to aging are characterized by mortality

rates that are steadier and more predictable, and the boom-bust cycles are consequently damped. Thus, even if aging tends to depress individual fitness and to lower average population levels, it may nevertheless be selected because it protects against local extinction when population levels swing low.

Here we focus on the potential for aging and PRLS to stabilize population dynamics in a computational model where death rates respond to population density with a time delay, causing population to overshoot in both directions (for small delay) or to fluctuate chaotically (larger delay) (Hutchinson 1948, May 1973). We show that aging helps to stabilize population dynamics, and that PRLS may contribute to minimizing the individual cost of demographic homeostasis.

Dynamics of the time-delayed logistic equation

The logistic equation is the oldest and simplest dynamic model of a population in an environment of limited carrying capacity K .

$$\frac{1}{N} \frac{dN}{dt} = r(1 - N/K)$$

where the left hand side is the logarithmic population growth rate, r is the maximal growth rate in the absence of crowding, and K is the steady-state population level. For N that is small compared to K , the solution exhibits exponential growth; and for $N > K$, the solution declines toward K . Solutions to the differential logistic equation are extremely well-behaved: N 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 (Li and Yorke 1975). The behavior of the logistic equation with finite time increments may be either smooth or turbulent, depending on the dimensionless parameter $r\Delta t$. For small values (up to about 2), the behavior is very much like the differential equation; for $2 < r\Delta t < 3$, the solution makes a transition from tame and well-behaved to full dynamic chaos, such that N jumps wildly about from one time step to the next (Bar Yam 1997). The onset of chaotic behavior is rapid and precipitous for values of $r\Delta t$ in the neighborhood of 2.7. For real biological systems, this translates to extinction.

In biological systems, r is the population growth rate, and the time step Δt corresponds to the response time of the ecology (Hutchinson 1948). Population growth may be supported in a period of 'overshoot' even though N exceeds the long-term carrying capacity K ; and conversely, an 'overspent' ecosystem requires a finite recovery time before it is capable of supporting a full complement of K individuals.

Consider, for example, a hypothetical population of grazing herbivores. The population is stabilized by scarcity of grass, and by predation from carnivores. These define a steady-state population level which is sustainable on long time scales. If the population rises above this sustainable level, then grass will become scarcer and predators will become more numerous, increasing the death rate from trophic levels above and below. But these ecological adjustments take time.

The grass may continue to feed the larger population, until it is eaten down to the roots; and a few predator generations must pass before their population adjusts to a more abundant food supply. During the time that the grass and predators are adjusting, the herbivore population may continue to grow beyond its sustainable value. The time scales of the species at adjoining trophic levels determine a response time for ecological feedback. It is this ecological response time that corresponds to Δt in the logistic difference equation.

Considerations of physiology and ecology offer no fundamental assurance that $r\Delta t$ must remain small (Rosenzweig 1973). In fact, individual selection pressure for higher fertility may cause birth rates in a population to rise, raising r while the response time of the underlying ecology (Δt) remains fixed. Demographic chaos acts swiftly, and can cause local extinctions on the time scale of only a few generations. This has the potential to be a very powerful form of group selection. We speculate that the reason we find persistent ecosystems in nature is not that population dynamics are inherently stable, but that demographic homeostasis has been the target of robust selection.

Description of the model

We have translated the logistic equation with time-delayed feedback into an individual-based computational model. In the classical differential equation, r is population growth rate, the difference between birth and death rates which are not disambiguated. Similarly, the quadratic term, the population's response to crowding, may be a lowering of the birth rate or an increase in the death rate. In our implementation, we have made the simplest assumption: the linear term corresponds to births, and it is independent of population size, while the quadratic term corresponds to deaths, and it is strictly proportional to population N . In other words, we assume that as population varies, fertility remains constant, and that crowding is the only source of mortality, so that the only mortality term is proportional to N/K .

In the basic model, taken from Mitteldorf (2006), a population is tracked as individuals. In each time step, each individual reproduces (clonally) with probability f , and dies with probability fN_d/K . N_d is the population size d time units in the past. (Typically, in our models $d=30$ time steps.)

Fertility f is a genetic characteristic of the individual. K is held constant such that the long-term average population size is 100 individuals. For the simple case of $d=0$ and a featureless life history, we have $K=100$; but in other variations of the model, K is adjusted to whatever level will insure a long-term average population size of 100. Details of this adjustment process are described below.

Model variations

We have varied this basic model to simulate life history characteristics, and explore their consequences. Four life history parameters have been used, singly or in combination.

