Chapter 1

The Evolution of Aging

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Inclusive fitness theory [1], better known as kin selection, has often been cited as an alternative to group selection as a way of explaining the evolution of altruistic behavior. However, an evolving understanding of inclusive fitness has seen it redefined, by its creator, in terms of levels of selection, leading to a blurring of the distinctions between the two. Hamilton [2] suggests that if a distinction is to be made between group and kin selection, the term 'group selection' should only be used when there is no reliance on kin associations.

Based on the early group selection model of Gilpin [3] for the evolution of predatory restraint, Mitteldorf [4] designed an ALife simulation that models the evolution of aging and population regulation. Mitteldorf sees the evolution of aging as a case of 'extreme' altruism " ... in the sense that the cost to the individual is high and direct, while the benefit to the population is far too diffuse to be accounted for by kin selection" [4, p. 346].

We demonstrate that Mitteldorf's simulation is dependent on kin selection, by reproducing his ALife simulations and then introducing a mechanism to remove all and only the effects of kin selection within it. The result is the collapse of group selection in the simulation, suggesting a new understanding of the relation between group and kin selection is needed.

1.1 Introduction

The evolution of an aging trait would appear to be in direct conflict with the individual selection concept of natural selection. Individual selection refers to selection of the organism with the greatest individual fitness, measured as ability to survive and reproduce, or simply the individual's expected number of descendants. It is easy to see that an organism exhibiting a trait, such as aging, which by definition reduces its own survivability, will leave fewer descendants than a competing organism without the trait. The benefits of an aging trait, if there are any, could only be received by organisms other than the organism exhibiting the trait. For this reason such a trait, if beneficial, must be altruistic. In order to give an explanation of such an altruistic adaptation, one must call upon a mechanism of selection which incorporates such altruistic benefits, or otherwise deny that it is an adaptation.

There have been two notable attempts at explaining such a mechanism, these are: group selection, proposed by Wynne-Edwards [5], and inclusive fitness theory, proposed by Hamilton [1]. Group selection differs from individual selection in that it is the *group* rather the *individual* organism that selection acts upon. Group fitness is measured as the group's ability to prolong the period before extinction (group survival) and to produce emigrants and pioneer new groups (group reproduction). Maynard Smith suggested that this could be measured simply as the probability of the group producing a successful pioneering emigrant before extinction [6]. Group selection would sometimes act in opposition to individual selection, especially with selfish traits which may indirectly cause the group's extinction, such as unsustainable resource usage. Inclusive fitness theory gives yet another definition for the term 'fitness'. It differs from individual fitness in a further augmentation by the benefits, and harms, caused to the fitness of neighbours, weighted by their relatedness. This *gene* selection mechanism is often labelled kin selection, as neighbours will often be related to the individual holding the gene, inheriting the same genes, and hence an altruistic gene will increase its own inclusive fitness by benefiting copies of itself in kin. Inclusive fitness theory, often cited as an alternative to group selection in explaining altruistic adaptations, has since been redefined by Hamilton & Price into levels of selective forces [7; 2]. That is, they use within and between group levels of selection force which are paralleled to individual and group selection respectively.

Alternative explanations for the presence of an aging trait include the idea that it is not an adaptation at all but rather the side effect of another beneficial adaption or that it is the manifestation of mutational load. Such alternative explanations are appealing as it is difficult to conceive of

an altruistic benefit which could outweigh the obvious direct cost to the organism's individual fitness. Mitteldorf [8] argues that although popular opinion is that aging has a non-adaptive explanation, the experimental evidence suggests otherwise. Mitteldorf [4] provides a group selection simulation which ascribes the benefit of aging to demographic homeostasis. This is, populations which live longer lives will exhibit chaotic population dynamics and will be more likely to become extinct. Individual selection will result in the selection of organisms which live longer and longer lives, eventually causing chaotic population dynamics, leading to the group's extinction. Mitteldorf claims this "tragedy of the commons" can never be addressed by individual selection and that the differential extinction of groups outweighs individual selection to enforce growth restraint through birth restraint and aging. Mitteldorf claims that these traits " ... constitute 'extreme' altruism in the sense that the cost to the individual is high and direct, while the benefit to the population is far too diffuse to be accounted for by kin selection" [4, p. 346].

