Simulation of the evolution of aging: effects of aggression and kin-recognition

Svetlana Krivenko[1], Mikhail Burtsev[2]

[1] Moscow Institute of Physics and Technologies, Institutsky per., 9, Dolgoprudny, RU-141700, Russia
[2] Keldysh Institute of Applied Mathematics, 4 Miusskaya sq., Moscow, RU-125047, Russia
[1] skrivenko@gmail.com, [2] mbur@ya.ru

Abstract. Current biological theory has no commonly accepted view on the phenomenon of aging. On the one hand it is considered as an inescapable degradation immanent to complex biological systems and on the other hand as outcome of evolution. At the moment, there are three major complementary theories of evolutionary origin of senescence – the programmed death theory, the mutation accumulation theory, and the antagonistic pleiotropy theory. The later two are rather extensively studied theoretically and computationally then the former one is paid less attention. Here we present computer multi-agent model of aging evolution compatible with theories of programmed death and mutation accumulation. In our study we test how presence of aggression and kin-recognition affects evolution of age dependent suicide which is an analog of programmed death in the model.

Keywords. Aging, senescence, evolution, simulation, model, artificial life, Weismann, programmed death, mutation accumulation, cooperation.

Introduction

One of the fundamental problems of biology is the problem of phenomenon of aging [1,2]. There are two alternative approaches to the explanation of senescence. The first assumes that the aging is an immanent feature of all living matter. For example, the disposable soma theory [3,4,5] considers senescence as decline in somatic maintenance and repair. The second approach to the problem of aging is constituted by the evolutionary theories.

Evolutionary theory has no straightforward account for the aging. It is obvious that the death makes reproduction and, as a consequence, further proliferation of organism's genes in a population impossible. Therefore, selection should favor increase of an organism's lifespan leading to a more numerous progeny. Quoting the pioneer of aging research August Weismann:

> "This brings us face to face with one of the most difficult problems in the whole range of physiology,-the question of the origin of death. As soon as we thoroughly understand the circumstances upon which normal death depends in general, we shall be able to make a further inquiry as to the circumstances

> which influence its earlier or later appearance, as well as to any functional changes in the organism which may produce such a result." [6 p.20].

Weismann put forward the theory of evolutionary emergence of death known today as the theory of programmed death or phenoptosis [7]. His hypothesis is:

> "I consider that death is not a primary necessity, but that it has been secondarily acquired as an adaptation. I believe that life is endowed with a fixed duration, not because it is contrary to its nature to be unlimited, but because the unlimited existence of individuals would be a luxury without any corresponding advantage." [6 p.24].

The hypothesis of programmed death is difficult to test; moreover Weismann proposed no plausible evolutionary scenario for emergence of "suicidal" adaptation.

At the middle of the 20th century other theories of senescence evolution were proposed. In accordance with the Medawar's theory of mutation accumulation [8-12] the aging is a result of accumulation of deleterious mutations which reveal their effect only in an old age. It is supposed that this kind of mutations is weakly affected by selection due to mortality caused by external factors. The other evolutionary theory of aging is the antagonistic pleiotropy theory suggested by Williams [13]. This theory relates aging to accretion of mutations which have the positive effect in a young age and the negative in an old age.

Theories of Weismann, Medawar and Williams propose independent causes of the senescence evolution. The hypothesis of programmed death assumes that phenoptosis itself has adaptive value. Mutation accumulation theory rests on the neutrality of senescence mutations. In the antagonistic pleiotropy theory a "death" side of mutation hitch-hike on an adaptive young age side. All three mechanisms are not incompatible and may comprise the holistic picture of aging evolution.

There are some theoretical studies of the senescence evolution based on the mutation accumulation and antagonistic pleiotropy approaches [2,11-12,14-18] but no recent studies in the framework of programmed death theory. We propose an evolutionary model of aging which allows exploration of the hypotheses of programmed death and mutation accumulation by means of computer simulations. In our model possible actions of individuals are independently affected by age, hence antagonistic pleiotropy is impossible. In the original formulation the theory of programmed death relates adaptiveness of senescence with cooperation, i.e. with "giving way" for new generations. Therefore, if this theory is accepted then evolution of aging should be affected by the strength of selection towards cooperation. So, we simulate evolution with varying strength of selection and ability for kin cooperation to observe their effect on plausibility of programmed death strategies emergence in the model.

Model

This computer model is a development of the previous one which was used for simulation of social evolution [20] and cooperation [19,21].