- a_m , the age at maturity measured in time-steps, is a genetically-programmed age before which the individual is both vulnerable and infertile. Fertility is zero, and

mortality is 3 times the corresponding value for adults. (The adult value is proportional to total population, according to the logistic equation.) a_m is 0 in some life histories and 3 in others.

- a_b , the age at death, represents a genetically-programmed, fixed life span. Individuals with this gene suffer mortality at age a_b , independent of crowding mortality. a_d is infinite in some runs, varying down to 16 time steps.
- a_f is an age after which fertility drops to zero. In order to explore the consequences of PRLS, we allow in some life histories for senescence that affects fertility only.
- a_v is the age of 'vitality senescence'. Individuals become weaker such that their probability of (logistic) death from crowding is enhanced from the adult value by a factor 5. (Thus they are even more vulnerable than juveniles, and can serve as a 'buffer population' which begins to die off well before population reaches its peak.)

A (genetic) life history is fully specified by these six genes: f , K , a_m , a_b , a_f , and a_v .

Validation and preliminary experiments with the model

Our baseline manipulation was simply to allow a gene f for fertility to evolve. As expected, f evolves monotonically upward, driving population dynamics into an unstable regime, and causing extinction. This is a robust result, independent of other life history characteristics and of time delay d . f will always continue to rise until extinction occurs. This feature of the model has a potential counterpart in nature, and it may be that many species have evolved fertility levels that are lower than their physiology allows in order to avoid overpopulation, as Wynne-Edwards (1962) speculated.

We varied the time delay d , and noted the value of fertility just before extinction occurs. As predicted, this maximum tolerated fertility is inversely proportional to d (Fig. 1).

This extinction behavior presents a challenge in constructing evolutionary models. One approach for exploring evolutionary dynamics of populations subject to extinction

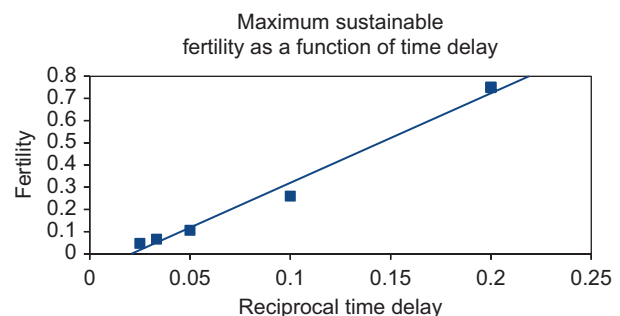


Figure 1. Fertility is permitted to evolve, with selection driving it up. High fertility then drives population volatility, leading to extinction. The last recorded value of fertility is plotted on the Y axis, and the reciprocal of time delay, $1/d$ is the X axis.

is to construct a metapopulation of quasi-independent sites, with migration among them. Using this approach Mitteldorf (2006), showed that a dynamic equilibrium could be established with a process of extinctions and recolonizations taking place within the metapopulation. When local values of fertility within a subpopulation evolve too high, the population becomes chaotic and quickly goes extinct, and the site is re-seeded from another subpopulation with lower fertilities. Thus a dynamic steady state becomes established, in which fertility is always rising within each subpopulation, but the metapopulation average stays below the critical level leading to global extinction. This model shows that this process can lead to the evolution of limits to fertility. Mitteldorf (2006) showed that under different conditions the same process can also lead to the evolution of limited life span, which also slows the population growth rate and stabilizes population dynamics.

The goal of the present investigation is to ask whether other life-history adaptations – PRLS in particular – can stabilize population dynamics, perhaps with less impact on individual level fitness than limits on life span or fertility alone. Our premise is that individual selection for higher fertility leads to higher population growth rates, until chaotic population dynamics drives the population to extinction. Extinction becomes a group-selective force for lower population growth rate. Fertility, life span, PRLS and other life history characteristics are forged by a combination of individual and group selection. The question we ask is what life history characteristics can act to stabilize the population dynamics with minimal impact on individual fitness.

To avoid making unnecessary assumptions we have chosen not to use a metapopulation model. Instead, we search for optimal life history parameters within the single-site model, increasing fertility to the highest level consistent with a standard low extinction risk, arbitrarily defined. These different, optimized life histories were then compared

in ‘tournaments’ in which life history types competed pairwise in evolutionary matches, beginning with a 50-50 mixed population and proceeding to fixation.

First experiment: fixed life span, with and without fixed fertility-span

Though the model is simple, the way in which it was run warrants some explanation. Simply allowing the life history genes to evolve was not an option, because this leads to extinction. Instead, we used a two-step process. In the first step, we varied life-history parameters, looking for combinations that led to sustainable population dynamics. In the second step, these selected (stable) life histories were compared pair-wise to search for those with highest individual fitness.