We argue that Mitteldorf's simulation is, despite Mitteldorf claims, reliant on kin associations and is therefore also a kin selection model. In Sections 1.2.1 – 1.2.3 we review the group selection debate in order to better understand the relationship between group and kin selection, and the mechanisms behind Mitteldorf's simulation experiments. In Section 1.2.4 we review Mitteldorf's group selection model and in Section 1.3 devise a method of removing kin selection from his model. In Sections 1.4.1 & 1.4.2 we replicate Mitteldorf's results with and without kin selection using our proposed method. Finally, we conclude with an analysis of the relationship between kin and group selection.

1.2 Background

1.2.1 *Group Selection*

The early generation of working group selection models, first proposed by Wynne-Edwards [5] and later adopted by Gilpin [3] among others, ascribed to group selection a major role in the evolution of population regulation. In these models it is the differing viability of the groups, together with their fecundity, that drives selection. Groups that contain selfish genes are more likely to become extinct and have less opportunity to produce emigrants to pioneer new groups. These types of models are reviewed by Maynard Smith [6] who describes a simplified version of Gilpin's predator-prey model (Figure 1.1).

Maynard Smith's model is divided into a number of discrete patches,

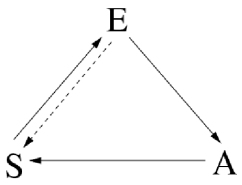

Fig. 1.1 States and transitions of the early group selection models.

each capable of supporting a single group. There are three different states each patch can take at a given time: empty (E), where the group population has become extinct; selfish (S), holding a group that contains *at least some* selfish individuals; or altruistic (A), holding a group that contains *only* altruistic individuals. Transitions occur between states due to extinction ($S \rightarrow E$) and migration (or mutation), either by re-population ($E \rightarrow S|A$) or by a selfish gene takeover ($A \rightarrow S$). In these models the main question is how the cooperation of the altruistic individuals will affect the factors of extinction and migration.

Maynard Smith observes that the fate of these models, when viewed in this manner, is dependent on a single parameter M, which is "the expected number of successful 'selfish' emigrants from an S patch during the lifetime of the patch" [6, p. 281]. A successful selfish emigrant is one that establishes itself and leaves descendants in a neighbouring E or A patch. If the expected number of emigrants from S patches is greater than one ($M > 1$) then the S patches will increase in frequency. Otherwise, the S patches will become extinct faster than they can found new groups and will therefore be selected out of the system. These models demonstrate that the mechanism of group selection is a logical possibility. However, it is debated whether or not the stringent conditions required for the evolution of an altruistic gene could be realised in nature [6; 9].

1.2.2 *Kin Selection*

Shortly after Wynne-Edwards' group selection mechanism was proposed, Hamilton [1] introduced inclusive fitness theory, which was initially seen as an alternative method of explaining the evolution of altruism. Inclusive fitness theory shifts the selection emphasis from the individual to the gene, be it held by the individual, or as a replica in another — it is the organism's individual fitness, augmented by the harms and benefits caused to the fitness of neighbours, weighted by their relatedness [1]. As the neighbours of the individual are also likely to be kin, the selection of the gene with the

greatest inclusive fitness is often termed kin selection. It is represented by Hamilton's rule [1], which holds that the criterion for the positive selection of a gene is:

$$\sum_i (b_i - c_i) r_i > 0 \tag{1.1}$$

where the subscript i denotes the i^{th} member of the species; r_i is the relatedness between actor and individual i; b_i is the benefit to the fitness of the individual i; and c_i is the cost to the fitness of the individual i. The relatedness r approximates the chance that a copy of the same allele at a given locus will be held by both the donor and recipient. For example, siblings have an equal chance of inheriting the alleles of either parent at a particular locus and hence have a relatedness of $\frac{1}{2}$. Considering the simple case of a gene which only bestows benefit on a sibling a at the cost of the gene carrier b, applying Equation 1.1, we can see that for a gene to be positively selected, $b_a \times \frac{1}{2} - c_b > 0$. That is, the benefit to the receiving sibling must be greater than twice the cost to the donor. The consequences of the theory are summed up by Hamilton [1] in two points,

(1) for a gene to receive positive selection it is not necessarily enough that it should increase the fitness of its bearer above the average if this tends to be done at the heavy expense of related individuals; and
(2) conversely, that a gene may receive positive selection even though disadvantageous to its bearers if it causes them to confer sufficiently large advantages on relatives.

