Cell cycles with telomerase synthesis for increases productivity farm animals

Veronika Borisova[1*], and *Sergey* Borisov[2]

[1]FSBEI HE "Smolensk State Agricultural Academy", Department of Agricultural Products Processing Technology, 10/2, Bolshaya Sovetskaya st., Smolensk, 214000, Russian Federation
[2]Municipal budgetary educational institution "Secondary school No. 28" of the city of Smolensk, 14, Bakunina str., Smolensk, 214000, Russian Federation

Abstract. The article considers the work of cells with infinite cycles. These cells are also capable of synthesizing telomerase with reverse transcriptase, which lengthens telomeres. Telomere lengthening leads to improved cell repair, as it is related to the telomeres length, indicating the period lived by the part of the cell cycle to which this cell belongs. The cells live longer when they are better regenerated. For measurements long of cell cycles, under telomeres are special genes of apoptosis. They orient apoptosis to long of telomere. With help of telomere, BNA not sticks together at division and cancer not start. So cell's mechanism of aging use for solutions problems with possible cancer tumors. These cells also cannot terminate the cycle with group apoptosis after reaching the Hayflick limit of 50 cell-divisions, which is needed to reduce the risk of developing cancerous tumours.

1 Introduction

This article has been written based on the analysis of the sources [1-3]. The objective of the research is studying the cycles of the cells synthesizing telomerase, examining the effect of telomerase on the cell repair and on the cycle lifespan.

To store information about the genetic structure of eukaryotic cells, linear molecules of the DNA chromosome are used [4]. In the 30s of the last century, different behavior of whole chromosomes and their fragments was discovered: instability of broken chromosomes, their fusion, and rearrangement were noted [5]. At the same time, it was assumed that this behavior is associated with the presence of special nucleotide sequences at the ends of chromosomes. They were called telomeres [6-8]. Telomeres consist of a special sequence of proteins and RNA located in a special sequence and form a nucleoprotein complex [9-10].

Also, has been studied functioning system of cells aging and mission telomere in it. Except this, has been studied work of cells mechanism of division and function of telomere in it. Beside has been discovered importance of system reparation in live of cells with

* Corresponding author: borisowaveronika@yandex.ru

synthesis of telomerase and artificial extension live of ordinary cells. Questions of the functioning of cell cycles with telomerase synthesis are discussed in the works [11-21].

2 Materials and methods

Spermatozoa, cancer and stem cells are capable of synthesizing telomerase. Their cell-division capability is increased because of good cell condition. Apart from those cells, the telomerase is also synthesized by other cells that should be renewed. The regular somatic cells do not synthesize telomerase because of several reasons. The telomerase production in somatic cells may bring the cell closer to transformation into the cancer cell from which the cancerous tumor can be developed. The other reason is that the telomeres are the benchmarks of cell repair and of cell-division cycle termination by dying off the last cell of the cycle, which has high risk of its transformation into a cancer cell. The breaking of DNA starts in the process of this cell division.

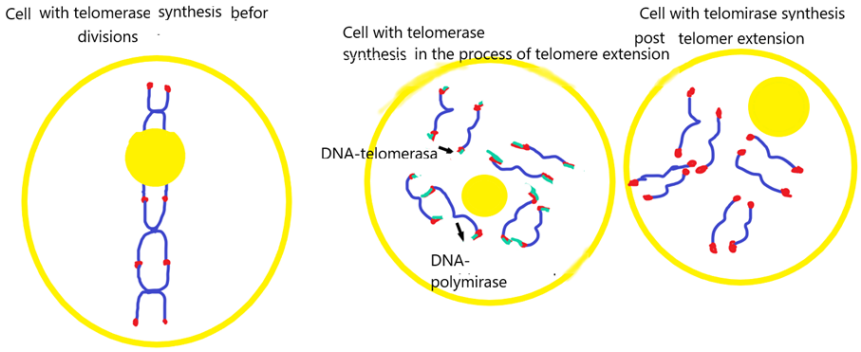

Fig. 1. Scheme of division cells with telomerase synthesis.

Telomerase is synthesized in the process of reverse transcriptase. It is a process during which the information is transcribed from RNA to DNA, but not from DNA to RNA. Telomeres have RNA and proteins in their composition. The cell cycle becomes longer if its telomeres are extended. This is due to the fact that the cells are repaired better and faster for a longer life and a longer cell-division cycle in this case. This is because the cell repair depends on the telomere lengths. If the long telomeres are available, the cell repairs itself after 25 years of human life just as it has repaired itself before, i.e. the cells are repaired just as well regardless of age. The cell-division capability is increased because of good cell condition. The cells producing telomerase live longer than the regular cells at the same stage of the cell-division cycle. However, they also accumulate mutations, just as regular cells do, because they are exposed to radiation and other external effects, and their mutated parts are repaired with DNA proteins just as it is done to regular somatic cells.