In the first experiment, maturation time a_m was taken to be zero. We defined three qualitative classes of life history:

1. No senescence: fertility does not vary with age, and the only mortality is logistic mortality, with probability proportional to (delayed) population.
2. A fixed life span: individuals that survive mortality associated with the logistic growth equation die when they reach the maximum life span a_d .
3. Loss of fertility occurring at a fixed age a_β followed by a fixed life span a_d that is greater than the age at which reproductive senescence occurs.

For each of these life histories, we searched through combinations of parameters (automatically and by hand), and identified those with the highest fertility levels consistent with demographic stability. We defined demographic stability arbitrarily as 85% of runs completing 25 000 time steps without extinction. (Other reasonable stability criteria were also tried, with qualitatively similar results. Insensitivity to stability criteria is probably due to the steepness of the onset of dynamic chaos.)

Table 1. Parameters of the first twenty life histories in the individual fitness tournament described for the first experiment.

No.	Wins	Losses	Type	% PR	Life history parameters					
					a_m	a_f	a_v	a_d	f	K
1	8209	4791	3	21	0	24	∞	34	0.060	208.19
2	7702	5298	3	16	0	22	∞	28	0.065	211.73
3	7578	5422	3	19	0	26	∞	38	0.060	174.11
4	7512	5488	3	20	0	24	∞	36	0.065	175.25
5	7426	5574	3	9	0	22	∞	24	0.060	266.43
6	7405	5595	3	17	0	26	∞	38	0.065	155.06
7	7299	5701	3	15	0	24	∞	30	0.060	203.74
8	7253	5747	3	17	0	26	∞	36	0.060	173.01
9	7198	5802	3	10	0	32	∞	42	0.060	134.29
10	7129	5871	3	7	0	23	∞	25	0.070	166.51
11	7123	5877	2	0	0	∞	∞	20	0.060	264.55
12	7091	5909	2	0	0	∞	∞	36	0.060	119.78
13	7062	5938	3	16	0	28	∞	40	0.060	154.52
14	7059	5941	3	12	0	26	∞	32	0.060	172.91
15	7051	5949	3	8	0	22	∞	24	0.065	209.14
16	7025	5975	3	6	0	40	∞	50	0.060	116.89
17	7014	5986	2	0	0	∞	∞	26	0.060	154.24
18	7006	5994	2	0	0	∞	∞	20	0.060	263.85
19	6994	6006	3	4	0	40	∞	46	0.060	116.96
20	6983	6017	3	9	0	21	∞	23	0.065	238.10

We constrained the search with values of carrying capacity, K , calibrated to make the long-term average population size equal to 100. Thus we are assuming that the ecology determines a fixed carrying capacity which cannot be exceeded indefinitely. Without this assumption, competition would have consistently favored increasing carrying capacities, and populations would have evolved arbitrarily high values of K .

We ran the basic model for a wide range of parameter sets, most of which resulted in prompt extinction. From these, we collected 131 stable life histories and pitted them against each other in a tournament. Each of the 131 was >85% stable, and each was calibrated to a long-term average population of 100 individuals. These 131 comprised 4 of type 1, 67 of type 2, and 60 of type 3. There were only 4 of type 1, and these were very similar, because the two criteria of stability and fixed average population determine the only two parameters (f and K) in a featureless, type 1 life history. Type 2 has an additional parameter (age at death) and type 3 has two additional parameters (age at fertility loss and age at death), so it would have been natural to include more type 3 than type 2 life histories in the competition; however, since our hypothesis favors type 3, we did not want to admit a possibility of biasing the results in favor of type 3 by including more type 3 entries in the tournament.

The tournament was ‘round-robin’, conducted as follows: each of the pairs of types ($1/2 \times 131 \times 130 = 8515$) was used to initialize a population of 100 individuals, 50 of each type. The delayed logistic model was run until one type evolved to fixation (which was generally just a few hundred time steps). The contest for each pairing was repeated 100 times. Thus, each life history type was matched in 100 trial bouts with each of the 130 other types, for a total of 13 000 competitions per type. Counts were maintained of victories and losses, and each of the life history types was ranked according to the number of victories (out of 13 000 total). These binary contests compared individual fitnesses in the context of the same ecology (the same time delay) in which they were originally selected. Conceptually, these competitions were addressing the issue of what life history and parameter sets had the highest individual fitness given the constraints that 1) they are able to persist for extended periods and 2) the long term mean population size is 100.

