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Molecular Approaches in Anticancer Drug Development: A focused on Targeted Therapies

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Abstract
This paper highlights the need for study into the development of effective customized targeted therapies with the potential to overcome pharmaceutical resistance while also decreasing toxicity and unwanted effects. Extensive structural studies, genetic and biochemical research, and the discovery of new druggable targets are essential components of the drug development framework necessary to achieve this aim. To further molecular focused therapy, new medications with different chemical entities or action mechanisms are required. Additional methods for cancer-specific control beyond director allosteric modulation include posttranslational modification and targeted protein degradation utilizing proteolysis-targeting chimera (PROTAC).
Keywords: Cancer, Drug, Targeted

Introduction

One of the main killers on a global scale is cancer. In 2020, there will be around 19.3 million new instances of cancer and over 10.0 million deaths attributed to the disease, according to the Global Cancer Observatory (GLOBOCAN). The projected 47% increase to 28.4 million instances of cancer-related burden (including incidence and mortality) in 2040 compared to 2020 is mostly attributable to aging, socioeconomic development, overweight status, and smoking. Consequently, creating effective cancer treatment regimens is crucial [1]. Various treatment techniques have been used in clinical settings to address cancer, including surgery, radiation therapy, systemic anticancer therapy, and others. The respectability of the tumor, the patient's biology, the patient's functional performance, the disease stage, and any comorbidities determine the sequence of administration. Cytotoxic chemotherapy, hormone pharmaceuticals, targeted treatment, antitumor immunotherapy, and other anticancer drugs are all part of systemic anticancer therapy, which tries to cure, palliate, ease symptoms, and enhance the quality of life for cancer patients. Cytotoxic chemotherapy is widely used in adjuvant, neoadjuvant, and palliative treatment [2]. It decreases the survival of rapidly proliferating cells by establishing covalent connections with DNA, RNA, and proteins, interrupting mitosis, and altering DNA and RNA synthesis. A more focused form of cancer treatment has recently emerged, which increases cancer cell selectivity in contrast to chemotherapy's less desirable side effects and damage caused by its nonselective impact on normally growing cells.

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Some examples of targeted therapy include hormonal agents like aromatase inhibitors and estrogen receptor (ER) antagonists, which have been used to treat hormone receptor (HR)-dependent breast cancer and male and female reproductive cancers, immune checkpoint inhibitors like antibodies against programmed cell death protein 1 (PD-1), programmed death-ligand 1 (PD-L1), or cytotoxic T-lymphocyte associated protein 4 (CTLA-4), which activate host antitumor immunity directly or indirectly, and targeted cytotoxic therapy that interferes with a specific cellular target, such as meth 10. In spite of the targeted treatments' anticancer efficacy, these medications are only suitable for individuals with identifiable driver mutations or abnormalities [3].

Objectives

1. To explore molecular mechanisms and pathways that can be exploited for targeted cancer therapies.
2. To evaluate the efficacy of novel targeted therapies in preclinical and clinical settings.
3. To investigate strategies for overcoming resistance mechanisms in targeted therapy.
4. To assess the potential of combination therapies integrating targeted approaches with conventional treatments.

Scope

This study focuses on:

- Identifying molecular targets, such as specific proteins, genes, and signaling pathways involved in tumor progression.
- Analyzing cutting-edge technologies like monoclonal antibodies, small-molecule inhibitors, and RNA-based therapeutics.
- Evaluating the application of biomarkers for patient stratification and treatment personalization.
- Highlighting challenges in drug delivery, resistance development, and translating research from bench to bedside.

Significance of The Study

A revolutionary change in cancer therapy, targeted therapies have the ability to be more precise while reducing adverse effects compared to traditional methods. A major advance in the field of customized medicine, this work paves the way for the creation of unique treatment regimens for each patient by analyzing their tumor patterns. This research may help overcome the present constraints of targeted treatment, which might lead to better results from the next generation of anticancer medications. Furthermore, the research highlights the need of integrating genetic diagnostics with treatment techniques to improve precision medicine in cancer.