One of reasons degradation of work cells with telomerase synthesis, which live in tissue, is degradation of intercellular matrix. When intercellular matrix make his work worse cells, which living at it make their work worse too. At degradation of intercellular matrix cells, receive different quantity of nutrition, cellars signals are conducted worse, new stem cells difference worse too.

Regular cell tissues has definite reserve of strength, mortgaged initially. At work of cell tissues their reserve of strength degradation. At young organism, this cells restoration and restoration intercellular matrix. But there is hidden wear and tear in the tissue, and at a

certain age these problems lead to a large amount of damage and development ends, and later aging begins.

Stem cells need telomerase synthesis. They are consumed as the cell cycle dies off. The stem cell must go to the place of the dead cycle, differentiate and form a new cell cycle. Stem cells synthesize telomerase, but after differentiation, they stop synthesizing telomerase to reduce the risk of cancer. With the help of telomerase, stem cells are able to divide a large number of times before differentiation and form new stem cells. For this reason, the tissue can receive stem cells for a longer time, which prolongs the life cycle of the body. During division, the stem cell divides into two stem cells, but one goes into differentiation, and the other remains stem. In this way, the number of stem cells in the tissue is maintained.

3 Discussion

Genetic structure mutations impair the functioning of organoids with abnormal proteins. Because of impaired functioning of its organoids, the cell works worse and dies faster. This leads to shortening of cell-division cycle through shortening of telomeres. Part of DNA strand is removed during cell repair. The removed part of DNA strand needs to be replaced. For this purpose, the information is taken from RNA during inverse transcription. This information has low accuracy, and thus, the abnormal gene goes into DNA.

Accumulation of cell debris is another reason for dying of cells producing telomerase. Cell debris includes free radicals, molecules, and 'broken' proteins generated during cell life. A free radical is a protein, a lipid, or other molecule that contacted with active form of oxygen or radiation. The atoms of their molecules are missing an electron, which they obtain from neighboring molecules. For that reason, new molecules with missing electron can be formed. Free radicals formed from protein, appear due to the following reasons: ribosome errors, contact with active forms of oxygen and radiation. They can produce sticky parts, which stick together upon contact with other proteins.

Telomerase works by completing part of the telomeres, and then DNA polymerase completes the other part. Telomeres are a specific set of nucleotides that do not contain gene code and, like other parts of DNA, can be subject to destruction due to free radicals or external influences, due to which they can be destroyed and bring about group apoptosis in the next cells of the cycle.

Carbohydrates are another problem for proteins. They are capable of attaching to proteins and be together with them. In addition to that, they are deformed and adopt other form. The cell has several methods of destroying these proteins, such as destruction or rectifying its form. Besides proteins, lipids also can become free radicals. A lipid is a joining of molecules that form cell membranes. They easily cross the cell walls and exist for a long time. They are also present in the mitochondria membranes. One of the problems, presented by cell debris, is obstructed transport inside and outside of the cell. Most of free radicals are synthesized in mitochondria, since they use oxygen for generating energy. Their structure also includes proteins and lipids. They disrupt the strand and information stored in it upon contact with DNA molecule. When the DNA strand is ruptured, the repairing proteins attempt to join DNA, which can lead to new errors. New errors can also be the result of unsuccessful attempt to correct existing abnormalities in DNA. These errors lead to regular cells being transformed into cancer tumors cells. A cell can degenerate into cancer tumor because of too many mutations, or incorrect joining of DNA strand by repairing proteins. Spermatozoa and cancer cells have causes of aging similar to those of regular cells. Cell-to-cell communications are also aging in cancer tumors.

The cells, synthesizing telomerase, do not control their division: spermatozoa are excreted from the body and do not form live tissue, the cancer cells do not control their

number. In case of spermatozoa, the synthesis of telomerase is required for their replenishment after excreting from the body. They feature high division rate, but they can't transform into cancer tumor because they are regularly excreted from the body and are short-living. The cancer cells are dangerous for the body, because they can quickly grow into cancer tumor. The more often the cell division occurs, the more intensely the telomeres are shortened. Hence, the cells are less capable of repairing, and are less capable of dividing for various reasons. The tissue cells and other division activators begin to work worse, and the cell divides less frequently. A body with young cells is capable of developing because it has no these problems. The same is true for the tissues synthesizing telomerase, because they also have long telomeres. The key difference of these cells from the regular ones is that they do not have Hayflick limit of 50 cell-divisions. Besides that, the regular cells are not capable of maintaining steady restoration after 25 years. The importance of destruction of old cells to avoid developing cancerous tumors is one of the reasons for the Hayflick limit.