On average life histories that included fertility senescence and a fixed life span (type 3) significantly outperformed both life histories with a fixed life span (type 2), and simple life histories with no senescence (type 1) (Mann–Whitney $p < 10^{-6}$). More significant is the comparison of the best exemplars of each type (Table 1). The top ten places in the tournament were all of type 3. Thus, based on the tournament results, the highest individual fitness was achieved while incorporating fertility senescence. Ranks 11 through 36 included 15 of type 3 and 11 of type 2. The best-performing life history of type 1 (no senescence) was ranked 37th in individual fitness, with 6792 victories out of 13 000.

The average percentage of post-reproductives in the population is a model-independent measure of the demographic significance of PRLS. Time-average proportion of post-reproductives ranged from 7% to 21%, with an average

of 14%. (The highest value, 21%, characterized the overall first-place life history.)

The results suggest that, within this model, simply suppressing fertility to achieve demographic stability is the highest-cost solution. ‘Drop-dead’ senescence does considerably better, with lower individual cost for comparable group benefit. Including a post-reproductive period, with a buffer population of ‘disposable’, non-reproducing individuals further lowered the individual cost of the same group benefit.

Second experiment: three-part life histories with juvenile period

In this second series of runs, we investigate a different kind of senescence, while allowing for a period of maturation at the front end of the life cycle. The juvenile period (standardized at $a_m = 3$ time steps) is characterized by reproductive immaturity (infertility) and a mortality rate that is 3 times that of adults. At a fixed age, a_v , adults become senescent, and their logistic mortality rises to 5 times that of adults ($5/3$ the mortality rate for juveniles). In contrast to the ‘drop-dead’ senescence implemented in the first experiment, all mortality in these trials is proportional to crowding, with different ages having different proportionality constants. The ratio 3:1:5 for rates of mortality was chosen somewhat arbitrarily, in order to allow the senescent class to die first and buffer the juveniles from crowding deaths. We define three qualitative types of life history (all three include the same juvenile period of 3 time steps):

1. No senescence, just as in the first experiment.
4. Vitality senescence at age a_v , defined by a 5-fold increase in the logistic constant for crowding mortality.
5. Fertility senescence (sudden infertility) beginning at age a_f , followed by vitality senescence at age a_v , with $a_v \geq a_f$.

In populations of type 1 and type 4, there are no post-reproductives, but type 5 allows for the possibility of a post-reproductive age class. Type 4 represents loss of vitality without loss of fertility, and type 5 represents loss of both fertility and vitality with age, at ages that may be equal or greater. (The remaining logical possibility – loss of fertility without loss of vitality – was also tried, but led to anomalous results: a stable solution that probably corresponds to nothing in biology. Old, infertile individuals accumulate as juveniles die off. The very old remain in a population sized well below steady-state levels, so that the death rate is small, but they are not reproducing, so the population can last in this state for a very long time.)

We conducted a round-robin tournament including 4 specimens of type 1, 20 of type 4 and 20 of type 5. Once again, there were many more of type 5 that we might have included, because type 5 depends on two parameters (a_v and a_f) where type 4 depends on only one (a_v); but we wished to avoid any risk of stacking the numbers in favor of type 5, and consequently included only the best 20.

All the type 5 life histories filled the top 20 spots. The top ten carried an average of 27% of their populations as post-reproductive (Table 2). Type 1, without senescence, occupied the next three slots, ahead of all the type 4 life histories. This suggests that simply weakening with age, without infertility, is not a winning strategy for maintaining population

Table 2. Parameters of the first twenty life histories in the individual fitness tournament described for the second experiment.

No.	Wins	Losses	Type	% PR	Life history parameters					
					a_m	a_f	a_v	a_d	f	K
1	2718	1582	5	34	3	24	30	∞	0.070	406.98
2	2716	1584	5	30	3	28	34	∞	0.060	350.67
3	2688	1612	5	20	3	26	26	∞	0.070	330.34
4	2679	1621	5	32	3	26	32	∞	0.065	368.48
5	2672	1628	5	28	3	28	32	∞	0.060	350.06
6	2644	1656	5	27	3	26	28	∞	0.065	369.11
7	2644	1656	5	25	3	24	26	∞	0.075	360.23
8	2626	1674	5	19	3	30	30	∞	0.060	297.62
9	2615	1685	5	27	3	24	24	∞	0.070	403.46
10	2595	1705	5	26	3	28	30	∞	0.060	349.04
11	2582	1718	5	17	3	32	34	∞	0.060	266.08
12	2565	1735	5	30	3	26	30	∞	0.065	367.02
13	2555	1745	5	30	3	24	26	∞	0.070	403.69
14	2504	1796	5	15	3	30	30	∞	0.065	272.42
15	2499	1801	5	21	3	30	32	∞	0.060	297.03
16	2499	1801	5	24	3	28	28	∞	0.060	347.62
17	2441	1859	5	25	3	30	36	∞	0.060	296.15
18	2429	1871	5	23	3	24	24	∞	0.075	356.46
19	2367	1933	5	30	3	18	18	∞	0.100	491.64
20	2329	6017	5	29	3	22	24	∞	0.080	402.41

