Editorial

Is Aging a Failure or a Conquest of Natural Selection?


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When, a few months ago, the Editor-in-Chief of *Current Aging Science*, Prof. Lahiri, offered me the possibility of organizing a thematic issue of the journal about aging, I immediately thought of what I should consider for the pivotal point of the subject:

"When Darwin published his theory of evolution by natural selection, the main criticism was the contrast between the astonishing power attributed to natural selection, capable of shaping the eye, the brain and numberless very sophisticated characters, and its apparent incapacity to avoid senescence decay. This question was terrible; a possible deadly blow for the new theory, and Darwin knew its great danger. He had two possible answers: 1) (*Non-adaptive hypothesis*) The first was to postulate that natural selection could shape the infinite marvels of the living beings but could not preserve a body from the damage caused by the time. This was a postulate without any proof and had the flavor of an *ad hoc* hypothesis. Moreover, among the species, there were enormous differences in aging rates. It was also necessary to assume that the limits in the powers of natural selection were different from species to species: another postulate without any proof and with the flavor of an *ad hoc* hypothesis. 2) (*Adaptive hypothesis*) The second possible answer, chosen by Darwin, was to propose that the age-related progressive decline of all functions had some supra-individual advantage. Darwin hypothesized that aging was advantageous for the species, but he knew very well that this explanation was not proved and therefore weak and easily disputable. Likely, he preferred it because it did not, as the first answer did, undermine the roots of evolutionism, but the explanation was a temporary setback that needed a careful reassessment" [1].

About 150 years after Darwin's epoch-making proposal, the question has not yet been agreed upon unanimously.

I am convinced that any discussion about aging must somehow be preceded by a valid answer to this question, which may be summarized thus:

"Is aging a failure or a conquest of natural selection?"

In regard to this subject, I invited contributions from a number of professed supporters of either of these alternative theses, as well as from other scientists working in the field of aging, whose opinions would certainly be of great value.

The invitation (which I also extended to myself), was put as follows:

> *"This question, a pivotal point of biology as a whole, is not merely an abstract theoretical topic with no practical effects.*
>
> *For the first thesis, aging is only the accumulation of disparate damage and the control of aging is, therefore, achievable only in part and at the cost of enormous effort.*
>
> *Conversely, if aging is genetically programmed, this means that we can envisage the possibility that the phenomenon may be brought entirely under our control.*
>
> *Therefore, the theoretical arguments and empirical data that support or refute the two hypotheses must be carefully evaluated, without any uncritical acceptance of either of the two alternatives.*
>
> *I think that this is an opportunity for a forthright discussion in which the two positions may be compared, not from the viewpoint of a pre-established indisputable thesis but by basing the arguments only on theoretical or empirical scientific topics."*

The works of those who have accepted the invitation and formulated a highly valuable contribution are included in this issue. Their contributions have been evaluated by qualified referees with the criterion that a paper should not be excluded solely because it supports the ideas of the opposing camp. It is not, therefore, the aim of the thematic issue to arrive at a conclusion shared by all, a goal which is perhaps still somewhat premature, but represents, rather, a place of open discussion where the two contrary positions may debate: the reader will weigh up both the validity of the individual contributions and the validity of this thematic issue as a whole.

Given that I am a participant in the debate, firmly on the side of programmed aging thesis, it would be wrong of me to add comments about the works put forward and the specific arguments in support of or against either of the two theses, or proposing intermediate hypotheses.

I may only say, with extreme confidence, that the future of aging research will judge who is in the right and who is in the wrong.

However, a short description of the specific subject of each work is necessary.

Aledo & Blanco [2] propose an intermediate interpretation between the two opposite theses that are the topic of this thematic issue. Aging is interpreted as a "stochastic process out of the reach of natural selection" but, as "those genetic pathways influencing the rate of aging" are "targets of natural selection"; short or long longevities are always an adaptation.

The work of Bartke [3] documents and confirms the sound hypothesis that the availability of nutrients and the levels of anabolic hormones during development influence the longevity.

Cohen's contribution [4], while stressing that "there are problems with the longstanding non-adaptive paradigm", maintains that evidence does not support the opposite idea about aging as adaptation.

De Grey [5] reviews the arguments and the evidence in support of programmed aging thesis and maintains that "the unfortunate truth" is the non-existence of any aging program. Therefore, the struggle against aging requires an elaborate "divide-and-conquer panel of interventions to repair the damage that the body inflicts upon itself throughout life as side-effects of its normal operation."

The first work of Goldsmith [6] maintains that "new evolutionary mechanics theories and new discoveries support programmed mammal aging as well as programmed lifespan limitation in non-mammals" and "describes some of the many ancillary circumstances" that hamper the attainment of a shared solution for "the programmed / non-programmed conundrum".

The other work of Goldsmith [7] highlights the great importance of the question debated in this thematic issue for the medical research.

Katcher [8] maintains that aging may be explained through "basic principles of mammalian aging", supported by experimental data, and without "the constraints of evolutionary theory". This would implicate that the control of aging both "at cellular and higher levels of biological organization" is feasible.

The paper of Libertini [9] presents a series, divided in 10 groups, of empirical data and theoretical arguments in support of programmed aging paradigm and in contrast with the opposite view.

Another supporter of programmed aging paradigm, Mitteldorf [10], maintains that the "most promising possibility is that the machinery responsible for maintenance of the vibrant and youthful state of the body is never really lost, but de-commissioned by hormonal signals in the aging body; restoring a youthful signaling environment should then be sufficient to prompt the body to restore itself".

Olovnikov [11] proposes an original theory where aging is determined and regulated by the loss of particular hypothetical cell organelles ("chronomeres" and "printomeres"), "whereas telomere shortening is a bystander of this process". Only time will tell if this theory, a radical approach to the problem of aging, is correct or not.

The paper of Potter [12] analyses the complex relations between diseases, allelic variants and aging and their effects on longevity.

Skulachev MV, Severin SS & Skulachev VP [13] see aging as a program that increases the capacity of evolution, or evolvability, and discuss the conditions (moderate food restriction, heavy muscle work, a lowering or a rise in the external temperature, small amounts of metabolic poisons, low doses of radiation and other deleterious events, geroprotective psychological factors) in which this program appears to be decelerated to get additional resources for survival.

Finally, Vanhaelen [14] maintains that aging is the consequence of an age-related progressive alteration of a dynamical equilibrium in the cellular environment, and that there is an optimized compromise between maintenance requirements and evolutionary constraints.

I must conclude this editorial with my sincere and strong thanks to the authors for their useful contributions to this issue, the reviewers for their anonymous but crucial work, and above all *Current Aging Science* (Bentham) and its Editor-in-Chief, Prof. Debomoy K. Lahiri, for the trust placed in me in the preparation of this special issue. I sincerely hope that this confidence will be reflected in the approval of the readers of Current Aging Science.

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