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Mammals die young!? Hypothetical age-dependent mechanism of self-destruction of mammals

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Abstract It is hypothesized that, in the evolutionary lineage from the mammal-like reptiles *Synapsida* to mammals, in addition to existing systemic and cellular mechanisms of aging arising appears particular age-related mechanism of self-destruction of organism mammals, which does not allow accumulation of individuals with the damage of genome in population. Age-related mechanism of self-destruction of organism mammals is a result of the evolutionary events linked with arising of phenomenon "postmitotic brain" in mammals.

Keywords: *aging, hypothesis, mammals, birds, RNA*

Introduction

The initial purpose of this work was, generalizing the most important facts and ideas, to show a possibility of further development a Dilman's ontogenetic model of aging. But in the process of work there was a need to revise some dogmas and common notions, and as a result, there was proposed a hypothesis/concept about an age-dependent mechanism of self-destruction of mammals (AMSM).

This hypothesis is about 10 years. The author decided to publish it only after Skulachev postulated his concept of phenoptosis. As well as after publication of reports about when the germ-line precursor cells are removed, life-span is increased dramatically the nematode *Caenorhabditis elegans* by Arantes-Oliveira et al [1] and Jiang et al [2], about, that accumulation of a ROS- induced changing occurs in neurons of pituitary gland and hypothalamus of cerebrum of human mainly. I.e. when it became clear that apparently hypothesis had a right to exist. In view of latitude of material, the work is presented in a most general way.

Premises and motivation.

In the proposed hypothesis Weismann's views recently reanimated by Skulachev about the aging and death, as "something secondary that appeared in the process of adaptation" are taken as the axiom [3]. In Skulachev's version, potential immortality has already considered not as luxury, but as mortal danger for population in contrast initial version of Weismann.

Even minimum quantity of individuals with the damaged genomes in population can have fatal consequences. If it be so, then evolutionary and reproductive advantages on the one hand are to get population better protecting from presence individuals with the damage genome, on the other hand must be occurred a selection of individuals, which better other were able to protect its gene from the damage. Such individuals must get old later and get chance leave the most numerous posterity. That is why in the process of evolution a mechanism that reduced to the minimum probability of changing itself a genetic program have to be arisen [4].

The propounded hypothesis is based on ideas of Dilman [5-7] who supposed that if stability of internal environment of organism is the obligatory condition of its existence, a programmed deviation of homeostasis is the condition of its development ("law of deviation of homeostasis"). In other words, age-dependent mechanism of juvenile postnatal development of organism exists, in the basis of which changing sensitivity of hypothalamus in responsiveness to feedback signals lies. During a period of adulthood, it enters in the pathological phase, and its further functioning brings about unfavourable changes in the level homeostasis, with the effect of formation of the main diseases. I.e. sensitivity of hypothalamus to the inhibitory influence of glucose, estrogens, corticosteroids and other factors that signalizing by way of a negative feedback control about state of the three main homeostatic system - energy, reproductive and adaptation is declining. So that with age cholesterol, insulin and cortisol levels in blood increases, toleration organism

to the glucose is also reduced, and other unfavourable changes occurs. A homeostatic disorder leads to the development of characteristic diseases for an elderly age or normal diseases of aging (term of Dilman, in humans it is the so-called main non-infectious diseases): -obesity, diabetes, atherosclerosis, cancrophilia, depression, metabolic immunodepression, hyper-tension, hyperadaptosis, autoimmune diseases and climax. In fact, Dilman considered an aging as the age-related formation and development of 10th normal diseases of aging and has offered a mechanism of their formation. Besides, he denied an age rate and enters a notion of ideal rate, - a characteristic rate of a homeostatic level during the most flowering age.

Developing these views, Dilman postulated the ontogenetic model of aging [8, 9] that was taken as the basis of the given hypothesis:

1. The realization of the body development program requires a programmed deviation of homeostasis.

2. In mammals, changing a threshold of sensitivity of hypothalamus in responsiveness to feedback signals in a postembryonic development plays lock-and-key role in the mechanism of arising a necessary deviation of homeostasis.

3. Conservation of this mechanism after the end of postnatal development is directly responsible for the transformation of program of development in the mechanism of aging and formation of age-connected diseases.