stability without sacrificing individual fitness. Simply lowering fertility works better. But best by far in this context is combination of fertility senescence and vitality senescence. The proportion of post-reproductives carried in the population was even higher than in experiment 1.

It is the combination of high fertility and large post-reproductive populations that created an advantage for both individual and group level fitnesses. The buffer population of post-reproductives is highest when the population is thriving and growing at an unsustainable rate. When the population is collapsing, the post-reproductives are the first to die, and the high fertility of the remaining (young) population helps to assure survival in hard times.

Third experiment: random parameters

In this series, the original stock of life histories was chosen by selecting random values between 12 and 50 for each of the life history ages, for a_f , a_v and a_d . These were filtered, using the same criteria as the first two experiments, and the first 101 that led to stable population dynamics were saved and entered into a round robin tournament. With random values for the critical ages, the parameter types did not always fall neatly into the five established categories. Among the contestants, there were 43 with some PRLS and 58 without. The time-average percentage of post-reproductives in the populations of the 43 was 12.2%.

Table 3. Parameters of the first twenty life histories in the individual fitness tournament described for the third experiment.

No.	Wins	Losses	Type	% PR	Life history parameters					
					a_m	a_f	a_v	a_d	f	K
1	6992	3008	2/4	0	3	44	30	43	0.070	211.74
2	6315	3685	5	25	3	23	26	34	0.070	462.96
3	6123	3877	5	23	3	23	26	34	0.075	398.30
4	5909	4091	3	22	3	22	34	31	0.090	343.12
5	5807	4193	2	0	3	43	33	22	0.065	476.54
6	5740	4260	3	32	3	24	47	42	0.075	353.61
7	5693	4307	3/5	21	3	23	30	32	0.085	332.16
8	5658	4342	3/5	11	3	23	20	44	0.095	305.17
9	5577	4423	3/5	14	3	32	39	43	0.070	236.01
10	5564	4436	3/5	26	3	23	28	41	0.080	356.03
11	5556	4444	2	0	3	43	33	22	0.070	397.95
12	5545	4455	3/5	11	3	30	31	42	0.075	245.10
13	5541	4459	3/5	22	3	23	30	32	0.080	356.35
14	5534	4466	2	0	3	29	34	20	0.095	323.57
15	5391	4609	3/5	19	3	25	32	36	0.085	287.55
16	5368	4632	2	0	3	43	33	22	0.075	352.78
17	5357	4643	3	19	3	29	36	49	0.075	254.50
18	5343	4657	3/5	19	3	23	26	34	0.085	330.48
19	5269	4731	2/4	2	3	28	25	29	0.080	257.82
20	5264	4736	2	3	3	37	43	39	0.075	204.19

The result (Table 3) was that all the first 20 tournament winners included some senescence, and 15 out of 20 included PRLS. The average percentage of post-reproductives among the top 20 was 13.5%. Eight of the top 10 included some PRLS, with an average percentage of post-reproductives equal to 17.4%.

Once again, PRLS appeared to be helpful but not absolutely necessary in order to create stable life histories with high individual fitness.

Discussion

The ubiquity of PRLS in nature defies prevailing theories for the evolution of senescence, and is difficult, if not impossible, to explain from selection on an individual level. We seek to resolve this conundrum by considering another conundrum faced by standard population genetic theory: selection among individuals always favors those that reproduce the fastest. This logically leads to maximal intrinsic rates of increase, r , for the population. Yet high values of r should lead to chaotic population dynamics, something that is rarely, if ever, seen in nature. For example, Costantino et al. (1997) observed chaotic dynamics in populations of *Tribolium castaneum* that had artificially manipulated birth and death rates, and noted that unmanipulated populations of this species are not chaotic. A third conundrum is that it appears that not all species have life histories optimized for maximal r . As an obvious example, most higher animals tolerate the two-fold cost of carrying males, suggesting that r is not the sole component of fitness. Wynne-Edwards (1962) documented many examples of natural population regulation, and though his theoretical reasoning was promptly refuted (Williams 1966), his observations as a naturalist have never been adequately explained, as they imply that individual fertility has been moderated for the sake of population regulation at the group level. We offer a hypothesis that addresses all three of these issues: that selection against chaotic life histories that lead to extinction has put an upper limit on the r that populations can attain. We suggest that demographic homeostasis may be a major target of natural selection that opposes individual selection and keeps rates of reproduction in check.

