Proceedings of the 6th International Conference on Biological Engineering and Medical Science
DOI: 10.54254/2753-8818/2026.38032

Rethinking Targeted Therapy in Glioblastoma: From Molecular Targets to Context-Dependent Therapeutic Vulnerabilities

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Abstract. Glioblastoma (GBM) is a highly aggressive brain tumor characterized by poor survival and frequent recurrence. Although numerous targeted drugs, including inhibitors of receptor tyrosine kinases (RTKs), cyclin-dependent kinases (CDKs), and poly(ADP-ribose) polymerase (PARP), have entered clinical trials, their therapeutic benefits remain limited, highlighting the gap between molecular targets and durable therapeutic vulnerabilities. This review summarizes the molecular mechanisms and clinical limitations of representative targeted therapies and examines how GBM heterogeneity, cellular plasticity, tumor microenvironment (TME), blood-brain barrier (BBB), pathway redundancy, and evolutionary adaptation contribute to treatment failure. Potential strategies, including multi-targeted therapy, precision oncology, AI-assisted target prioritization, and improved BBB-penetrating drug delivery, are discussed. Overall, targeting compensatory pathways across multiple cellular states may be more effective than focusing on a single fixed target. Future research should identify biologically interconnected targets, clarify TME-associated cellular plasticity, integrate multi-omics into AI-assisted target selection, and optimize BBB-penetrating delivery systems to improve therapeutic efficacy and durability.

Keywords: Glioblastoma, Targeted drugs, Tumor heterogeneity, Cellular Plasticity

1. Introduction

Glioblastoma (GBM) is one of the most fatal brain tumors, with limited survival advantages and poor prognosis. The survival rate of GBM shows no significant improvement with conventional treatments like surgical dissection and then applying chemotherapy and radiotherapy, and the survival rate remains poor even with targeted therapy [1]. This raises the urgent requirements of increasing patient survival rate. Although scientists have identified many potential targets of GBM, they have not shown significant improvement in patients' outcomes in clinical trials. Targeted therapies demonstrated positive outcomes in treating other cancers but not GBM, raising confusion about why the results are disappointing in GBM.

Targeted therapy has a clear and reasonable logic: identifying abnormal gene expression patterns for regulating crucial signaling pathways, reducing tumor proliferation and survival in cancer cells selectively. There have been many related pathways and targets identified in GBM, including

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DOI: 10.54254/2753-8818/2026.38032

epidermal growth factor receptor (EGFR) and platelet-derived growth factor receptor (PDGFR) [1]. Cyclin-dependent kinases (CDKs) and PRAP [2]. The abnormal regulation of related pathways is important for tumor growth and survival. Blocking those pathways in tumor cells should be devastating to tumor progression and eventually kill tumor cells, like GBM cells. However, the case of GBM means the molecular understanding is not equal to clinical success.

The main biological barriers related to resistance and recurrence are intra-tumoral heterogeneity, cellular plasticity, tumor microenvironment (TME), and pathway redundancy for adaptation. GBM cells exist in many different cell population types and cellular states, each having different transcriptional patterns, and switching between different cell population types or cellular states is available, making it difficult to kill all cells based on one or a few targets [3]. Also, with multiple pathways to change gene regulation, GBM cells can depend on other pathways to survive if not all pathways are blocked, corresponding to the challenges to kill all cell populations [4]. In addition to the cellular level, the immunosuppressive and pro-tumoral hypoxia TME reduces the effectiveness of drugs, and the blood-brain barrier (BBB) hinders drug delivery [4, 5]. Those interconnected mechanisms increase the difficulty of completely eliminating GBM cells, so resistant cells will grow and refill the population, eventually leading to tumor recurrence.

Therefore, a more comprehensive target selection method is needed, which has a multi-layer focus on dysregulation in tumor cells and essentiality across dynamic cellular types and states, especially those related to therapy resistance and recurrence. Understanding the current limitations of targeted therapies and integrating the biology of GBM into consideration of future strategies are crucial to construct a better target selection framework for future success in clinical trials.

