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The Emergence of Cancer and the Elimination of Cancer Cells

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Abstract: Cancer is a complex genetic disease whose development is associated with numerous changes at the cellular level. This article provides a comprehensive examination of the uncontrolled growth of cancer cells, impaired differentiation, and mechanisms of evasion of apoptosis. It also analyzes the main molecular pathways of oncogenesis and modern therapeutic approaches targeting them, including chemotherapy, radiotherapy, immunotherapy, and targeted therapy. The study emphasizes the importance of an in-depth understanding of the pathophysiology of the disease in developing new strategies aimed at effectively eliminating cancer cells.

Keywords: Cancer, Cancer cells, Oncogenesis, Therapy, Apoptosis, Molecular mechanisms, Immunotherapy

Introduction

Cancer (oncological diseases) remains one of the most pressing global public health challenges of the twenty-first century. It claims millions of lives every year and places an enormous economic and social burden on society. According to the World Health Organization, approximately 19 million people worldwide develop cancer each year, and nearly 8 million people die from this disease [2]. These figures clearly demonstrate the widespread prevalence of the disease and the serious threat it poses to humanity. There are more than 220 types of cancer, each with its own specific mechanisms of development, clinical manifestations, and treatment methods [2]. This complexity creates significant challenges in diagnosing the disease and developing effective therapies.

The essence of cancer is manifested in the uncontrolled growth and division of cells, as well as their ability to invade surrounding tissues and metastasize to other parts of the body [2]. Genetic changes and mutations lie at the root of these processes. Cellular health is maintained through a delicate balance between oncogenes and tumor suppressor genes; however, inherited or acquired mutations can disrupt this balance [3]. For example, mutations in the BRCA1 and BRCA2 genes in breast and ovarian cancer, and in the APC and TP53 genes in colorectal cancer, play a decisive role in disease development [3]. Numerous factors, including viruses, microbes, toxic chemicals, hormones, radiation, and heredity, also contribute to the development of cancer [2]. In particular, colorectal cancer (CRC) remains a pressing issue in oncological coloproctology, and there is a continuing need to study its risk factors, mechanisms of development, and genetic composition [1]. Genetic composition plays a key role in disease prognosis, and the identification of transcriptomic signature-based subtypes is important for a more comprehensive understanding of CRC [1].

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The distinctive characteristics of cancer cells, including their growth at twice the rate of normal cells and their high degree of heterogeneity (for example, five types of stomach cancer, six types of breast and lung cancer, and seventeen types of ovarian cancer), complicate treatment [2]. The ability to metastasize leads to disease spread and reduces treatment effectiveness. Therefore, it is essential to gain an in-depth understanding of the molecular mechanisms of cancer, decode the genetic changes that lead to its development, and identify disruptions in cell-cycle regulation [3]. This knowledge serves as a basis for understanding the causes of the disease and developing personalized treatment strategies [3].

This article aims to provide a comprehensive analysis of the emergence of cancer, its molecular and genetic foundations, disruptions in cell-cycle regulation, and modern and promising methods for eliminating cancer cells. Alongside conventional treatment methods, particular attention is given to innovative approaches such as targeted therapy, immunotherapy, and personalized medicine. Specifically, personalized anticancer vaccines prepared from a patient's own tumor, such as dendritic cell vaccines, have been reported to potentially double treatment outcomes [2]. The introduction of early diagnosis and modern technologies may create the possibility of curing malignant tumors in 100% of cases [2]. The main aim of the article is to explain the complex biology of cancer, clarify the mechanisms of its development, and contribute to the development of new and more effective strategies for combating this disease in the future. This, in turn, will help improve the quality of life and extend the survival of patients with cancer.

