Driving cancer innovation forward

Investor Presentation

July 2026

Forward-Looking Statements

Various statements in this presentation concerning BioLineRx's future expectations constitute "forward-looking statements" within the meaning of the Private Securities Litigation Reform Act of 1995. These statements include words such as "anticipates," "believes," "could," "estimates," "expects," "intends," "may," "plans," "potential," "predicts," "projects," "should," "will," and "would," and describe opinions about future events. These include statements regarding management's expectations, beliefs and intentions regarding, among other things, the expectations with regard to clinical trials of motixafortide and GLIX1, expected timing of clinical readouts, the expected cash runway, and BioLineRx's business strategy. These forward-looking statements involve known and unknown risks, uncertainties and other factors that may cause the actual results, performance or achievements of BioLineRx to be materially different from any future results, performance or achievements expressed or implied by such forward-looking statements. Factors that could cause BioLineRx's actual results to differ materially from those expressed or implied in such forward-looking statements include, but are not limited to: the clinical development, commercialization and market acceptance of GLIX1 and motixafortide including the degree and pace of market uptake of APHEXDA for the mobilization of hematopoietic stem cells for autologous transplantation in multiple myeloma patients; the initiation, timing, progress and results of BioLineRx’s preclinical studies, clinical trials and other therapeutic candidate development efforts; BioLineRx’s ability to advance GLIX1 and motixafortide into clinical trials or to successfully complete its preclinical studies or clinical trials; whether the clinical trial results for GLIX1 and motixafortide will be predictive of real-world results; BioLineRx’s receipt of regulatory approvals for GLIX1 and motixafortide and the timing of other regulatory filings and approvals; whether access to GLIX1 and motixafortide is achieved in a commercially viable manner and whether GLIX1 and motixafortide receives adequate reimbursement from third-party payors; BioLineRx’s ability to establish, manage, and maintain corporate collaborations, as well as the ability of BioLineRx’s collaborators to execute on their development and commercialization plans; BioLineRx’s ability to integrate new therapeutic candidates and new personnel, as well as new collaborations; the interpretation of the properties and characteristics of BioLineRx’s therapeutic candidates and of the results obtained with its therapeutic candidates in preclinical studies or clinical trials; the implementation of BioLineRx’s business model and strategic plans for its business and therapeutic candidates; the scope of protection that BioLineRx’s is able to establish and maintain for intellectual property rights covering its therapeutic candidates and its ability to operate its business without infringing the intellectual property rights of others; estimates of BioLineRx’s expenses, future revenues, capital requirements and its need for and ability to access sufficient additional financing; risks related to changes in healthcare laws, rules and regulations in the United States or elsewhere; competitive companies, technologies and BioLineRx’s industry; BioLineRx’s ability to maintain the listing of its ADSs on Nasdaq; statements as to the impact of the political and security situation in Israel on BioLineRx’s business which may exacerbate the magnitude of the factors discussed above. These and other factors are more fully discussed in the "Risk Factors" section of BioLineRx's most recent annual report on Form 20-F filed with the Securities and Exchange Commission on March 23, 2026. In addition, any forward-looking statements represent BioLineRx's views only as of the date of this release and should not be relied upon as representing its views as of any subsequent date. BioLineRx does not assume any obligation to update any forward-looking statements unless required by law.

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BioLineRx investor highlights

Validated and successful track record of clinical development and regulatory approval – with focus on advancing targeted therapies for cancers with high unmet needs

GLIX1, our new lead asset, targets a broad range of cancers with reduced TET2 activity

- Novel MOA focused on DNA damage repair
- Potential for strong synergy demonstrated in combination with PARP inhibitors
- Initial development in glioblastoma (GBM), followed by other cancers
- In GBM: strong rationale and potential to overcome common reasons for failures
  - Excellent blood-brain barrier penetration
  - Cytotoxic to patient-derived neurospheres (glioma stem cells)
  - Efficacious in orthotopic GBM models and a TMZ-resistant PDX model
- Phase 1/2a study in GBM initiated, with part 1 dose escalation results expected in H1 2027

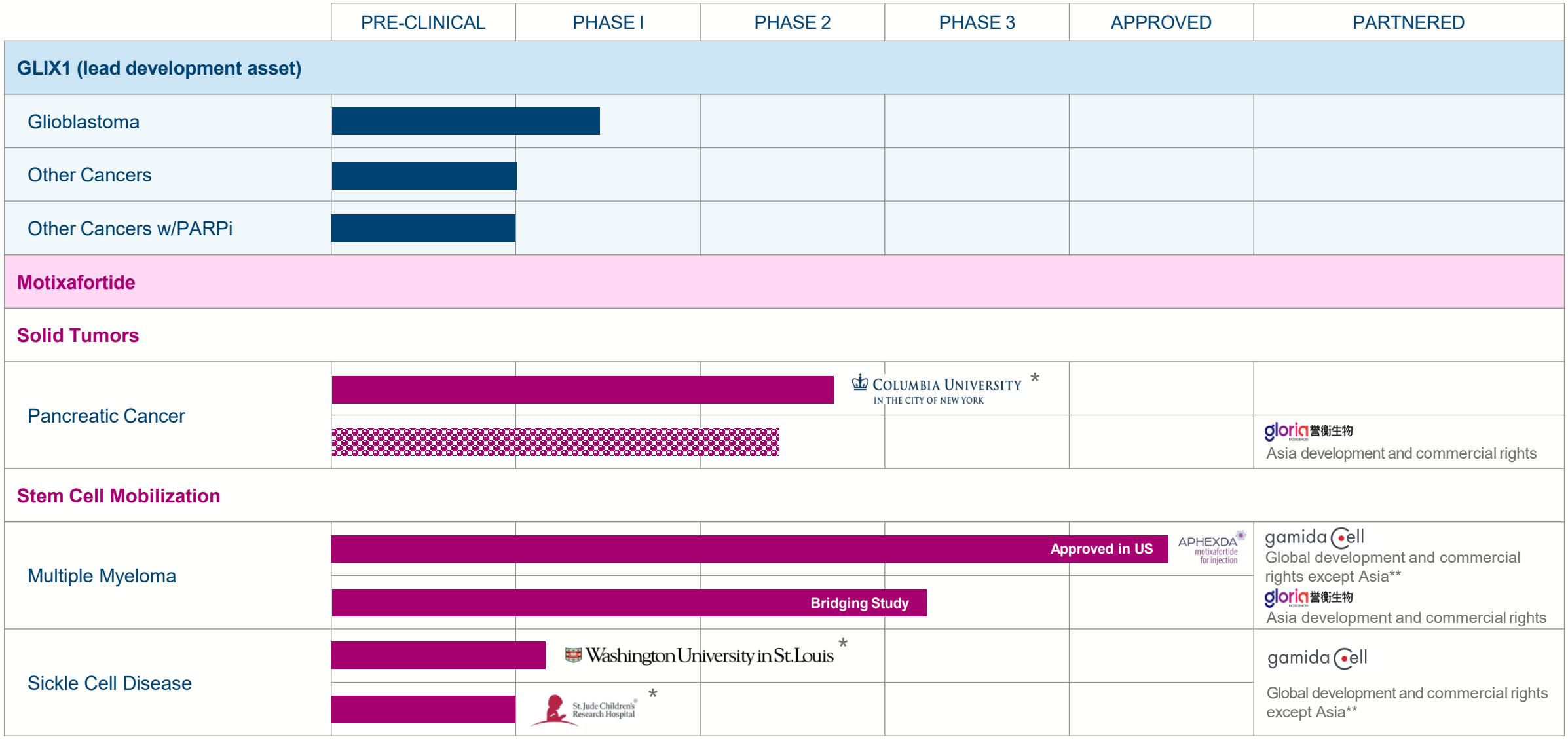
Motixafortide in metastatic pancreatic cancer (PDAC)

