Species Selection of Aging for the Sake of Diversity

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Abstract

In their paper, "Punctuated Equilibria: An Alternative to Phyletic Gradualism", Eldredge and Gould (1972) argue that most evolution occurs during geologically rapid speciation events, with species exhibiting stasis the vast majority of the time. Gould (2002, 2007) demonstrates that an implication of Punctuated Equilibrium is that selectionist theory is expanded to the level of species - defining species as basic units of macroevolution. In our paper, we demonstrate the evolution of aging rates in a species selection scenario. We have developed an ALife simulation environment with mating governed by evolving compatibility signatures, resulting in the formation of reproductively isolated subpopulations (i.e., species). Given a co-evolving parasite population, heterogeneity in a host subpopulation is beneficial for the health of that subpopulation. This can result in group selection pressure at the species level for the evolution of altruistic traits, such as a faster aging rate.

Introduction

The mechanism of aging has perplexed those who have attempted to understand it in a Darwinian framework. The wide and enduring variations in longevity across species suggests that aging rate is the result of natural selection. The theory of evolution by natural selection holds that many of the traits we observe in organisms are the result of adaptations to the environments of their ancestors. However, any possible adaptive benefit of faster aging cannot accrue to its bearer, who will likely have fewer offspring. Therefore, the evolution of aging requires a selection mechanism beyond the individual. Such selection mechanisms have been proposed by Wynne-Edwards (1962), in the form of group selection theory, and Hamilton (1964), in the form of inclusive fitness theory. In previous papers we showed that these mechanisms are interdependent in a prominent simulation model of the evolution of aging (Woodberry et al., 2005, 2007).

Bell (1982) posited that the diversity created by sexual recombination provides a group benefit in the co-evolution arms race between hosts and parasites. In a similar manner we argue that aging benefits the group by increasing population turnover and, thus, genetic diversity. Here we reinforce our hypothesis by demonstrating its practicality in a species selection scenario. There have been investigations into the implications of Punctuated Equilibrium on selectionist theory — identifying species as basic units of macroevolution, due to their stability (and hence individuality) between punctuation events (see Gould, 2002, 2007). On the individuality of species, Gould (2002) says: "So long as most new species arise by branching (speciation) rather than by transformation (anagenesis), species can be individuated by their uniquely personal duration, bounded by birth in branching and death by extinction. "

In this paper, we describe the design and use of an ALife simulation containing co-evolving host and parasite populations. Host agents have mating signatures, which are used to determine mate compatibility, and vulnerability signatures, which govern the infection process. Mutation of the signatures may cause the host population to diversify into reproductively isolated subpopulations, i.e., to speciate. The parasite population, which has infection and virility signatures, flourishes when the host population's vulnerability signatures are genetically uniform, creating a positive selective pressure for the evolution of aging for the sake of species diversity. Here we further our argument for the evolution of aging by demonstrating it in a species selection scenario — which arguably is more realistic than classical group selection.

Background

The Evolution of Aging

Aging is defined as the general deterioration of an organism, and its eventual death, by internal causes (Williams, 1957). The rates at which different species age is a perplexing phenomenon. The divergence is extraordinary, ranging from a few hours for some phytoplankton cells (Agustí et al., 1998) to a few days for some insects to thousands of years for the bristlecone pine tree. Furthermore, these different rates have themselves not varied greatly during recorded history, so far as we can tell. The genetic control of aging is beginning to come into view, with multiple genes already identified as participating in aging rates (e.g., Belenky et al., 2007). All

of this seems to suggest that aging rates have evolved because of their adaptive value. However, the obvious fitness costs of fast aging on individuals would cause strong direct selection pressure against it, suggesting that aging may be a side effect of some more essential characteristic, i.e., that it is non-adaptive. Historically, both adaptive, e.g., Weismann (1889), and non-adaptive, e.g., Williams (1957) and Medawar (1952), explanations of aging have been proposed. Recently there has accumulated compelling experimental evidence that aging *is* an adaptation (Mitteldorf, 2004; Bredesen, 2004; Skulachev, 1997). This has lead to a resurgence of research into possible adaptive benefits of aging, including our own.