In the model presented here we demonstrate how senescence and fertility loss in particular can evolve in life histories that are under selection at two levels: individual selection for higher r and population level selection for sustainable population dynamics. We examine a class of ecological instabilities related to demographic chaos that occurs when r rises above the ecology's response time. When a population exceeds the level (K) that is sustainable in the long run, the ecosystem goes into deficit and takes time to recover. When the net growth rate of a population exceeds the ecosystem's recovery time by about a factor of two, population fluctuations become severe, and extinction is a risk.

In many circumstances it may be that physiological or ecological constraints prevent a species from evolving high values of r , and the danger of population chaos is moot. However, in other circumstances it may be that species can achieve reproductive rates that are high enough that chaotic population dynamics can occur. When this happens there

will be selection at the population level as those populations with high r go extinct. By moderating growth rates this population level selection directly opposes individual selection pressure for higher fertility.

In our numerical experiments, we have tested different life history strategies both for demographic sustainability and for individual competitiveness. We have used tournaments including thousands of paired contests to evaluate the individual fitness of several hundred strategies, all of which are pre-filtered for sustainability. We conducted three such tournaments, using different styles of senescence.

In the first tournament, senescence was defined as sudden death at a genetically programmed age. In the second, senescence was defined as a five-fold increase in the logistic mortality rate after a certain age. In the third tournament, ages at onset of each of the three forms of senescence were allowed to vary randomly. Each of the three tournaments included options for 1) no senescence, 2) death without loss of fertility, and 3) loss of fertility followed by death. In each tournament, the most successful life history strategies included loss of fertility. This suggests that a post-reproductive period is particularly effective in creating an optimal compromise when individual selection for high growth rate conflicts with group selection for sustainable growth.

The contests were designed to be well-matched, with the result that no competitor won more than 70% of its pairings. This is because two of the most potent components of fitness were equalized by design in all of the contestants. These are 1) age of reproductive maturity, and 2) long-term average population. Both these traits are under strong directional selection, with no antagonism between individual- and group-level selection. We presume that age of maturity is fixed by physiological limits on growth rate, and pegged at its minimum value by directional selection. And long-term average population is fixed by the carrying capacity of the ecosystem at a value that cannot be exceeded. Thus, the parameter sets that constituted contestants in each tournament were all characterized by the same age at maturity and the same long-term average population.

We chose to use a selection tournament among stable life histories rather than a metapopulation model that included group selection for population stability because there are a large number of possible ways to construct a metapopulation model, and experience shows that their results are highly sensitive to unknowable details of the metapopulation structure. We decided that the tournament among possible solutions would be clearer, and would provide insights into the general process of adaptation to multilevel selection without being constrained by the details of a specific model of multilevel selection.

In our model, life histories that include reproductive senescence are able to compete successfully with long-lived life histories because the former have higher fertility early in life. In this respect, our model bears a strong resemblance to the better-established theory of antagonistic pleiotropy (Williams 1957, Rose 1991). In both the present model and in antagonistic pleiotropy, shorter life spans are balanced by higher fertility. The logic behind these theories is slightly different, however: the theory of antagonistic pleiotropy assumes that there are negative genetic correlations between reproduction and lifespan, because the same genes that

support high fertility also cause damage that leads to aging. Thus individual selection favors individuals that maximize fertility at the expense of a shorter lifespan. In the present model, the negative correlation is not assumed but derived from the requirement of stable population dynamics. The evolution of decrease in lifespan and fertility senescence in this model results from population level selection against the chaotic dynamics that come with high growth rates.

Aging and reproductive senescence are ubiquitous in animals, and post-reproductive life span is apparently more common than previously considered. The dominant school of evolutionary theory typically recognizes only selection at the individual level. Longer-term constraints on reproductive rates are rarely considered. Within this paradigm, the reasoning of Fisher (1930) and Williams (1957) is correct: affirmative mechanisms of aging could never have evolved through a process of natural selection at the individual level, and fertility senescence should not evolve except in special cases such as the grandmother hypotheses of humans (Peccei 2001). Without strong group selection, the non-adaptive models of aging such as antagonistic pleiotropy, mutation accumulation (Medawar 1952, Edney and Gill 1968), and the disposable soma (Kirkwood 1977) may be the most plausible models for evolution of aging. However, experimental contradictions to these theories abound (Goldsmith 2003, 2008, Bredesen 2004, Mitteldorf 2004b, 2010), and this may be reason enough to consider chaotic population dynamics as a candidate for the strong group selection that is necessary to evolve aging and PRLS as adaptations.

