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Why humans become old

Abstract

The article considers the telomere theory which authors were awarded the Nobel Prize in 2009. The aging process is explained from the point of view of Information-Wave Medicine (IWM). The aging program is shown to be a multi-level genetic structure controlled by the body's internal biological clock.

Content

Body aging

Today, there are dozens of human body aging hypothesis, but, unfortunately, there is no anti-aging, that is, life extension technology. I have considered the main hypotheses in the [4,5], but another popular theory recognized today – the "cell aging theory" – should not be left unmentioned. A Russian scientist A.M. Olovnikov hypothesized cellular aging in the 70s of last century. According to the hypothesis "bacteria non-aging is due to the ring shape of their DNA and the fact that telomeric sequences in stem and cancer cells are protected by constant - every time cell divides - lengthening with a particular enzyme - tandem-DNA-polymerase (today named - telomerase)". However, the scientist took no step further and even abandoned his hypothesis later. American scientists advanced further when addressing to a similar issue and in 2009, three of them were awarded the Nobel Prize in Physiology and Medicine "for discovery of how telomeres and telomerase enzyme protect chromosomes". The essence of this discovery is that the scientists have found that the end parts of chromosomes are protected by special protein structures called telomeres (composed of two Greek words: *telos* - end and *meros* - part). It was found that the telomeres have their maximum length at birth. Each time a cell divides the telomeres get shorter. The process of length reduction is associated with the cell life duration. When the telomeres completely disappear the cell dies, there are two variants of such death. The first one is the so-called apoptosis,

i.e. self-implementation of the program, which regulates when the cell is to stop its activities; the second one is a complete violation of the functional state of the cell, resulting in various pathogenic focus inside the cell. It was also found that the telomere shortening rate differs for different organisms.

Winner of the 2009 Nobel Prize Carol Greider has discovered that there is a special structure in cells called telomerase. The telomerase is an enzyme that prevents shortening of telomeric parts of chromosomes. However, since the amount of telomerase is small a catalyst has been genetically engineered. When introduced into the body it significantly increases telomerase activity. According to scientists, the use of this catalyst component has made it possible to stop cells aging or even reverse this process.

Another phenomenon caused by the telomere properties should be noted as well. The scientists have developed a life expectancy test, which caused a commercial boom in the United States. By paying 500 dollars a person can learn, according to the length of his telomeres, about the life time the Nature assigned to him.

We have studied the above said theory using information-wave medicine methods. We should say that today we cannot explain some of the results obtained and take them for granted. Perhaps, someone of our followers will be able to do it.

First, our study has confirmed that there are telomeres at the ends of chromosomes from birth. It is surprising that the telomeres have been found only at the ends of the even chromosomes. Another inexplicable surprising fact is that the presence of the telomeres in a human body is associated with the age. We tested 20 people aged under 100 and found that the telomeres are exist in the body only till the age of 80. In other words, both 80-year-old people and an officially recognized Israeli long-liver Yisrael Kristal in the age of 112 had the telomeres only when they were under 80. How and why they are absent from the long-livers' bodies stays unanswered today.

Another amazing fact is that we have found no telomere activity in any abnormal chromosome, as they are part of the chromosome and lose their activity when the main "body" of the structure mutates.

Another issue related to the telomere theory seems to be not well-grounded. The fact is that embryo chromosome set also contains corresponding amount of telomeres. However, some stem cells, neuronal precursors, should contain their own telomeres in their chromosome set as well. According to classical science, neuron death is caused by inability of their division, rather than telomeres shortening.

On the basis of the IWM concept developed by the author a viral genetic theory of aging has been created. As well as other IWM technologies this technology is based on the hypothesis of the information field of the Universe. This term means the existence of an information-wave structure, which remembers a state of any process and/or object at any given moment since the emergence of life on Earth. How can we prove it? By using one of radiesthesia technologies, properties of any medicine, such as aspirin, are written onto a temporary storage medium (aluminum foil) from the information field of the Universe. Then, the information properties of the aspirin tablet are transferred to an identical clean temporary medium. Equality of spectral copies justifies the use of the information transferred from the information field of the Universe. It should be noted that the existence of the field was first mentioned in ancient Indian philosophy called "Akashic Records". When applying the technologies in the IWM to check the presence of some pathogenic genes of a particular individual, we also appeal to the said information field of the Universe. In a similar way we could found an aging gene in the human body. This gene exists in a child genome at its birth and stays inactive till a certain age. The aging gene activation is associated with the so-called somatotropin growth hormone. Once the human body is completely developed the anterior pituitary lobe stops somatotropin production, which simultaneously activates the aging gene. This occurs approximately in the age of 27-30. For the sake of simplicity we have written above that the aging gene contains two levels. Further studies have shown that the structure of the gene is more complex and consists of seven levels. Moreover, different levels contain different amounts of genetic viruses jointed into groups, which we call genoviruses.