In Woodberry et al. (2007) we posited an adaptive explanation of aging. We argued that aging has a group fitness benefit which can outweigh the individual fitness cost. Groups with shorter individual life spans turn over faster and consequently have greater genetic diversity. In co-evolution scenarios, e.g., predator-prey and host-parasite interactions, groups with greater diversity will be less easily exploited, creating a stronger and healthier population.

Group and Kin Selection

An adaptive explanation of aging requires a selection mechanism accounting for the potential selection of altruistic traits. The papers of Wynne-Edwards (1962) on group selection and Hamilton (1964) on inclusive fitness theory attempted to give a mathematical analysis supporting selection mechanisms beyond individual selection. Maynard Smith (1976) went on to create a model demonstrating the logical possibility of group selection. He showed that the turnover of groups, via extinction and pioneering, can favor altruistic groups which, because of their altruism, have greater group lifespans and so greater opportunity to found new groups. This enables scenarios where cheaters, even while having a fitness advantage within groups, do not take over the population. Inclusive fitness (or kin selection) theory, by contrast, shifts the focus downwards from the individual to the gene, whether held by the individual or, as a replica, by a relative. The inclusive fitness of a gene is just the organism's individual fitness augmented by the harms and benefits caused to the fitness of others, weighted by their relatedness, i.e., the probability of their carrying the same allele (Hamilton, 1964). Inclusive fitness theory, or kin selection, has become widely accepted, especially as an explanation for the evolution of altruistic behavior. The group selection concept, however, has remained contentious. It has been doubted, for example, whether the selection pressure for selfish behavior within groups can be overcome in nature by selection pressure for altruism between groups. In Woodberry et al. (2005) we argued that group selection may be dependent upon kin selection, rather than in opposition to it, as most would have it. That is, kin selection may well provide selection pressure within groups for an altruistic trait that is also being selected for at the group level, when the latter selection pressure would be insufficient for evolutionary stability of the trait on its own.[1]

Species

The concept of species, as a taxonomic classification, remains central to biology and a host of related fields. The definition of species remains controversial, as there is an inherent vagueness in its application, e.g., asexual species, ring species and hybrids. The most generally accepted definition of species, which we follow, is a reproductively isolated sub-population (Mayr, 1963) — that is, a group of actually or potentially interbreeding populations that are reproductively isolated from other such groups. Studies of speciation are based on geographic circumstances: allopatric and peripatric speciation rely on geographic isolation, whereas sympatric and parapatric speciation are based on the emergence of new species with little, or no, geographic isolation. In our simulation, as there are no barriers to migration, speciation must be described in the latter terms.

Punctuated Equilibrium

Eldredge and Gould (1972) drew attention to what they saw as a mistaken view that evolution can only occur gradually and, indeed, can only occur at a constant, continuous rate — a concept they labelled Phyletic Gradualism. They argued instead that most evolution occurs during geologically short-term speciation events, with species exhibiting approximate stasis the vast majority of the time. They claimed that this punctuated equilibrium view of evolution is more consistent with the observations made in the fossil record.

Under the Punctuated Equilibrium concept, once a species becomes static and defined, it takes on a kind of individuality. It has a lifespan; it has the opportunity to reproduce through speciation; and, in the end, it will disappear. This supports a metaphorical similarity with individual reproduction and, therefore also, with individual fitness (Gould, 2002). But the similarity is more than metaphorical with group selection, for this just is a kind of group selection. Species become units of selection, competing with other species within the biosphere for the opportunity to create new species and to avoid early extinction; this creates a species selection mechanism which falls under the group selection model described above, and which caters for the evolution of altruistic traits.

