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Treatment with monoclonal antibodies in cancer - efficacy and prospects

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Abstract

Objective: This review paper examines the effectiveness and prospects of monoclonal antibody therapies in oncology. The development of these therapies has revolutionized targeted cancer treatment due to the specificity and molecular precision of the antibodies. The article discusses their mechanisms of action, clinical applications and new trends, with a special focus on the role of personalized therapies.

Materials and Methods: The review covers key studies on monoclonal antibody therapies, analyzing their mechanisms of action, such as antibody-dependent cellular cytotoxicity (ADCC) and complement-dependent cytotoxicity (CDC). The paper also considers the integration of these therapies with other treatments, such as chemotherapy and immunotherapy.

Main results: Monoclonal antibodies have shown high efficacy in the treatment of various cancers, including breast, ovarian and lung cancers, by targeting specific antigens such as HER2 and PD-1/PD-L1. Advances in bispecific antibodies, drug-antibody conjugates and personalized biomarkers are further improving treatment outcomes. Challenges such as resistance and side effects are being addressed through genetic engineering and innovative drug delivery systems.

Conclusions: Monoclonal antibody therapies have revolutionized cancer treatment, offering precise and personalized therapeutic approaches. Further research into combination therapies and new antibody technologies promises to overcome current limitations and expand their therapeutic potential.

Keywords: Monoclonal bodies, combination therapy, anti-cancer therapy, HER2, BRCA

Introduction

The development of oncology medicine in recent decades has brought innovative treatments that are changing the approach to cancer therapy. Among them, a special place is occupied by monoclonal antibodies, which, thanks to their specificity and ability to precisely affect molecular targets, are becoming a tool of crucial importance in targeted therapy.

Their production technology, pioneered by the discovery of hybridomics in the 1970s, has enabled the use of these compounds in the treatment of various types of cancer. Initial successes, such as the development of the OKT3 antibody, paved the way for widespread use in medicine. This thesis discusses the mechanisms of action of monoclonal antibodies, their clinical application, as well as new research directions and prospects for their development, focusing on the efficacy and possibilities for personalizing therapy.

The Development of Monoclonal Antibody Therapy in Oncology

Monoclonal antibodies continue to be a beacon of hope for targeted cancer therapies, offering unmatched precision and effectiveness. What sets these therapies apart is their unique combination of features: incredible specificity, long serum half-life, high affinity for their targets, and their ability to leverage the immune system's natural responses. These proteins have become a cornerstone of modern medicine because they are stable, highly adaptable, and uniquely structured to bind both antigens and immune receptors. Additionally, their high susceptibility to protein genetic engineering opens the door to even greater advancements [1].

The story of monoclonal antibody therapies began with a major breakthrough in the 1970s, when G. Köhler and C. Milstein developed hybridoma technology to produce monoclonal antibodies in mice. Their groundbreaking work earned them the Nobel Prize in Physiology or Medicine in 1984. A few years later, in 1979, Kung and colleagues introduced the world to OKT3 (Ortho Kung T3), the first monoclonal antibody designed to recognize the CD3 surface antigen on human T cells. This antibody, later marketed as muromonab, became the first mouse monoclonal antibody approved for human therapy, receiving FDA approval in 1986 [2].

All monoclonal antibodies share a common suffix, “-mab,” in their international names, along with an indication of their specific type. One exciting area of development is the creation of bispecific monoclonal antibodies. These innovative antibodies are designed with two arms: one targets tumor antigens, while the other is aimed at enhancing the immune system’s ability to destroy cancer cells. To achieve this dual specificity, bispecific antibodies are often engineered by chemically coupling two different antibodies, creating a molecule with four binding sites (Fab fragments). Each pair of Fab fragments binds to a distinct molecule. Alternatively, these antibodies can be produced by fusing two hybridomas that generate antibodies with different specificities. The ultimate goal of these therapies is to more effectively engage the host immune system in fighting cancer cells [3].

The use of monoclonal antibodies in oncology is particularly effective because of their ability to selectively target well-defined molecules on tumor cells. This interaction blocks pathways that fuel tumor growth and oncogenesis. Mechanistically, monoclonal antibodies destroy tumor cells through immune responses like antibody-dependent cell-mediated cytotoxicity (ADCC) or complement-dependent cytotoxicity (CDC). They can also enhance tumor cell death by triggering apoptosis, modulating ligand-receptor interactions, or inhibiting growth factor signaling pathways. In some cases, they even interfere with angiogenesis, preventing the formation of new blood vessels that feed tumor growth.