Anticancer Nanoformulations

Low water solubility, poor bioavailability, and weak tumor targeting capabilities are common challenges in efficiently delivering small molecule anticancer medicines to solid tumors. Nanotechnology-driven formulations are one potential approach to the safe and effective treatment of cancer with small molecule drugs. Nanoparticles laden with drugs may reach tumor sites in enough concentration via active or passive absorption routes that start in the systemic circulation [22]. Figure 2.4 shows the enhanced permeability and retention (EPR) effect, which is a common passive targeting mechanism in tumor microenvironments including leaky vasculatures associated to angiogenesis and faulty lymphatics. In contrast, nanoparticles that adapt to the tumor's environment—including its acidity, enzyme overexpression, oxygen levels, reactive oxygen species, redox potentials, levels, and other characteristics—are the focus of active targeting. Folic acid molecules, aptamers, antibodies, and cell penetrating peptides are examples of targeting moieties that are often linked to nanoparticle architectures in order to train the response to such stimuli. Active targeting can occasionally be accomplished by employing environmental cues like heat, light, voltage, ultrasound, magnetic fields, etc. There are a few main kinds of nanoparticles that target tumors, such as those made of polymers, lipids, or other substances [23]. Figure 2.5 shows a variety of nanocarriers made of polymers. Polymeric micelles, which function as amphiphilic nanocarrier systems, contain hydrophobic drugs within a core that is formed by the hydrophobic portion of the polymer. While low-molecular-weight surfactants can also be used to create micelles, polymeric micelles offer enhanced drug solubilization and loading capabilities, increased stability, and controlled drug release power. Block copolymers with

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distinct hydrophilic and hydrophobic domains are the polymers used. Alternately, the hydrophilic portion of the polymer might form a core and, in the event that it is required, contain the hydrophilic medication. Amphiphilic diblock copolymer monomethoxy PEG-block-poly(D,L-lactide) is the building block of Genexol-PM, a paclitaxel formulation that is Cremophor EL free and polymeric in micelle form. Amphotericin B and the biological surfactant sodium deoxycholate are included in the lyophilized formulation known as fungizone. After being reconstituted, it creates micelles that are based on surfactants[24].

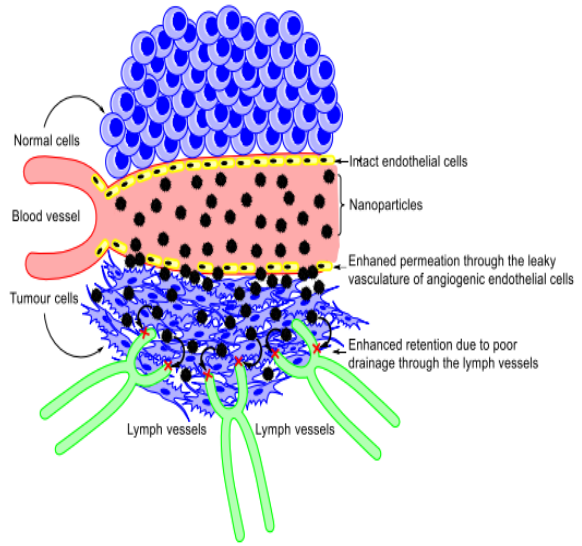

Figure 2.4 The tumor microenvironment presents an opportunity for medicine delivery via enhanced permeability and retention.

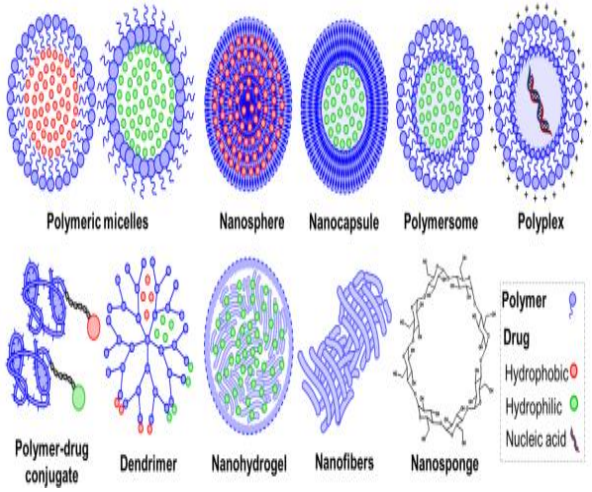

Figure 2.5 Different kinds of nanocarriers made of polymers

The polymeric matrix is uniformly dispersed throughout the homogenous structure of polymeric nanospheres. Medicines that are hydrophilic or hydrophobic can either be physically adsorbed onto the surfaces of the nanospheres that are produced or entrapped in this matrix. In the polymeric cubic phase, bicontinuous nanospheres are being studied as an alternative to lipid cubosomes. One common platform technology for topical medication administration is TyroSpheres, which are manufactured from tyrosine generated triblock copolymers [25].