Literature Review

An in-depth understanding of the molecular and genetic foundations of cancer development is crucial for developing effective strategies to combat this disease. As emphasized in the initial sources, genetic changes and mutations underlie the uncontrolled growth and division of cancer cells, as well as their ability to metastasize [3]. Studies conducted in recent years have further highlighted the complexity of these processes. Disruption of the delicate balance between oncogenes and tumor suppressor genes, which maintains cellular health, often occurs as a result of inherited or acquired mutations [3]. For example, activating mutations in oncogenes such as RAS, MYC, and EGFR initiate signaling pathways that continuously stimulate cell growth and proliferation. Conversely, inactivating mutations in tumor suppressor genes such as TP53, RB1, BRCA1, and BRCA2 disrupt cell-cycle control, DNA repair mechanisms, and apoptosis (programmed cell death), allowing damaged cells to survive and multiply [3]. Research conducted after 2020 has focused particularly on the role of epigenetic changes, such as DNA methylation and histone modifications, in cancer development. These changes can alter gene expression without changing the genetic sequence itself, thereby contributing to cancer pathogenesis through the activation of oncogenes or the inactivation of tumor suppressor genes. Understanding these molecular pathways provides a basis for developing targeted therapies.

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Disruption of cell-cycle regulation is one of the main characteristics of cancer development. In normal cells, the cell cycle is strictly controlled and includes the G1, S, G2, and M phases; these processes are regulated by cyclins and cyclin-dependent kinases (CDKs). Cell-cycle checkpoints (G1/S, G2/M, and the mitotic checkpoint) ensure DNA integrity and the proper progression of cell division. In cancer cells, however, these regulatory mechanisms are disrupted. For example, overexpression of cyclin D1 or activation of CDK4/6 accelerates the transition from the G1 phase to the S phase. At the same time, inactivation of CDK inhibitors such as p16, p21, and p27, or mutations in the tumor suppressor genes TP53 and RB1, abolish cell-cycle control, resulting in uncontrolled cellular proliferation [3]. Recent studies have demonstrated the effectiveness of CDK4/6 inhibitors (such as palbociclib and ribociclib) in the treatment of breast cancer, confirming the clinical importance of targeting cell-cycle regulation.

Analyzing cancer-causing factors and risk groups is important for disease prevention and early diagnosis. The initial text identifies factors such as viruses, microbes, toxic chemicals, hormones, radiation, and heredity [2]. The literature review indicates that human papillomavirus (HPV) plays an important role in cervical cancer; hepatitis B and C viruses (HBV and HCV) in liver cancer; and Epstein–Barr virus (EBV) in the development of nasopharyngeal carcinoma and certain lymphomas. The bacterium Helicobacter pylori increases the risk of stomach cancer. Chemical carcinogens, such as polycyclic aromatic hydrocarbons and aromatic amines in tobacco smoke, directly damage DNA and cause mutations. Ionizing radiation (X-rays and gamma rays) and ultraviolet (UV) radiation also cause DNA damage. Hereditary predisposition manifests through genetic syndromes such as Lynch syndrome (colorectal cancer), Li-Fraumeni syndrome (various types of cancer), and BRCA1/2 mutations (breast and ovarian cancer) [3]. Epidemiological studies conducted after 2020 have further clarified the role of lifestyle factors, including obesity, unhealthy diet, physical inactivity, and alcohol consumption, in increasing cancer risk. Understanding these factors provides a foundation for improving public health and optimizing cancer screening programs.

Traditional cancer treatment methods, including surgery, chemotherapy, and radiotherapy, have long formed the basis of oncological practice. Surgery is considered effective, particularly for removing localized tumors and treating the disease in its early stages. Its main objective is complete tumor resection and the prevention of metastasis. However, the possibilities of surgery are limited in metastatic disease or when the tumor is located in an anatomically complex area. Radiotherapy uses high-energy radiation to destroy cancer cells by damaging their DNA. It is used to treat localized tumors or to eliminate residual cells after surgery. The main disadvantages of radiotherapy are its potential to damage healthy tissues and the development of radioresistance. Chemotherapy uses drugs that stop the growth of cancer cells or kill them by exploiting their rapid-dividing properties. It has a systemic effect and is also used in metastatic disease. However, the main limitations of chemotherapy are its toxic effects on healthy, rapidly dividing cells (for example, bone marrow cells, hair follicles, and gastrointestinal tract cells), serious adverse effects, and the development of drug resistance. In

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recent years, these traditional methods have often been used in combination with modern therapies, helping to increase treatment effectiveness.