To further boost their therapeutic efficacy, monoclonal antibodies can be conjugated with other agents, such as radioisotopes, toxins, cytostatic drugs, or cytokines. These combinations allow for a more potent and targeted attack on cancer cells, expanding the potential of immunotherapy [4].

The importance of monoclonal antibodies in personalizing cancer treatment.

A Shitara K study published in 2020 focusing on the efficacy and safety of pembrolizumab chemotherapy in patients with advanced gastric cancer in first-line treatment indicates that the drug is more effective than chemotherapy in treating advanced G/GEJ adenocarcinoma, with fewer side effects. The greatest benefit of pembrolizumab has been reported in patients with:

- PD-L1 CPS of 10 or higher
- MSI-H tumors

In patients with PD-L1 CPS of 1 or higher, pembrolizumab was no worse than chemotherapy. In contrast, when combined with chemotherapy, pembrolizumab did not provide a clinically significant benefit over chemotherapy treatment alone [5].

A study by Emese Zsiros on the use of monoclonal antibodies in the treatment of recurrent ovarian cancer has shown that the combination of pembrolizumab with bevacizumab and oral metronomic cyclophosphamide is safe and well tolerated in patients with recurrent ovarian cancer. This finding suggests that this drug combination may be a promising treatment option for this group of patients. The researchers plan to further analyze the biosamples to better understand the synergistic mechanisms between immunotherapy (ICI), anti-angiogenic therapy and regulation of T-cell depletion. A deeper understanding of these mechanisms may enable the application of immunotherapy to more patients in the future. The study included 30 patients with platinum-resistant disease, and the most common histologic subtype was high-grade serous carcinoma. The average number of prior lines of chemotherapy was 3.4 for all patients, and 3.8 for patients with platinum-resistant disease. 14 patients had prior exposure to bevacizumab, and five patients were using oral cyclophosphamide. No significant differences were observed between the platinum-resistant and platinum-sensitive disease populations with respect to age, race, performance status, PD-L1 or BRCA status. None of the participants had prior exposure to anti-PD-1 or other ICI antibodies [6].

Rita Nanda's phase 3 study of the inclusion of monoclonal therapy in women with early-stage breast cancer showed that the addition of pembrolizumab to standard neoadjuvant chemotherapy doubled the estimated pathologic complete response (pCR) rates for both HR-positive/ERBB2-negative and triple-negative breast cancer. This suggests that checkpoint blockade in women with early-stage high-risk, ERBB2-negative breast cancer may be an effective therapeutic strategy. Pembrolizumab was the first of 10 drugs to complete the study in the HR-positive/ERBB2-negative group. All patients in the study showed progressive biomarker emissions. The final estimated pCR rates, assessed in March 2017, were:

- 44% for pembrolizumab versus 17% for the control group in the ERBB2-negative cohort,
- 30% for pembrolizumab versus 13% for the control group in the HR-positive/ERBB2-negative cohort,
- 60% for pembrolizumab versus 22% for the control group in the triple-negative cohort.

Pembrolizumab also affected the distribution of RCBs (responses according to clinical criteria) toward a lower disease burden in all study groups. The most common adverse effects were immune-related endocrinopathies, especially thyroid abnormalities (13.0%) and adrenal insufficiency. Achieving a pCR seemed predictive of long-term outcomes, as patients with a pCR after pembrolizumab and chemotherapy had high rates of event-free survival. These promising results suggest that pembrolizumab may be a valuable addition to standard neoadjuvant chemotherapy for the treatment of early-stage, high-risk, ERBB2-negative breast cancer [7].

Mechanism of action of monoclonal antibodies in the fight against cancer.

Current immunotherapy strategies typically aim to activate anti-tumor T-cell immunity, although recent years have also focused on macrophage-dependent innate anti-tumor immunity. Neutrophils exhibit marked antitumor activity and can induce phagocytosis, trogocytosis, and direct cytotoxic elimination of tumor cells. In addition, therapeutic tumor-targeted monoclonal antibodies induce anti-tumor immune responses mediated by all cells expressing the innate Fc receptor, including neutrophils. Indeed, neutrophil depletion significantly reduced the efficacy of monoclonal antibody treatment and increased tumor progression in various preclinical studies. Moreover, infusion of neutrophils in mouse models reduced tumor progression. However, evidence of neutrophils' anti-tumor activity is fragmentary and comes mainly from in vitro tests or mouse models, and reports of neutrophils' anti-tumor activity in humans are still being developed [8].