- The CheMo4METPANC randomized Phase 2b combination trial of motixafortide in PDAC is ongoing
- Study is being run in collaboration with Columbia University, and supported by Regeneron and BioLineRx
- Interim futility analysis expected in 2026

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Pipeline assets

*Investigator-initiated study
**Rights exclude solid-tumors
Studies in Planning



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GLIX1: A first-in-class, oral, small molecule with a novel mechanism of action applicable across a broad range of cancer indications

GLIX1 is positioned to address unmet needs for novel and more effective cancer treatments

First-in-class, novel mechanism of action, applicable to a broad range of cancers – targeting DNA damage repair

Oral route of administration

Excellent pre-clinical safety profile, allowing long-term treatment and potential combination treatments

Proven blood-brain-barrier penetration

Potential strong synergy in combination with PARP inhibitors

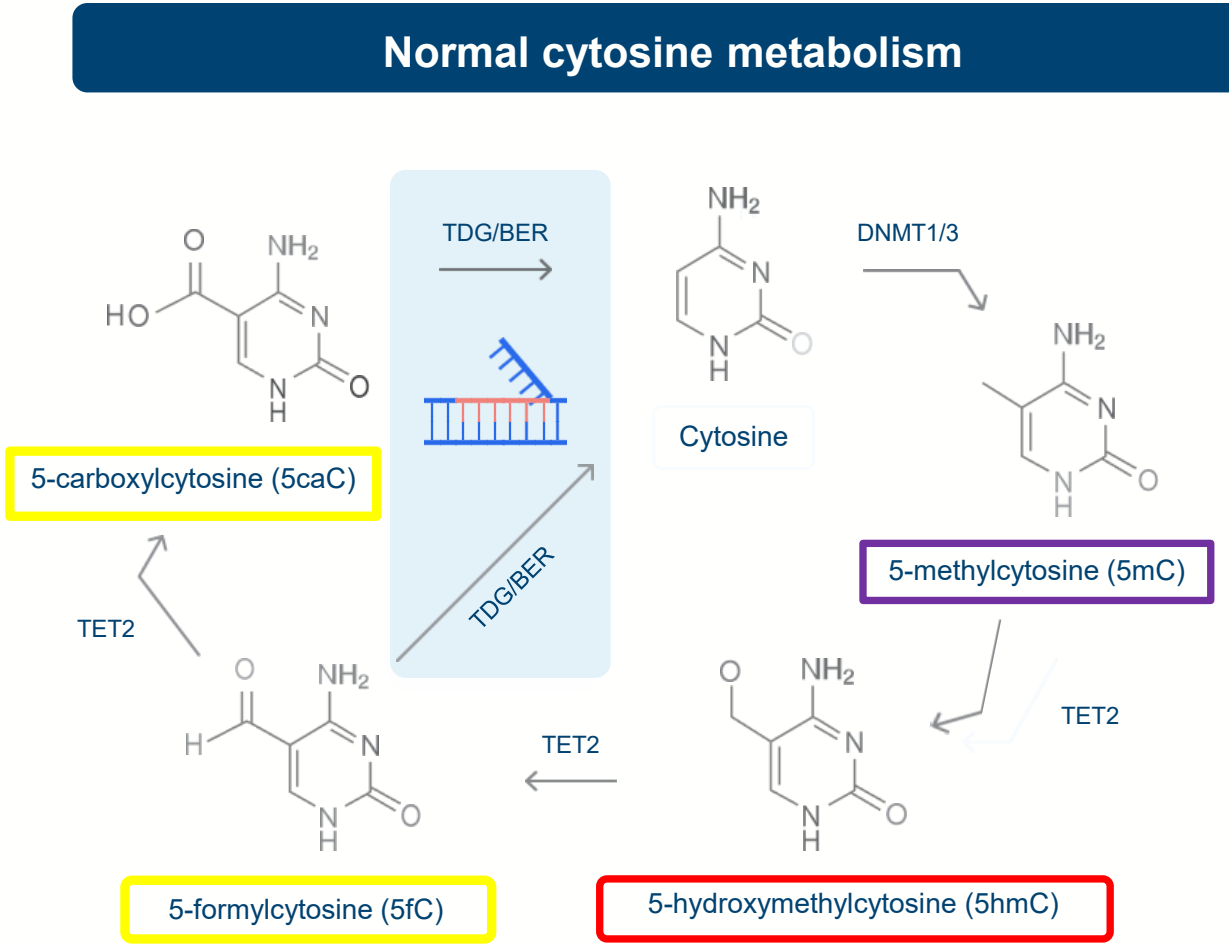
Glioblastoma, the first target indication, expected to provide data to support development in CNS and additional cancers

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GLIX1 mechanism of action

GLIX 1 activates Ten-Eleven Translocation 2 (TET2) that is commonly inhibited in cancer (1/3)

- TET2 initiates the DNA **demethylation** cycle by oxidizing 5-methylcytosine (5mc) to 5-hydroxymethylcytosine (5hmc), resulting **in single-stranded DNA breaks**[1]. These single-stranded DNA breaks are well tolerated in normal cells



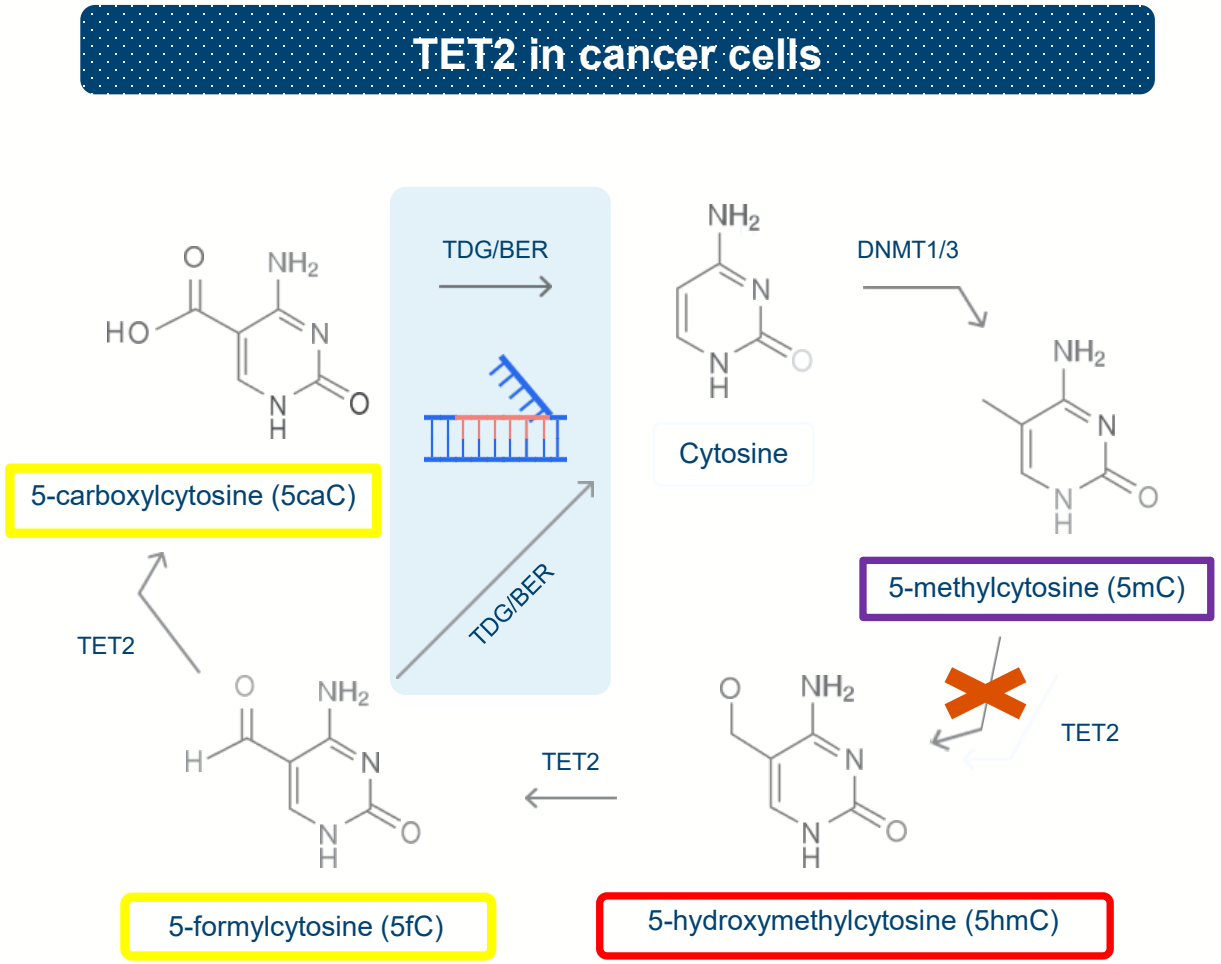
1.Jinrong Zhu et al, Cancer Bio Med, 2023, 21(2):111–116; 2. Wenxin Da et al, Clinical and Translational Oncology, 2024, 26:2156–2165; 3. Johnson KC et al, Nature Communications, 2016, 7, 13177; 4. Luisa Cimmino et al, Cell, 2017, 170(6):1079-1095