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References

- Abrams, P. A. 2000. The evolution of predator–prey interactions: theory and evidence. – *Annu. Rev. Ecol. Syst.* 31: 79–105.
- Austad, S. 1993. Retarded senescence in an insular population of Virginia opossums. – *J. Zool. Lond.* 229: 695–708.
- Bar Yam, Y. 1997. Dynamics of complex systems. – Addison-Wesley.
- Bijma, P. et al. 2007. Multilevel selection 1: quantitative genetics of inheritance and response to selection. – *Genetics* 175: 277–288.
- Blurton Jones, N. H. et al. 1989. Studying costs of children in two foraging societies: implications for schedules of reproduction. – In: Standon, V. F. (ed.), *Comparative socioecology of mammals and man*. Blackwell, pp. 365–390.
- Borrello, M. 2010. Evolutionary restraints: the contentious history of group selection. – Univ. of Chicago Press.
- Bredesen, D. E. 2004. The non-existent aging program: how does it work? – *Aging Cell* 3: 255–259.
- Brody, S. H. et al. 1923. The rate of senescence of the domestic fowl as measured by the decline in egg production with age. – *J. Gen. Physiol.* 6: 41–45.
- Cohen, A. A. 2004. Female post-reproductive lifespan: a general mammalian trait. – *Biol. Rev. Camb. Philos. Soc.* 79: 733–750.
- Costantino, R. F. et al. 1997. Chaotic dynamics in an insect population. – *Science* 275: 389–391.
- Edney, E. B. and Gill, R. W. 1968. Evolution of senescence and specific longevity. – *Nature* 220: 281–282.
- Ferriere, R. and Fox, G. A. 1995. Chaos and evolution. – *Trends Ecol. Evol.* 10: 480–485.
- Fisher, R. A. 1930. *The genetical theory of natural selection*. – Clarendon Press.
- Gaulin, S. 1980. Sexual dimorphism in the human post-reproductive life-span: possible causes. – *J. Human Evol.* 9: 227–232.
- Gilpin, M. E. 1975. Group selection in predator–prey communities. – Princeton Univ. Press.
- Goldsmith, T. 2003, 2008. *The Evolution of Aging*. – Azinet <www.azinet.com/aging/Aging_Book.pdf>
- Goodnight, C. J. and Stevens, L. 1997. Experimental studies of group selection: what do they tell us about group selection in nature? – *Am. Nat.* 150 Suppl 1: S59–79.
- Goranson, N. et al. 2005. Resolving an adaptive conundrum: reproduction in *Caenorhabditis elegans* is not sperm-limited when food is scarce. – *Evol. Ecol. Res.* 7: 325–333.
- Gosden, R. G. et al. 1983. Imminent oocyte exhaustion and reduced follicular recruitment mark the transition to acyclicity in aging C57BL/6J mice. – *Biol. Reprod.* 28: 255–260.
- Hawkes, K. 2003. Grandmothers and the evolution of human longevity. – *Am. J. Human Biol.* 15: 380–400.
- Hawkes, K. O. C. and Blurton Jones, N. 1997. Hadza women's time allocation, offspring provisioning, and the evolution of long postmenopausal life spans. – *Curr. Anthropol.* 38: 551–577.
- Hawkes, K. et al. 2011. A reappraisal of grandmothing and natural selection. – *Proc. Biol. Sci.* 278: 1936–1941.
- Heckel, D. G. and Roughgarden, J. 1980. A species near its equilibrium size in a fluctuating environment can evolve a lower intrinsic rate of increase. – *Proc. Natl Acad. Sci. USA* 77: 7497–7500.
- Herndon, J. G. and Walker, L. C. 2010. The grandmother effect and the uniqueness of the human aging phenotype. – *Gerontology* 56: 217–219.
- Holmes, D. J. and Ottinger, M. A. 2003. Birds as long-lived animal models for the study of aging. – *Exp. Gerontol.* 38: 1365–1375.
- Hutchinson, G. E. 1948. Circular casual systems in ecology. – *Ann. N. Y. Acad. Sci.* 50: 221–246.
- Kachel, A. F. et al. 2011a. Grandmothing and natural selection. – *Proc. Biol. Sci.* 278: 384–391.
- Kachel, A. F. et al. 2011b. Grandmothing and natural selection revisited. – *Proc. R. Soc. B* 278: 1939–1941.
- Kirkwood, T. 1977. Evolution of aging. – *Nature* 270: 301–304.
- Li, T. Y. and Yorke, J. A. 1975. Period three implies chaos. – *Am. Math. Monthly* 82: 985–992.
- May, R. M. 1973. *Stability and complexity in model ecosystems*. – Princeton Univ. Press.
- Maynard Smith, J. 1976. Group selection. – *Q. Rev. Biol.* 51: 277–283.
- McAuliffe, K. and Whitehead, H. 2005. Eusociality, menopause and information in matrilineal whales. – *Trends Ecol. Evol.* 20: 650.
- Medawar, P. B. 1952. An unsolved problem of biology. – H. K. Lewis & Co.
- Minois, N. et al. 2005. Advances in measuring lifespan in the yeast *Saccharomyces cerevisiae*. – *Proc. Natl Acad. Sci. USA* 102: 402–406.
- Mitteldorf, J. 2004a. Chaotic population dynamics and the evolution of aging. – In: Pollack, J. et al. (eds), *9th Int. Conf. on Artificial Life*. MIT Press, pp. 346–351.
- Mitteldorf, J. 2004b. Aging selected for its own sake. – *Evol. Ecol. Res.* 6: 1–17.
- Mitteldorf, J. 2006. Chaotic population dynamics and the evolution of aging: proposing a demographic theory of senescence. – *Evol. Ecol. Res.* 8: 561–574.