2. Current targeted therapy

2.1. RTK inhibitors

RTKs regulate many downstream genes controlling cellular behavior, including growth and other metabolism, so dysregulation of RTKs would lead to abnormal cellular behavior, eventually becoming cancerous cells [1]. EGFR amplification is one of the most frequent alterations under RTKs, while PDGFR is the second dominant, with subunits crucial for embryonic development and blood-vessel formation [1]. Although their pathogenesis mechanisms are well-understood, the clinical benefits translated are still limited, as many FDA-approved RTK inhibitors, primarily for other cancers, did not show significant improvement in mean survival of GBM patients in clinical trials [1].

2.2. Cell-cycle targeting (CDK) inhibitors

Overexpression of CDKs is quite frequent in many cancers, especially CDK4/6, increasing inactivation of the tumor suppressor retinoblastoma protein (Rb) and eventually promoting expression of genes required for the G1 to S phase transition of the cell cycle [6]. Targeting CDK4/6 can inhibit tumor growth by blocking the cell cycle, and there have been approved CDK4/6 inhibitors for treating breast cancers, justifying the logic of target selection [6]. Despite being promising, the clinical benefits are still limited, with a phase-2 clinical trial of a CDK4/6 inhibitor, palbociclib, on recurrent GBM adult patients showing 95% of evaluable patients having tumor progressing within six months and a median progression free survival (PFS) of only 5.14 weeks , which is lower than the PFS of GBM recurrent patients receiving re-radiation (5.3 months) [7, 8].

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2.3. DNA damage/apoptosis ---PRAP inhibitors

Acquiring the ability to escape from apoptosis is a hallmark of cancer [9, 10], and sensing DNA damage is part of the DNA damage response that can trigger apoptosis. Therefore, the poly-ADP-ribose polymerase (PARPi), which can lead to replication-associated double-strand breaks, can increase cell lethality and is also combined with temozolomide and radiotherapy to increase the cytotoxic effects in GBM treatment [2]. Although PRAPi has become standard treatment of some solid tumors and is promising for treating GBM, there is no significant evidence that combining PRAPi with standard GBM treatments shows clinical improvements [2].

There are multiple molecular targets with reasonable rationale but fail to produce significant clinical benefits. Therefore, identifying and understanding the molecular mechanisms of candidate targets is not sufficient, and the key problem is not a lack of targets. Instead, incorporating more GBM biology into consideration will help the development of future therapies, and understanding how characteristic GBM biology limits the targeted therapies is the first and important step.

3. Factors limiting the clinical benefits of current targeted therapies

3.1. Intratumoral heterogeneity

Intratumoral heterogeneity indicates tumor composition of GBM is complex, with differences in genetic expression and thus forming different cellular states. Targeted gene alterations may only be present in some but not all cells, so eliminating tumor cells is difficult. According to Neftel et al., there are four main cellular states in GBM: (1) neural-progenitor-like (NPC-like), (2) oligodendrocyte-progenitor-like (OPC-like), (3) astrocyte-like (AC-like), and (4) mesenchymal-like (MES-like) states, and the relative frequency of cells in each state can vary and is influenced by amplifications of the CDK4, EGFR, and PDGFRA genes and the mutations in the NF1 locus, respectively [3]. This means each cellular state depends on unique pathways, having different sensitivity to a targeted drug. As a result, the mono-target drugs may only affect cells in limited cellular states. Unsensitive cells would survive and develop a population without depending on targeted genes, eventually leading to resistance and tumor recurrence. This can explain why mono-target drugs, like those targeting EGFR or PDGFR, demonstrated limited clinical improvements, corresponding to one gene alteration not being present in all GBM cells and also demonstrating complex co-existence patterns: EGFR is present in around 57.4% and PDGFRA is 13.1%, while half of tumors with alteration of PDGFRA also have EGFR alterations (42.4%) [9]. Therefore, intratumoral heterogeneity challenges traditional, mono-targeted drugs as the target gene does not universally exist in all cells.