The two-dimensional artificial world in our model is divided into cells, which either contain a resource bundle or are empty. An empty cell can acquire a resource

bundle with a certain probability per time step and lose it when resource is consumed by an agent. Agents are characterized by a set of receptors and effectors connected by a neural net. Each effector is responsible for a particular action. Agents can do nothing (rest), consume the resource bundle if it is present (eat), produce offspring (divide), go forward to a neighbour cell (move), make a turn to left or right (turn), attack another agent if present in the same cell (attack), and commit suicide (die). All actions spend energy taken from the agent's internal store. If internal energy is completely depleted, the agent "naturally" dies. The least energetically demanding action is rest, the most demanding is attack. Consumption of resource increases the internal store of energy subject to an upper limit (the maximum energy that can be stored). When an agent divides, one offspring is created and placed in the same cell as the parent. The parent then transfers half of its energy to the offspring. When one agent hits another, the victim loses an amount of energy, which is gained by the attacker (energetic costs of actions provided in the table 2).

Sensory inputs of agents include its internal store of energy, whether there are resources in the agent's field of vision (the cell it is in, the neighbour cell in front of the agent, and the cells on the right and left), and how many other agents are in the field of vision. Each agent has external phenotype that is coded by a vector of integer values (markers). The markers do not influence behaviour but function only as indicators of similarity. The euclidian distance between an agent's markers and the markers of another agent in the cell (a potential subject for attack) is also a sensory input. An agent perceives its own age. Value of an age input grows with actual age of an agent until it reaches maximum at double average population age. After the value of age sensory input remains constant. Behaviour of an agent is controlled by a simple one-layer neural net. Both weights of the neural net and external markers are inherited by the offspring when an agent divides, subject to a set rate of mutation.

Behaviour of agents is governed by a simple control system in which each output associated with a specific action is connected to sensory inputs from the environment or the internal state of the agent. The control system is linear and functions similarly to a feed-forward neural network with no hidden layer. To calculate the output vector **O** of values, the input vector **I** is multiplied by a matrix of weights **W**, which are constrained to lie in the range $[-W_{\max}, W_{\max}]$:

$$O_j = \sum_i w_{i,j} I_i \ . \qquad \textbf{(1)}$$

At each time step, the agent performs the action associated with the maximum output value (note that the order in which agents act is randomly shuffled every step). The input vector **I** is populated with information about the presence of resource and other agents in the field of vision (the cell where the agent is, the neighbour cell in front of the agent, and the cells on the right and left), the level of internal resource, the euclidean distance between marker vectors of the agent and its partner for potential interaction, and own age. A full list of input variables and their definitions are given in Table 1. At the start of simulation, an initial population was formed from the agents with the same matrix of weights **W**. All the weights in this matrix were set to zero except for three that defined the following simple strategy: move if a resource bundle is in the forward cell; eat if a resource is in the current cell; divide otherwise.

Correspondence between outputs and actions, and how changes of the internal resource r depend on actions are summarized in Table 2.

To speed up simulations, all variables were integers. For all simulations, the size of the world was 900 cells, W_{max} was 1,000, r_{max} was 5,000, the dimension of the marker vector was 10, and its values were bounded by $[-W_{max}, W_{max}]$.

If the agent executes the action 'divide', its offspring is placed in the same cell. The genome of the offspring is constructed in the following way: first, for every weight of the control system, a random value uniformly distributed on the interval $[-0.03W_{max}, 0.03W_{max}]$ is added; (2) for every component of the marker, a random value uniformly distributed on the interval $[-0.15\ W_{max}, 0.15W_{max}]$ is added.

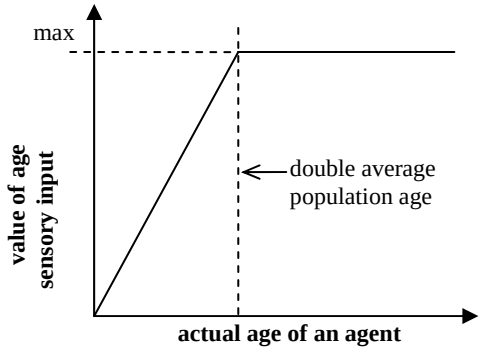

Fig. 1. Dependence of value of age sensory input on actual age of an agent.

