Volume 3, Issue 5, May 2025 ISSN (E): 2938-3765

MOLECULAR BIOLOGY OF CANCER AND BIOLOGICAL FOUNDATIONS FOR EARLY DIAGNOSIS

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Abstract
This article analyzes the cellular and molecular causes of cancer, including genetic mutations, oncogenes, intracellular signaling pathways, and disruptions in apoptosis processes. It also examines the biological characteristics of cancer cells and the potential for early detection using molecular methods. The article discusses the biological basis for early diagnosis and personalized approaches to cancer diagnostics, focusing on biomarkers and genetic tests.

Keywords: Oncogene, apoptosis, mutation, biomarker, molecular diagnostics, cancer cell, genome, transduction.

Introduction

Cancer is one of the most significant and complex health challenges faced by humanity today. It is not a single disease but rather a collection of related disorders characterized by the uncontrolled growth and spread of abnormal cells. Understanding the molecular biology of cancer is essential for developing effective diagnostic, therapeutic, and preventive strategies. Recent advances in molecular genetics and cellular biology have transformed our understanding of the mechanisms underlying cancer development, providing new insights into its initiation, progression, and metastasis.

At its core, cancer arises from genetic mutations that disrupt the normal regulation of cell growth, division, and death. These mutations can activate oncogenes, inactivate tumor suppressor genes, or interfere with cellular signaling pathways, leading to the unchecked proliferation of malignant cells. In addition to genetic changes, epigenetic modifications and alterations in the tumor microenvironment also play critical roles in cancer progression.

A key characteristic of cancer cells is their ability to evade the natural process of programmed cell death (apoptosis) and to sustain continuous growth signals. This is often supported by the activation of telomerase, an enzyme that maintains chromosomal integrity and allows cells to divide indefinitely. Furthermore, cancer cells can stimulate the formation of new blood vessels (angiogenesis) to ensure a continuous supply of oxygen and nutrients, facilitating tumor growth and spread.

Early detection of cancer is crucial for improving patient outcomes. Molecular diagnostic methods, including biomarker analysis, next-generation sequencing (NGS), and liquid biopsy, have become

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essential tools for identifying genetic alterations and monitoring disease progression. These techniques enable personalized treatment approaches, targeting specific molecular abnormalities unique to each patient's cancer.

In this article, we will explore the molecular mechanisms that drive cancer development, the role of genetic and epigenetic changes, and the importance of early diagnosis based on biomarkers and genetic testing. Understanding these biological foundations is critical for the development of more precise and effective cancer therapies, ultimately improving survival rates and patient quality of life.

Cancer remains one of the most significant global health challenges, characterized by uncontrolled cell growth and the potential to spread to other parts of the body. Understanding the molecular basis of cancer is essential for developing effective diagnostic and therapeutic strategies. This article discusses the genetic and molecular mechanisms driving cancer development and highlights the biological foundations critical for early diagnosis.

Molecular Basis of Cancer Development

Cancer arises from the accumulation of genetic mutations and epigenetic alterations that disrupt the normal regulation of cell growth and division. These changes can affect key regulatory genes, including oncogenes, tumor suppressor genes, and genes involved in DNA repair. The main molecular mechanisms involved in cancer progression include:

1. Oncogenes and Tumor Suppressor Genes

Oncogenes are mutated forms of normal cellular genes (proto-oncogenes) that promote uncontrolled cell growth and division. Examples include the RAS, MYC, and HER2 genes, which, when activated, can lead to excessive signaling for cell proliferation. In contrast, tumor suppressor genes, like TP53, RB1, and BRCA1, normally function to inhibit cell growth and promote DNA repair. Loss or inactivation of these genes removes the critical checks on cell division, promoting tumor formation.

2. DNA Repair Defects and Genomic Instability

Deficiencies in DNA repair mechanisms can lead to the accumulation of genetic mutations. For example, defects in the BRCA1 and BRCA2 genes, which are critical for double-strand DNA break repair, significantly increase the risk of breast and ovarian cancers. Genomic instability resulting from such defects accelerates the accumulation of oncogenic mutations and promotes tumor progression.

3. Epigenetic Alterations

Epigenetic changes, including DNA methylation, histone modification, and non-coding RNA expression, also play a significant role in cancer. Aberrant DNA methylation can silence tumor suppressor genes, while changes in histone modification can disrupt normal chromatin structure, affecting gene expression.

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4. Tumor Microenvironment and Immune Evasion

The tumor microenvironment, consisting of cancer cells, stromal cells, immune cells, and extracellular matrix components, plays a critical role in cancer progression. Tumors can manipulate this microenvironment to evade immune detection, promote angiogenesis, and resist therapy.