Simulation Design

To test hypotheses about aging, we designed a multi-agent ALife simulation environment. Co-evolving populations of host and parasite agents interact within overlapping neighbourhoods on a board, sharing food sources and potentially

[1] Multilevel selection theory asserts the compatibility of multiple levels of selection, rather than their interdependency (Wilson and Sober, 1994).

reproducing sexually. Table 1 provides an overview of the simulation parameters, which are discussed in depth below. When designing simulations, it is necessary to consider the trade-off between the complexity of the model and its completeness. Although more complex models are harder to analyse, simpler models could neglect important mechanisms that allow validation against real systems (Grimm et al., 2005). In our design process, we tried to find a satisfactory trade-off for our simulation.

Table 1: Simulation Parameters

Parameter	Comment
Epoch Length	100 cycles
Run Length	100 epochs
Board Size	120×120 cells
Neighbourhood Size	3×3 cells
New Food	$N(1, \frac{1}{10})$ units
Initial Health	20 units
Parental Health Investment	10 units/parent
Health Energy Overhead	1 unit/cycle
Max Health	80 units
Mature Health	60 units
Accident Rate	0.1
Parasite Generate Rate	0.0001
Signature Length	100 bits
Signature Mutator	0.005
Initial Expiry Gene	20
Expiry Mutator	$N(Expiry, \frac{Expiry}{50})$
Initial Airborne Gene	0.05
Airborne Mutator	$N(Airborne, 0.001)$
Airborne Co-ordinate Jump	N(0,10)

World

Time: Simulation runs are divided into a number of epochs for statistics collection. During each epoch, the simulation world and agents are updated over a number of cycles, with statistical information collected and saved to file at the end of the epoch. The methods of agent and world updating are discussed in depth below.

Board: The simulation board consists of a square grid of cells, wrapped so that the edges meet, forming a torus shaped world. Each cell contains an occupant population, unlimited in size, and a food store. Each cycle the food store is replenished with new food, as determined by a normal distribution, and energy is recycled from the previous cycle, i.e., uneaten food and recycled agent energy (discussed later). The cell contents interact with the nine cells in the Moore neighbourhood — recycled food is distributed, evenly, to neighboring cells; and agents feed, mate and migrate freely within their neighbourhood.

Agents

Host Agent: The host agents are the focus of the simulation. Each host agent occupies the cell into which they were born. Each cycle all host agents have an opportunity, in a randomly selected order, to eat and reproduce, after which they are tested for death conditions. Figure 1 shows the algorithm used for updating hosts, discussed in detail below. The simulation maintains for each host agent its age, health, and chromosome. Age is initialised at zero and incremented each cycle. The health is incremented whenever the host agent successfully eats and is decremented each cycle by an energy overhead and also by a parental investment whenever the host agent reproduces. The chromosome is inherited at birth and, along with the states of the other variables and environment, determines agent behaviour.

Each cycle the host agent eats, unless its health is below zero or greater than a maximum value. A cell is selected, randomly, from the cells neighboring the agent's occupancy cell, and all the contents of that cell's food store are transferred to the host's health.

Host agents are genderless, but reproduce predominantly sexually. After the agent has eaten, it is tested against a health threshold; if the agent has sufficient health, it attempts to reproduce. In addition to avoiding suicidal mating, this forces agents to mature before reproducing, since new agents will lack sufficient health. When reproducing, the agent first checks its neighbourhood for any mate requests by compatible agents (compatibility is discussed later). If one is found, the agents reproduce sexually and two offspring are created. If the agent fails to find a mate, but its health exceeds its maximum health threshold, it will reproduce asexually. The initial health of the offspring is the sum of parent health donations.

There are three causes of death. The host agent dies if:

1. its health falls below zero;
2. its age exceeds a genetically determined expiry age (discussed later); or
3. it dies of external, accidental causes, as determined by an accident probability each cycle.