3.2. Cellular plasticity

Beyond different cellular states existing in GBM tumors, being able to transition between cellular states further increases the difficulty of targeted drugs. This ability is referred to as cellular plasticity, which is unlocked in many cancers to escape from terminal differentiation states with disruption of differentiation: dedifferentiation (returning to progenitor state), blocked dedifferentiation (blocked from reaching terminal state), and transdifferentiation (switching to a different cell lineage) [10]. GBM cells were shown to have the potential for cellular state transition, influenced by genetic alterations and the tumor microenvironment (TME) [3]. Since different cellular states have different genetic alterations, they may depend on different pathways, and the

DOI: 10.54254/2753-8818/2026.38032

addition of targeted drugs may induce a transition from sensitive to resistant cellular states to survive from targeted drugs, supported by transdifferentiation events shown in melanoma, which does not directly prove this mechanism for GBM but implies the potential for GBM to utilize [11]. In this case, this transition means the characteristic genetic alterations defining each cellular state change, and thus cellular plasticity challenges the fixed-targets design of targeted drugs with dynamic, variable targets.

3.3. TME and blood-brain barrier (BBB）

Apart from the cellular level, TME also limits the effectiveness of targeted drugs. According to Sherma et al., GBM cells can modify the TME to promote proliferation, invasiveness, and other pro-tumorigenesis characteristics; The acidosis and hypoxia due to the increasing reliance on anaerobic respiration (Warburg effect) are examples of GBM metabolism changing the microenvironment, which creates an immunosuppressive TME and reduces the effectiveness of targets related to immune cell functions [5]. Also, TME affects transition between different cellular states, so changing the microenvironment by either tumor cell metabolism or drug molecules potentially contributes to cellular plasticity. Even if the drug molecules are effective, the limited and unequal tumor exposure to drugs still hinders anti-tumor progress [3]. For instance, CDK4/6 inhibitors, like Abemaciclib, ribociclib, and palbociclib approved for breast cancer subtype treatment poorly penetrate BBB [6]. This suggests BBB challenges the drug delivery system and may represent the bottleneck in passing clinical trials. Eliminating all GBM cells becomes difficult, and surviving cells would refill the population. Therefore, the dynamic environmental factors influencing and influenced by GBM cells impose challenges on traditional, cellular-level targeted drugs

3.4. Pathway redundancy and evolutionary adaptation

Macroscopically, the presence of multiple pro-tumorigenesis biological pathways also challenges mono- and fixed-targeted drugs, as blocking one or a few fixed pathways cannot eliminate all GBM cells. Those pathways include RTK-related, Rb, TP53, and PI3K/Akt signaling pathways, spanning the cell cycle, proliferation, and DNA damage response regulation [1, 4]. When introducing a drug targeting RTKs, cells may switch to other cellular states or lineages with different transcriptional patterns, thus switching to other pathways and developing different sensitivity to the drug to escape, causing therapeutic resistance [3, 10]. The expansion of the population can also drive tumor recurrence. As a result, GBM cells can adapt to the environment, with redundant pathways provided by the multiple, inter-switchable cellular states, possessing resistance to traditional, fixed, and mono-targeted drugs along with the TME.

4. Potential strategies

4.1. Multi-targets therapy

The pathway redundancy and evolutionary adaptation enabled by the cellular plasticity suggest the lack of a universally present target in all GBM cells. Therefore, targeting multiple pathways is a potential solution, and selecting the optimal combination of targets is important to block redundant pathways. Ideally, selected pathways should be compensatory, ideally both function for tumor progression but with different mechanisms, to block GBM cells from escaping treatment by triggering synthetic lethality. However, more detailed aspects like toxicity, optimizing combination,

DOI: 10.54254/2753-8818/2026.38032

and delivery effectiveness across BBB are still under investigation. Also, recent clinical trials for multi-target inhibitors indicates that neither mono-target (namely erlotinib and gefitinib) nor combining different (multitarget) small molecule inhibitors that target various pathways showed a significant increase in overall survival (OS) in GBM patients [12]. Therefore, multi-targets therapy is promising, while still requiring further research to bring clinical benefits.