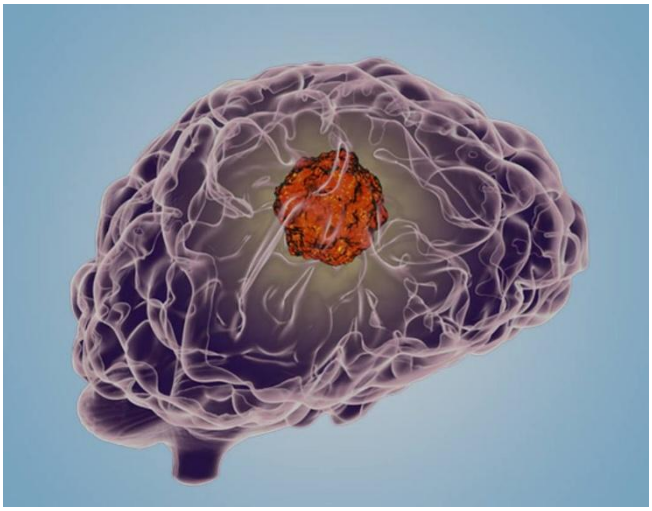
Biological Foundations for Early Diagnosis

Early cancer detection significantly improves patient outcomes. Biomarkers play a critical role in early diagnosis, allowing for the identification of cancers at their earliest stages. Key approaches for early cancer detection include:

- **Liquid Biopsies**: Detecting circulating tumor cells (CTCs), cell-free DNA (cfDNA), and exosomes in blood samples.

- **Genetic and Epigenetic Markers**: Identifying specific mutations, methylation patterns, and non-coding RNAs associated with cancer.

- **Protein Biomarkers**: Measuring tumor-associated antigens like PSA for prostate cancer and CA-125 for ovarian cancer.



Neovascularization (Formation of New Blood Vessels)

Cancer cells induce the formation of new blood vessels in their surrounding tissue. This provides them with increased oxygen and nutrients, supporting their rapid growth.

Invasion and Metastasis

Some types of brain cancer can invade surrounding tissues. While metastasis to other organs is rare, the spread within the brain itself can be rapid.

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Clinical Symptoms:

- Headache (worsens in the morning)
- Vomiting (without nausea)
- Short-term memory loss
- Ringing in the ears, vision or hearing problems
- Dizziness, loss of balance
- Changes in mental state, depression
- Muscle weakness on one side of the body

Conclusion:

Brain cancer is one of the most dangerous and complex oncological diseases for human life, associated with factors like gender, age, and genetics. Biologically, it develops as a result of genetic mutations in normal neuroglial or neuronal cells, disruption of apoptosis, and activation of oncogenes. Cancer cells in brain tissue have the ability to grow uncontrollably and invade surrounding tissues. During this process, new blood vessels (angiogenesis) form, providing tumor cells with oxygen and nutrients. Given that any tumor in the brain can disrupt essential brain functions, early diagnosis and effective treatment strategies are critically important.

References

1. Hanahan, D., & Weinberg, R. A. (2011). The Hallmarks of Cancer: The Next Generation. *Cell*, 144(5), 646–674.
2. Vogelstein, B., & Kinzler, K. W. (2004). Cancer genes and the pathways they control. *Nature Medicine*, 10(8), 789-799.
3. Kinzler, K. W., & Vogelstein, B. (1997). Cancer-susceptibility genes. *The Journal of Clinical Investigation*, 100(3), 639–646.
4. Kauffman, E. C., & Oltvai, Z. N. (2003). Molecular biology of cancer. *Journal of Clinical Oncology*, 21(23), 4461-4469.
5. Chong, J. C., & Parchment, R. E. (2006). Molecular diagnostics of cancer: Biomarkers and molecular techniques. *Nature Reviews Cancer*, 6(2), 140-145.
6. Bartek, J., & Lukas, J. (2007). Chk1 and Chk2 kinases in checkpoint control and cancer. *Cancer Cell*, 11(5), 510-519.
7. Mulligan, G. J., & Carter, S. (2008). Cancer genomics: From gene discovery to clinical applications. *The Lancet Oncology*, 9(7), 632-641.
8. López-Otín, C., & Hunter, T. (2010). The regulatory crosstalk between cancer and aging. *Nature Reviews Molecular Cell Biology*, 11(3), 201-214.
9. Zhang, L., & Xu, Z. (2015). Molecular pathogenesis of cancer: Advances in cancer biology and therapeutic strategies. *Annual Review of Cancer Biology*, 1(1), 45-68.
10. Takahashi, T., & Yuasa, Y. (2004). Molecular approaches to early diagnosis of cancer. *Journal of Cancer Research and Clinical Oncology*, 130(8), 505-515.
11. Шайхова, Г. И., Отажонов, И. О., & Рустамова, М. Т. (2019). Малобелковая диета для больных с хронической болезнью почек. *Экспериментальная и клиническая гастроэнтерология*, (12 (172)), 135-142.