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GLIX 1 activates Ten-Eleven Translocation 2 (TET2) that is commonly inhibited in cancer (2/3)

- TET2 initiates the DNA **demethylation** cycle by oxidizing 5-methylcytosine (5mc) to 5-hydroxymethylcytosine (5hmc), resulting **in single-stranded DNA breaks**[1]. These single-stranded DNA breaks are well tolerated in normal cells

- In cancer cells, hypermethylated regions are common and **TET2 is inhibited by oncometabolites**, leading to increased DNA methylation (5mc) in close genomic proximity[3]



1.Jinrong Zhu et al, Cancer Bio Med, 2023, 21(2):111–116; 2. Wenxin Da et al, Clinical and Translational Oncology, 2024, 26:2156–2165; 3. Johnson KC et al, Nature Communications, 2016, 7, 13177; 4. Luisa Cimmino et al, Cell, 2017, 170(6):1079-1095

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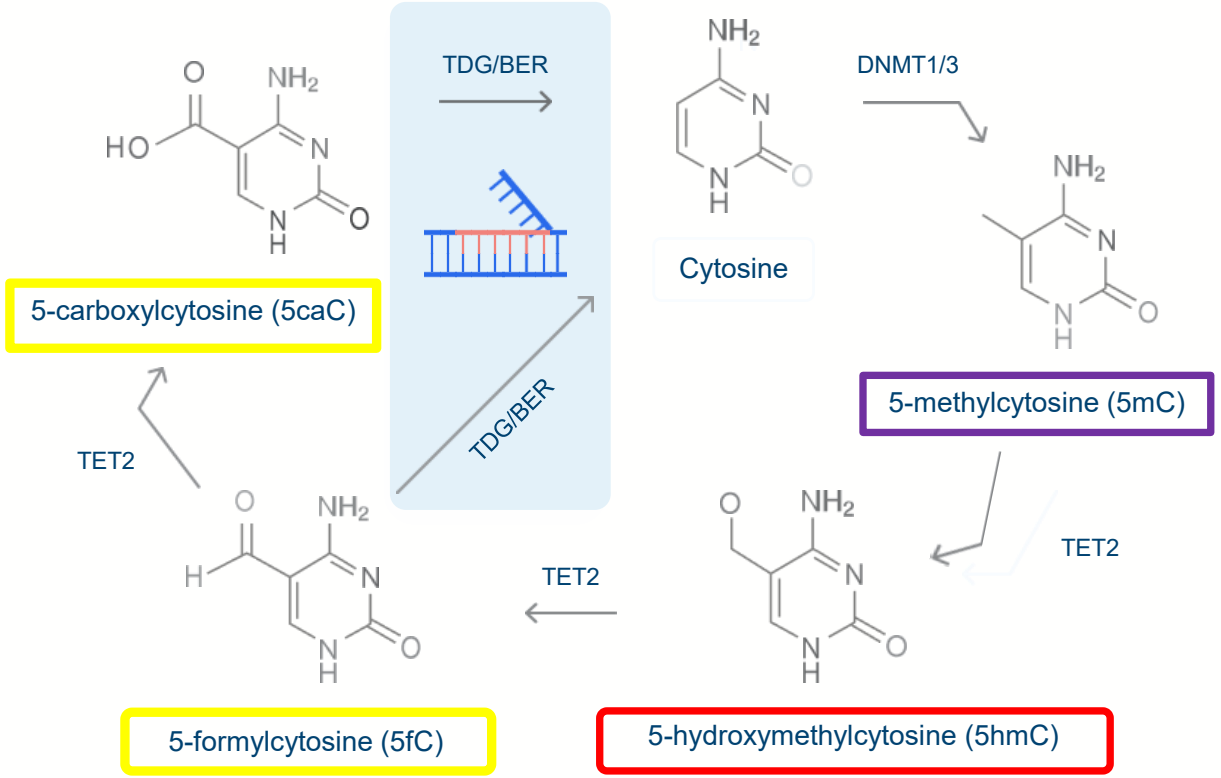
GLIX 1 activates Ten-Eleven Translocation 2 (TET2) that is commonly inhibited in cancer (3/3)

- TET2 initiates the DNA **demethylation** cycle by oxidizing 5-methylcytosine (5mc) to 5-hydroxymethylcytosine (5hmc), resulting **in single-stranded DNA breaks**[1]. These single-stranded DNA breaks are well tolerated in normal cells

- In cancer cells, hypermethylated regions are common and **TET2 is inhibited by oncometabolites**, leading to increased DNA methylation (5mc) in close genomic proximity[3]

- Restoration of TET2 activity creates many single-stranded DNA breaks at these heavily methylated regions, resulting in **double-stranded DNA breaks**, which **overwhelm the repair capacity of the cells, killing the cancer cells**[4]