These first two causes of death are necessary to have an ecologically plausible test environment for examining theories of the evolution of aging. The implementation of accidental death is not a strict requirement of the simulation — however, it makes the simulation more realistic, by weakening selection pressures in favour of faster aging in a way we know operates in real populations. Having a closed ecosystem requires us to remove dead agents from the board and recycle any remaining energy held as health through the growth of new plant food.

```
decrement health
increment age
if parasite to be generated then
   generate and save parasite
if health < 0 OR age > expiry age OR accident then
   remove agent
   recycle health energy
else
   if health < max health then
      attempt eat action
   if health > mature health then
      if mate available then
         reproduce sexually
      else if health > max health then
         reproduce asexually
   save agent
```

Figure 1: Host Update Algorithm

Host Chromosome: The host agent chromosome contains:

- an expiry age gene;
- a mate compatibility bit string signature; and
- a vulnerability bit string signature.

The expiry age gene is used at conception to determine an expiry age for the agent, by sampling a normal distribution with variance proportional to its magnitude. It is inherited from a randomly selected parent with a chance of mutation, according to a normal distribution. As this gene has no side-effects, it is expected that fast aging would always be selected against unless the scenario provides aging its own selective value.

The mate compatibility signature is used to determine whether two agents are capable of mating. Compatibility is determined by testing whether the Hamming distance between the strings is greater than a fixed mating variance threshold (see Figure 2). The signature is inherited (via crossover) with a chance of mutation flipping each bit copied. This mechanism allows for the diversification of the mate signatures and thus the emergence of sexually isolated subpopulations, i.e., new species.

The vulnerability signature is used as an interface for parasite interaction. It is inherited and mutated in the same fashion as the mate compatibility signature. Its function is discussed in detail in the parasite section below.

Parasite Agents: The parasite agents live off the host agent population. There may be an unlimited number of parasites living off a single host; however, if the host has non-positive health, it cannot carry any more parasites, and will die when next updated. Figure 3 shows the algorithm

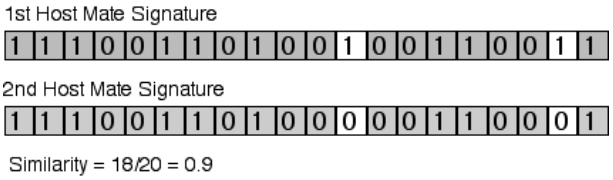

Figure 2: Example interaction between two host mate signatures. In this case, if the mate threshold parameter is set at a value lower than, or equal to, 0.9, the agents may mate, and thus are of the same species. (Note: all signatures in the simulation are 100 bits in length, not 20 as in this illustration.)

used for updating parasites, which we now discuss. Each cycle all parasite agents are transmitted to a new host in their neighbourhood, if there is one, which is randomly selected if there is more than one. Occasionally a parasite will become airborne (with a probability determined by a gene in its chromosome) and is transmitted to a random cell on the board and a random host within that cell, if there is one. The co-ordinates of the destination cell are determined by sampling a normal distribution. If the new location is unoccupied, the parasite fails to attach with a host and dies. Becoming airborne provides the parasite population the opportunity to infect new populations.

When a transmission is successful, airborne or otherwise, the parasite agent attempts, twice, to steal health from its host and use it to clone offspring (one for each successful health steal), which will act during the next cycle. Infection and reproduction (i.e., virility) are based on an interaction between the parasite and host chromosomes, discussed below. After the parasite has attempted reproduction, it dies. To ensure that the parasite population is never completely eradicated, there is a small probability a new parasite will be generated for every host agent updated.

```
transmit parasite to new host
if successful infection then
   for all reproduction attempts do
      if host health > 0 AND successful virility test then
         decrement health of host
         clone and save offspring
```

Figure 3: Parasite Update Algorithm

Parasite Chromosome: The parasite chromosome has three components:

- an infection bit string signature;
- a virility bit string signature; and
- an airborne probability gene.

The success of infection and parasite reproduction is determined via the interaction of the parasite's infection and virility signatures and the host agent's vulnerability signature. The probability of a successful infection is determined by the following function of the Hamming distance between the parasite's infection signature and the host's vulnerability signature:

$$P(infect) = \sqrt{\frac{Hamming\ Dist(infect, vulner)}{Signature\ Length}} \quad (1)$$

Likewise, the probability of successful reproduction is determined by inserting the Hamming distance between the parasite virility signature and the host vulnerability signature in the same function (see Figure 4). Both signatures are inherited from the parent parasite with a probability of mutation flipping each bit value.