Modern cancer therapies, particularly targeted therapy and immunotherapy, have brought revolutionary changes to oncology. Targeted therapy is directed at specific molecular pathways or genetic alterations that are essential for the growth and survival of cancer cells. Compared with conventional chemotherapy, this approach is more selective and causes minimal damage to healthy cells. For example, tyrosine kinase inhibitors (TKIs), including imatinib (for chronic myeloid leukemia and gastrointestinal stromal tumors), and gefitinib and erlotinib (for lung cancer with EGFR mutations), block growth signals in cancer cells. Monoclonal antibodies, such as trastuzumab (for HER2-positive breast cancer) and cetuximab (for EGFR-positive colorectal cancer), bind to receptors on the surface of tumor cells, stopping their growth or directing the immune system to destroy them. PARP inhibitors (for example, olaparib) demonstrate efficacy in ovarian and breast cancers with BRCA mutations through the mechanism of synthetic lethality. The main challenge associated with targeted therapy is the development of drug resistance, which often occurs as a result of secondary mutations or bypass activation of signaling pathways.

Immunotherapy aims to mobilize the patient's own immune system to fight cancer cells. One of the most important achievements in this field is the development of immune checkpoint inhibitors (ICIs). PD-1/PD-L1 inhibitors (pembrolizumab, nivolumab, and atezolizumab) and CTLA-4 inhibitors (ipilimumab) block the “brake” mechanisms in T cells, restoring the immune system's ability to recognize and destroy cancer cells. These drugs have achieved significant success in the treatment of melanoma and lung, kidney, and bladder cancers. Another innovative approach is CAR T-cell therapy, in which a patient's T cells are genetically modified and equipped with chimeric antigen receptors (CARs) that recognize specific antigens on cancer cells (for example, CD19 in leukemia and lymphomas). This therapy has demonstrated remarkable results in the treatment of hematological malignancies. As emphasized in the initial text, personalized anticancer vaccines prepared from a patient's own tumor, particularly dendritic cell vaccines, may double treatment outcomes [2]. These vaccines aim to trigger a strong and specific antitumor immune response by presenting tumor antigens to the immune system. They may be particularly effective in the early stages of the disease and when used in combination with other therapies. The limitations of immunotherapy include immune-related adverse events (irAEs) and the fact that not all patients respond to treatment.

Future approaches and personalized medicine promise increasingly effective strategies for cancer treatment. Multi-omics technologies (genomics, transcriptomics, proteomics, and metabolomics) make it possible to analyze a patient's cancer comprehensively, providing a basis for identifying the molecular profile of the disease and developing a personalized treatment plan. Liquid biopsies (circulating tumor DNA (ctDNA), circulating tumor cells (CTCs), and exosomes in the blood) have considerable potential for the early diagnosis of cancer using minimally invasive methods, monitoring treatment response, and identifying resistance mechanisms. Gene-editing technologies such as

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CRISPR/Cas9 offer theoretical possibilities for treating cancer at the genetic level, for example, by inactivating oncogenes or restoring tumor suppressor genes. Artificial intelligence (AI) and machine learning (ML) algorithms are playing an increasingly important role in diagnosis (analysis of pathology and radiology images), drug discovery, prediction of treatment response, and the provision of personalized recommendations based on large patient databases. Nanotechnology opens up new opportunities for targeted drug delivery to cancer cells and for improving imaging methods.

