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BIOCHEMICAL REGULATION OF THE CELL CYCLE AND ITS DISRUPTIONS IN TUMORS

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Abstract
The cell cycle is a complex and dynamic system that regulates cell growth, DNA replication, and division. This process is controlled through cyclins, cyclin-dependent kinases (CDKs), CDK inhibitors, and checkpoints. CDKs, when combined with cyclins, signal the cell to progress to the next phase of the cell cycle, making their activity a central mechanism for controlling cell division. If these regulatory mechanisms are disrupted, cells divide uncontrollably, leading to tumor (cancer) development. This article analyzes the biochemical regulation of the cell cycle, its genetic and molecular mechanisms, as well as the consequences of regulatory disruption in tumor cells, based on various statistical studies and experiments. The article contributes to a deeper understanding of diseases associated with cell cycle dysregulation and supports the advancement of oncological research and therapeutic strategies.

Keywords: Cell cycle, cyclins, cyclin-dependent kinases (CDKs), CDK inhibitors, checkpoints, cell division, tumor cells, cancer, genomic instability, DNA replication

Introduction

The cell cycle is a complex, organized, and dynamic mechanism that regulates cell division and growth in eukaryotic cells. It consists of the G1 phase (growth), S phase (DNA synthesis), G2 phase (preparation for division), and M phase (mitosis), with each phase closely linked to the cell's molecular state and

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metabolic activity. These processes are biochemically controlled through cyclins, cyclin-dependent kinases (CDKs), CDK inhibitors, and checkpoints. The cell cycle system ensures genomic stability, detects and repairs mutations, and guarantees orderly cell division.

Disruption of the cell cycle, particularly excessive CDK activity, loss or mutation of tumor suppressor genes such as **p53**, leads to uncontrolled cell proliferation. This forms the molecular basis of tumor (cancer) development. Proto-oncogenes and oncogenes provide stimulatory mechanisms for cell division, whereas tumor suppressor genes regulate and restrict cell division. Therefore, the molecular control of the cell cycle and its dysregulation is of central importance in oncology research, diagnostics, and the development of therapeutic strategies.

Main Body

1. Healthy Cell and Its Cycle

A healthy cell carries out growth, division, and apoptosis in an orderly and tightly regulated manner. These processes are controlled by the cell cycle, which consists of the G_1 phase (growth), S phase (DNA synthesis), G_2 phase (preparation for division), and M phase (mitosis). At each stage, the cell monitors its metabolic status, genomic stability, and molecular signals. This regulation is ensured through cyclins, cyclin-dependent kinases (CDKs), CDK inhibitors, and checkpoints.

- CDKs (cyclin-dependent kinases) govern cell division at the molecular level. By binding to cyclins, they signal the cell to progress to the next phase. For example, the CDK4/6 + Cyclin D complex facilitates the transition from G_1 to S phase, promoting DNA replication.
- CDK inhibitors (p21, p27, p16) restrict CDK activity, maintaining the cell cycle under tight control.
- Checkpoints at the G_1/S and G_2/M phases evaluate the cell's condition and halt division if DNA is damaged or if cellular stress is detected.

Through this tightly coordinated system, healthy cell division is orderly, ensures genomic stability, and prevents the accumulation of mutations and genomic instability.

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2. Cellular Transformation in Cancer: Mechanisms, Etiology, and Pathogenesis

When cell cycle control is disrupted, cells enter uncontrolled division, leading to tumor development. This process occurs through several molecular and genetic factors:

- **Proto-oncogenes and Oncogenes:** Proto-oncogenes promote growth and division in healthy cells. Their mutation or overactivation converts them into oncogenes, resulting in uncontrolled cell proliferation. Examples include **RAS, MYC, and Cyclin D**, which act as oncogenes in various cancer types.
- **Tumor Suppressor Genes and p53:** Tumor suppressor genes slow down cell division and promote apoptosis. The most well-known is **p53,** which halts the cell cycle or triggers apoptosis when DNA is damaged. If p53 is mutated, cells continue dividing with damaged DNA, representing a key molecular pathway in cancer development.
- **Disruption of CDKs and CDK Inhibitors:** Excessive CDK activity or insufficient levels of CDK inhibitors (CKIs) leads to uncontrolled cell cycle progression and increases genomic instability.
- **Checkpoint Dysfunction:** Malfunction of **G_1/S** and **G_2/M checkpoints** allows cells to continue dividing despite DNA damage.

These mechanisms collectively initiate tumor development by promoting uncontrolled cell proliferation, impairing apoptosis, and increasing genomic instability.

Brain Cancer and Cell Cycle Dysregulation

Brain cancer, particularly **gliomas** and **glioblastomas**, represents some of the most complex and aggressive tumors that arise from disrupted cell cycle control. In healthy brain cells—**neuroglial cells**—division is tightly regulated. These cells halt division when DNA is damaged, activate apoptosis when necessary, and maintain genomic stability.

However, during brain tumor development, multiple molecular and genetic factors act together to promote **uncontrolled cell proliferation**.

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Mutation of Proto-Oncogenes and Oncogene Activation

In healthy cells, **proto-oncogenes** promote growth and division in a controlled manner. However, mutations or excessive activation convert them into **oncogenes.** For example**, RAS** and **MYC oncogenes** in neuroglial cells become hyperactive, leading to uncontrolled cell proliferation. This contributes to rapid cell growth and tumor development.