1.2.3 *Price Equation*

Inclusive fitness theory was often cited as an alternative to group selection in explaining the evolution of altruistic behaviour until Price [7] and Hamilton [2] reformulated it into equations which identified different levels of selection: within- and between-group levels. Hamilton defines his groups unusually, as sharing *equally* the benefits of altruism, creating an important difference between his groups and real groups. As there is no preference for holders of the same genes, such as kin, altruistic genes are always selected against within the group, as any selfish free riders receive the same benefits as everybody else without paying the cost. This assumption is also made implicitly in the group selection models discussed earlier (Section 1.2.1). For an altruistic gene to be positively selected, the magnitude of between-group selection must be greater than within-group selection. That is, groups with a higher frequency of altruists can perform better by increas-

ing group fitness and hence increasing the relative size of altruistic groups in the population. If this increase outweighs the decrease in frequency of altruists within each group, the gene will increase in global frequency. The more varied the frequency of altruists across the groups, and the more benefit bestowed by the altruists on the group, the greater this between-group selection will be. Figure 1.2 (adapted from [10]) illustrates this effect in a population divided, for a period, into two groups with a varied frequency of altruists, represented by slices of pies. Within-group selection causes the altruistic "pie slice" to shrink in both groups. However, between-group selection causes an increase in the altruistic "pie size" resulting in an increase in the overall frequency of altruist genes.

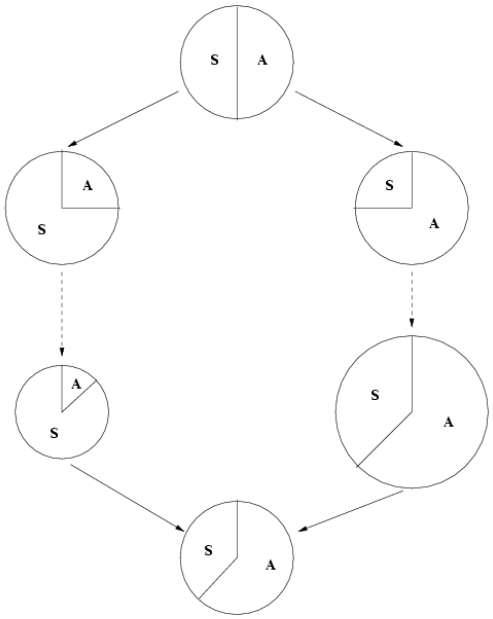

Fig. 1.2 Within- and between-group selection.

The relationship between the Price equation and group selection is considered by some as simply one of mathematical convenience [6]. On the other hand, Wilson [11] uses the equation as the basis of his 'multilevel selection', justifying redefining groups as 'trait groups' which might only exist for a part of their life cycle. As for conventional, partially isolated groups, Hamilton [2] points out that relatedness, due to complex kin associations, eventually builds up to:

$$r_g = \frac{1}{2E+1} \tag{1.2}$$

independent of group size, where r_g is the mean intra-group relatedness; E is the absolute number of emigrants per group, per generation. That is, virtually closed groups become highly related units, regardless of size.

1.2.4 *Mitteldorf's Aging Simulation*

Mitteldorf's simulation [4] is based on the early group selection models of Wynne-Edwards [5] & Gilpin [3], in which group extinction is the driving force (see Section 1.2.1). The simulation experiments demonstrate the evolution of population regulation through aging and birth restraint, which Mitteldorf claims " ... constitute 'extreme' altruism in the sense that the cost to the individual is high and direct, while the benefit to the population is far too diffuse to be accounted for by kin selection" [4, p. 346].

Mitteldorf's model is composed of a grid of 16x16 cells, each capable of holding a single group of approximately 100 individuals, which migrate to neighbouring cells at a rate of 10^{-5} individuals per cycle. Mitteldorf employs a logistic equation to model death by overcrowding:

$$\frac{dx}{dt} = bx(1 - \frac{x}{K}) \tag{1.3}$$

where $\frac{dx}{dt}$ is the population growth rate; x is the population size; b is the birth rate; and K is the steady state population level. When the population size is less than the steady state level ($x < K$) the population exhibits exponential growth, whereas, when $x > K$ the population exhibits exponential decline. Populations governed by this equation are normally well-behaved, approaching K asymptotically either from above or below. When a small delay is introduced into these equations, instead of approaching K asymptotically x will overshoot and oscillate. If this delay is further increased, the behaviour of the group undergoes a transition into dynamic chaos, resulting in fluctuations that cause extinction. The solution of population regulation, either by restraining birth rate or increasing aging rate, is permitted to evolve, by asexual reproduction, in Mitteldorf's simulations. If the individuals fail to regulate population growth, the group will experience chaotic fluctuations, causing it, and all its members, to become extinct.