1. Mechanisms of action of monoclonal antibodies:

• ADCC (antibody-dependent cellular cytotoxicity): Monoclonal antibodies bind to antigens on the surface of cancer cells, activating neutrophils that release toxic substances such as reactive oxygen species and proteolytic enzymes to destroy cancer cells.

• ADCP (antibody-dependent cellular phagocytosis): These antibodies can opsonize tumor cells, facilitating their phagocytosis by neutrophils.

• Trogocytosis: Neutrophils also play a role in attacking fragments of the tumor cell membrane, leading to tumor damage.

2. Modification of the tumor microenvironment:

• Antibodies that block inhibitory signals, such as CD47 (the "don't eat me" signal), enable neutrophils to phagocytose tumor cells more efficiently.

• Monoclonal antibodies can be combined with cytokines such as IFN-α, which enhance the ability of neutrophils to destroy cancer cells.

3. Interaction with Fc receptors:

• IgG- and IgA-type antibodies interact differently with receptors on neutrophils, which affects the efficiency of activation of the anti-tumor response. For example, IgA antibodies can more efficiently activate neutrophils compared to IgG.

4. Potential constraints and challenges:

• The tumor microenvironment, including the presence of immunosuppressive cytokines like TGF-β, can limit the effectiveness of neutrophils. Blockade of these factors increases the antitumor activity of neutrophils.

5. Monoclonal antibodies are thus an effective tool in cancer therapy, acting through a variety of mechanisms involving the immune system. [8]

Antigenic specificity and mode of binding to cancer cells using HLX10 as an example.

Monoclonal antibodies work by activating the immune system, as an example (mAbs) that block the PD-1 receptor, HLX10. This is a fully humanized IgG4-class antibody that binds to the PD-1 receptor and blocks interaction with its ligands (PD-L1 and PD-L2). This blockade reverses tumor immunosuppression, enabling T-cell reactivation and anti-tumor activity. It works by blocking the PD-1/PD-L1 interaction, effectively reversing immunosuppression and enhancing the anti-tumor response. Its unique binding mode and high antigenic specificity make it a potential candidate for more effective cancer therapies in combination with other agents [9].

1. Blocking PD-1/PD-L1 and PD-1/PD-L2 interactions:

• HLX10 inhibits the binding of PD-1 to its ligands PD-L1 and PD-L2, which reverses T-cell depletion.

• The mechanism of blockade is based on spatial overlap between the PD-1 epitopes bound by HLX10 and the ligands, preventing their simultaneous binding.

2. Comparison with other antibodies:

• HLX10 binds to PD-1 with higher or similar efficacy compared to clinically approved antibodies (Pembrolizumab, Nivolumab).

• HLX10 shows better ability to induce T-cell proliferation and secretion of cytokines (e.g., IL-2, IFN-γ).

3. Effects on the tumor microenvironment:

• HLX10 activates T cells, leading to an increased anti-tumor response in in vivo models.

• When combined with an anti-VEGF antibody (e.g., Bevacizumab), it enhances the effects of therapy by normalizing blood vessels and improving T-lymphocyte infiltration into the tumor [9].

Mechanisms of antibody-dependent cytotoxicity.

Antibody-dependent cellular cytotoxicity (ADCC), is an immune mechanism in which target cells, such as cancer cells, are coated with antibodies. This in turn recruits effector cells of the immune system to kill the target cell by non-phagocytic mechanisms. Antibodies act as bridges, binding to antigens on the surface of the target cell via their Fab fragments and linking effector cells via their Fc fragments.

NK cells are the main effector type that mediate ADCC, but other myeloid types, such as monocytes, macrophages, neutrophils, eosinophils and dendritic cells, are also capable of doing so.

The effector cells induce target cell death by releasing cytotoxic granules, Fas signaling and initiating reactive oxygen species. The clinical efficacy of most targeted monoclonal antibodies depends mainly on NK cells. Preclinical studies and clinical trials have confirmed that ADCC is an important factor influencing the efficacy of monoclonal antibodies in vivo. FcγR polymorphisms, which affect antibody binding strength, correlate with clinical outcomes in patients receiving monoclonal antibody therapy. Patients with high-affinity FcγRs that mediate stronger ADCC have better clinical outcomes. The ADCC function of antibodies can be enhanced by:

1. Altering the Fc portion of the monoclonal antibody to increase its binding affinity to the activating FcγRIIIA through targeted mutagenesis,
2. Fc domain glycosylation change,
3. Fc domain fucosylation removal.