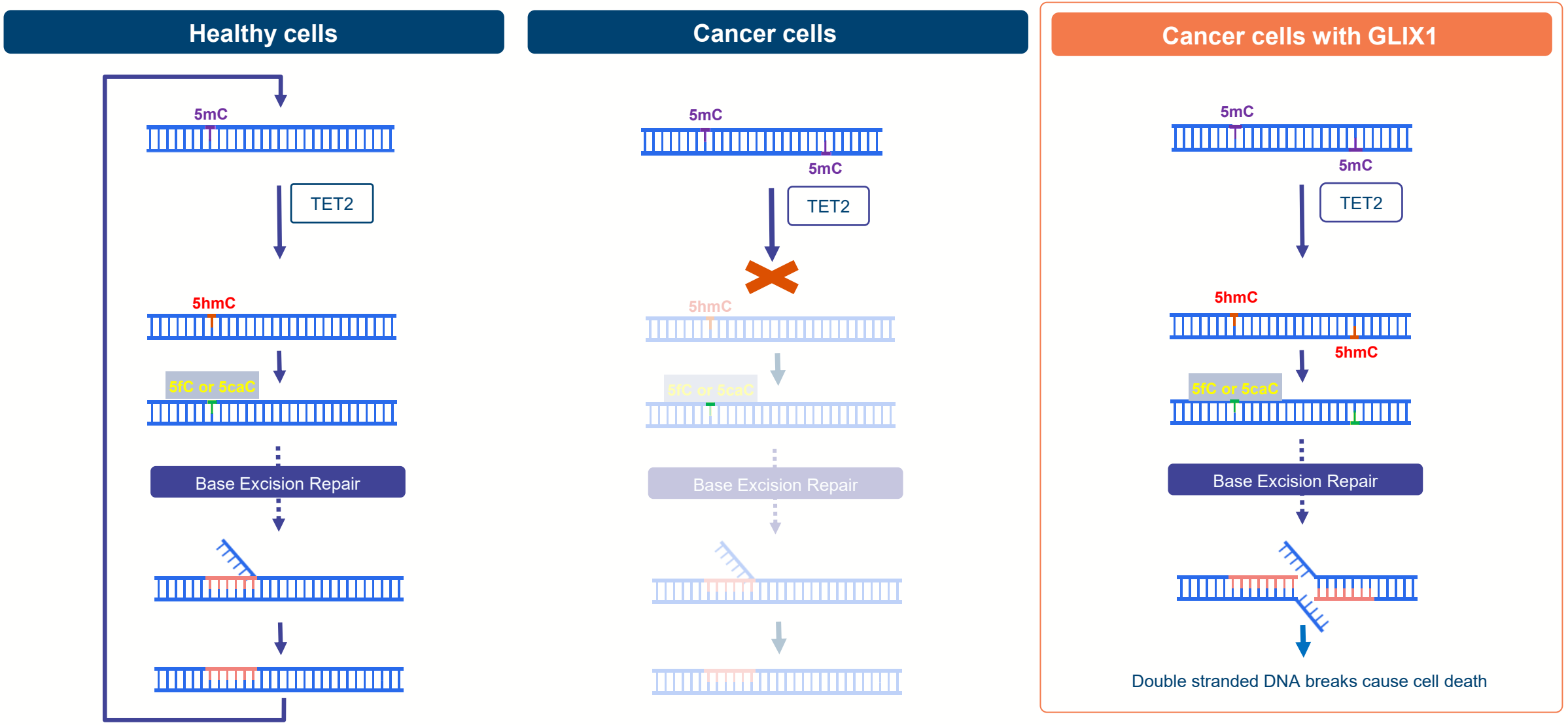
GLIX1 restores TET2 activity in cancer cells



1.Jinrong Zhu et al, Cancer Bio Med, 2023, 21(2):111–116; 2. Wenxin Da et al, Clinical and Translational Oncology, 2024, 26:2156–2165; 3. Johnson KC et al, Nature Communications, 2016, 7, 13177; 4. Luisa Cimmino et al, Cell, 2017, 170(6):1079-1095

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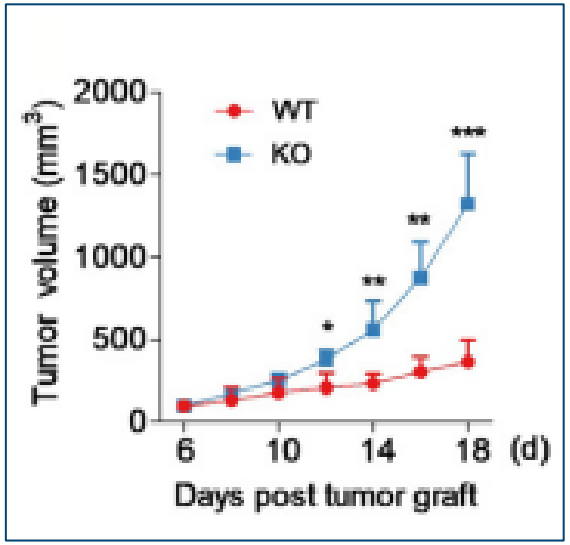
GLIX1-driven TET2 activity overwhelms DNA repair in cancer cells



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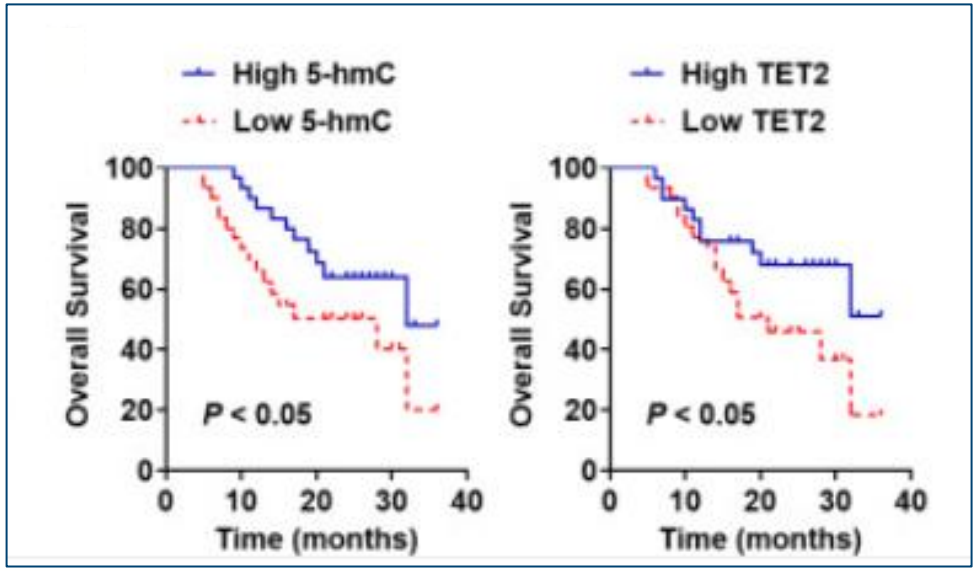
TET2 as a tumor suppressor

- **TET2 knockdown** results in tumor growth[1]
- TET2 knockdown significantly promotes, while TET2 overexpression inhibits, the proliferation, migration and invasion of cancer cell lines[2].



Deletion of Tet2 in C57BL/6 mice promotes syngeneic tumor growth. Tumor-bearing mice and tumors after subcutaneous injection of Hepa1-6 hepatoma cells into WT and Tet2 −/− (KO) mice. Mean tumor volumes in WT and Tet2-/- mice (n = 6). Reproduced from [1].

- **Low TET2** expression and Low 5-hmC levels are correlated with **shorter overall survival in several cancers**.[2,3,4,5,7,9]
- **Restoring TET2 expression** was shown to reduced tumorigenic activity [6,8,]



The correlation between the 5-hmC and TET2 level with the prognosis of lung cancer patients. Reproduced from [2].

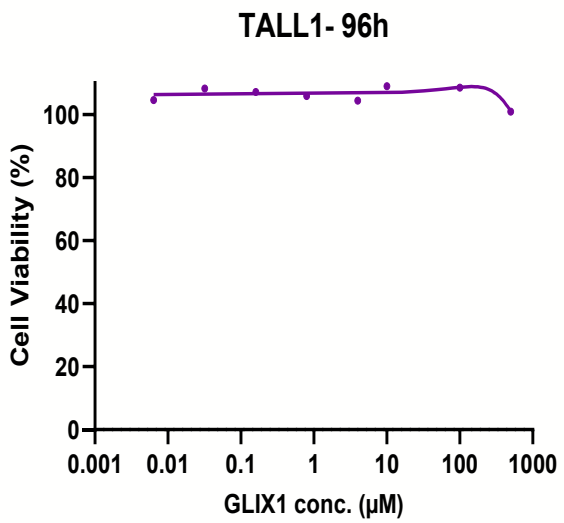
1) Shuangqi et al, 2020 2) Cheng et al, 2023 3) Huang et al. 2025 4) Zhang et al., 2015 5) Tucker et al., 2018 6) Cimmino et al., 2017 7) Lopez-Bertoni H et al., 2022. 8) Gracia et al., 2018 9) Chen S, et al., 2020.