In Mitteldorf's paper, he describes three simulations: the first is a calibration run, determining, in the absence of aging, the maximum sustainable birth rates; the second run permitted aging rates to evolve whilst the birth

rate was kept constant; and in the third run both aging and birth rates where permitted to evolve independently. In this paper we review only experiments concerning the evolution of an aging rate, holding birth rates fixed. Mitteldorf's simulation works because, below a certain threshold rate of aging, groups become extinct faster than they can export their members, as can be seen from Maynard Smith's analysis, $M < 1$ (see Section 1.2.1). This threshold rate of aging is determined from the migration rate — at higher migration rates the threshold rate will be lower, as groups require a shorter "lifetime" to export their members.

1.3 Methods

Our Mitteldorf simulation replica uses a 16x16 grid of cells (patches) with a steady state population level (K) of 100. Each cycle every agent had a fixed chance of reproducing asexually (b) with a probability of 0.045; and migrating to a neighbouring cell with a probability of 10^{-5}. The agent also had a chance of dying either by cell crowding or old age. The chance of a death by crowding is given by a probability proportional to the population sharing the site after a time delay of 50 cycles was applied (see Equation 1.3). Otherwise the agent would die of aging once it exceeded its genetically determined natural age of death. Each agent had: a position; an age incremented each time step; and an age of natural death, which was determined from a chromosome holding an evolving aging rate, a Gompertz function [12]. The Gompertz function is used in actuarial science to determine the probability a newborn will survive to an age, t. It is given by the function:

$$S(t) = e^{\frac{I(1-e^{Gt})}{G}} \tag{1.4}$$

where I is the intrinsic vulnerability, fixed at 0.001 in our simulations; and G is the Gompertz value, which is permitted to evolve. A lower Gompertz aging rate equates to a longer life. This aging rate was inherited and mutated with a probability of 10^{-3} by a normal distribution with variance of 0.01.

In order to test the reliance of Mitteldorf's model on kin selection, it required the identification and removal of kin associations to determine the effects on the model. Maynard Smith states that " ... kin selection can operate whenever relatives live close to one another, and hence can influence one another's chances of survival and reproduction" [6, p. 279]. This suggests that to remove kin selection it is simply enough to ensure that there is no correlation between the locations of the parent and child. This

could be done simply by selecting a location at random to spawn each new child. However, in the case of the Mitteldorf model (Figure 1.1) randomly spawning children to empty patches would negate the effects of migration founding new groups. To address this problem we adopt the mechanism of a compulsory adoption queue. As new children are born, they are placed at the end of the adoption queue and in their place a child is taken from the front of the queue and placed in the same cell as its new adopting parent. This way we can switch kin selection "on" or "off" and ensure that all other factors, such as group density, remain unaffected.

1.4 Results

In order to demonstrate Mitteldorf's simulation's reliance on kin selection, we replicated his simulation experiments (see Section 1.2.4), first without aging and then evolving an aging rate. Next, we repeated the simulation runs, implementing an adoption queue (see Section 1.3) in order to discern the impact of kin associations and kin selection. In our simulations we concern ourselves exclusively with the evolution of aging rate, while holding the birth rate constant.

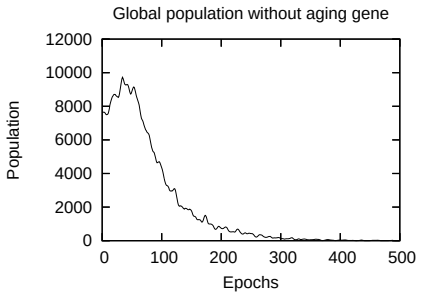

Fig. 1.3 Without aging the global population is quickly driven extinct through chaotic population dynamics within groups.

1.4.1 *Simulations Replicating Mitteldorf's Results*

We initially replicated Mitteldorf's simulation experiments (see Section 1.2.4): first simulating the group chaotic population dynamics without aging genes, and the expected result of eventual global extinction; and second with aging genes, and the expected evolution of a steady aging rate.