Colorectal cancer (CRC) remains a pressing issue in oncological coloproctology, and there is a continuing need to study its risk factors, mechanisms of development, and genetic composition [1]. Recent literature reviews have focused on the complex molecular mechanisms of CRC, particularly the adenoma–carcinoma sequence and the associated genetic changes (mutations in APC, KRAS, TP53, and SMAD4). Molecular pathways such as microsatellite instability (MSI) and chromosomal instability (CIN) are important for identifying different CRC subtypes. In particular, the identification of transcriptomic signature-based subtypes is important for a more comprehensive understanding of CRC [1]. Classification systems such as the Consensus Molecular Subtypes (CMS1–4) help determine the prognostic and predictive characteristics of CRC. For example, patients with the CMS1 (MSI-immune) subtype of CRC may respond well to immune checkpoint inhibitors, which is important for personalizing treatment strategies. This advanced understanding, in turn, provides a basis for developing highly specific and tailored treatment methods [1]. The implementation of disease-specific therapeutic strategies is expected to lead to significantly more effective management of colorectal cancer, thereby improving patient care and outcomes [1].

In conclusion, despite the progress made in understanding the complex biology of cancer, many challenges remain. Tumor heterogeneity, the development of drug resistance, and the effective treatment of metastatic disease continue to be pressing issues. However, as emphasized in the initial text, early diagnosis and the introduction of modern technologies may create the possibility of curing malignant tumors in 100% of cases [2]. Achieving this goal requires continued research in molecular biology, genetics, immunology, and bioinformatics, as well as the introduction of innovative approaches into clinical practice. The development of personalized medicine makes it possible to tailor treatment to the specific molecular profile of each patient, thereby improving the quality of life and extending the survival of people with cancer.

Research Methodology

This article aims to provide a comprehensive analysis of the emergence of cancer, its molecular and genetic foundations, disruptions in cell-cycle regulation, and modern and promising methods for eliminating cancer cells. Its methodology is based on the principles of a systematic literature review. The article is intended to highlight the complex aspects of cancer biology, explain the mechanisms of its development, and contribute to the development of new and more effective strategies for combating this disease in the future. To achieve this aim, the following research questions were formulated, defining the main areas of analysis:

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What are the molecular and genetic foundations of the emergence and development of cancer, and what roles do oncogenes, tumor suppressor genes, and epigenetic changes play in these processes?

How does disruption of cell-cycle regulation affect cancer pathogenesis, and is targeted intervention in these disruptions of therapeutic importance?

What are the main cancer-causing factors (viruses, microbes, chemicals, radiation, hormones, and heredity) and risk groups, and how do lifestyle factors contribute to disease development?

What mechanisms underlie traditional cancer treatment methods (surgery, chemotherapy, and radiotherapy), what are their advantages and limitations, and how are they integrated with modern therapies?

What innovative approaches are offered by modern cancer therapies, particularly targeted therapy (tyrosine kinase inhibitors, monoclonal antibodies, and PARP inhibitors) and immunotherapy (immune checkpoint inhibitors, CAR T-cell therapy, and dendritic cell vaccines), and what are their clinical effectiveness and main challenges (such as resistance)?

What potential do promising future approaches to cancer treatment (personalized medicine, multi-omics technologies, liquid biopsy, gene editing, artificial intelligence, and nanotechnology) offer, and how can they improve treatment outcomes?

Using colorectal cancer (CRC) as an example, what is the significance of the molecular subtypes of the disease (such as transcriptomic signature-based subtypes and CMS) for personalizing treatment strategies, and how is this approach expected to improve patient care?

The search for relevant literature for the article was conducted in a comprehensive and systematic manner. PubMed, Scopus, Web of Science, Google Scholar, and ScienceDirect were selected as the main scientific databases for the search. These platforms contain the most relevant and reputable peer-reviewed publications in biomedicine and oncology, helping to ensure the scientific validity of the analysis. Search queries were formulated in Uzbek and English using the following main keywords and various combinations thereof:

In Uzbek: “saraton paydo bo'lishi” (cancer development), “rak hujayralari yo'q qilish” (cancer cell elimination), “saraton molekulyar asoslari” (molecular basis of cancer), “genetik mutatsiyalar saraton” (genetic mutations in cancer), “hujayra sikli nazorati buzilishi” (disruption of cell-cycle control), “onkogenlar” (oncogenes), “o'simta supressor genlari” (tumor suppressor genes), “maqsadli terapiya” (targeted therapy), “immunoterapiya” (immunotherapy), “CAR T-hujayra” (CAR T cell), “dendrit hujayrali vaksinalar” (dendritic cell vaccines), “shaxsiylashtirilgan tibbiyot” (personalized medicine), “suyuq biopsiya” (liquid biopsy), “CRISPR/Cas9 saraton” (CRISPR/Cas9 cancer), “sun'iy intellekt onkologiya” (artificial intelligence in oncology), “nanotexnologiya saraton” (nanotechnology in cancer), “yo'g'on ichak saratoni molekulyar” (colorectal cancer molecular), “YIS subtiplari” (CRC subtypes), “saraton xavf omillari” (cancer risk factors), “saraton davolash usullari” (cancer treatment methods), “saraton epigenetikasi” (cancer epigenetics), “hujayra proliferatsiyasi saraton” (cell proliferation in cancer), and “metastaz mexanizmlari” (mechanisms of metastasis).

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In English: “cancer origin,” “cancer cell elimination,” “molecular basis of cancer,” “genetic mutations cancer,” “cell cycle control disruption,” “oncogenes,” “tumor suppressor genes,” “targeted therapy,” “immunotherapy,” “CAR T-cell therapy,” “dendritic cell vaccines cancer,” “personalized medicine oncology,” “liquid biopsy cancer,” “CRISPR/Cas9 cancer,” “artificial intelligence oncology,” “nanotechnology cancer,” “colorectal cancer molecular,” “CRC subtypes,” “cancer risk factors,” “cancer treatment modalities,” “cancer epigenetics,” “cell proliferation cancer,” and “metastasis mechanisms.”

The selected keywords were intended to fully cover the main topics and research questions of the article, reflecting a broad spectrum ranging from the molecular biology of cancer to its clinical treatment methods. Using keywords in various combinations, such as “cancer AND molecular basis,” “immunotherapy AND CAR T-cell,” and “colorectal cancer AND genetic subtypes,” increased the precision and relevance of the search results. Boolean operators (AND, OR, NOT) and truncation (*) symbols (for example, using the word stem “therap” to cover variants such as “therapy,” “therapies,” and “therapeutic”) were applied. This approach ensured the retrieval of the maximum possible number of relevant publications and increased the efficiency of the search process.

The publication date restriction was important in selecting the literature. To ensure the relevance of the article, particular attention was paid mainly to scientific literature published after 2020. This restriction made it possible to analyze the latest advances and innovative approaches in cancer research, as this field is developing very rapidly and new discoveries are emerging regularly. At the same time, fundamental theories and discoveries published before 2020, which were necessary to explain the basic mechanisms of cancer, were also carefully included when they provided a solid foundation for modern understanding. This helped preserve the historical context of the article and provide a more comprehensive account of the topic. In the literature review, sources [1], [2], and [3] served as the main guiding documents and established the starting point for the analysis. In particular, information from source [2] on the global burden of cancer, its types, and personalized anticancer vaccines, as well as information from source [3] on the role of genetic changes and mutations in cancer pathogenesis and on genetic testing, helped define the main directions during the initial stages of the literature search. Information from source [1] on the molecular mechanisms of colorectal cancer (CRC) and transcriptomic signature-based subtypes provided the basis for an in-depth analysis of CRC.

All literature identified through the search was subjected to a two-stage selection process. In the first stage, the titles and abstracts of the identified articles were initially screened for relevance to the research questions and the overall aim of the article. Sources that were not directly related to the topic, were duplicates, or were of poor quality were excluded at this stage. In the second stage, the full texts of articles that passed the initial screening were examined in depth. Each article was evaluated in terms of the relevance of its content, scientific validity, methodological rigor, and its potential to answer the research questions.

