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# COMMON PATHOGENETIC PATHWAYS OF CANCER AND AGING

***Jumayev Abdulaziz,Axatov Islombek, Rashidov Jahongir, Umarov Shoxjahon, Parmonov Oybek, Abdirashidova Gulnoza***
*Students of group 301 of the Faculty of Treatment No. 2 and Senior Lecturer, Department of Pathological Physiology Samarkand State Medical University,*

**Abstract.**A growing body of experimental evidence has gradually shown that aging and carcinogenesis share many common molecular pathways. During aging, the accumulation of genetic and epigenetic changes, reduction in telomere length, progressive impairment of DNA damage repair mechanisms, regulation of glucose metabolism, cell cycle control, and stem cell self-renewal lead to the induction of senescence or apoptosis and loss of replicative capacity. The same cellular dysfunctional mechanisms that characterize the aging process have been found to be involved, with varying levels of evidence, in carcinogenesis and are discussed herein.
**Keywords:** carcinogenesis, aging, apoptosis, inflammation

Aging is the biggest risk of cancer development. About 77% of all cases of cancer are diagnosed in people aged 55 years and older, and after the late average age, the probability of developing cancer in men is 50%, and women are 35%. Thus, with an increase in the aging population, it is expected that cancer will become an even more serious problem. Some recent innovative studies and epochal discoveries revealed what turned out to be a complex and multifaceted link between cancer and aging, and caused great interest in this area of research. Evidence that P53 tumor suppressor can affect both cancer development and early aging, were obtained as a result of the study of mutant mice with a hyperactive P53 genome, which were extremely resistant to cancer, but It is noteworthy, they also demonstrated signs of premature aging. Another study showed that aging fibroblasts contribute to the growth of precancerous and malignant epithelial cells of a person in culture. Scientists from the Center for Research of Cancer Hutchinson found that genetic instability, the main sign of malignancy, accompanies the aging of mother cells in yeast, shedding light on how cancer can occur with aging; The authors suggested that nuclear genome can be initially unstable with age. It appears more and more evidence that some of the constantly active molecular mechanisms that protect cells from cancer transformation and other diseases are increasingly violated with age and can be involved in the aging of the body as a whole and in carcinogenesis.

Oxidative stress system. One class of theories of aging is based on the concept that damage caused by either normal toxic byproducts of metabolism or ineffective repair mechanisms accumulates throughout life and causes aging.

Reactive oxygen species (ROS) refer to free radicals and oxidants, which are molecules so extremely reactive that they can damage every cellular component. ROS are continually generated and eliminated in the biological system.

Cellular respiration and mitochondrial DNA. ROS resulting from cellular respiration occurring in the mitochondria have been hypothesized as a major source of damage that leads to aging. One hypothesis is that the rate of mitochondrial ROS generation may act as a determinant of longevity. The lower rate of mitochondrial ROS generation, together with the lower rate of lipid peroxidation in long-lived animals compared to short-lived ones, may be the main reasons for their slow aging.

Mechanisms of Telomere Repair. Telomeres are short tandem repeating DNA sequences found at the ends of eukaryotic chromosomes that function to stabilize the integrity of chromosome ends. Telomeres compensate for incomplete semiconservative DNA replication at chromosome ends, so without telomeres, cells would lose the end of their chromosomes and the essential information it contains. Protection against homologous recombination and non-homologous end joining (NHEJ) constitutes the essential "capping" role of telomeres.

Apoptosis and Aging Pathways. The relationship between cellular aging and apoptosis pathways and the processes of aging and carcinogenesis is complex. Apoptosis, programmed and genetically controlled cell death, can benefit the body by eliminating defective cells and protecting against cancer. Tissue depleted of many cells due to high levels of apoptosis can exhibit a "senescent phenotype." Transgenic mice with defective mtDNA repair will have a shortened lifespan, increased apoptosis, and decreased cellular function.

Insulin-like signaling pathway. The link between glucose metabolism and aging began with studies of calorie restriction with adequate nutrition (CRAN). CRAN is not the same as dieting or fasting, but involves reducing calorie intake while maintaining optimal intake of essential nutrients. Consuming a low-calorie, nutritionally balanced diet works remarkably well in a wide range of animals, increasing lifespan and prolonging good health.

Mechanisms of DNA repair. Eukaryotic cells, exposed to millions of DNA lesions per day from both normal metabolic activity and environmental factors, activate protective pathways by inducing a variety of proteins involved in DNA repair, cell cycle checkpoint control, protein trafficking, and degradation. DNA repair refers to the set of constantly active processes by which the cell identifies and repairs DNA damage.

DNA methylation. Somatic epigenetic inheritance depends on enzymes that control DNA methylation status and histone acetylation. DNA methylation, in particular, is a key epigenetic modulator regulating chromatin stability and gene expression, and typically occurs at CpG sites ("CpG islands") in vertebrates. Methylation of CpG dinucleotides located in gene regulatory regions (GRRs) often results in gene silencing.

Cell cycle control mechanisms. Cell cycle checkpoints are control mechanisms that ensure the fidelity of cell division in eukaryotic cells. These checkpoints verify that the processes of each phase of the cell cycle have been accurately completed before progression to the next phase.

It is generally believed that decreased activity of checkpoint components predisposes cells to chromosomal instability, aneuploidy, and malignant transformation.

Stem cell division. Due to their ability to self-renew and differentiate, adult stem cells are an in vivo source for replacing cells that are lost daily from tissues with high turnover rates during the life of an organism.

Adult stem cells are a good model for studying aging because they suffer from the consequences of aging, which leads to a decrease in the ability to self-renew and differentiate correctly.

**Conclusions**

Growing scientific evidence highlights the multifaceted and intriguing links that exist between the biological mechanisms underlying the aging process and cancer development. During aging, the accumulation of many intertwined cellular changes leads to the induction of senescence, apoptosis, and loss of replicative capacity: genetic alterations such as DNA damage and mutations and chromosome rearrangements; epigenetic changes such as abnormal DNA methylation; shortening of telomeres, etc.

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