What molecular mechanisms in cells of brain bring about too sad changes to the mammal organism? There is an opinion that many processes of cellular aging are cell-autonomous i.e., and independent of external signals. But, cell-autonomous and systemic aging may coexist or trigger each other [10].

A number of researchers suppose that aging of multicellular organisms is determined by mitochondria of postmitotic cells and slowly renewed tissues because of the accumulation of mutagenous molecules mitochondrial DNA (mtDNA) [11, 12].

Phenomenon "postmitotic brain" of mammals is not only the best illustration of these models, but also the place where the accumulation of mutagenous molecules of mtDNA is a simple function of time and velocity of metabolism. Such conditions are created due to the following:

a) the mammal brain has very low apoptosis degree of neurons during life-span of organism [13];

b) neurons do not divide and are not renewable in the adult mammal organism. Only minimal regeneration of neurons is possible in the central nervous system.

It is not a secret that the dogma that brain is truly postmitotic is open to question, and it will be discussed below. But, to all appearance, the intensity of processes of neurogenesis and renewal is so low that cannot cause a significant correction to the scheme presented above.

With time, as a result of accumulations of mutagenous mtDNA molecules among most part of a population of neurons (including and hormone-synthesizing), defects of oxidative phophorylation with the effect of reducing a producing of adenosine triphosphate (ATP) appear and disturbances of functions and biogenesis of mitochondria, level of reactive oxygen species (ROS) production and accumulation of damaged structures of these neurons increases. As a rule, neurons with significant damage of mtDNA are subjected to a process of apoptosis in the first instance. In view of this, pool of hormone-synthesizing neurons in the process of aging also decreases by means of apoptosis. This process has a self-speed up nature. If this scheme would be integrated in the ontogenetic model of aging of Dilman, in this integrated aging model, the accumulation of damage mtDNA molecules in hormone-synthesizing neurons is necessary to consider as activate trigger and biological clock of juvenile development and aging [14].

Hence, a ROS-induced changing of an energy balance of neurons, can change sensitivity of neurosecreting brain elements in responsiveness to feedback signals, especially to hormonal stimulus, and apoptosis by the way reduction of pool neurosecreting neurons enlarges a functional load on the remainder, all this can lead to changing of the level of neurosecretion.

So, it is logical to suppose that the process of aging of mammals is closely connected with postmitotionary state of many neurons of their brain.

It is well known that amongst classes of the subphylum *Vertebrata* "postmitotic brain" is a unique phenomenon and it is found only among *Mammali*a.

Neuronal precursor cells persist in the adult vertebrate forebrain, residing primarily in the ventricular/subventricular zone (SZ). In vivo, SZ precursors yield progeny, which may die or give rise to glia. Yet, they may also generate neurons, which are recruited to restricted regions such as the avian telencephalon and mammalian olfactory bulb [15].

Classical studies of scientists since the end of the 18 th till nowadays showed a possibility of both reparative and physiological regeneration of nervous tissues (neurons) in cold-blooded vertebrates - fish, amphibians, reptiles [16-19].

To all appearance, the warm-blooded vertebrates - birds and mammals completely have lost the capacity of reparative regeneration.

Physiological regeneration of a nervous tissue of the warm-blooded vertebrates is a ticklish question, particularly in respect of mammals.

Opinions on this subject possible can be divided in such way:

Radical – to certain stage embrional or early postnatal development neurons are proparagated by mitosis, but after this stage -by amitosis. Pool of brain neurons can renew owing to a proliferation of cambial cells in the brain, which under certain conditions generate valuable neurons [16].

Cautiousness and dominant at present - except the olfactory bulb and hippocampus, new neurons are not normally generated in the adult mammal brain [19-21].

Conservative - in adult mammals, neurons have lost the capacity to form new neurons by division [22].