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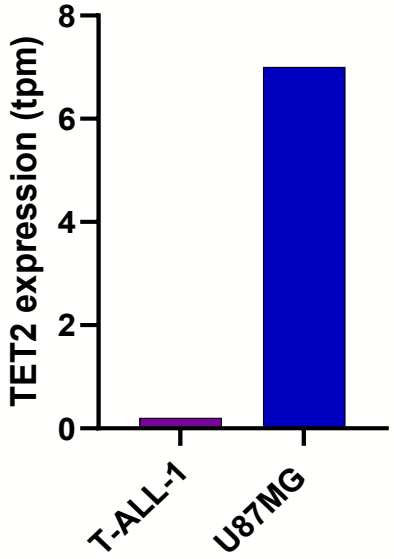
GLIX1 is active only in the presence of TET2

TET2 deficient cell lines (TALL-1) are resistant to GLIX1 treatment

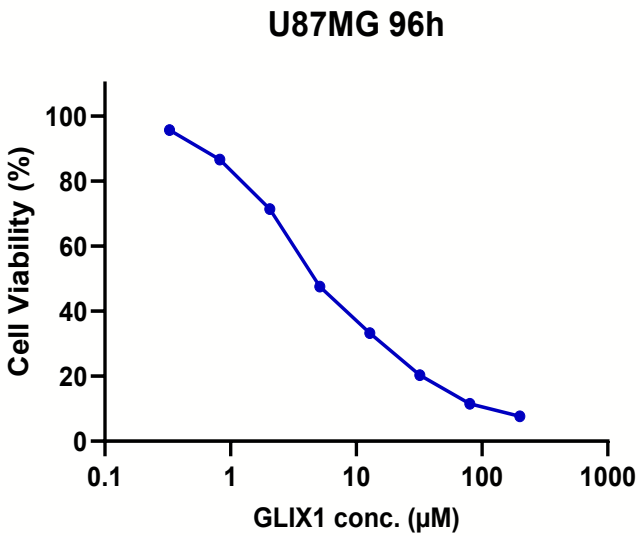
TET2 deficient cell line



TET2 expression



TET2 expressing cell line

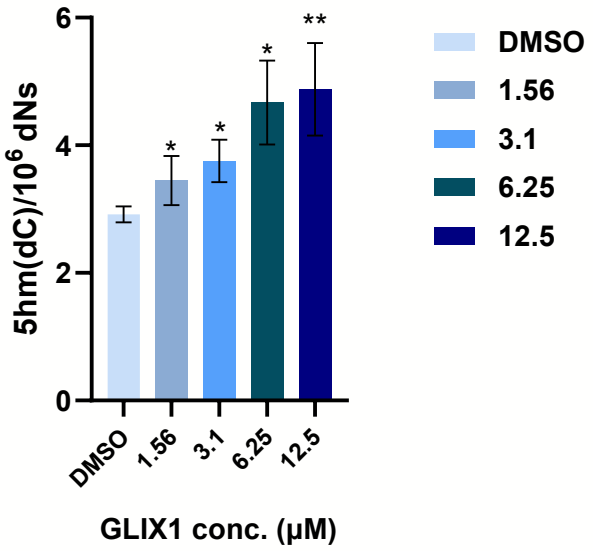


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Pre-clinical studies confirm MOA

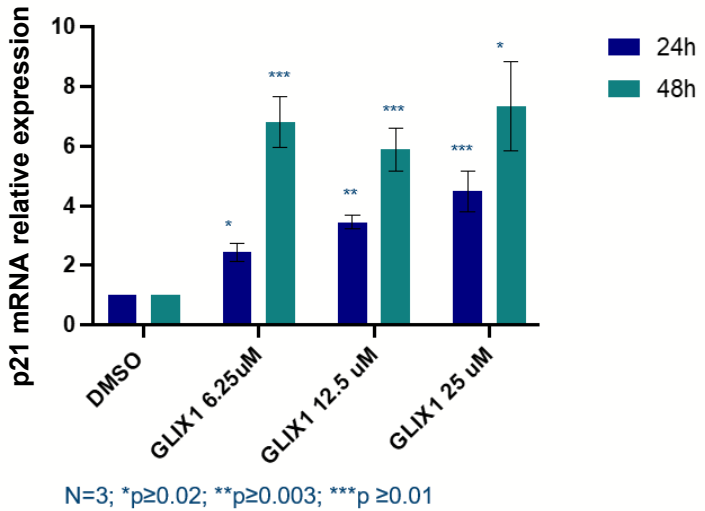
Studies confirm increase in 5hmC levels after GLIX1 treatment, leading to DNA damage and cancer cell death

GLIX1 enhances TET2 activity resulting in increased TET2 product (5hmC)



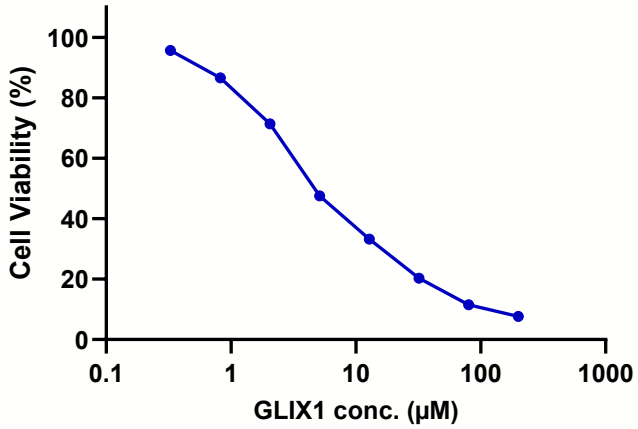
Tissue cultured analysis confirms increased **5hmC** levels after GLIX1 treatment, with dose response

5hmC is processed to DNA damage, overwhelming the repair capacity of the cancer cell



Restoring the **5hmC** levels in cancer cells leads to DNA damage

DNA damage cause cancer cell death



Treatment with GLIX1 results in cancer cell death

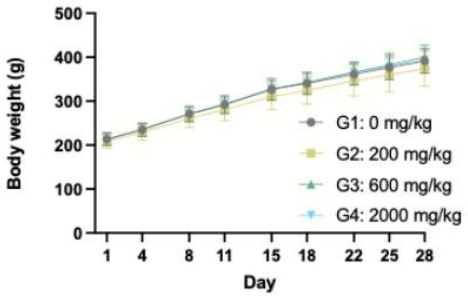
All assessments were conducted in the U87MG glioblastoma cell line.