Polymeric nanoparticles with structural similarities to core-shell complexes are referred to as nanocapsules. Polymeric coatings encase drugs that are hydrophilic or hydrophobic and contain oil or water in the middle. Therefore, the vesicular structures are nanocapsules. Polymeric nanocapsules have been used to deliver active ingredients in cosmetics topically, according to reports [26]. Polymersomes are vesicles made by self-assembling amphiphilic block copolymers. Enclosing an aqueous cavity, these vesicles may capture hydrophilic drugs. They are frequently prepared with methods like phase inversion and film rehydration. Vesicles made of polymer are more durable, resistant to damage, more flexible than liposomes. Nonviral vectors for gene or medication delivery may be found in polyplexes, which are complexes of cationic polymer with DNA or RNA polyanions. Low immunological reaction, unlimited gene size, and simplicity of manufacturing are only a few of the benefits they exhibit compared to viral vectors [27].

Targeted Drug Delivery

Conventional chemotherapeutic medication distribution impacts normal and tumoral cells equally. Conventional chemotherapeutics face a number of biological hurdles on their way to the target site of action, which may lead to drug inactivation or the development of undesirable side effects in various organs and tissues. A substantial dosage of the medicinal substance is necessary to attain the targeted therapeutic concentration in a particular organ or tissue [39]. Adverse effects may occur in large quantities when therapeutic drugs are inadvertently exposed to healthy tissue. The pharmacological agent's tissue selectivity is a major issue. To prevent the

therapeutic drug from harming healthy tissue, it must be delivered selectively to the affected area. One emerging technique to increase treatment effectiveness while limiting negative effects is tumor targeted administration of conventional medicines. According to Torchilin (2000), drugs may be more effectively delivered to specific areas of the body, limiting their buildup in healthy tissues. It doesn't rely on where or how it's administered[40]. A targeted delivery system including cancer chemotherapeutics has the potential to increase the effectiveness of the drugs by a number of means, one of which being the following:

- Improved drug delivery due to increased solubility
- Increased drug accumulation in tumor tissue
- Stable and controlled drug release
- Extended circulation half-life (t1/2)
- Protection of chemotherapeutic agents from degradation
- Better cellular uptake
- Less toxicity

According to Ehrlich and Himmelweit (Ehrlich and Himmelweit, 1956), in 1906, Paul Ehrlich envisioned drug targeting as a "magic bullet"—a two-part entity that could identify and engage with a target before delivering curative activity to that target. In order to be successful, a targeted medication delivery system must be able to evade, retain, target, and release. Depending on the route chosen for the drug delivery system, multiple delivery methods are required to target therapeutic agents to specific areas. There are a few of ways to target drugs: passively and actively [41].

Passive Targeting

The drug carrier is carried into the tumor interstitium by the leaky vasculature in passive drug targeting and accumulated there (Fig. 2.8). Architectural anomalies and a high percentage of vascular density in tumor blood arteries are caused by imperfections like an increased number of proliferating endothelial cells, irregular branching, and blind loops with twisted shapes. In addition, solid tumors often have damaged lymphatic arteries, which means that macromolecules aren't being cleared out of the tumor interstitium as efficiently and thus drainage is ineffective. According to Maeda et al. (2001)[42], this passive occurrence is often referred to as the "Enhanced Permeability and Retention" (EPR) effect. Nowadays, when it comes to cancer treatment targeting strategies, the EPR effect is regarded as the best. Nanocarriers need to stay in the bloodstream for long enough to deliver enough medicine to the tumor for EPR effect passive cancer targeting to work. The limitations of passive drug targeting are real. The level of passive drug targeting is greatly affected by tumor vascularization, angiogenesis, interstitial fluid pressure, and other factors. The level of vascular permeability inside a tumor might vary, according to Peer et al. (2007). Massey and Schnitzer (2010) [43] state that EPR effects could vary based on whether a tumor is developing orthotopically or subcutaneously. Furthermore, the EPR effect is absent in a large number of hypovascular tumor tissues. When extravasation happens in solid tumors, the drug's chances of being internalized and distributed evenly throughout the tumor decrease. This is because the drug or nanocarrier may have trouble passing through the interstitial spaces due to the increased pressure in the fluid (Heldin et al., 2004). In order to provide anticancer medications to the correct individuals at the appropriate time via passive drug targeting, extensive knowledge on the tumor kind and the tumor's vascularization structure is required. To improve drug localization in tumors and tumor cell uptake, third-generation nanocarriers based on customized targeting were developed [44].