It was considered for a long time that birds have also a phenomenon "postmitotic brain", but studies of Goldman have shown, that similar judgement has not a real base under itself [23]. The neurogenesis continues in the brain of adult birds. These cells are born in the ventricular zone of the lateral ventricles. Young neurons then migrate long distances guided, in part, by radial cell processes and become incorporated throughout most of the telencephalon [24]. It is well known that, radial cells, which in mammals disappear as neurogenesis ends, persist in the adult avian brain. The presence of radial glia in the adult avian forebrain and their apparent absence in mammals is related to neurogenesis in adulthood, which occurs in birds and much less or not at all in mammals [25]. In this way, databases on this subject have not been well documented and inconsistent. One can be affirmed with certainty, that:

1. Neurogenesis continues amongst all classes of the subphylum *Vertebrata* throughout life.
2. Central nervous system (C.N.C.) of birds and mammals consists of postmitotic neurons in adulthood.
3. C.N.C. of the normal adult mammals has very low degree of age-related decline in neuron number through neuron death by way apoptosis and low renewing level pool of neurons by way neurogenesis [13].
4. In contrast the normal adult avian C.N.C. has high degree as apoptosis so and regeneration of neurons by way neurogenesis. Classical example - seasonal change of neurons quantity in songbirds.

Proceed from these considerations, it is highly reasonable to carry out comparison of the maximum longevity between species of *Mammalia* that have a phenomenon of "postmitotic brain" and species of other classes *Vertebrata* capable to regenerate neurons. The subphylum *Vertebrata* is presented either by cold-blooded and warm-blooded classes of animal. Considering difficulties of comparison between cold-blooded and warm-blooded animal in the maximum longevity, in this case, it is the most reasonable to conduct comparisons between homeotherm classes, i.e. the maximum longevity of *Mammalia* species to compare with the maximum longevity of *Ave*s classes species. It is accepted to consider that interspecific collations unfit for any proof, but they are a remarkable method to define hypotheses and their preliminary examination [26]. So, in this case, a similar comparison is quite appropriate.

For birds and mammals three traits have independent correlation with life-span: body weight, brain weight and metabolic rate [27, 28].

For mammals well marked relationship of life-span and the mass of brain is more distinctive, than with the mass of body [29, 30]. However, there is a tendency for birds to live longer than mammals with similar brain and body size despite their higher body temperatures and metabolic rates, that from standpoints of existed theories of aging is a phenomenon which hard to understand [31]. As it is clear from the results of collations, such a tendency is rather essential, for example, among animals which have weigt about 30 g, mammal species have maximum life-span from 2-h to 12 once smaller, than birds of same weight of body (see Table 1).

These circumstances give an occasion for supposition that the phenomenon of "postmitotic brain" is exactly the reason of a shorter life-span of mammals, as compared with birds.

Though it is completely impossible, exclude that similar vitality a result exceptional cellular resistance to oxidative damage. At least once, it is established for some types of cells organism of adult birds [32].

Hypothesis (concept) about age-dependent mechanism of self-destruction of mammals.

Analyzing the above and well-known biology data, I suppose, that a population which has mechanisms of purification from undesirable individuals which became dangerous for this population have to get the evolutionary advantages. Purification of population from the undesirable individuals by a way of a programmed suicide a phenomenon is widespread and it is found even in bacteria [33-35]. A phenomenon of similar nature should be attributed to arised in *Eukariota* in the evolutionary lineage from *Protozoa* to *Metazoa* phenomena of aging and age-related indivertible death. At present, the phenomenon of aging is