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GLIX1 safety

GLP tox studies in rats and dogs demonstrate safety at high doses

Rats



In-life Phase Parameters

No mortalities and no test-item clinical signs

No ocular abnormalities

No neurobehavioral changes

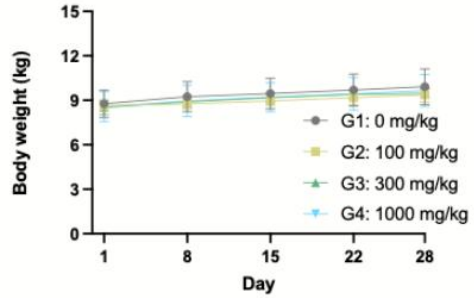
No changes in body weight and food consumption

- Poorly formed feces (dogs 1000 mg/kg) - non-adverse as not associated with decreased BW and FC

Safety Pharmacology

No effect on CNS, cardiovascular or respiratory system

Dogs



No body weight change over 28 days

Clinical Pathology

No changes in hematology

No changes in coagulation

No changes in clinical chemistry

No changes in urinalysis

Anatomic Pathology

No changes in terminal fasting body and organ weight

No test item-related findings in gross pathology

No target organ toxicity identified and MTD not reached up to the highest doses tested

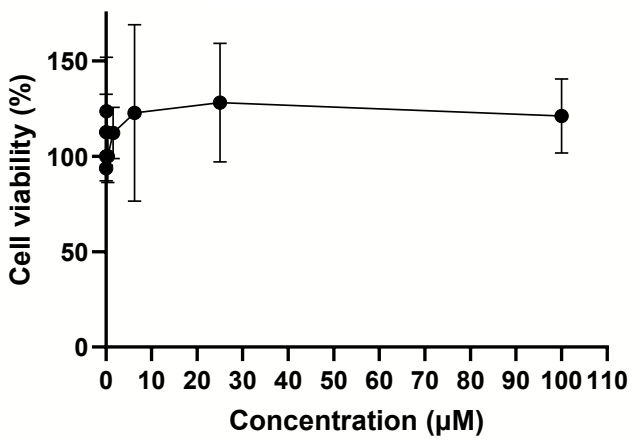
2000 mg/kg in rats (Human equivalent dose (HED) ~ 20000 mg/day) and 1000 mg/kg in dogs (HED ~ 30000 mg/day)

Note: PK saturation starting at 200 mg/kg in rats (HED ~2000 mg/day) and 100 mg/kg in dogs (HED ~3000 mg/day)

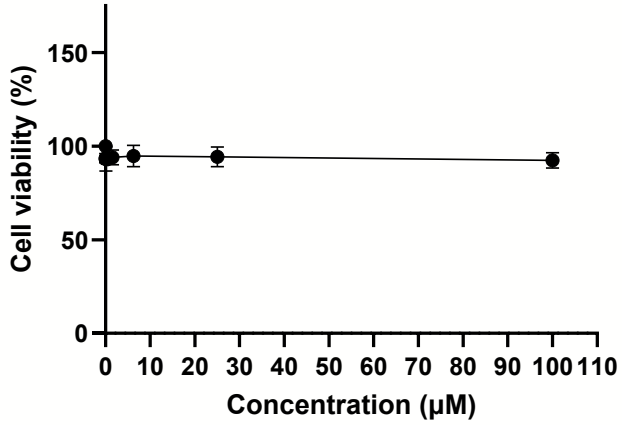
GLIX1 has no toxicity in normal cells

Lack of cytotoxicity demonstrated in human peripheral blood mononuclear cells (PBMCs)

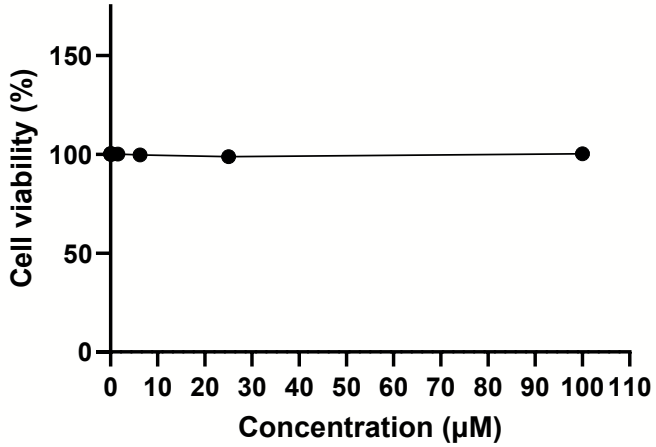
PBMC A



PBMC_C



PBMC_M

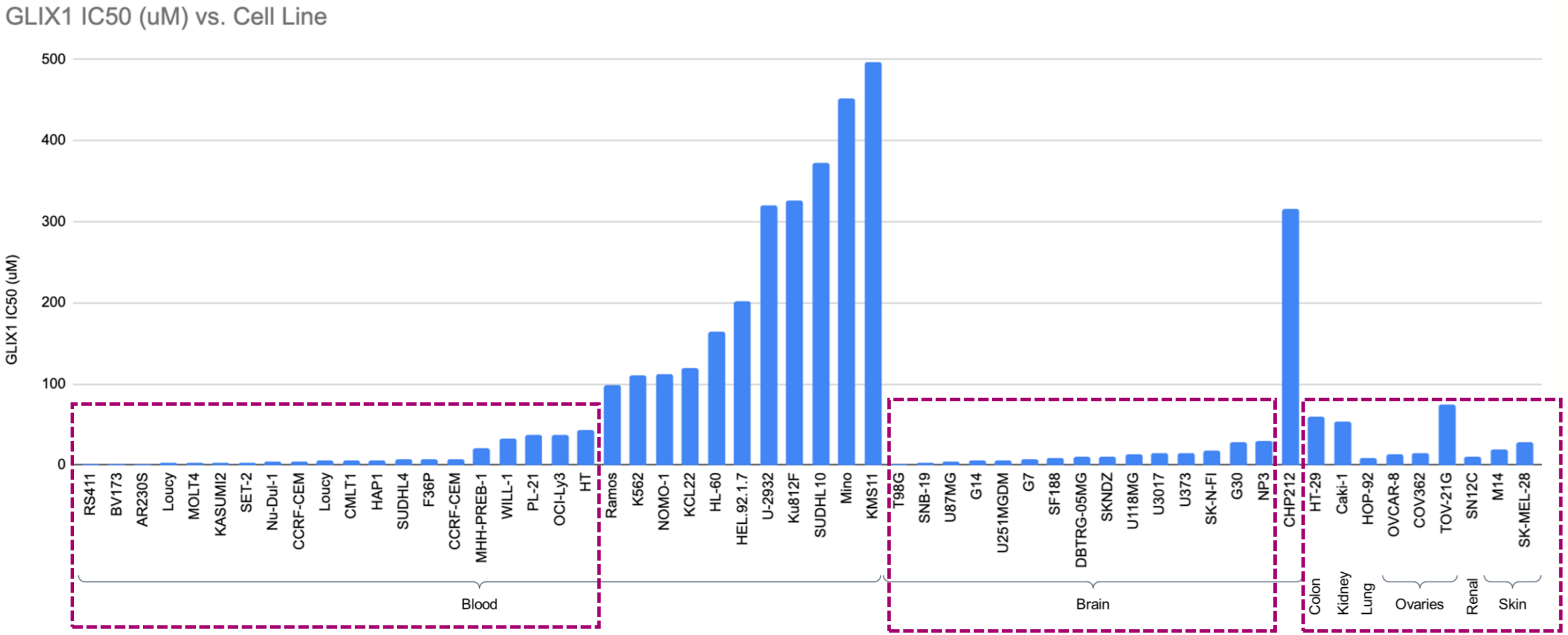


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GLIX1 efficacy

GLIX1 shows efficacy in numerous different cancer cell lines

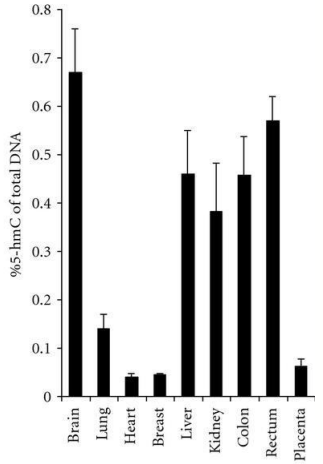
High potency of GLIX1 (reflected by low IC50) demonstrated in a number of different cancer cell lines