It is important to realize that the genoviruses as they viewed from the IWM point of view are not "biological objects", but only a set of information-wave structures. In other words, the genoviruses are the information-wave pathogenic structures, which cause the same pathological focus in the body as live virus groups similar to them in terms of radiation.

Thus, the first level contains a genovirus consisting of 21 adenoviruses. According to virologists, there are up to 80 types of biological adenoviruses. However, only some of them are pathogenic. From all pathogenic adenoviruses we can single out a group of the so-called "children's" adenoviruses, which cause various infections in children including type 1 diabetes. As to the adenoviruses comprising the aging gene, they all relate to non-pathogenic viruses. In other words, they

do not cause pathogenic focus, but only slowly deteriorate the functional state of the cells they get into. Since they get into all of the body's cells, the aging gene deteriorates the functional state of the entire body.

There are seven adenoviruses in the genovirus of the second level of the aging gene, five adenoviruses in the third level, four – in the fourth level, three – in the fifth level, two – in the sixth level. A genovirus of the seventh level contains a herpesviridae virus and oncovirus. We can state that the activation of all levels of the aging gene is gradual under the influence of the body's biological clock and extends over the first half of life. We believe that each level activates within two to three subsequent years. The 7th level of the gene activation (secondary activation) is related to the hormonal rearrangement of the entire body. This corresponds to the age of about 45 to 50. Simultaneously, local immunity is suppressed in all organs and systems where there is the 7th level gene. Considering that this genovirus contains pathogenic genes, we can assume that the secondary activation significantly deteriorates the functional state of practically all organs. Thus, for example, even small deviations in the functional state of pancreas from its normal condition causes type Two diabetes in most cases.

We have found that the secondary activation of the aging gene causes mutation of the 31st chromosome followed by abnormal changes in it. The mutating structures are genoviruses contained in the last level of the aging gene, i.e. oncovirus and herpesviridae virus.

From the above it follows that the earlier we annihilate the aging gene, the longer a person can live. There are two ways of the aging gene annihilation. Firstly, prior to its activation, secondly, removing it from the genome of prospective parents before conception. Of course, due to several reasons it is impossible to check both technologies on humans. Mice are very convenient for checking the said technologies, since their life expectancy in captivity is 6-7 years on the average, which makes it possible to monitor their aging.

Finalizing the description of the aging gene effect on the functional state of the body, we cannot but mention one feature of this gene inherent in long-livers. It should be noted that according to classical science long-livers are people aged 90 years and older. When checking their aging gene it was found that it has not been activated throughout their life. We believe that this is only due to failure of the biological clock of a particular individual. Since human rhythms are regulated by the so-called pineal gland (epiphysis), we have firstly checked the functional state of this brain structure. It turned out that all long-livers have a pathological focus in their pineal gland, preventing aging gene activation.

This hypothesis is proven by the fact that the aging gene of all long-livers over 90 years old at the time of their death contained all the above mentioned viruses on all the levels. A person who reads the article may have reasonable question – notwithstanding everything said above in outward appearance long-livers look old rather than young. We believe that life extension is not directly related to rejuvenation of the body. Here is why. So that a person could stay young, if the aging gene is not activation, there should be no other reasons deteriorating body's functional state. But such reasons always exist. These include genetic and infectious diseases, impact of harmful radiations, etc.

Conclusion

At the end, I would like to express some thoughts about interconnection of anti-aging process and rejuvenation of the body. As mentioned above, living under impact of the environment a person cannot stay outwardly young for the following reasons. Human skin is effected with sun rays, wind, environment condition, meals, lifestyle. All these reasons are associated with many other factors. Therefore, even if we are capable to reverse the aging process, we will not reverse outward appearance completely. Meanwhile, the biological age of the body, or rather, the difference between chronological and biological age, determines the condition of the body. However, there is another parameter that determines the condition of the body – its biological age. An old person can feel young and vice versa. Therefore the issue of how to combine longevity with the outward appearance is, unfortunately, not definite and is subject to serious study. But that is another topic.

References

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