1.4.1.1 *Without Aging:*

Figure 1.3 was generated by averaging the results of 20 simulation runs, each lasting until all groups had become extinct, less than 500 epochs (×10 cycles). In these runs the simulation described in Section 1.3 was used, with all deaths by aging switched "off", removing all effects of the aging gene. This means that all agent deaths are only attributable to group crowding. Figure 1.3 shows that the groups, in agreement with Mitteldorf's findings, eventually drive themselves, through chaotic population fluctuations, into extinction.

1.4.1.2 *With Aging:*

Figures 1.4, 1.6 and 1.8 (i.e. the left side of the full page of figures) were generated by averaging the results of 20 simulation runs, each lasting 5000 epochs (×10 cycles). In these runs the simulation described in Section 1.3 was used. Figure 1.4 shows that the global population evolves to a steady state at approximately 6000 agents, indicating that, on average, approximately $\frac{2}{3}$ of patches are empty at any point in time. Figure 1.6 shows the percentage of all deaths that are attributable to aging, approximately 20%. Figure 1.8 shows the evolving rate of aging, the population evolves a Gompertz value, G, of 0.2 which equates to an expected age of natural death of approximately 50 cycles. These results are in accord with Mitteldorf's findings.

1.4.2 *Simulation Without Kin Selection*

To test the importance of kin selection on the model, we performed runs using the simulation described in Section 1.3 with kin selection turned "off", generating the Figures 1.5, 1.7, and 1.9 (i.e. the right side of the full page of figures), by averaging the results of 20 simulation runs, each lasting until all groups had become extinct, less than 1500 epochs (×10 cycles). As can be seen from Figure 1.9 the aging rate is quickly selected against, resulting in the increase of crowding deaths seen in Figure 1.7. Figure 1.5 shows the population eventually dying out, as in the runs without aging, unable to sustain itself with the high growth rates and consequent chaotic population fluctuations.

When kin selection is "on", we would expect the members of the same group to be more genetically related to each other than to members of different groups. Conversely, when kin selection is "off", we would expect that members of the same group will be just as closely related to members of different groups as to each other. In order to test this we measured the genetic relatedness between agents as the difference between their evolved

Gompertz values, G, performing a t-test to compare the means between groups. When kin selection was "on", the means of genetic relatedness of members of the same group and members of different groups were found to differ significantly ($t(59.4) = -28.26, p \ll .05$). When kin selection was "off", while there was some difference in the mean genetic relatedness of members of the same group and members of different groups, the difference was not statistically significant ($t(785) = 1.90, p > .05$).

1.5 Conclusion

Of the controversy over group selection and the importance of kin selection for the evolution of altruism Hamilton remarked:

> Because of the way that it was first explained, the approach using inclusive fitness has often been identified with 'kin selection' and presented as an alternative to 'group selection' as a way of establishing altruistic social behavior by natural selection. ... Kinship should be considered just one way of getting positive regression of genotype in the recipient, and that it is positive regression that is vitally necessary for altruism [2].

The idea is that inclusive fitness is more general than kin selection and might arise by other mechanisms than kin selection. What is needed for group selection of altruistic behavior is the positive association between group fitness and the altruistic genes required by the Price Equation. Such an association can arise by kin selection or by other means. But however it arises, it will result in differential group fitness leading to a spreading of altruism.

Our results are certainly consistent with this view. We would say that group selection is supervenient upon kin selection in the simulations we have conducted; that is, there are multiple possible ways of realizing group selection, kin selection being one [13]. Kin selection can lead to within group selection for altruistic behavior, so long as the groups are *not* Hamilton's groups which miraculously share the benefits of altruistic behavior identically across all group members (see [14] for an example). And kin selection can lead to between group selection for altruistic behavior, as our current study demonstrates. But the association between longevity of groups and altruism could in principle happen otherwise, for example, by chance or (at least in the case of artificial simulations) by intervention of a Designer. In our simulations, and so also in Mitteldorf's simulations, neither of these alternatives apply; indeed, there was no alternative to kin selection in making group selection operative. We believe that, although

kin selection is not logically necessary for group selection, it is practically necessary: nature also provides no alternative basis for differential group survival and reproduction. Hamilton's other suggestion that we reserve "group selection" for situations where kin selection is inoperative [2] would empty the term of all practical application.