Table 1. List of input variables and their definitions

Input variable*	**Value**
I_1	Bias constant, k
I_2, I_3, I_4, I_5	k if there is resource bundle in the field of agent's vision; 0 in the opposite case
I_6, I_7, I_8, I_9	cN_c, where c is a constant, N_c is the number of agents in the given cell of the field of agent's vision
I_{10}	Value of internal resource, r
I_{11}	$r_{max}-r$
I_{12}	$\sqrt{\sum_i (\overline{m}_i - m_i)^2}$, where $\overline{m}$ is a centroid of markers of all agents at the current cell
I_{13}	$\frac{k \cdot \sqrt{\sum_i (m_i^p - m_i)^2}}{2M_{max}}$, where m^p is a marker of partner to interact
I_{14}	see fig. 1.

*Note that I_1 is a constant and that I_2 to I_5 are binary variables (k is a functional analogue of unity and was set equal to r_{max}, where r_{max} is the maximal possible value of stored internal resource minus the energy storage capacity).

Table 2. The energetic costs of an agent's actions*

Output vector	**Action**	**Change of internal resource *r*†**
O_0	Rest	$-0.001r_{max}$
O_1	Turn left	$-0.002r_{max}$
O_2	Turn right	$-0.002r_{max}$
O_3	Consume the resource bundle	$+0.04r_{max}$‡
O_4	Move	$-0.004r_{max}$
O_5	Divide	$-0.004r_{max}$§
O_6	Fight (randomly chosen agent in the cell)	The cost of attack is 0.1 r_{max}; the gain is $+0.2r_{max}$ if internal resource of the victim is $r_n \geq 0.2r_{max}$ and $+r_n$ otherwise; the victim loses $-0.2\ r_{max}$.
O_7	Die	$r = 0$

*Note that r_{max} is the energy storage capacity.
†This scheme of setting parameter values reflects our assumption that the energetic cost of movement (move, turn left, and so on) is greater than the cost of resting, whereas the cost of attack is much greater than the cost of movement. Note that energetic losses are indicated with a minus sign and gains with a plus sign.
‡Food appears in the cell with the constant probability of 1/80 and the amount of resource in the bundle was $0.04r_{max}$.
§When the agent divides it spends $0.004r_{max}$; half of the remaining energy is then transferred to the offspring.

Results

To study how strength of selection and ability to cooperate affect the evolution of aging we have conducted simulations with four modifications of the basic model. These four variants were produced by combination of variation in selection pressure implemented by switching on or off attacking action and ability to cooperate by switching on or off kin recognition.

With each modification of the model a series of runs were conducted. At the beginning of every run the world was populated with the same initial population (as described in the previous section) but with different seeds for random number generator. All the weights from self-age input and to "die" action were set to zero, so there were no bias in the population toward any particular age dependent and death strategy. Amount of the resources in the world allows population density up to ~0.75 agent per cell.

During the runs every agent executing "die" action was monitored. This allowed to locate runs in which the ages of suicides were accreted to the close range around

double average age in the population. Particular example of such run is presented on the figure 2. There is no correlation between the age of action “die” execution and double average age in the population at the on set of evolution. Then after a short transient around $3\ 10^6$ time step the death strategy emerges. Now the agents usually murder themselves at ages about double average population age, i.e. about maximal individually perceivable age (see fig.1). It is notable that the suicide strategy persists during considerable amount of evolutionary time which indicates presence of stabilizing evolutionary mechanisms.

The runs where the death strategy was evolved can be clearly identified by plotting distribution of suicide ages normalized to the double average population ages. Typical distributions for runs with emerged senescence, with no age related strategy and for the control run with disabled age input are presented on figure 3. Distribution for the run where no age dependent suicide evolved is similar to the run without possibility to perceive self-age and hence control behavior appropriately. On the other hand distribution for the case with evolved suicide strategy has remarkable peak at the maximal individually perceivable age.

With the aid of suicide ages distribution we found how many runs for each of four model modifications demonstrate evolution of death strategy (see fig. 4). The modification with attack action and kin recognition enabled had the highest plausibility of death strategy evolution (7 out of total 11 runs). When agents can fight each other but can not recognize kin the suicide strategy evolved in smaller fraction of runs (6 out of total 13 runs). The rest two “peaceful” modifications of the model had lowest proportion of outcomes with evolved senescence (6 out of total 13 runs).