4 Results

The parent cell genocode with all accumulated mutations is transferred to the daughter cells during cell division. The last cell of the cell-division cycle has the largest number of mutations transferred to it from the previous cells and accumulated for the lifetime. This cell has a high risk of being transformed into cancer cell. To make sure it does not happen, the cell should stop splitting and terminate the cell-division cycle. Since telomeres are the benchmarks of cell-division cycle length, they do not restore in regular cells. The cell-division cycle should be suppressed to prevent accumulation of mutations that can lead to cancer development. The termination of cell-division cycle is guided by telomeres. The cycle is suppressed if telomeres do not have the length required for the next division. It is necessary for preventing DNA strand rupture and eliminating the possibility of cancer. In this case the cell will not start breaking the ends of DNA strands instead of telomeres and will not obtain additional mutations. This cell should kill itself to not be able to divide and produce a cancerous cell. If a new cell is produced from the last cell of the cell-division cycle, it might transform into a cancer cell (for the reason of a large number of mutations), start synthesizing telomerase in unlimited quantities, and develop into a cancer tumor. The reason of telomere shortening is the inability of DNA polymerase to copy DNA strand entirely. Thus, the telomeres are shortened, if they are available, in the process of cell division. Without telomeres, the ends of DNA strands would have shortened, which could lead to mutations. The cancer cells need telomerase in order not to die, as regular cells do. The regular cells trigger apoptosis for all cells of the cycle to terminate their division, since after 50 divisions they do not have telomeres fit for it. If these cells were capable of division, the rupture of DNA strands could have started, which could cause mutations. These cells have infinite division cycles.

With the help of external influences, it is possible to activate the synthesis of telomerase in ordinary cells when creating certain environmental conditions, such as the consumption of vitamins and other environmental influences. Telomerase synthesis in ordinary cells of farm animals can be induced using genetic correction and specialized food additives. With an overdose, it is possible to develop a cancerous tumor, but with the right dose, it is possible to reduce aging and reduce the risk of cancer. It is also possible to strengthen the reparation system to prolong life. This is an important part of rejuvenation, because cells and cell cycles with telomerase synthesis live longer and have an increased risk of developing cancer.

5 Conclusion

The effect of telomerase on the cell repair routine and on the duration of cell-division cycle have been research in this research. The reasons of long cell-division cycles of cancerous cells and spermatozoa have been found. The information obtained in the study can be used for prolonging human life and developing new methods of cancerous tumor treatments.

As a result of the study, the effect of telomerase on cell recovery, cycle length was established, and the cause of the long cycle of cancer cells, stem cells and sperm was also established. This article examines ways to extend the life of farm animals. With the help of information obtained during the study, it is possible to extend the life of humans and other farm animals. In some species of farm animals, with increasing life expectancy, agricultural productivity indicators improve. By rejuvenating animals such as cows, their milk production will increase due to the extension of their life. By enhancing the DNA repair system to reduce the risk of cancer developing from old cells with a large number of mutations and little telomerase synthesis in ordinary cells. This can lead to prolongation of the life of the cell cycle of the entire organism. You can also use tissue cleansing of old cells using accelerated apoptosis, which can lead to prolongation of the life of all tissue cells. In addition, a connection was established between cell aging and the synthesis of telomerase and the clogging of cells with cellular debris and other molecules formed during the life of cells. It is impossible for a cell to prolong its life indefinitely, because it will mutate and eventually become cancerous, or with a very high-quality DNA repair system, it will lose the opportunity to evolve. You can also strengthen the system of repair and cleansing of the cell from cellular debris, which will also prolong the life and improve the functioning of the cell and the entire tissue as a whole. In addition to the synthesis of telomerase, DNA polymerase can be transcribed onto telomeres. This method of prolonging the life of tissue weakens the evolution of organisms.

New methods of treating cancer tumors using genetic correction and the introduction of genes that prohibit the synthesis of telomerase and stop the endless number of divisions and the death of the cycle when telomeres end. In addition, the presence of apoptosis genes under telomeres was established and the operation of the cell aging mechanism and the role of telomeres in the life cycle were studied.

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