Aging Happens by Default

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Abstract

With rare exceptions theories proposed to explain aging focalized in general on one specific aspect of the functioning of the organism. This does not seem reasonable, as the cause of such a general phenomenon cannot be pinpointed. One has to look for the phenomenon of aging in terms of the broad requirements needed for life to persist; the most fundamental requirement is energy expenditure, which inevitably follows the second law of thermodynamic. Hence aging is an unavoidable event, there is no alternative it is inherent to the process of existing.

Keywords: Energy requirements; Thermodynamic; Second law; Asymmetric cell division; Quantum reactions

Introduction

Theories in general considered the phenomenon either as the result of wear and tear, a depletion of a potential, a programmed type of event, or of some kind of advantage for the survival of the population where natural selection would play the main role. A theory like the protein error hypothesis has a cultural origin it is based on the belief that the two sister cells were identical. However, when division was studied in individual eukaryotic cells it was found that DNA synthesis and cell division are asymmetric (Macieira-Coelho 1982, 1995, 2007) leading to cumulative modifications. The apparent order observed when one studies a phenomenon globally disappears when it is studied at a lower scale. When a cell population divides there is a whole distribution of heterogeneity, which can keep going for a while. But eventually the system collapses the distribution of DNA between sister cells becoming chaotic [2-4].

When in post-mitosis a steady state cannot be reached either; at the molecular level, all is fuzziness, uncertainty, and probabilistic, which increase the further one goes down in the scale. A cell that would metabolize remaining exactly identical without leaving any modifications behind cannot exist. At the molecular level metabolism depends on energy transduction for the induction of the right molecular conformations to perform a biochemical reaction. Unavoidably there is a probability that not all molecules have the adequate functional shape to achieve the best result, there is a distribution of conformations with different efficacies [5]. The biology of conformation wears down during aging [6], which is one of the reasons why functions become increasingly less adequate. The deterioration of conformational flexibility occurs also at the macroscopically level leading to the structural reorganization of the organs [7].

When one goes further down at the molecular level uncertainty becomes more pronounced, at the particle level life is driven on the...
quantum edge [8]. For instance, DNA replication depends on base pairing which is provided by hydrogen bonds obtained with shared protons (the nuclei of hydrogen atoms). This base pairing is ruled by quantum mechanics, i.e. by uncertainty that originates infidelity [8]; enzyme reactions are also driven on the quantum edge.

Change is inevitable in biology, which leads to a decline in the probability of perpetuating the organism. The second law states that all systems spontaneously change in such a way as to decrease their capacity for subsequent change. A system driven by the utilization of energy has to follow the second law with entropy increasing inexorably. Hence there is no alternative to aging.

References

1. Bonafé M, Valensin S, Gianni W, Marigliano V, Franceschi C (2001) The unexpected contribution of immunosenescence to the levelling off of cancer incidence and mortality in the oldest old. Crit Rev Oncol Hematol 39: 227-233.

2. Macieira-Coelho A, Bengtson A, Van der Ploeg M (1982) Distribution of DNA between sister cells during serial subcultivation of human fibroblasts. Histochemistry 75: 11-24.

3. Macieira-Coelho A (1995) Chaos in DNA partition during the last mitoses of the proliferative life-span of human fibroblasts. FEBS Lett 1995 358: 126-128.

4. Macieira-Coelho A (2001) Asymmetric distribution of DNA between daughter cells with final symmetry breaking during aging of human fibroblasts. Prog Mol Subcell Biol 45: 227-242.

5. Cantor CR, Schimmel PR (1980) Biophysical chemistry. Part I: The conformation of biological macromolecules. WH Freeman 296.

6. Macieira-Coelho A (2016) Slowing down of the cell cycle during fibroblast proliferation. Cellular Aging and Replicative Senescence, Switzerland 4: 29-47.

7. Macieira-Coelho A (2014) Aging Facts and Theories. Control of cell replication during aging, Karger publishers, Basel, Switzerland 39: 24-44.

8. Al-Khalili J, McFadden J (2014) Life on the edge: The coming of age of quantum biology.