Active drug targeting

The nanocarrier surface with a peripherally attached targeting moiety or ligand is depicted in Figure 2.8. Active targeting, or selective engagement with certain receptors that are overexpressed in the target area, is made possible by this. According to Shi et al. (2011) and Cheng et al. (2012), the ligand is chosen to bind to receptors or surface molecules that are overexpressed in sick organs, tissues, cells, or subcellular domains in comparison to normal cells [45].

Designing actively-targeted nanoparticles drug carriers is a challenging task due to the numerous factors that affect their effectiveness, such as the NPs' architecture and size (Jiang et al., 2008), ligands' types, conjugation, and density (Gu et al.,

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2008), and carriers' administration method (Bertrand and Leroux, 2012). The effectiveness of the ligand-receptor interaction is influenced by a number of cellular factors. These include, among other things, the rate of internalization of the surface receptor after ligand binding, the ratio of target cell receptor expression to non-target cell expression, and the accessibility of the receptor on the surface of the target tissue. According to research by Wen et al. (1995) and Capone et al. (1984), tumor targeting receptors' surface expression might change over time, and their distribution inside tumors is not always uniform.

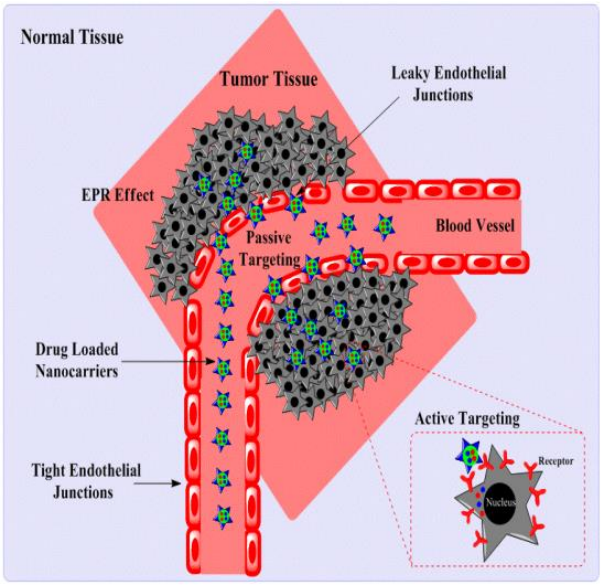

Fig. 2.8: An illustration of how the ligand-receptor interaction and the EPR effect localize nanoparticles in solid tumors

Receptor Tyrosine Kinase Inhibitors

Inhibitors targeting the EGFR family

The human EGFR family consists of ErbB1/EGFR, ErbB2/HER2, ErbB3/HER3, and ErbB4/HER4. These proteins are members of the ErbB family. Except for HER2, which is unable to bind ligands, the EGFR family of proteins is activated when ligands such as EGF, epiregulin, transforming growth factor-α (TGF-α), and neuregulins bind to them. About 25% of women with breast cancer have either an overexpression or an amplified form of the HER2 gene. Cancers of solid tissues may develop when an EGFR kinase-activating mutation, such as an exon 19 microdeletion, a cytoplasmic tyrosine kinase domain mutation at the L858R position, or a reduced extracellular domain (EGFRvIII), is present [93]. The ligand-independent aberrant EGFR activation is caused by these code mutations. In East Asian females who do not smoke and who have non-small cell lung cancer (NSCLC), it is common to find exon 19 microdeletions and L858R point mutations. In most cases, glioblastoma will have EGFRvIII. Additional EGFR mutations that confer resistance to EGFR TKIs have been discovered in non-small cell lung cancer (NSCLC). These mutations include E884K, D761Y, T854A, and exon 20 insertion [94].