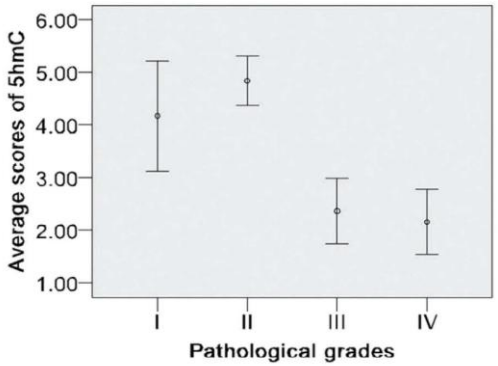
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Glioblastoma (GBM) chosen as initial indication supported by strong rationale

5hmC levels are significantly reduced in GBM

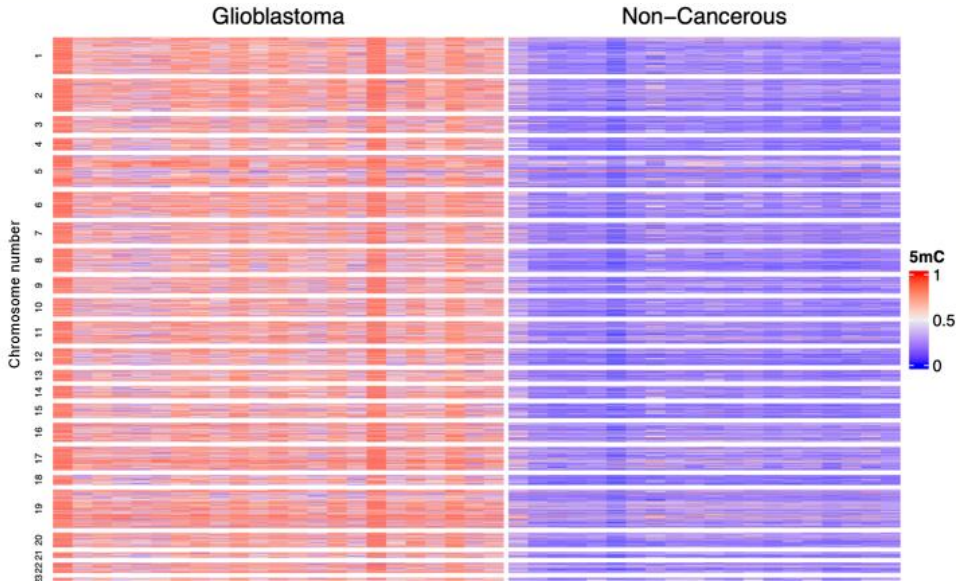


Healthy human brain tissue has high levels of 5hmC.[1]



Higher grade gliomas, have lower 5hmC levels.[2]

GBM has significantly more 5mC in close proximity in the genome vs normal cell lines



Healthy **Cancer**

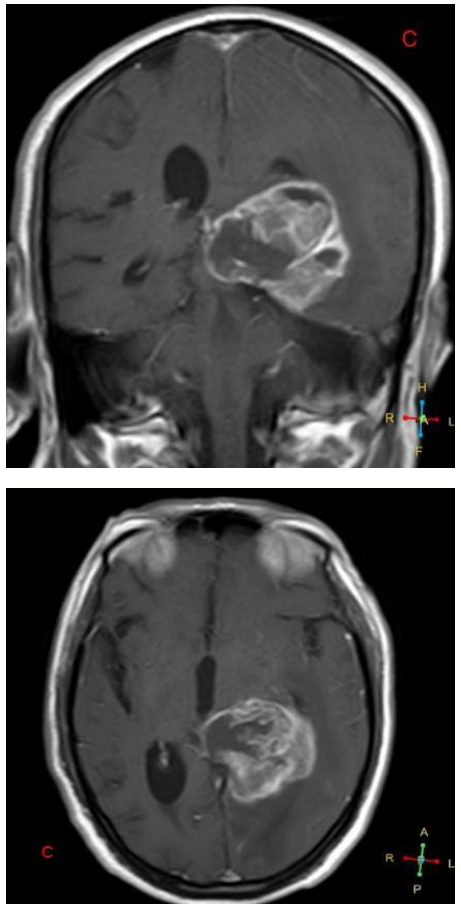
5hmC Level → 5mC Level

1- Journal of Nucleic Acids, 2011 Jun 9; 870726. 2- Nature, 2016 Feb 11;6:20882

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GBM is one of most challenging cancers with no effective treatment options

- GBM is the **most common** and most aggressive primary brain tumor
- Poor prognosis with **median survival of 12–18 months**[1,2]
- SoC for newly diagnosed GBM is surgery + radiotherapy + Temozolomide (TMZ)[3] established in 2005
  - Demonstrated marginal increase in OS:14.6 months with TMZ vs. 12.1 months without TMZ
  - **Mainly improves outcome for patients with methylated MGMT promoter** (less than half of all GBM patients)
- **No established SoC for recurrent GBM**

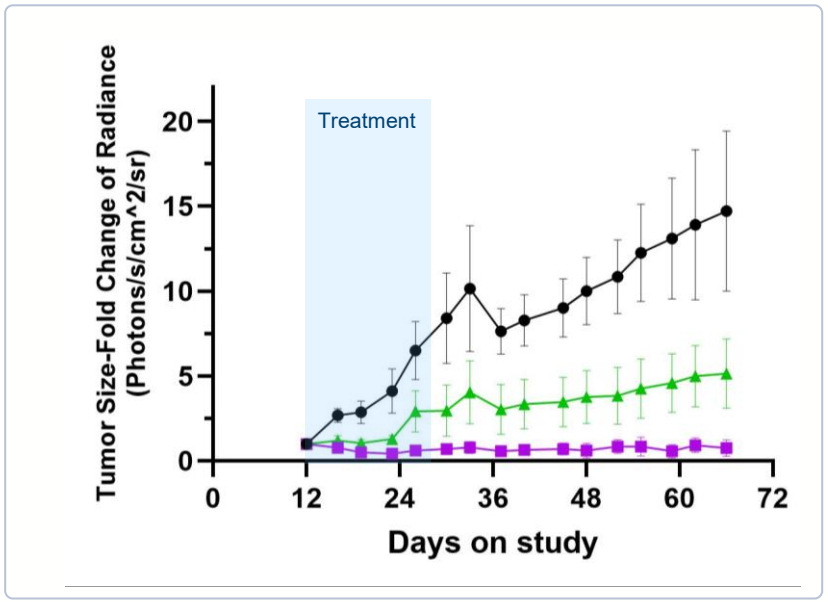


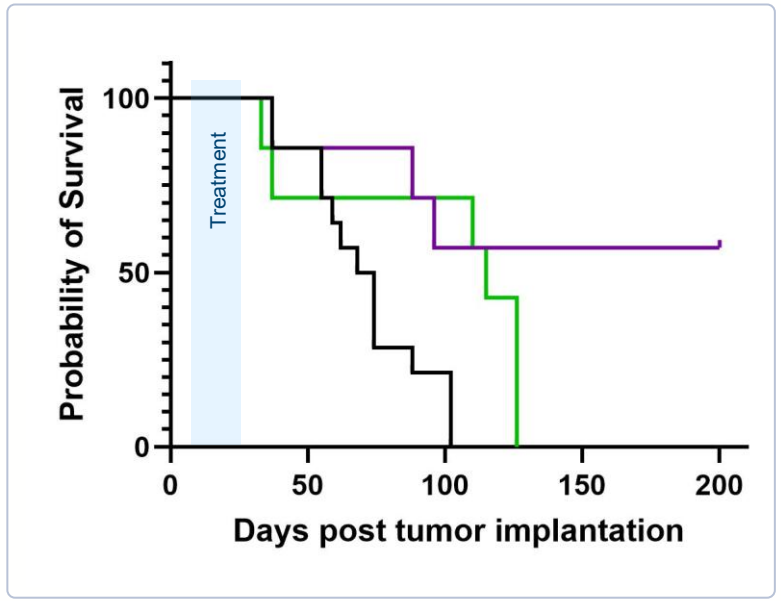
1. Biomedicines, 2021 Mar 22;9(3):324
2. SEER 2017
3. N Engl J Med 2005;352:987-996