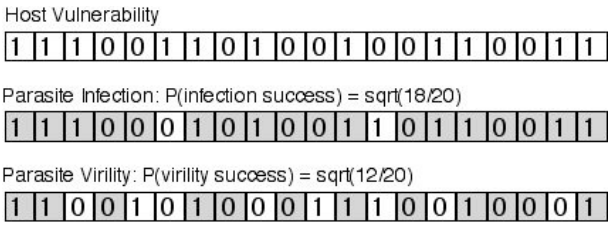

Figure 4: Example interaction between the host vulnerability signature and the parasite infection and virility signatures. (Note: all signatures in the simulation are 100 bits in length, not 20 as in this example.)

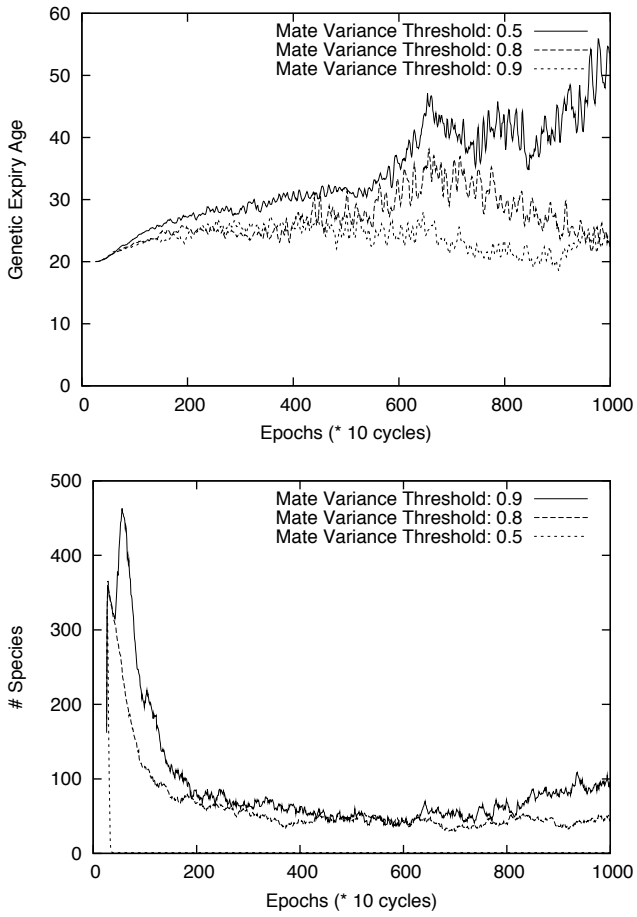

Figure 5: Figures tracking the evolution of (a) genetic expiry age and (b) number of species, for select cases of mate variance. Each of these plots represents a single simulation run. Resulting genetic expiry age and number of species are summarised in Figures 6(a) & 6(b).

Experiments

Evolution of Aging

In Woodberry et al. (2007) we demonstrated that our hypothesis of aging for the sake of diversity can be correct in a classical group structured simulation; here we extend that work, demonstrating its possibility also in a species selection scenario. In order to explore the effects of species groups on the evolved aging rate, simulations were run with a variety of mate variance thresholds (see Figures 5(a) & 5(b)) — a low threshold will allow host agents with greatly differing signatures to mate, reducing the number of species, whereas greater thresholds are more restrictive and thus produce a greater frequency of speciation. The resultant evolved genetic expiry ages and number of species for a range of mate variance thresholds are summarised in Figures 6(a) & 6(b).

From Figure 6(a) we can see that, as expected, the number of species present in the simulation increases as the limiting mate variance threshold increases. From Figure 6(b) we can see that as the mate variance threshold increases, and thus the number of species and the strength of inter-species competition increase, the expiry age gene evolves for shorter life spans. It is noteworthy that even when there is only one species, and thus no inter-species competition, the expiry age gene evolves to an equilibrium; this must be based simply on a background kin selection pressure, since there is no group or species structure. The species selection pressure acting on top of kin selection drives the aging rates higher — i.e., it drives lifespans downwards.