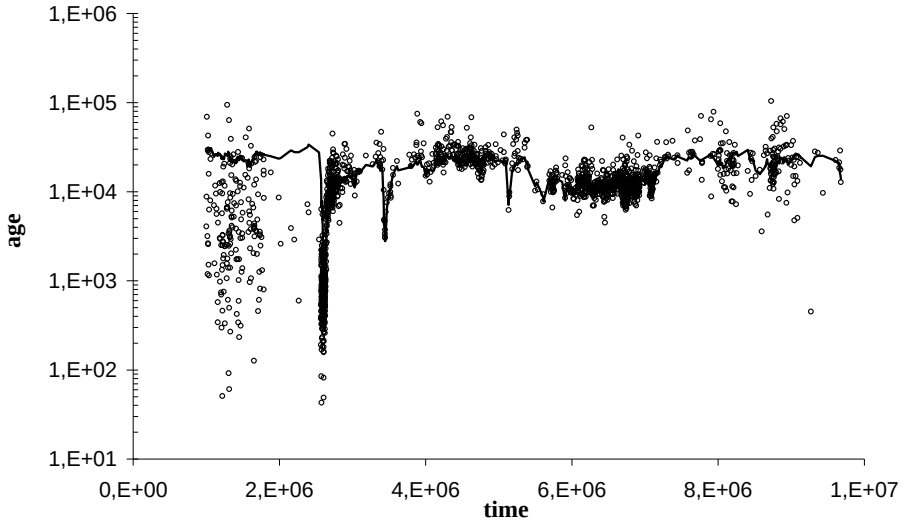

Fig. 2. The dynamics of suicide ages (circles) and double average population age (solid line). The age scale is logarithmic.

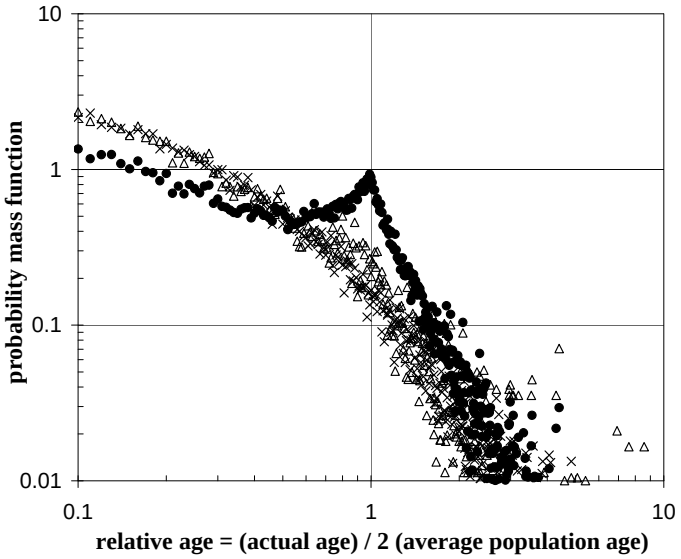

Fig. 3. Distributions of suicide ages normalized to the double average population age for the model without age input (crosses), for the run without evolved death strategy (triangles), for the run with evolved death strategy (solid circles).

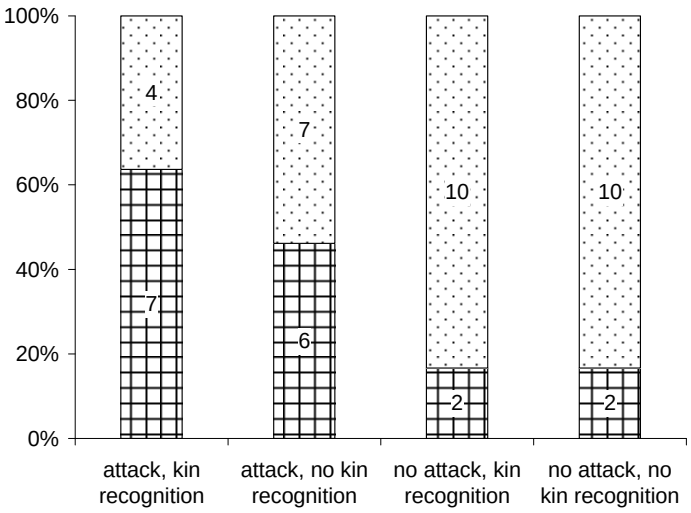

Fig. 4. Simulations outcomes for four modifications of the model. Each bar represents total of runs with evolved suicide strategy (grided area) and without it (dotted area). There is number of runs inside each area.

Discussion

The results of simulations can be outlined as follows:

- Age dependent death strategy evolved in the simulations without any predisposition or bias with only basic assumptions of agent's abilities to perceive self-age and commit suicide.
- Evolved death strategy demonstrated persistence over evolutionary time, i.e. evolutionary stability.
- Plausibility of death strategy emergence in the course of evolution in the model depends on the selection pressure (on agents' possibility to fight each other) and possibility of cooperation (through kin recognition).

The first result means that age dependent suicide strategy can invade population of immortal individuals, and the only prerequisite is presence of some mechanisms for determining self-age and phenoptosis.