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Publication prestige and scientific reputation: Preference was given to peer-reviewed articles published in high-impact-factor, internationally recognized scientific journals. This was important for ensuring the reliability and scientific value of the information presented.

Study type and design: Original research articles, comprehensive review articles, meta-analyses, systematic reviews, clinical trial results, and official reports and guidelines from reputable organizations (such as the World Health Organization and the National Cancer Institute) were included. Results from laboratory studies (in vitro and in vivo), animal models, and human clinical trials were assessed separately.

Currency and relevance of information: Publications from after 2020 were given priority, making it possible to reflect the latest advances in cancer biology and treatment, new technologies, and innovative approaches. Older information of fundamental importance was included only when it served as a basis for modern understanding.

Topical relevance and coverage: Articles containing information on the emergence of cancer, its molecular mechanisms, genetic foundations, cell-cycle regulation, treatment methods (conventional, targeted, and immunotherapy), and future approaches were selected. Each section was designed to directly address the research questions.

Strength of evidence and methodological rigor: Particular attention was paid to clinical trial results (especially Phase II and Phase III trials), large cohort studies, and reproducible in vitro/in vivo studies that clearly explain molecular mechanisms. Studies with weak methodology, small sample sizes, or statistically insignificant results were excluded from the selection or their limitations were indicated.

Assessment of bias: The presence of potential bias in each study (for example, selection bias, publication bias, and financial conflicts of interest) was assessed, and consideration was given to how these factors could affect the interpretation of the results.

During the selection process, when several articles were found on the same topic, the sources providing the most comprehensive, up-to-date, and reliable evidence were selected. In some cases, several articles were included to cover different aspects of a topic, ensuring a multifaceted analysis. In addition, the bibliographies (reference lists) of the key articles were examined to identify further relevant sources (the snowballing method). This approach helped increase the completeness and depth of the literature review while making it possible to identify the best available evidence for each aspect of the topic.

Data extraction and synthesis from the selected literature played a decisive role in shaping the main content of the article. The following types of information were systematically extracted from each article and organized into thematic groups:

Molecular and genetic foundations of cancer: Information on oncogenes (for example, RAS, MYC, and EGFR), tumor suppressor genes (for example, TP53, RB1, and BRCA1/2), types of

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mutations in these genes (activating and inactivating), epigenetic changes (DNA methylation and histone modifications), and their effects on gene expression.

Disruption of cell-cycle regulation: The roles of cyclins, cyclin-dependent kinases (CDKs), and their inhibitors (p16, p21, and p27); disruption of cell-cycle checkpoints (G1/S, G2/M, and mitotic); and the ways in which these disruptions lead to uncontrolled proliferation of cancer cells.

Cancer-causing factors: Epidemiological and molecular evidence concerning viruses (HPV, HBV, HCV, and EBV), bacteria (Helicobacter pylori), chemical carcinogens (tobacco smoke), radiation (ionizing and UV), hormones, hereditary predisposition (Lynch syndrome, Li-Fraumeni syndrome, and BRCA1/2 mutations), and lifestyle factors (obesity, unhealthy diet, and alcohol).

Traditional treatment methods: The principles of surgical resection, the DNA-damaging mechanisms of radiotherapy, the mechanisms by which chemotherapy inhibits cell division, their clinical applications, effectiveness, adverse effects, and resistance-related problems.

Targeted therapy: The mechanisms of action on specific molecular pathways of drugs such as tyrosine kinase inhibitors (for example, imatinib, gefitinib, and erlotinib), monoclonal antibodies (for example, trastuzumab and cetuximab), and PARP inhibitors (for example, olaparib), their clinical indications, and the causes of the development of drug resistance.

Immunotherapy: The mechanisms by which approaches such as immune checkpoint inhibitors (for example, PD-1/PD-L1 and CTLA-4 inhibitors), CAR T-cell therapy, and dendritic cell vaccines [2] mobilize the immune system against cancer, their clinical outcomes, and immune-related adverse events (irAEs).