reduced to the small number of single cellular and organism processes defining rate of aging such as apoptosis, age- associated accumulation of mutations in mtDNA molecules, defects in cell cycle control, mitotic dysregulation, genome instability, telomere shortening, age-associated change in level of the neurohumor regulation and other cellular and organism pathologies [10]. Aging and death are not only the final acts in the development of any multicellular organism, but also a necessary condition of their existence and evolutionary progress. Generally, these processes in *Vertebrata* ensure aging and death of organism, but in the evolutionary lineage from the mammal-like reptiles, *Synapsida* to mammals in addition AMSM appears, which protects population from the accumulation of individuals with a damaged genome. AMSM damages systems of life ensuring of organism, whereby interrupts its current. To all appearance, it occurs at the moment of beginning of the first signs manifestation of negative influence of processes of cellular aging to organism and long before their terminations. AMSM is the result of evolutionary events, which caused the disappearance of cells of radial glia in adult mammal brain and rise of a "postmitotic brain"phenomenon. Postmitotic hormone-synthesizing neurons of brain in the process of aging accumulate mutagenous molecules mtDNA, as a result the level of ROS production and accumulations of damaged by them structures of these neurons increases. Perhaps, these slow ROS-linked processes are a trigger and biological clock for AMSM and they cause the change of sensitivity of neurosecreting brain elements in responsiveness to feedback signals. Furthermore, neurons with significant damage of mtDNA are subjected to a process of apoptosis. In view of this, a pool of hormone-synthesizing neurons in the process of aging also decreases by means of apoptosis. As a result, it changes the production, processing, and degradation of neuroendocrine hormones and neuropeptides in hormone-synthesizing brain elements, i.e. by altering regulation of physiological processes through the neuroendocrine system. In the organism elements of "homeostatic anarchy" arise which in one's turn, has to determine the formation of characteristic diseases of the old age, because of which organism has to die. Similar mechanism for a better preservation of genomes from damage is only in mammals and seems to be absent amongst other *Vertebrat*a.

The recent discoveries are greatly complementing and made some changes to the above-stated notions.

In Jiang et al study, the established fact is that in the hypothalamus from aged mice increased expression levels of enzymes involved in mitochondria respiratory chain relative to young mice reveal.

The expression of four NADH-ubiquinone oxidoreductase subunits, two cytochrome- C-oxidase subunits, and three ATP synthase subunits was increased more than 2-fold ones. High levels of the expression of mitochondrial respiratory enzymes suggest that the production of hypothalamic ROS increases in aged mice. No similar changes were found in aged cortex, suggesting that different brain tissues may age at different rates because of the tissue-specific metabolic rate variability or physiological environment differences [2]. Possibly, such results are also to witness that , the accumulation of ROS-induced damaging in mtDNA of neurosecreting neurons of cerebrum is a regulated function.

If for the simplicity of narration a terminological system and notions of the germ-plasma theory of August Weismann is used , it is necessary to suppose that all events of stopping of the organism functioning occur in cells of the somatoplasm mammals. But, apriory, the injury of a genetic program in all cells of somatoplasm can not have a considerable importance for the conservation and an evolutionary progress of population. The genofond of population can worsen only by mutations in cells of germ plasm of organism.

The rhetorical question: Maybe the process of the mutagenous molecules of mtDNA accumulation in the pool hormone-synthesizing neurons of brain is the function from the amount (rate) accumulations of mutations in cells germ plasm organism. This is in turn supposes that an orchestrated set of hormonally regulated germ plasm/ somatoplasm interactions will exist well. I.e. presence of, on the one hand, hormonal factors secreted by germ plasm in blood-stream, but with other – presence of intracellular mechanism defines the rate of damaged mtDNA accumulation of that it being controlled of this hormonal factor(s).

It is difficult to presuppose, that cell can define a degree a mutagenous damage own genome, and in response to these damaging secrete certain hormonal factor. The other situation is more probable. In the case of a rise of favourable conditions for damage in genome of cells of germ - plasm of organism, such as, accumulation mutagenous metabolites, poisons, some diseases and other factors some cells germ plasm or somatoplasm will be secreting this hormonal factor in blood-stream, which starts up intracellular mechanism, that defines a rate of accumulation of damaging mtDNA hormone-synthesizing neurons or

this mechanism starts up the presence or the certain concentration certain mutagenous metabolites.

At present the similar scheme has been offered and has been proved for the nematode *Caenorhabditis elegans*.

Investigation of stem cells these worm it was found, deleting the stem cells, predecessors of sexual cells inevitable brings about increasing a life span of a worm. It is deleting of stem cells that to such result to lead, rather then developing from them later sexual cells.

It is proposed that the germ-line stem cells affect life span by influencing the production of, or the response to, a steroid hormone that promotes longevity.

Proves.

It is necessary to consider some facts, which characterize the degree of accuracy and adequacy of given hypothesis.