One of the several EGFR TKIs that have been produced in the last few decades may be used to treat patients with non-small cell lung cancer (NSCLC) who have kinase-activating mutations. Erlotinib and gefitinib, two first-generation EGFR-TKIs, interact with the ATP-binding pocket of either active or inactive EGFR [95]. Afatinib and dacomitinib, two examples of irreversible EGFR inhibitors, are members of the second generation of EGFR TKIs. These drugs bind covalently to the ATP-binding pocket of EGFR8. Initial and subsequent generations of EGFR-TKIs have shown promising results for patients with EGFR kinase-activating mutations. However, if the EGFR T790M mutation in exon 20 is present, which affects approximately half of NSCLC patients, these medications may become resistant.

Other Targeted Anticancer Agents

Newly authorized or tested forms of targeted therapy include the following: epigenetic modulators (such as inhibitors of DNA methyltransferase, histone deacetylase, EZH2, and isocitrate dehydrogenase), smoothened inhibitors, Bcl-2 inhibitors, PARP inhibitors, proteasome inhibitors, and so on.

PARP inhibitors

DNA damage response (DDR) and DNA repair pathway regulation are both reliant on PARP family members. Binding of PARP proteins to DNA lesions causes PARylation of DNA damage response components and chromatin. PARP1 and PARP2 are the most prominent members of this family. This technique leads to DNA repair by introducing effectors that repair damaged DNA, such as XRCC. After autoPARylation is finished, DNA repair proteins are brought in to continue the

process after PARP separates from DNA. An important part of the conservative homologous recombination repair (HRR) process, which is used to heal double-strand DNA breaks, are the tumor-suppressor genes BReastCAncer gene 1 (BRCA1) and BRCA2 (BRCA1/2). Multiple malignancies, including breast, ovarian, pancreatic, and prostate cancers, have been associated with mutations in the BRCA1/2 genes. Damage to DNA may occur as a result of nonconservative DNA repair processes, such nonhomologous end joining, when the BRCA1/2 gene is mutated, which prevents BRCA from performing its HRR function. The use of PARP inhibitors in the treatment of BRCA-deficient cells causes genomic instability and the death of cancer cells because these cells are prone to blocking the DNA repair machinery. Some have proposed that tumors including BRCA mutations might be treated by targeting PARP, since the inhibition of PARP and BRCA1/2 mutations form a synthetic lethal interaction. Ovarian, breast, and prostate cancers that carry a BRCA mutation have been treated with a variety of oral PARP inhibitors, including olaparib, rucaparib, niraparib, and talazoparib.

Conclusion

Tailor-made treatments are a game-changer in the fight against cancer because they enable more precise treatment with fewer undesirable side effects than ever before. Targeted treatments attempt to lessen the likelihood of harm to healthy tissues caused by conventional treatments like radiation and chemotherapy by disrupting specific biochemical processes and mechanisms that are only found in cancer cells. In addition to better treatment results, this method improves patients' quality of life by reducing the likelihood of side effects.

The aforementioned study emphasizes the significance of utilizing molecular insights to identify, validate, and develop novel therapeutic targets. The discipline has made great strides in combating some of the most aggressive and treatment-resistant malignancies by using modern technologies including RNA-based therapies, small-molecule inhibitors, and monoclonal antibodies. These therapies have already been quite precise, but with the addition of biomarkers for patient stratification, they may be even more precisely adjusted to each tumor's specific genetic and molecular makeup.

In spite of these advancements, there are still challenges to be overcome before tailored medicines can be used completely. The molecular complexity of cancer, the heterogeneity of tumors, and the development of resistance mechanisms are major obstacles. Resistance, in particular, remains a significant issue due to the fact that cancer cells frequently evolve to avoid treatment interventions. In addition, there is a technological issue that needs to be resolved: delivering targeted medicines to tumor regions as little as possible while minimizing systemic exposure. There is still a huge chasm between what we find in the lab and what we can use in the clinic. Financial, logistical, and regulatory obstacles that hinder translational research frequently delay the availability of novel treatments for patients in need. Concerns over equality in global healthcare are heightened by the high research costs and subsequent price of these medicines, which further restrict their accessibility, especially in resource-constrained places.