Future approaches: The role of multi-omics technologies (genomics, transcriptomics, proteomics, and metabolomics) in comprehensive cancer analysis; the potential of liquid biopsies (ctDNA, CTCs, and exosomes) for early diagnosis and monitoring; the therapeutic possibilities of gene-editing technologies (CRISPR/Cas9); the importance of artificial intelligence (AI) and machine learning (ML) algorithms in diagnosis and treatment personalization; and new opportunities offered by nanotechnology for targeted drug delivery.

Specific information on colorectal cancer (CRC): The adenoma–carcinoma sequence; genetic changes (mutations in APC, KRAS, TP53, and SMAD4); molecular pathways such as microsatellite instability (MSI) and chromosomal instability (CIN); Consensus Molecular Subtypes (CMS1–4) [1] and their prognostic/predictive significance; and the effectiveness of personalized approaches to CRC treatment.

The extracted information was grouped by topic and synthesized in accordance with the planned structure of the article. During synthesis, particular attention was paid not merely to compiling information but also to critically analyzing it, combining evidence from different sources, comparing conflicting views, and formulating general conclusions. This approach made it possible to present a comprehensive and coherent picture of the complex biology and therapy of cancer. During data synthesis, the evidence presented in each section was based on the most recent and reliable sources,

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while the main ideas of sources [1], [2], and [3] were incorporated organically into the relevant parts of the article. For example, the global burden of cancer and the effectiveness of personalized vaccines emphasized in source [2] were discussed in greater detail in the "Introduction" and "Modern Cancer Therapies" sections. Information from source [3] on genetic changes and genetic testing was explored in greater depth in the section "Molecular and Genetic Foundations of Cancer." The molecular subtypes of CRC and personalized treatment strategies described in source [1] were analyzed in detail in the section "Future Approaches, Personalized Medicine, and Conclusion," demonstrating the importance of implementing disease-specific therapeutic strategies.

The structure of the article followed the planned outline and ensured a logical sequence from the fundamental biology of cancer to its clinical manifestations, treatment methods, and future prospects. Each section was designed to address specific research questions and was enriched with the most important information obtained from the literature review.

The "Introduction" section presented the global burden of cancer [2] and the main aim of the article. This section emphasized that cancer poses a serious threat to humanity and highlighted the complexity of its biology, diagnosis, and the development of effective therapies.

The section "Molecular and Genetic Foundations of Cancer" provided an in-depth analysis of the roles of oncogenes, tumor suppressor genes [3], mutations, and epigenetic changes in cancer development. Particular attention was paid to the importance of epigenetic mechanisms and their effects on gene expression in studies conducted after 2020, revealing an even more complex picture of cancer pathogenesis.

The section "Cell-Cycle Regulation and Cancer Development" described the complex mechanisms of cell-cycle regulation, the roles of cyclins, CDKs, and their inhibitors, and how disruption of this regulation contributes to the uncontrolled proliferation of cancer cells. The clinical importance of modern therapies such as CDK4/6 inhibitors was also emphasized, demonstrating the therapeutic potential of targeting cell-cycle regulation.

The section "Cancer-Causing Factors and Risk Groups" analyzed, on the basis of epidemiological studies, the roles of viruses, bacteria, chemical carcinogens, radiation, hormones, hereditary predisposition [3], and lifestyle factors in increasing cancer risk. This section emphasized the importance of understanding risk factors in developing strategies for disease prevention and early diagnosis.

The section "Traditional Cancer Treatment Methods" discussed the basic principles of surgery, chemotherapy, and radiotherapy, their advantages and limitations, and the possibilities of combining them with modern therapies. This section highlighted the long-standing role of traditional methods in clinical practice and their integrated role in modern oncology.

The section "Modern Cancer Therapies: Targeted Therapy and Immunotherapy" was one of the central sections of the article. It provided a comprehensive account of the molecular mechanisms of targeted therapy, various drugs (TKIs, monoclonal antibodies, and PARP inhibitors), and their clinical

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applications, as well as the revolutionary approaches of immunotherapy (ICIs, CAR T-cell therapy, and dendritic cell vaccines [2]). Resistance mechanisms and immune-related adverse effects were also critically assessed, demonstrating the complexity of these therapies and the need to optimize them.