Thus, and as the results of Section 1.4.2 show, Mitteldorf's model is dependent on the kin associations within its groups and therefore is a kin selection model. The operations of kin selection may be too diffuse to be seen by the naked eye, but they can easily be seen in t-tests over simulation runs, which we consider the more reliable instrument.

We can see that Mitteldorf's groups are in fact highly related, as new groups are founded by small groups: indeed, in Mitteldorf's asexually reproducing, sparse and low migrating rate model, most groups will be founded by a single individual and have little or no contact with other groups throughout its lifetime. This results in groups of individuals which are practically clones of each other. We can also see from Hamilton's observation (see Section 1.2.3) that the genetic relatedness builds up in virtually closed groups to approximately $\frac{1}{2E+1}$. Both these points, separately and combined, argue strongly that Mitteldorf's groups are highly related units.

Future work. As a follow-on to this study, we are developing a simulation which tests the Weismann hypothesis [15], an adaptive explanation of the evolution of aging. The Weismann hypothesis attributes the benefits of aging to "making room for the young". That is, a population that ages will turnover faster, promoting genetic diversity and adapting more nimbly to a changing environment. In our simulation a host population co-evolves with a disease population. Individual selection within host groups results in the evolution of longer living hosts and consequently groups with less diversity. Groups of hosts with less diversity are more easily exploited by the disease population and are less successful at producing emigrants. This differential success of groups counterbalances the selection against aging within groups, resulting in the evolution of a steady aging rate.

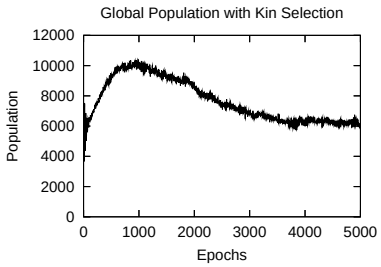

Fig. 1.4 With aging and kin selection the global population evolves to a steady state of approximately 6000 agents.

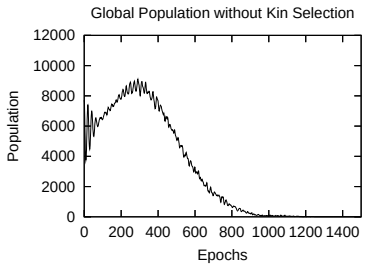

Fig. 1.5 Without kin selection the global population is quickly driven extinct through chaotic population dynamics within groups after the aging gene has been selected out of the system.

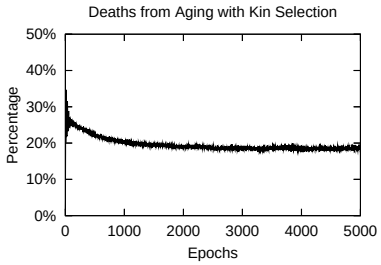

Fig. 1.6 With kin selection the type of deaths evolves to a steady state with approximately 20% of deaths attributable to aging.

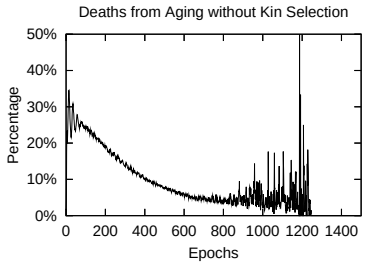

Fig. 1.7 Without kin selection the aging gene is selected against, resulting in the increase of crowding death as cause of agent death.

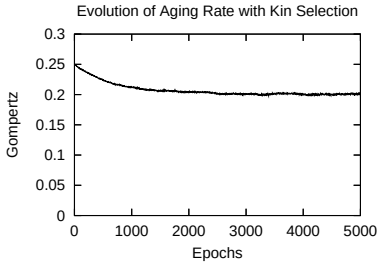

Fig. 1.8 With kin selection an aging rate evolves to a steady Gompertz value of 0.2 (approximately 50 cycle lifetime).

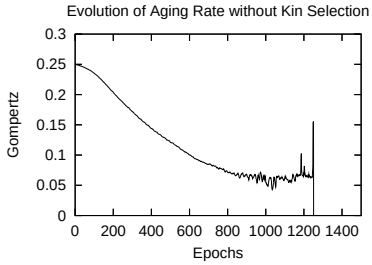

Fig. 1.9 Without kin selection the aging gene is quickly selected out of system.

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