This study demonstrates the necessity of teamwork to address these issues. To ensure sure these treatments are available and effective, researchers, doctors, politicians, and the pharmaceutical industry need to collaborate. To overcome current obstacles and propel the next wave of advances in targeted treatment development, new technologies like nanotechnology for drug transport, CRISPR for accurate genetic editing, and artificial intelligence for drug discovery are showing tremendous promise.

The concepts and data presented in this study may serve as a basis for subsequent research in molecular-targeted oncology. By maintaining our commitment to cutting-edge research, fostering collaborations across disciplines, and prioritizing patient-centered care, we can work toward developing treatments that improve the quality of life for cancer patients worldwide. The ultimate goal is still the same: to make cancer less of a killer and more of a disease that can be treated so that personalized medicines can eventually replace chemotherapy and radiation.

Future Perspectives

Many tyrosine kinase inhibitors have been developed, tested, and used clinically for the treatment of cancer thanks to advances in molecular diagnostics, genome-wide analysis, and our understanding of cancer biology. A number of significant challenges prevent targeted therapy from becoming a successful cancer treatment, including toxicity, drug-resistant tumor recurrence, and poor effectiveness. This highlights the need for further study into the development of effective customized targeted therapies with the potential to overcome pharmaceutical resistance while also decreasing toxicity and unwanted effects. Extensive structural studies, genetic and biochemical research, and the discovery of new druggable targets are essential components of the drug development framework necessary to achieve this aim. To further molecular focused therapy, new medications with different chemical entities or action mechanisms are required. Additional methods for cancer-specific control beyond direct or allosteric modulation include posttranslational modification and targeted protein degradation utilizing proteolysis-targeting chimera (PROTAC). Optimal combinatorial therapeutic strategies utilizing molecular targeted therapy, either alone or in combination with other anticancer treatments (like immune checkpoint inhibitors and chemotherapy), are necessary for increased efficacy, decreased toxicity, and minimal drug resistance. It is also critical to find small molecule inhibitors that can block the signaling pathways that lead to cancer cell growth and medication resistance. Targeting cancer cells specifically is just as crucial as developing methods to target normal cells nonspecifically, which leads to toxicity and side effects in targeted treatments. An excellent example of this is the new family of inhibitors that target KRASG12C. It was previously believed that KRAS is an unattainable target due to the failure of farnesyltransferase inhibitors in clinical studies. To sidestep the drawbacks of blocking wild-type KRAS, new research took use of cysteine's strong reactivity to create chemicals that bind covalently to KRAS via the mutant cysteine residue and allosterically prevent GTP from binding to KRAS. This method not only inhibits wild-type KRAS but also mutant KRAS without occupying the surface GTP/GDP binding pocket. The discovery of many effective KRASG12C inhibitors has been spurred by this ground-breaking study, and they have all gained clinical approval. Furthermore, preclinical testing has been conducted on medicines that target several mutant KRAS types, including KRASG12D.

References

1. Barouch-Bentov, R.; Sauer, K., Mechanisms of drug resistance in kinases. Expert OpinInvestig Drugs 2011, 20 (2), 153-208.
2. Wu, P.; Nielsen, T. E.; Clausen, M. H., FDA-approved small-molecule kinase inhibitors. Trends Pharmacol Sci 2015, 36 (7), 422-39.
3. Zhang, J.; Yang, P. L.; Gray, N. S., Targeting cancer with small molecule kinase inhibitors. Nat Rev Cancer 2009, 9 (1), 28-39.
4. Chen, Y.-f.; Fu, L.-w., Mechanisms of acquired resistance to tyrosine kinase inhibitors. Acta Pharmaceutica Sinica B 2011, 1 (4), 197-207.
5. Siveen, K. S.; Prabhu, K. S.; Achkar, I. W.; Kuttikrishnan, S.; Shyam, S.; Khan, A. Q.; Merhi, M.; Dermime, S.; Uddin, S., Role of non receptor tyrosine kinases in hematological malignances and its targeting by natural products. Mol Cancer 2018, 17 (1), 31.
6. Pottier, C.; Fresnais, M.; Gilon, M.; Jerusalem, G.; Longuespee, R.; Sounni, N. E., Tyrosine kinase inhibitors in cancer: breakthrough and challenges of targeted therapy. Cancers (Basel) 2020, 12 (3), 731.
7. Fletcher, J. I.; Williams, R. T.; Henderson, M. J.; Norris, M. D.; Haber, M., ABC transporters as mediators of drug resistance and contributors to cancer cell biology. Drug Resist Updat 2016, 26, 1-9.
8. Milane, L.; Ganesh, S.; Shah, S.; Duan, Z. F.; Amiji, M., Multi-modal strategies for overcoming tumor drug resistance: hypoxia, the Warburg effect, stem cells, and multifunctional nanotechnology. J Control Release 2011, 155 (2), 237-47.