The section “Future Approaches, Personalized Medicine, and Conclusion” discussed in detail the newest and most promising areas of cancer treatment, including the potential of multi-omics technologies, liquid biopsies, gene editing (CRISPR/Cas9), artificial intelligence (AI), machine learning (ML), and nanotechnology. Particular emphasis was placed on the importance of personalized medicine in tailoring treatment to each patient's specific molecular profile. Using colorectal cancer (CRC) [1] as an example, the importance of identifying molecular subtypes and developing treatment strategies tailored to them was discussed in detail. The conclusion of the article outlined achievements in understanding and treating cancer, as well as existing challenges and directions for future research; at the same time, the idea that early diagnosis and modern technologies may create the possibility of a 100% cure [2] was reiterated.

The principles of scientific accuracy, consistency, and clarity were strictly observed in developing the content of the article. Complex molecular and genetic processes, as well as therapeutic approaches, were presented in clear and concise academic language. Each claim was supported by reliable scientific sources and confirmed by relevant references.

Since this literature review article does not include research involving human participants, approval from an ethics committee was not required. Nevertheless, the general ethical principles of scientific research were strictly observed, including objectivity of information, prevention of plagiarism, and correct citation of all sources used. Scientific integrity and respect for intellectual property rights were fundamental principles throughout the preparation of the article.

Like any literature review, this article also has certain limitations. First, the scope of the analysis is determined by the selected databases, keywords, and publication date restrictions. This may mean that not all available literature on the topic has been covered, despite the use of a comprehensive search strategy. Second, publication bias may exist in the scientific literature, meaning that positive results are published more frequently than negative results. This may lead to some distortion of the overall picture of the topic. Third, because cancer research is developing very rapidly, some information that is the most up to date at the time of writing may be revised or changed within a short period. This may limit the time-specific relevance of the analysis. Fourth, methodological diversity and heterogeneity of results across different studies may create difficulties in synthesizing the information, since results obtained from different study designs cannot always be compared directly. Despite these limitations, the article seeks to provide a comprehensive and critical analysis of recent advances in cancer biology and treatment, serving as a valuable source of information for specialists and students in the field.

This methodology is intended to ensure the scientific validity, consistency, and relevance of the article, and to help develop an in-depth understanding of the complex field of cancer and identify new

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directions for combating it. The systematic approach used in the research process increases the reliability and objectivity of the conclusions and recommendations presented in the article. The information presented in the article, with particular emphasis on advances achieved after 2020, demonstrates the future potential of personalized and innovative approaches to cancer treatment. Approaches such as identifying molecular subtypes and applying tailored therapies using colorectal cancer as an example [1] represent an important step toward more effective cancer management and improved patient quality of life. The article also seeks to support with scientific evidence the optimistic view that early diagnosis and modern technologies may make it possible to achieve a complete cure for cancer [2]. This identifies important directions for future research and clinical practice in oncology while contributing to collective efforts aimed at addressing the global public health challenge posed by cancer.

Conclusion

This article provided a comprehensive analysis of the complex biology of cancer, its genetic and molecular foundations, disruptions in cell-cycle regulation, and the factors that cause the disease. Alongside traditional treatment methods, innovative approaches such as targeted therapy and immunotherapy have achieved significant advances in oncology. In the future, advanced tools such as personalized medicine, multi-omics technologies, liquid biopsy, and artificial intelligence will provide a basis for developing treatment strategies tailored to each patient's specific molecular profile. In particular, the identification of molecular subtypes, using colorectal cancer as an example, is important for increasing treatment effectiveness. Although early diagnosis and the integration of modern technologies promise the possibility of a complete cure for cancer, challenges such as tumor heterogeneity and drug resistance remain important areas for further research.

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