Supposedly the line of mice with the inactive AMSM is raised. Proceed from considerations set below in "the First proof "of this article by way of simple interpolating with a maximum longevity of birds of such weight, it could be possibly defined that the maximum life-span of individuals of this mice line will be about 10-20 years. Let's name this period the “potential life-span of mammals”. But researchers will be able to observe animal of this lines only within maximum longevity of wild type, (as it is well known, it is about 3-h years), so mice will be fatally killed by means of some devices not later than these 3-h years. Practically they will be killed else very young animals! The author supposes that differences between this fantastic situation and reality are negligible. To all appearance, AMSM indivertibly kills a mammal organism before it achieves its 1/3 periods of «potential life-span of mammals». It follows, that a potential longevity of tissue of mammals exceed in 3 - 4 times of the maximum longevity of their organism!

The hypothesis/concept of AMSM has the virtue of making very specific predictions, and for this reason, it is vulnerable to observational disproof.

The First proof. Since concept of AMSM supposes that anyone of old mammal individuals possesses comparatively young somatic tissues. Therefore, hypothetical predictions are following:

1. In the field of transplantation of internal organs and tissues in mammals, that possibly will be observed in the following phenomena:

a) during serial transplantation of normal organs and tissues in young, isogenic hosts, longevity of these organs and tissues must exceed in several times longevity of mammal organism, i.e. they must be outlive not one host;

b) in mammals, received from old donors organs and tissues after transplantation in organism of the young recipient are to restore their own juvenile quality, even in case when a donor’s organ ceases its physiological functioning in the organism of donor. For example: non-functional ovaries, womb and others;

c) in mammals, received from young donor organs and tissue after transplantation in organism of the old recipients are to lost their own juvenile quality, and in some case it ceases its physiological functioning in the organism of recipient.

So, studies, conducted on inbred and syngeneic mice, have shown that transplantat skins flaps received from the old host accreted young host. When second host got older, transplantat transplanted the third younger host and etc. Viability and proliferate potential of cells were saved during 7-8 years, it much more exceeds the life span of mice comparably to maximum life-span of birds of such sizes as a mice. However, after 5-6 serial transfers transplantat decreased in his size, with each following grafting proliferation of cells it was reduced, and in the end tissue degenerated.

Similar result was received for transplantat tissue of an epithelium of mammary gland. Transplantat saved viability for six years in young organism [36-38].

At the transplantation of ovary from old rats to young ovaryotomyzed estrous function is restored, whereas ovary of young animals stopped its existence in organism of old rats [39, 40].

Moreover, in the field of liver transplantation an opinion prevails that advanced donor age is not a contraindication to liver transplantation if careful assessment of donors is made on a case-by-case basis [41].

2. In the field of cells and tissue cultures. A correlation between the age of donor and the replicative life-span of cells in culture has to be a little significant or not at all in all observed cases. Really, what can researcher expect if he compares the replicative life-span of cells in culture from cell lines established from donors of different ages but which does not exceed a first third of “potential life-span of mammals”?

This supposition is confirmed by numerous literary data. Else, since Goldstein [42] day's it is known that the replicative life-span of normal human fibroblasts received from healthy donors in culture does not correlate with donor age [43]. Several studies of rodent skin fibroblasts appear to support the existence of a small, though significant, inverse correlation between donor age and replicative life-span [44-46].

The Second proof. What can a researcher observe, in case if a work of AMSM is stopped, for example, by resection of neurosecreting brain elements and artificially to retain a level of homeostasis in organism on the most optimum level. In this case, the hypothetical prediction is following: maximum longevity mammals will be at the best reach extreme values of «potential life-span of mammals», and at the first stages the functioning of internal organs and organism normalizes. As it is well known, the fragment of such investigation has undertaken by W. Denckla. Conducting experiments with rats, he found that beside old animal with the distant pituitary gland and received thyroxine, an effect of rejuvenation occurs, which is shown in the functioning of cardiovascular and immune system and even outwardly, in the escalated growing of wool. These rats not only look "younger", as well as they are given to biochemical and physiological investigation corresponded to significantly younger animal [47].

This detailed hypothetical prediction agrees with experiment.