4.2. Precision medicine

Due to the heterogeneity nature of GBM, different patients have different GBM cell composition in their tumors, suggesting that their tumor vulnerabilities also differ due to different gene expression patterns of different cell states. Designing a targeted drug to effectively treat all patients is difficult due to the lack of a universal target existing in tumors of all patients, eventually leaving some GBM cells survive and expand to drive tumor recurrence and resistance. Therefore, acknowledging that different patients have different targets and personalizing the targeted drug based on their tumor molecular profile can potentially tackle the challenge. For instance, patients with BRAF V600E-mutated GBM benefit from BRAF/MEK-inhibition, since the drug targets the correct molecular alterations and related pathway in those patients [13]. However, not all targeted therapy is effective due to factors like poor tumor penetration and pathway redundancy; further consideration for bioavailability, TME, and targeting pathways (not just single target) are required to improve the efficacy [13]. Finding the correct candidate targets with precision ontology is necessary, while still requiring further research on optimization in drug delivery and considering biology of GBM.

4.3. AI-assisted target prioritization

Nowadays there are many potential targets for GBM, including EGFR, CDK, and others mentioned before. Then selecting promising targets for further development is the next critical step. Integrating multi-layer information, for instance, from gene expression pattern and essentiality, would assist this process. With initial candidate targets from bulk-RNA seq which identifying dysregulated genes on average, the next step is to focus on cellular level. Since GBM tumor is heterogenous, identifying individual GBM cells' molecular alterations provides insights for target selection. Researchers have shown utilizing single-cell technology, including transcriptomics, would be helpful in revealing GBM subtype functions and vulnerabilities. Also, target validation with methods like CRISPR is necessary to identify genes actually functioning in tumor progression and resistance. By integrating relevant information, researchers can use AI to generate a ranking that help prioritizing the most promising targets, including those genes that are highly expressed in bulk-RNA seq, single-cell transcriptomics of specific resistant-related cell population or across multiple cellular states, and also proved to cause tumor cell death after knock-down. AI framework could be modified to assign scores and weight for each necessary sections and rank based on the overall scores. AI can also integrate pathological and clinical information apart from multi-omics, suggesting its potential to include GBM biology, like the instability of targets, the lack of universal targets, complex tumor microenvironment and BBB, into target prioritization framework [14]. Combining this with precision medicine would optimize the results as it is more patient-specific but also consider whole layers.

DOI: 10.54254/2753-8818/2026.38032

4.4. Improving drug delivery

Even if the drug molecules are well-designed to target multiple compensatory pathways or targets expressed across multiple cell types based on precise tumor profiling, being unable to reach the tumor sites with sufficient concentration still limits the effectiveness. The BBB limits drug penetration to lipophilic, low molecular weight (MW) molecules, making most small molecule drugs and nearly all large molecule drugs fail to pass through it [15]. TMZ is a lipophilic and small MW molecule that theoretically could penetrate BBB effectively, but the drug exposure on GBM tumor remains poor [15]. This promotes the development of more advanced drug delivery systems or modifications that enable drug molecules to cross BBB and reach the GBM tumor, including BBB-penetrated drugs and nanoparticle. The BBB-penetrated drugs include conjugating drug molecule with known anti-tumor effects to a chemical moiety that increases its solubility or cell permeability and some chemical engineering utilizing barrier entry mechanism, like carrier-mediated transport, with the latter group having a drug that shows promising early clinical trial results but awaiting phase I and II results [15]. The Nanoparticle system allows molecule entrance without modifying the drug molecule, despite having limitations including low tumor retention and suboptimal drug release control [15]. Improving drug penetration with better delivery across BBB is a crucial and promising strategy, while further research is needed to conquer existing challenges.