- Mitteldorf, J. 2010. Evolutionary origins of aging. – In: Fahy, G. M. et al. (eds), *Approaches to the control of aging: building a pathway to human life extension*. Springer, pp. 87–126.
- Mueller, L. D. and Joshi, A. 2000. *Stability in model populations*. – Princeton Univ. Press.
- Muir, W. M. 1996. Group selection for adaptation to multiple-hen cages: selection program and direct responses. – *Poult. Sci.* 75: 447–458.
- O’Connell, J. et al. 1999. Grandmothering and the evolution of homo erectus. – *J. Human Evol.* 36: 461–485.
- Ottinger, M. A. and Balthazart, J. 1986. Altered endocrine and behavioral responses with reproductive aging in the male Japanese quail. – *Horm. Behav.* 20: 83–94.
- Packer, C. et al. 1998. Reproductive cessation in female mammals. – *Nature* 392: 807–811.
- Pavelka, M. S. and Fedigan, L. M. 1999. Reproductive termination in female Japanese monkeys: a comparative life history perspective. – *Am. J. Phys. Anthropol.* 109: 455–464.
- Peccei, J. S. 2001. A critique of the grandmother hypotheses: old and new. – *Am. J. Human Biol.* 13: 434–452.
- Reznick, D. et al. 2006. The evolution of senescence and post-reproductive lifespan in guppies (*Poecilia reticulata*). – *PLoS Biol.* 4: e7.
- Rose, M. 1991. *Evolutionary biology of aging*. – Oxford Univ. Press.
- Rosenzweig, M. L. 1973. Exploitation in three trophic levels. – *Am. Nat.* 107: 275–294.
- Sober, E. and Wilson, D. S. 1998. *Unto others: the evolution and psychology of unselfish behavior*. – Harvard Univ. Press.
- Stevens, L. et al. 1995. Multi-level selection in natural populations of *Impatiens capensis*. – *Am. Nat.* 145: 513–526.
- Swenson, W. et al. 2000. Artificial ecosystem selection. – *Proc. Natl Acad. Sci. USA* 97: 9110–9114.
- Thomas, W. R. et al. 1980. Chaos, asymmetric growth and group selection for dynamic stability. – *Ecology* 61: 1312–1320.
- Turelli, M. and Petry, D. 1980. Density-dependent selection in a random environment: an evolutionary process that can maintain stable population dynamics. – *Proc. Natl Acad. Sci. USA* 77: 7501–7505.
- Ward, E. J. et al. 2009. The role of menopause and reproductive senescence in a long-lived social mammal. – *Front. Zool.* 6: 4.
- Williams, G. 1957. Pleiotropy, natural selection, and the evolution of senescence. – *Evolution* 11: 398–411.
- Williams, G. 1966. *Adaptation and natural selection*. – Princeton Univ. Press.
- Wynne-Edwards, V. 1962. Animal dispersion in relation to social behavior. – Oliver & Boyd, Edinburgh.