The Third proof. It is well known that ionising radiation damages both chromosomes as whole, and separate genes in particular, as well as causes a ruin of cells. The mechanisms through which these changes are produced are not yet understood completely, but each change is thought to be the result of chemical alterations, which are initiated by radiation as it randomly traverses the cell. Certainly, mtDNA in the same way will inevitably be damaged under the action of ionizing radiation. In case if really amongst *Vertebrata* only mammals have AMSM, where cumulating of damaging mtDNA neurons is a trigger of aging – then they must possess the high radiosensitivity. Since AMSM "is doomed" under the action of ionizing radiating to intensify a rate of aging, and hereunder shorten life-span. Really – a supposed picture complies with a real. It is well known that amongst *Vertebrata* mammals have increased radiosensitivity and even small doses of ionizing radiation shorten life-span. Dose of radiation in 300 R, which can cause a radiation sickness amongst majorities of mammal species, for birds it practically harmless, but for the development of radiation sickness for some species of snakes the necessary dose is an order above, than for the majority of mammal species.

Discussion

The major conclusion from the present hypothesis is that significant increasing of mammal's life-span of (in 3-4 times) is possibly reached by their return in considerable extent a lost ability in process of evolutions to the adult neurogenesis. Caution is needed in the interpretation of these conclusions, since our present limited knowledge of aging allow author of the present hypothesis, is founded only very strongly on implicit assumptions, though this conclusion needs to be verified by experimental study.

The rhetorical question locates modern science by means for the achievement like purpose?

Problem of restoration of the lost ability to generate new neurons in mammals was interesting and it continues to be interesting to many researchers, but real and unique results (and as it seems to author, are undeservedly forgotten) in this direction was obtained only by the group of Prof. Polezhaev.

The data of this group clearly shows [16-18], that trauma, products of degradation of nervous tissues of cerebrum or high polymeric quickly prepared exogenous organo- (brain)-specific RNA (eoRNA) from tissues of brain inducing in nervous tissues of brain processes of mitosis, amitosis, apoptosis including and neurons. This eoRNA injection to the brain causes a strong degradation and dedifferentiation of nervous tissues. Part of cells perishes. The remainder of glial and nervous cells would be activating and would be rejuvenating. Intensive division of neurons mitotic and amitotic by the way are to be observed.

In the case of very strong influence a mitotic division of neurons appears, which, however, in many cases has an abortive character – it gets to stage of metaphases, whereupon cells perish.

Glial cells have intensive division mitotic and amitotic by the way.

Moreover, stimulated influences strongly activate processes of produced cambial type of cells from ependyma and subependymal layer, their migration to the white matter and the gray matter and differentiation of part from them in nervous cells.

Last is particularly clearly seen in white mater and molecular layer of cortex of cerebral hemispheres.

Something like in the brain of birds observed [48], as results bilateral electrolytic lesion of nucleus ectostriatum in forebrain of adult ring dove *(Streptopelia risori*a).

It is considered that acting factor eoRNA is a fraction small nucleolar RNAs (snRNAs). [49, 50] snRNAs are a subclass of non-protein-coding RNAs and exist in the cell mainly in the manner of ribonucleoprotein particles.

There is an opinion, that introns and other non-coding RNAs form the primary control architecture that underpins eukaryotic differentiation and development. RNA-mediated gene regulation is widespread in higher eukaryotes and complex genetic

phenomena like RNA interference, co-suppression, transgene silencing, imprinting, methylation, and possibly position-effect variegation and transvection, all involve intersecting pathways based on connected to RNA signaling [51, 52].

Significantly, recent works showed that in neurons of brain a number brain-specific snRNAs exists [53]. What could be the biological role of the newly described brain-specific snRNAs?

Interpreting of the results of studies of Prof. Polezhaev group from standpoints of these beliefs about a role of snRNA, it is possible to suppose that when receiving eoRNA an phenol by the way, seems to be liberated sequences of snRNA, which under injection have caused dedifferentiation in nervous tissues and mitosis of neurons in mammals. The identification such the snRNA sequence would have enormous scientific and therapeutic value.

Instead of conclusion. Since the present hypothesis touches practically all aspects of the phenomenon of aging, author, limited by the frames of scientific article, tried
to focus one's attention on a matter of further verification of this hypothesis.

Certainly, many facts and phenomena can directly or indirectly witness in favour of existence of particular age-related mechanism of self-destruction of organism mammals, or are at least to interpret as such.