5. Discussion

5.1. Evaluation of potential strategies

The above strategies address known aspects of GBM biology related to limitation in targeted drugs to different extent. Multi-target drugs address pathway redundancy and adaptive evolution, which is enabled by intra-tumoral heterogeneity and cellular plasticity. This requires understanding of complex compensatory relationship between multiple pathways and also consideration of toxicity to normal cells. Precision ontology addresses the heterogeneity between patients, but GBM cells are adaptively evolving, indicating the needs of constant sampling and drug adjustment. AI-assisted target prioritization provides most promising candidate targets justified by multi-layer data, clinical information, and publications, potentially providing targets that exist across multiple cell states and are less affected by immunosuppressive TME to address the heterogeneity, cellular plasticity, and TME. However, the reliability is largely affected by the quality of data and publication. Finally, improving drug delivery primarily targets blood brain barrier but lack of consideration of other GBM biology. Some strategies overlap in some issues but they are mostly complementary, so combining all into a consistent workflow may optimize the effectiveness. For instance, AI-assisted target prioritization identifies most promising candidate targets, and then profiling the patient's tumor to select most patient-relevant targets. After that, multi-target personalized drug molecules are designed and the nanoparticle or modifications on anti-tumor molecules are used to maximize drug penetration.

5.2. Limitations

Despite considering most GBM biology driven clinical failure of targeted drugs, the above strategies do not specifically address the TME. The AI can prioritize targets that are less associated with immune cell function, but these would limit the potential choices. Also, TME is dynamic, and the change in TME is related to the transition between cell states (cellular plasticity). With limited

DOI: 10.54254/2753-8818/2026.38032

information about how cell states composition would change, it is difficult to ensure the selected targets for the multi-target drugs can cover the molecular alterations of all GBM cells. The above strategies do not actively adjust TME and cannot predict the cell state transition, which limits the effectiveness as some cells may still escape the treatment and expand.

5.3. Future direction

The proposed framework above still requires detailed refinement in each step. Future study may focus on incorporating more data sets, like proteomics and metabolomics, and designing the practical scoring and weighing system for AI to find multiple targets directly addressing GBM biology. Incorporating longitudinal tumor profiling data to find GBM cells evolving trajectory and taking that into personalized drug consideration can address the dynamic and evolving GBM cell population structure. Also, future study should fill in gaps about TME and its relationship to GBM cell plasticity, potentially developing strategies that either allow researchers to predict the possible cell composition based on known TME or adjust TME through drugs. Longitude study about TMW is also required due to its dynamic nature.

6. Conclusion

This essay explains the reasoning behind limited clinical translation of GBM targeted drugs despite having many potential targets, by analyzing the dynamic and inter-connected GBM biology, including heterogeneity, cellular plasticity, TME, BBB, pathway redundancy and adaption, and discuss the advantages and limitations of potential strategies, providing a structured understanding of GBM biology and its relationship to underperformance of targeted drugs. The essay identifies the absence of universally expressed targets and the instability of targets limits the effectiveness of current targeted drugs, especially those based on an isolated target, and also concluded that integrates designing multi-targeted drugs, precision ontology, AI assisted target prioritization, and improving drug delivery across BBB to complementarily target GBM biology, suggesting a promising combinatory framework for GBM treatment. However, the proposed framework remains limited on addressing TME and related cellular plasticity, due to insufficient current strategies that primarily target TME. Future study should focus more on identifying multiple biologically-related targets and capturing the dynamic TME and tumor cell evolving trend to address the unstable targets, instead of devoting to mono and fixed-target drugs, developing the AI-assisted framework with more datasets, and improving the current BBB penetration drug delivery system to increase the drug retention or other relevant aspects.

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