We conducted additional experiments to analyse the effect of varying the parasite virility (see Figure 7). We would expect virility to evolve via kin and group selection mechanisms in real parasite populations, however for simplicity we chose a parametric implementation of virility, enabling these experiments. These results show that when the parasites are over-virulent, the host population is quickly driven to extinction and consequently the number of species drops to zero. When the parasites are under-virulent, they fail to maintain a foothold in the host population. In consequence of this,

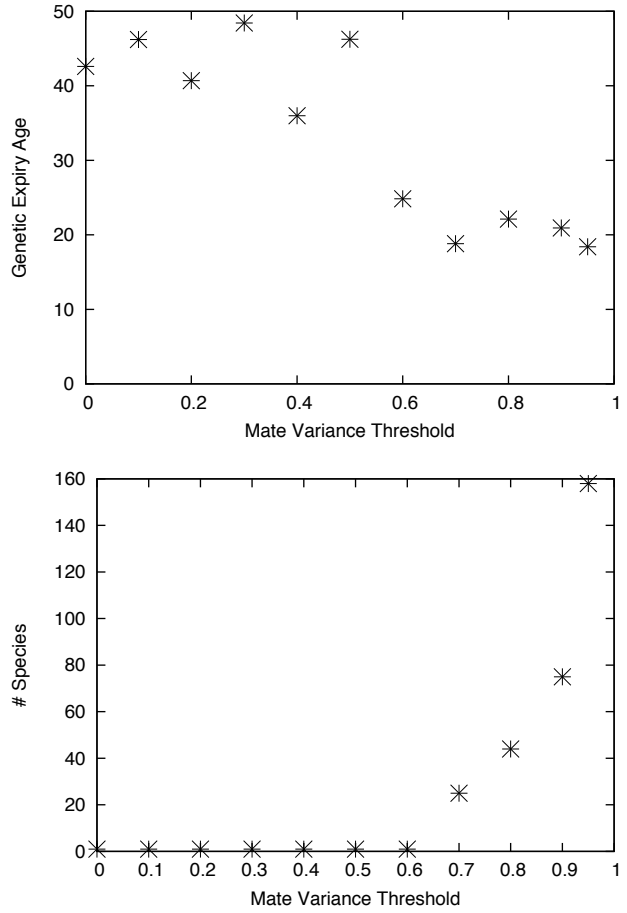

Figure 6: Resulting (a) number of species and (b) evolved expiry age, after simulation runs completed, with varied mate variance.

there are fewer dead-zones (where the parasites have killed off the host population), and the host population, which consumes the same amount of food regardless, spreads across the board, becoming less dense, which results in a greater rate of speciation.

Conclusion

The evolution of aging remains a puzzling phenomenon. Attempts to explain varying aging rates via individual selection have led many biologists to propose that it is non-adaptative — a side effect of some other beneficial trait — however, experimental evidence points to it being an adaptation. We argue that a primary benefit of aging is the generation of genetic diversity, which is of particular value in co-evolution scenarios. The evolution of altruistic traits such as adaptive aging requires an explanation of a selection mechanism that goes beyond the individual, such as kin and group selection. Punctuated equilibrium theory strongly suggests that species can support group selection. We have demonstrated with our