Resistance to monoclonal antibody therapy often includes impaired responses of cytotoxic effector cells of the immune system. New research is focusing on analyzing the mechanisms of action of monoclonal antibodies to identify new approaches to enhance clinical efficacy, including engineering strategies that enhance ADCC activity [10, 11].

A review of the major monoclonal antibodies used in cancer treatment.

As we wrote earlier, monoclonal antibodies are widely used to treat various types of cancer. They work by blocking receptors on cancer cells, binding substances that are ligands for these receptors, or binding proteins on the surface of cancer cells, which triggers immune system mechanisms that lead to the destruction of cancer cells.

Examples of the use of monoclonal antibodies in cancer treatment:

• Alemtuzumab is used in chronic lymphocytic leukemia (CLL) and prolymphocytic leukemia.

• Bevacizumab is used to treat advanced colorectal cancer, breast cancer, ovarian cancer, renal cell carcinoma and non-small cell lung cancer.

• Blinatumomab is used in relapsed and refractory B-cell acute lymphoblastic leukemia (ALL).

• Brentuximab vedotin is used in classical Hodgkin's lymphoma and non-Hodgkin's lymphoma (NHL) CD30+ T-cell lymphomas.

• Cetuximab and panitumumab are used to treat colorectal cancer with metastatic and normal RAS genes (cetuximab and panitumumab) and head and neck cancers (cetuximab).

• Daratumumab is used to treat plasmocytic myeloma (MM).

• Elotuzumab is used in refractory and relapsed MM.

• Gemtuzumab ozogamycin is used in CD33+ acute myeloid leukemia (AML).

• Ozogamycin inotuzumab is used in relapsed and refractory B-cell ALL.

• Pertuzumab is used to treat advanced breast cancer in combination trastuzumab and docetaxel.

• Vedotin polatuzumab is used in relapsed and refractory diffuse large granular B-cell lymphoma (DLBCL) in patients who are ineligible for hemopoietic cell transplantation, in combination with bendamustine and rituximab.

• Rituximab, obinutuzumab, ofatumumab are used to treat non-Hodgkin's lymphoma (NHL) B-cell lymphoma, CLL, non-classical Hodgkin's lymphoma and ALL B-cell lymphoma.

• Sacituzumab gowitekan is used to treat breast cancer.

• Trastuzumab is used to treat breast cancer with known HER2 receptor overexpression or HER2 gene amplification and HER2-positive gastric adenocarcinoma.

• Trastuzumab derukstekan/Trastuzumab emtansine is used to treat breast cancer with overexpression of HER2 protein or amplification of the HER2 gene - second and subsequent line treatment in advanced stages.

Unfortunately, the use of monoclonal antibodies is associated with some side effects. These include:

• allergic reactions,
• cytokine release syndrome,
• myelosuppression,
• peripheral neuropathy,
• liver dysfunction,
• heart failure,
• thrombocytopenia,
• anemia.

In some cases, other side effects may also occur, so it is important to discuss all potential risks and benefits with your doctor before starting treatment.

Contraindications to the use of monoclonal antibodies are usually related to individual patient characteristics, such as:

• hypersensitivity to the drug,
• pregnancy,
• breastfeeding,
• other diseases.

Advantages and limitations of monoclonal antibody therapy.

Monoclonal antibodies have emerged over the past decade as a preferred treatment or adjunct to chemotherapy because of their high specificity of reactivity and affinity for recognizing the target antigen, minimal side effects, and favorable pharmacokinetic and pharmacodynamic properties. The high specificity of mAb to its targets provides an attractive and effective option for developing therapeutic and molecular drug targets [13].

Early clinical trials with mAb-based therapies were very primitive and disappointing. The first mAb evaluated clinically against cancer was mouse mAb. Although there were fascinating indications that mAb therapy could be effective, problems associated with administering mouse mAb to humans limited their utility and clinical applications. Challenges included an increased immune response against the therapeutic mAb, very rapid clearance of the mAb from the body, and the suboptimal ability of the mouse mAb to interact with the human immune system in a way that leads to immune destruction [14, 15].

This problem was solved by cleaving the immunogenic Fc region of the immunoglobulin molecule or by recombinant methodologies. Recombinant methods have mainly focused on producing chimeric antibodies containing a mouse antibody recognition unit and a human Fc region or using a human IgG molecule with inserted mouse complementarity-determining residues to preserve antibody specificity.
Monoclonal antibodies have been very successful in diagnostics. mAb-based diagnostic reagents have the potential to identify abnormal target cells, infectious agents or disease response elements [13, 14, 15].