However, it must be admitted that in view of insufficiency of database of the modern biological science some facts and phenomena presented in section "proofs" do not constitute any kind of formal proof in the sense this term is normally understood.

So, to the author opinion, hypothesis will be a consider confirmed, if it is established the fact that: if the method of restoration of lost ability to generate new neurons in mammals is designed, and the life-span of animals with this restored ability will increase in 3-4 times.

Table 1. Maximum Longevity of Animals.

	Life-span in years	The mean weight of organism
Mammals		
@Golden hamster *(Mesocricetus auratus)*	1,8	15-30 g
Shrew *(Sorex fumeus)*	2	20 g
@House mouse *(Mus musculus)*	3	30 g
Vole *(Microtus arvalis Pall)*	2	10-47 g
Philippine tarsier (*Tarsius syrichta*)	12	95-165 g
@Gray squirrel *(Sciurus carolinensis)*	15	180-1000 g
# Tree shrew (*Tupaia*)	7	275 g
# Common marmoset (*Callithrix jacchus*)	15	413 g
*Guinea pig *(Cavia porcellus)*	7.5	600 g
# Squirrel monkey (*Saimiri sciureus*)	21	630 g
@Domestic cat *(Felis catus)*	21	2-5 kg
Rabbit *(Oryctolagus)*	4.7	2-9 kg
Alpine hare *(Lepus timidus)*	5-7	5.5 kg
*Siamag *(Symphalangus syndatylus)*	23	9-13 kg
#Chimpanzee *(Pan troglodytes)*	43	45 kg
*Chimpanzee *(Pan troglodytes)*	60	50 kg
Grizzly bear *(Ursus horribilis)*	31	780 kg
@Elephant Indian *(Elephas*	57	5000 kg

maximus)		
Aves		
*Whitethroat *(Sylvia borin)*	24	10 g
*Spotted flycatcher *(Muscicapa striata)*	12.5	12-18 g
@Kanary *(Serinus canaria)*	24	11 g
*European robin *(Erithacus rebucula)*	12	17 g
# Common swallow *(Hirundo rustica)*	21	20 g
@Great tit *(Parus major)*	9	20 g
@Nightingale *(Luscinia luscinia)*	3.8	25 g
*House sparrow *(Passer domesticus)*	11.5	32-35 g
* Skylark *(Alanda arvensis)*	20	40 g
*Blackbird *(Turdus merula)*	20	70 g
@Blue jay *(Cyanocitta cristata)*	4	150-200 g
@Domestic pigeon *(Columba livia domestica)*	35	240-360 g
*Common raven *(Corvus corax)*	69	800-1500 g
*Herring gull *(Larus agrentatus)*	49	1500 g
*Eagle owl *(Bubo bubo)*	68	2100-3200 g
*Bateleur *(Terathopius ecaudatus)*	55	2-3 kg
*Common crane *(Grus grus)*	43	4-5.5 kg
*Bewick's swan *(Cygnus columbianus bewickii)*	24.5	5-6 kg
*Golden eagle *(Aquila chrysaetos)*	46	3-6.5 kg
*Griffon vulture *(Gyps fulvus)*	38	6-8 kg
*White pelican *(Pelecanus onocrotalus)*	51	9-11 kg
*Andean condor *(Vultur gryphus)*	52-65	10-12 kg
*Emu *(Dromaius novaehollandiae)*	28	45-55 kg
*Ostrich *(Struthio camelus)*	40	50 kg

Given tables are taken from following sources: @ -Britannica CD,[54]; # - Cutler,[55].*- Life of animals. [56-57];

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Чому ссавці помирають молодими? Залежний від віку гіпотетичний механізм феноптозу (самознищення) ссавців

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Резюме Висунута гіпотеза має за мету прояснити деякі малозрозумілі феномени біології старіння. Зокрема, чому максимальна тривалість життя птахів набагато більша в порівнянні із ссавцями співставних розмірів незважаючи навіть на те, що птахи мають найвищий ступінь рівня метаболізму серед живих істот. Гіпотетизується, що ссавці на відміну від птахів та інших хребетних мають залежний від віку механізм "самознищення".

Ключові слова: старіння, гіпотеза, ссавці, птахи, РНК.