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webofjournals.com/index.php/5

12. Отажонов, И. О. (2010). Характеристика фактического питания и качественный анализ нутриентов в рационе питания студентов высших учебных заведений. *Врач-аспирант*, *43*(6.2), 278-285.
13. Отажонов, И. О., & Шайхова, Г. И. (2020). Фактическое питание больных с хронической болезнью почек. *Медицинские новости*, (5 (308)), 52-54.
14. Islamovna, S. G., Komildjanovich, Z. A., Otaboevich, O. I., & Fatihovich, Z. J. (2016). Characteristics of social and living conditions, the incidence of patients with CRF. *European science review*, (3-4), 142-144.
15. Отажонов, И. О. (2011). Заболеваемость студентов по материалам углубленного медосмотра студентов, обучающихся в высших учебных заведениях. Тошкент тиббиёт академияси Ахборотномаси. *Тошкент,(2)*, *122126*.
16. Гинатуллина, Е. Н., Шамансурова, Х. Ш., Элинская, О. Л., Ражапова, Н. Р., Ражабова, Н. Т., & Тожиева, З. Б. (2016). ТОКСИКОЛОГИЧЕСКАЯ ОЦЕНКА МЕДИКО-БИОЛОГИЧЕСКОЙ БЕЗОПАСНОСТИ СЫРЬЯ ДЛЯ ПРОИЗВОДСТВА НОВОГО ВИДА ПРОДУКЦИИ–БЫСТРО РАСТВОРИМОГО ЧАЙНО-МОЛОЧНОГО НАПИТКА. *Рациональное питание, пищевые добавки и биостимуляторы*, (1), 43-47.
17. Назарова, М., & Тажиева, З. (2024). ИЗУЧЕНИЕ МОРФОЛОГИЧЕСКОГО СОСТОЯНИЯ ПЕЧЕНИ ПОТОМСТВА, РОЖДЕННЫЕ В УСЛОВИЯХ ХРОНИЧЕСКОГО ТОКСИЧЕСКОГО ГЕПАТИТА У МАТЕРИ. *Journal of science-innovative research in Uzbekistan*, *2*(12), 233-240.
18. Исмоилова, З. А., Тажиева, З. Б., & Ражабова, Н. Т. COVID-19 ЎТКАЗГАН БОЛАЛАРДА ЎТКИР БУЙРАК ШИКАСТЛАНИШИНИ ҚИЁСИЙ БАҲОЛАШ. *ДОКТОР АХБОРОТНОМАСИ ВЕСТНИК ВРАЧА DOCTOR'S HERALD*, 72.
19. Shalaeva, E., Janabaev, B., Matmurotov, Q., Kasimov, U., Pulatov, U., Bobabekov, A., & Bozorboev, M. (2016, June). 1-year clinical outcomes in patients with Parkinsonism syndrome with/without type 2 diabetes. In *MOVEMENT DISORDERS* (Vol. 31, pp. S62-S63). 111 RIVER ST, HOBOKEN 07030-5774, NJ USA: WILEY-BLACKWELL.
20. Shalaeva, E., Saner, H., Babadjanov, B., Pulatov, U., Matmurotov, Q., & Shalaeva, A. (2015, August). Prognostic value of coronary artery calcium score for major perioperative cardiovascular complications in type 2 diabetic patients undergoing trans-femoral amputation. In *EUROPEAN HEART JOURNAL* (Vol. 36, pp. 928-928). GREAT CLARENDON ST, OXFORD OX2 6DP, ENGLAND: OXFORD UNIV PRESS.
21. Атажанов, Т. Ш., Бабаджнаов, Б. Д., Матмуротов, К. Ж., & Саттаров, И. С. Анализ эффективности малоинвазивных методов в лечении диабетической гангрены нижних конечностей. *Раны и раневые инфекции*, 20-21.
22. Shalaeva, E., Janabaev, B., Babadjanov, B., Matmurotov, Q., Kasimov, U., Pulatov, U., & Bobabekov, A. (2016). Severity of coronary artery stenosis in patients with critical peripheral artery disease undergoing high amputation. *Atherosclerosis*, *252*, e141-e142.
23. Shalaeva, E., Janabaev, B., Matmurotov, Q., Kasimov, U., Pulatov, U., & Bobabekov, A. (2016). Severity of atherosclerotic lesions and foot synovial tendon complex injury as factors of sepsis development in patients with diabetic foot. *Atherosclerosis*, *252*, e137-e138.

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webofjournals.com/index.php/5

24. Бабаджанов, Б. Д., Матмуротов, К. Ж., Моминов, А. Т., Бабабеков, А. Р., Атаков, С. С., & Атажанов, Т. Ш. (2015). Эффективность внутриартериального введения флуконазола при лечении осложненных форм диабетической стопы. *ООО «Maxliyo-shifo» &*, *2014*, 28-30.
25. Babadjanov, B. D., & Matmurotov, K. J. (2019). Efficacy of minimally invasive procedures in the treatment of lower extremities diabetic gangrene. *European science review*, *2*(1-2), 79-82.
26. Матмуротов, К. Ж., & Жанабаев, Б. Б. (2011). Влияние микобактериальных ассоциаций на кратность повторных оперативных вмешательств при диабетической гангрене нижних конечностей. *Врач-аспирант*, *46*(3.3), 394-399.

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