9. Zamboni, W. C.; Torchilin, V.; Patri, A. K.; Hrkach, J.; Stern, S.; Lee, R.; Nel, A.; Panaro, N. J.; Grodzinski, P., Best practices in cancer nanotechnology: perspective from NCI nanotechnology alliance. Clin Cancer Res 2012, 18 (12), 3229-41.
10. Gmeiner, W. H.; Ghosh, S., Nanotechnology for cancer treatment. Nanotechnol Rev 2015, 3 (2), 111-122.
11. Uthaman, S.; Huh, K. M.; Park, I.-K., Tumor microenvironment-responsive nanoparticles for cancer theragnostic applications. Biomater Res 2018, 22, 22.
12. Allison, R. R.; Mota, H. C.; Bagnato, V. S.; Sibata, C. H., Bio-nanotechnology and photodynamic therapy—State of the art review. Photodiagnosis and Photodynamic Therapy 2008, 5 (1), 19-28.
13. Fernandes, C.; Suares, D.; Yergeri, M. C., Tumor microenvironment targeted nanotherapy. Frontiers in pharmacology 2018, 9, 1230.
14. Zhang, Y.; Huang, Y.; Li, S., Polymeric micelles: nanocarriers for cancertargeted drug delivery. AAPS PharmSciTech 2014, 15 (4), 862-71.
15. Allen, S. D.; Bobbala, S.; Karabin, N. B.; Scott, E. A., On the advancement of polymeric bicontinuous nanospheres toward biomedical applications. Nanoscale Horizons 2019, 4 (2), 258-72.
16. Kothamasu, P.; Kanumur, H.; Ravur, N.; Maddu, C.; Parasuramrajam, R.; Thangavel, S., Nanocapsules: the weapons for novel drug delivery systems. Bioimpacts 2012, 2 (2), 71-81.
17. Xiao-ying, Z.; Pei-ying, Z., Polymersomes in nanomedicine - a review. Current Nanoscience 2017, 13 (2), 124-29.
18. Ulasov, A. V.; Khramtsov, Y. V.; Trusov, G. A.; Rosenkranz, A. A.; Sverdlov, E. D.; Sobolev, A. S., Properties of PEI-based polyplex nanoparticles that correlate with their transfection efficacy. Mol Ther 2011, 19 (1), 103-12.
19. Farjadian, F.; Ghasemi, A.; Gohari, O.; Roointan, A.; Karimi, M.; Hamblin, M. R., Nanopharmaceuticals and nanomedicines currently on the market: challenges and opportunities. Nanomedicine 2018, 14 (1), 93-126.
20. Hegde, N.; Velingkar, V.; Prabhakar, B., An update on design and pharmacology of dendritic poly(L-lysine). International Journal of Peptide Research and Therapeutics 2019, 25 (4), 1539-62.
21. Dalwadi, C.; Patel, G., Application of nanohydrogels in drug delivery systems: recent patents review. Recent Pat Nanotechnol 2015, 9 (1), 17-25.
22. Kajdič, S.; Planinšek, O.; Gašperlin, M.; Kocbek, P., Electrospun nanofibers for customized drug-delivery systems. Journal of Drug Delivery Science and Technology 2019, 51, 672-81.
23. Sherje, A. P.; Dravyakar, B. R.; Kadam, D.; Jadhav, M., Cyclodextrin-based nanosponges: A critical review. Carbohydr Polym 2017, 173, 37-49.

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