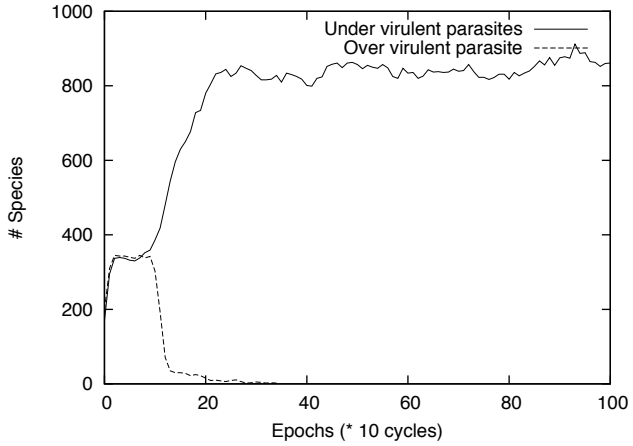

Figure 7: Experiments conducted varying the virulence of the parasites, i.e., changing the exponential in Equation 1, to 0.4 (under-virulent) and 0.6 (over-virulent).

simulation that species-level selection of an altruistic trait, in particular faster aging rates, can indeed occur.

References

Agustí, S., Satta, M., Mura, M., and Benavent, E. (1998). Dissolved esterase activity as a tracer of phytoplankton lysis: Evidence of high phytoplankton lysis rates in the northwestern mediterranean. *Limnology and Oceanography*, 43:1836–1849.

Belenky, P., Racette, F., Bogan, K., McClure, J., Smith, J., and Brenner, C. (2007). Nicotinamide riboside promotes sir2 silencing and extends lifespan via nrk and urh1/pnp1/meu1 pathways to nad+. *Cell*, 129:473–484.

Bell, G. (1982). *The Masterpiece of Nature: The Evolution and Genetics of Sexuality*. Croom Helm, London.

Bredesen, D. (2004). The Non-Existent Aging Program: How Does it Work. *Aging Cell*, 3:255–259.

Eldredge, N. and Gould, S. J. (1972). Punctuated Equilibria: An Alternative to Phyletic Gradualism. In Schopf, T. J. M., editor, *Models in Paleobiology*, pages 82–115, San Francisco. Freeman, Cooper and Company.

Gould, S. (2002). *The Structure of Evolutionary Theory*. Harvard University Press.

Gould, S. (2007). *Punctuated Equilibrium*. Harvard University Press.

Grimm, V., Revilla, E., Berger, U., Jeltsch, F., Mooij, W., Railsback, S., Thulke, H., Weiner, J., Wiegand, T., and DeAnglis, D. (2005). Pattern-Oriented Modeling of Agent-Based Complex System: Lessons from Ecology. *Science*, 310:987–991.

Hamilton, W. (1964). The Genetical Evolution of Social Behaviour, I and II. *Journal of Theoretical Biology*, 7:1–52.

Maynard Smith, J. (1976). Group Selection. *Quarterly Review of Biology*, 51:277–283.

Mayr, E. (1963). *Animal Species and Evolution*. Harvard University Press, Cambridge.

Medawar, P. (1952). *An Unsolved Problem in Biology*. H.K. Lewis, London.

Mitteldorf, J. (2004). Aging Selected for its Own Sake. *Evolutionary Ecology Research*, 7:1–17.

Skulachev, V. (1997). Ageing is a Specific Biological Function Rather Than the Result of a Disorder in Complex Living Systems: Biochemical Evidence in Support of Weismann's Hypothesis. *Biochemistry (Moscow)*, 62:1191–1195.

Weismann, A. (1889). *Essays Upon Heredity and Kindred Biological Problems*. Clarendon Press, Oxford.

Williams, G. (1957). Pleiotropy, Natural Selection, and the Evolution of Senescence. *Evolution*, 11:398–411.

Wilson, D. and Sober, E. (1994). Reintroducing group selection to the human behavioral sciences. *Behavioral and Brain Sciences*, 17:585–654.

Woodberry, O., Korb, K., and Nicholson, A. (2007). A Simulation Study of the Evolution of Aging. *Evolutionary Ecology Research*, 7:1077–1096.

Woodberry, O., Korb, K. B., and Nicholson, A. E. (2005). The Evolution of Aging. In *The Second Australian Conference on Artificial Life (ACAL 2005)*, Sydney, Australia.

Wynne-Edwards, V. (1962). *Animal Dispersion in Relation to Social Behavior*. Oliver and Boyd, Edinburgh.