Disruption of Tumor Suppressor Genes

The **p53 gene** functions to detect DNA-damaged cells, halt the cell cycle, and activate apoptosis. If **p53 is mutated** or loses its function, cells continue to divide even with damaged DNA. This increases genomic instability and represents a central molecular pathway for tumor development.

Dysregulation of CDKs, CDK Inhibitors, and Checkpoint Systems

The **CDK4/6 + Cyclin D complex** drives cells from the G_1 phase to the S phase. Excessive CDK activity or insufficient levels of **CDK inhibitors (p21, p27, p16)** lead to uncontrolled cell cycle progression. Additionally, dysfunction of **G_1/S and G_2/M checkpoints** allows DNA-damaged cells to continue dividing.

As a result, normal neuroglial cells proliferate uncontrollably, apoptotic mechanisms fail, and high-grade brain tumors such as **glioblastomas** develop. This process is **etiologically and pathogenetically complex,** involving genetic mutations, epigenetic changes, overactivation of growth factors, and immune system suppression.

Clinical Studies and Statistics

In **glioblastomas, CDK4/6 activity** is often found to be excessively high, leading to rapid cell proliferation. Mutations in the **p53 gene** are detected in approximately **50–70% of patients,** contributing to aggressive tumor progression. The activity **of proto-oncogenes (RAS, MYC)** increases the risk of tumor development by **2–4 times.**

Studying these molecular mechanisms has led to the development of **CDK4/6 inhibitors,** therapies that suppress oncogene activity, and strategies to restore p53

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function. These approaches show promising results in controlling brain cancer, slowing cell division, and limiting tumor growth.

Statistics

In 2022, approximately **19.98 million new cancer cases** were reported worldwide, and **9.74 million patients** died from cancer. The most common cancers in men were: **lung (~2.5 million), prostate (~1.47 million), colorectal (~1.07 million), stomach (~0.63 million), and liver (~0.60 million).** In women, the most common cancers were: **breast (~2.30 million), lung (~0.91 million), colorectal (~0.86 million), cervical (~0.66 million), and thyroid (~0.61 million).**

The leading causes of cancer-related deaths were: **lung (~1.8 million), colorectal (~0.9 million), liver (~0.76 million), breast (~0.67 million), and stomach (~0.66 million)**.

According to projections by the **World Health Organization**, by 2050, new cancer cases may reach approximately **35 million,** representing a **+75% increase** compared to 2022, with deaths estimated at around **18–19 million patients** (GLOBOCAN, IARC, WHO 2022; The Guardian 2024).

Conclusion

Aging and cell cycle dysregulation are complex, multifactorial biological phenomena, and understanding them at the molecular and genetic levels is crucial for the prevention of cancer and other age-related diseases. When cell cycle control is disrupted, **proto-oncogenes become activated**, the activity of **tumor suppressor genes**, particularly **p53**, is impaired, and **CDKs and checkpoint systems** become dysfunctional, leading to uncontrolled cell proliferation and tumor development.

Global statistics indicate that in 2022, approximately **20 million new cancer cases** were reported, with around **9.74 million deaths**, the most common types being **lung, breast, colorectal, and liver cancers**. Projections suggest that by 2050, new cases may reach **~35 million**, with deaths estimated at **18–19 million**.

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A healthy lifestyle and preventive measures can significantly reduce cancer risk. These include: **regular physical activity, balanced nutrition, maintaining a healthy weight, limiting tobacco and alcohol use, a diet rich in antioxidants to reduce mutations, prevention of viral infections (HPV, Hepatitis B), stress management, and regular medical check-ups**. At the molecular level, therapies that regulate the **cell cycle** and promote **apoptosis** also represent promising approaches for cancer prevention.

Thus, understanding the **cell cycle** and its **molecular mechanisms** is not only essential for the detection and treatment of cancer but also holds scientific and practical importance for prevention and the promotion of a long and healthy life.

References

1. AbduIov, I. A., & Xalbekova, X. U. (2026). Cell Biology (in Uzbek). Turan Edu Publishing.
2. Ministry of Health of the Republic of Uzbekistan. (2024). Cell Life Cycle, Cell Division, and Mitosis. Official Educational Guide.
3. Saidmurodova, Z. A., & Halimova, S. A. (2022). Cell proliferation and related concepts. Eurasian Journal of Medical and Natural Sciences.
4. Zhong, J., Liu, J., Tang, X., Zhou, W., Song, G., Zeng, Y., Zhang, X., & Zhou, J. (2025). Cell cycle proteins: Linking the cell cycle to tumors. Oncology Research, 33(6), 1335–1346.
5. Morgan, D. (2007). The Cell Cycle: Principles of Control. New Science Press.
6. p53 — Tumor suppressor protein. (n.d.). Wikipedia.
7. CDKN2A — Cyclin Dependent Kinase Inhibitor 2A (p16/p19ARF). (n.d.). Wikipedia.
8. World Health Organization. (2022). Global Cancer Statistics (GLOBOCAN Fact Sheets). International Agency for Research on Cancer.
9. The Guardian. (2024, February 1). Global cancer cases to rise by more than 75% by 2050, WHO predicts.