New perspectives and the future of monoclonal antibody therapy.
New perspectives and the future of monoclonal antibody therapy are focused on several key areas:
Combination with immunotherapy:

• Promising results suggest that the combination of bevacizumab, an angiogenesis inhibitor, with atezolizumab, an immune checkpoint inhibitor, has benefits in terms of disease progression-free survival (PFS) and overall survival (OS) in patients with non-small cell lung cancer (NSCLC) [16],

• These benefits were seen regardless of PD-L1 expression status and were even greater in patients with high expression of the effector T-cell gene, indicating active adaptive immune responses [16],

• A number of phase III trials are evaluating bevacizumab in combination with immune checkpoint inhibitors in ovarian cancer [16].

Combination with PARP inhibitors:

• The combination of bevacizumab with PARP inhibitors aims to increase the sensitivity of tumor cells to the cytotoxic effects of PARP inhibitors, while mitigating the hypoxic effects of VEGF inhibition [16],

• PARP inhibitors are approved for the treatment of cancers with BRCA mutations and BRCA-like tumors, such as ovarian cancer and breast cancer, and are being investigated in many other oncology indications [16],

• The PAOLA-1 trial showed a PFS benefit with the combination of bevacizumab and the PARP inhibitor olaparib for maintenance treatment of ovarian cancer [16].

Drug-antibody conjugates (ADCs):

• ADCs combine traditional chemotherapeutic drugs with highly selective, targeted monoclonal antibodies [17].

• Trastuzumab, the first monoclonal antibody to target HER2, has become a mainstay of anti-HER2 therapy [17].

• Trastuzumab-drug conjugates (T-DCs) have shown promising results in the treatment of HER2-low breast cancer and other solid tumors [17].

• Trastuzumab deruxtecan (T-Dxd) has shown significant safety and efficacy benefits over other approved treatments for HER2-negative and HER2-low breast cancer, HER2-positive gastric/gastroesophageal cancer, and HER2-mutated lung cancer [17].

Future directions for ADC research include:

• Combination treatment strategies: to enhance T-DC activity and overcome future resistance mechanisms [17],

• Testing combinations with other drugs, such as immunotherapy, chemotherapy and targeted therapy [17],

• Developing new dual-load ADCs to combat breast tumor heterogeneity and drug resistance [17].

The future of monoclonal antibody therapy looks promising. New combination treatment strategies, the development of ADCs and a better understanding of the mechanisms of action of these drugs are opening up new possibilities for patients with a variety of cancers.

Combination therapies.

Combination therapies are the combination of a monoclonal antibody with another active substance translations of such a combination are:

Combining cetuximab with immune checkpoint inhibitors (ICBs): Cetuximab, a monoclonal antibody targeting EGFR, has efficacy partially attributed to ADCC, which can combine innate and adaptive anti-tumor immune responses. ADCC-mediated destruction of tumor cells releases tumor cell-specific proteins that, when presented by antigen-presenting cells to cytotoxic T cells, lead to a more effective anti-tumor response. Patients with head and neck squamous cell carcinoma (HNSCC) with a sustained response to cetuximab have a sustained specific anti-tumor immune response. With the advent of immune checkpoint inhibitors that can further enhance such immune responses, it has been hypothesized that ICB may act synergistically with cetuximab. There is growing evidence to support combining anti-PD-1/PD-L1 monoclonal antibodies with cetuximab in patients with HNSCC. In addition, combinations of pembrolizumab or avelumab with cetuximab are currently in clinical trials. Combination of trastuzumab with ICB: Similarly, the use of ICBs in breast cancer to augment anti-HER2 monoclonal antibody therapy is a promising strategy. Preclinical evidence suggests that resistance to trastuzumab monotherapy can be overcome by combining with ICBs. Based on these findings, several clinical trials have been conducted to investigate the association between ICBs and monoclonal antibodies targeting HER2. Preliminary results from the phase I/II PANACEA trial, which tested pembrolizumab in combination with trastuzumab for the treatment of breast cancer patients with HER2 overexpression, indicated synergy in a subgroup of PD-L1+ patients. Combination of CTLA-4 and PD-1 inhibitors: monoclonal antibodies targeting CTLA-4 and PD-1 act on different immune checkpoints, meaning that their combination may lead to a synergistic enhancement of T-cell responses. In preclinical studies in mice, the combination of monoclonal antibodies directed against CTLA-4 and PD-1 performed significantly better than each antibody used alone. Similar results were obtained in clinical trials involving patients with metastatic melanoma, in which combination therapy with ipilimumab and nivolumab proved more effective than monotherapy with either drug alone. The FDA has approved the combination of ipilimumab and nivolumab for the treatment of melanoma. Clinical trials evaluating ipilimumab and nivolumab in other types of cancer are ongoing.