The second result is a sign of presence of evolutionary processes stabilizing death strategy. These can be some evolutionary advantage for aging agents and neutral mutation accumulation. Analysis of simulation runs strongly supports the claim of adaptivness because the number of agents committing suicide is rather large which means that there should be selection against suicide, and, hence, the death strategy cannot be neutral in this case. The other thing which plays not in favor of neutrality is that evolution of age dependent suicide in the model is affected by aggression and cooperation which theoretically should have no direct impact on the process of neutral mutation accumulation.

Simulations results for different modifications of the model demonstrate that strongest factor affecting the evolution of senescence in our set up is an aggression. As we expected, two model's modifications with enabled attack action have significantly higher fraction of runs with emergence of death strategy. We also anticipated the strong effect of ability to cooperate; here the outcome is more controversial. If aggression is present in the population then kin recognition seems to increase plausibility of senescence evolution, else it has no or small effect. It should be noted that simulations were conducted with rather low population density which is a condition for weak selection toward cooperation in the model as was found in the previous work [21]. Higher population densities (>2) should provide stronger kin-selection and might increase effect of cooperation on evolution of age dependent suicide in the model. We plan to test it in our future work as continue with more detailed analysis of evolved death strategies.

Acknowledgments

References

1. Wachter, K.W., Finch, C.E., Between, Zeus and the Salmon: The Biodemography of Longevity. National Academy Press, Washington (1997)
2. Goldsmith, T.C.: The Evolution of Aging. Azinet LLC (2006) [http://www.azinet.com/aging/Aging_Book.html].
3. Kirkwood, T.B.L.: Evolution of ageing. Nature, 270 (1977) 301–304.
4. Kirkwood, T.B.L., Holliday, R.: The evolution of ageing and longevity. Proc. R. Soc. London Ser. B Biol. Sci., 205 (1979) 531–546.
5. Kirkwood, T.B.L., Austad, S.N.: Why do we age? Nature, 408 (2000) 233–238.
6. Weismann, A.: Essays Upon Heredity and Kindred Biological Problems. Clarendon Press, Oxford (1889)
7. Skulachev, V.P.: The programmed death phenomena, aging, and the Samurai law of biology. Exp. Ger. 36 (2001) 995-1024.
8. Medawar, P. B.: Old age and natural death. In: Medawar, P. B., ed.: The Uniqueness of the Individual, New York: Basic Books, (1958) 17-43.
9. Medawar, P. B.: An Unsolved Problem in Biology. In: Medawar, P. B., ed.: The Uniqueness of the Individual, New York: Basic Books, (1958) 44-70.
10. Rose, M.R.: Evolutionary Biology of Aging. Oxford University Press, New York (1991).
11. Gavrilov, L. A., Gavrilova, N. S.: Evolutionary Theories of Aging and Longevity. TheScientificWorldJOURNAL, 2 (2002) 339-356.
12. Hughes, K. A., Reynolds, R.M.: Evolutionary and Mechanistic Theories of Aging. Annual Review of Entomology 50 (2005) 421-445
13. Williams, G. C.: Pleiotropy, natural selection and the evolution of senescence. Evolution 11 (1957) 398-411.
14. Penna, T. J. P.: A bit-string model for biological aging. Journal of Statistical Physics, 78 (1994) 1629-1633.
15. Stauffer, D.: Life, Love and Death: Models of biological reproduction and aging. Electronic proceedings of the workshop: Monte Carlo and Structure Optimization Methods for Biology, Chemistry and Physics (1999).
16. Moss de Oliveira, S.: Evolution, Ageing and Speciation: Monte Carlo Simulations of Biological Systems. Brazilian Journal of Physics, 34 (2004) 1066-1076
17. Dzwinel W., Yuen D.A.: Aging in Hostile Environment Modeled by Cellular Automata with Genetic Dynamics. International Journal of Modern Physics C, 16 (2004) 357–376.
18. Lee, R.D.: Rethinking the evolutionary theory of aging: Transfers, not births, shape senescence in social species. PNAS 100 (2003) 9637–9642
19. Burtsev, M.S.: Tracking the Trajectories of Evolution. Artificial Life, 10 (2004) 397-411.
20. Burtsev, M.S.: Artificial Life Meets Anthropology: A Case of Aggression in Primitive Societies. In: M. Capcarrere et al. (Eds.): ECAL 2005, LNAI, 3630 (2005) 655 – 664.
21. Burtsev, M.S., Turchin, P.V.: Evolution of cooperative strategies from first principles. Nature, 440 (2006) 1041-1044.