Combining PD-1 inhibitors with other checkpoint inhibitors or agonist antibodies: Anti-PD-1 monoclonal antibodies are most commonly used in combination strategies due to their more favorable toxicity profile compared to anti-CTLA-4 monoclonal antibodies. Checkpoint inhibitors LAG3 and TIM3 are often co-expressed with PD-1 on depleted T cells. ICB LAG3 in combination with anti-PD-1 is being tested in clinical trials in glioblastoma multiforme (NCT02658981) and other cancers (NCT02460224). Similar clinical trials are underway for the combination of anti-TIM3 and anti-PD-1 antibodies in liver cancer (NCT03680508) and several other solid tumors (NCT03744468).

Another promising combination strategy involves combining ICBs with agonist antibodies that activate stimulatory receptors. 4-1BB is a costimulatory receptor found on T cells and NK cells, and clinical trials evaluating 4-1BB agonist antibodies in combination with anti-PD-1 monoclonal antibody therapy are ongoing (NCT02253992 and NCT02179918). An agonist antibody to the glucocorticoid-induced TNF-related protein receptor (GITR), which promotes T-cell activation, has also proven effective in combination with nivolumab. Additional monoclonal antibody combinations that include agonist antibodies to OX40 expressed only on activated T cells are the subject of several clinical trials (NCT01714739 and NCT01750580) [10, 11].

Personalizing treatment based on biomarkers.

The NeoPACT trial evaluated the efficacy of an anthracycline-free chemoimmunotherapy regimen based on carboplatin and docetaxel in combination with pembrolizumab in patients with triple-negative breast cancer (TNBC). The study found that several biomarkers, including sTILs (stromal tumor infiltrating lymphocytes), PD-L1, DDIR signature (DNA damage immune response) and TIM signature (tumor immune microenvironment) were independently predictive of a complete pathological response (pCR). High pCR rates (exceeding 70%) were noted in subgroups with positive immune biomarkers. The I-SPY2 trial also examined the effect of adding pembrolizumab to standard neoadjuvant chemotherapy in women with early-stage breast cancer at high risk of recurrence. Pembrolizumab was administered in combination with paclitaxel followed by AC (doxorubicin and cyclophosphamide) and was found to more than double the estimated pCR rates for both hormone receptor-positive/ERBB2-negative breast cancer and triple-negative breast cancer. Identifying patients most likely to benefit from such treatment is crucial. Biomarkers such as sTILs, PD-L1, DDIR and TIM can be used to personalize therapy, ensuring that patients receive the most appropriate treatment for their specific situation [7, 18].

Summary and future research directions.

Monoclonal antibodies represent one of the most advanced tools in cancer treatment, offering an effective, precise and individualized therapeutic approach. Their unique properties, such as their high specificity and ability to trigger an immune system response, make them the cornerstone of modern oncology. Despite significant challenges, such as treatment resistance and side effects, research into new generations of antibodies and their combinations with other therapies offers hope for further enhancing their efficacy.

The development of drug-antibody conjugate technologies and the identification of predictive biomarkers are just some of the promising directions that could improve patients' quality of life and the efficacy of therapies. Thus, the future of monoclonal antibody therapy looks extremely promising, opening up new possibilities in the fight against cancer.

Disclosure

Author's contribution

Conceptualization: Bartosz Omasta; Methodology: Katarzyna Kamińska-Omasta Software: Kuba Borys Romańczuk; Check: Szymon Przemysław Stolarczyk and Daria Rybak; Formal analysis: Kinga Furtak and Olga Krupa; Investigation: Paulina Dorota Pietrukaniec and Magdalena Agata Czerska; Resources: Zofia Martyna Wójcik and Bartosz Omasta; Data curation: Daria Rybak; Writing - rough preparation: Olga Krupa; Writing - review and editing: Bartosz Omasta and Kinga Furtak; Visualization: Zofia Martyna Wójcik and Szymon Przemysław Stolarczyk; Supervision: Katarzyna Kamińska-Omasta; Project administration: Paulina Dorota Pietrukaniec and Kuba Borys Romańczuk; Receiving funding - no specific funding.

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