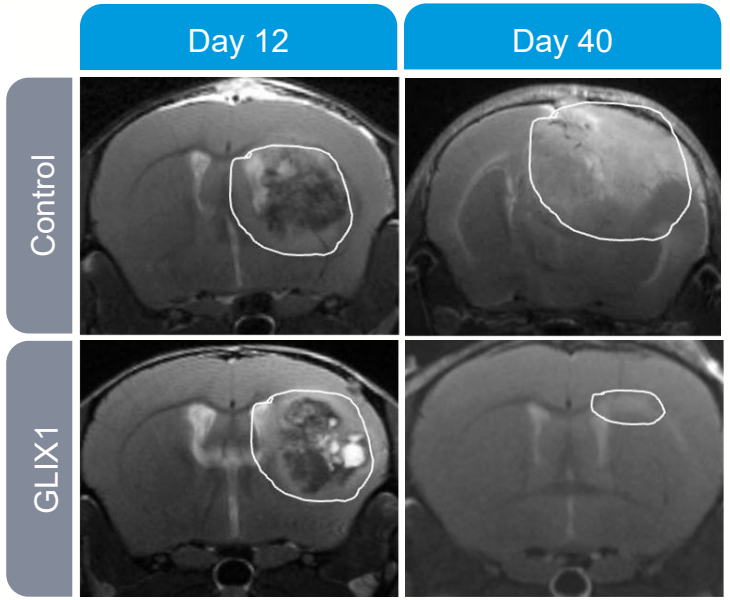
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GLIX1 demonstrated anti-tumor activity and prolonged survival in initial orthotopic GBM model

Potent anti-tumor activity in orthotopic GBM xenograft model







Control N=14 TMZ 1.5mg/kg QD N=7 GLIX1 1000mg/kg BID N=7

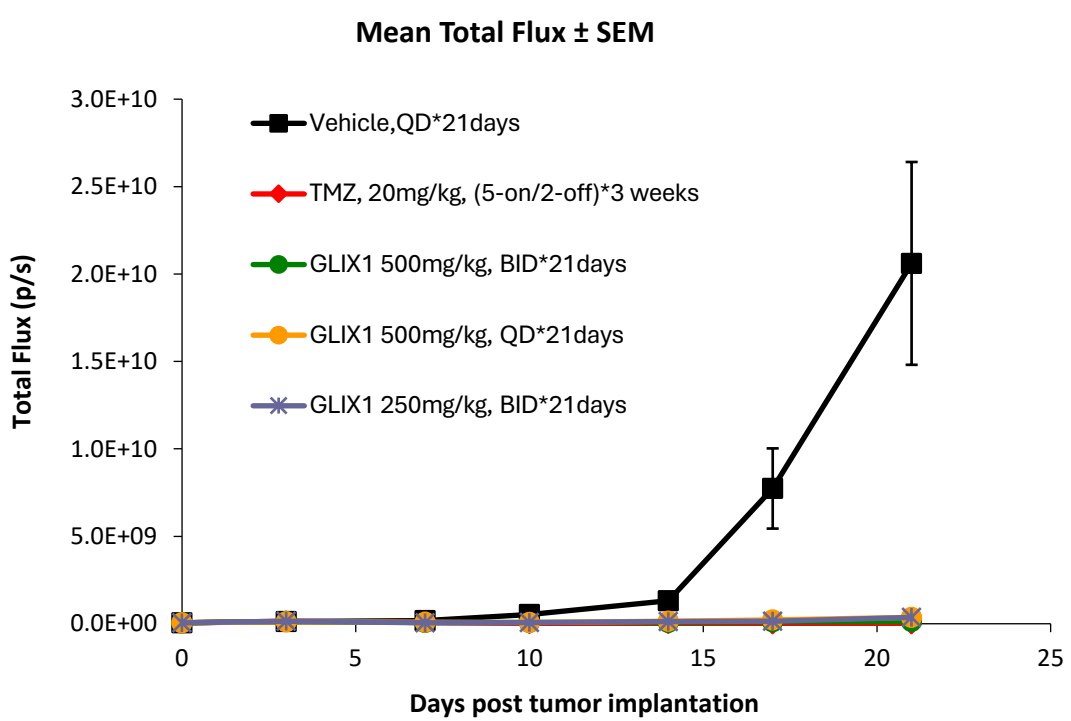
Stanford MEDICINE

Nude mice inoculated with SNB-19-Luc cells; treatment administrated orally, started at day 12 for 2 weeks for GLIX1 and for 6 days for TMZ

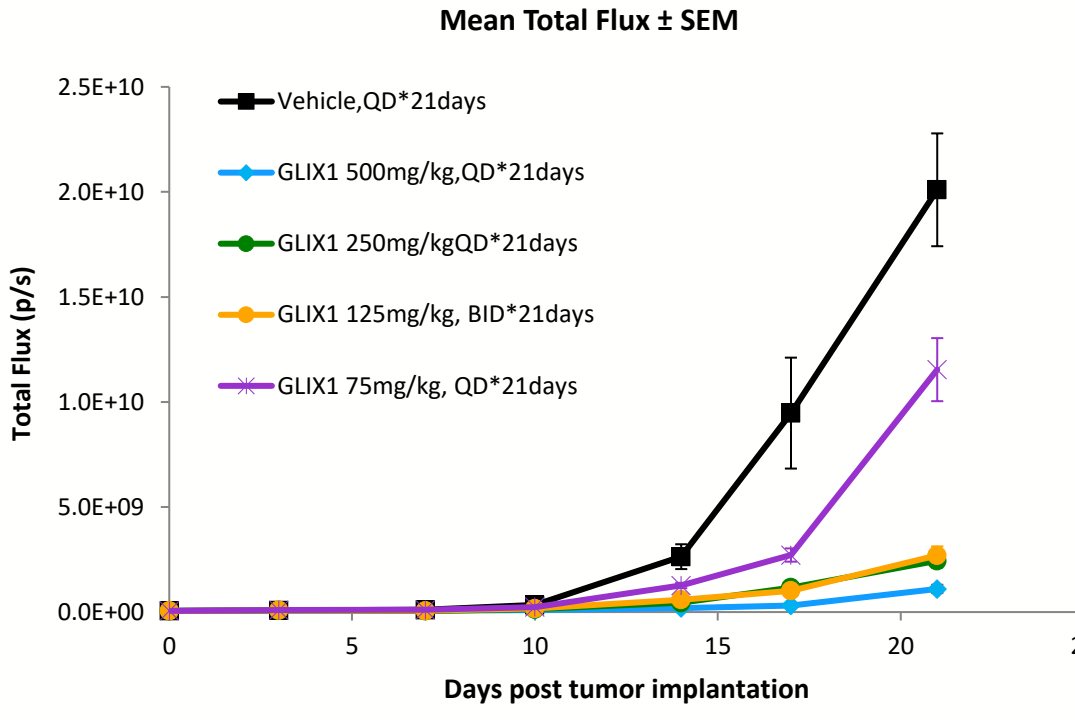
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GLIX1 demonstrated robust efficacy in dose-dependent manner in additional orthotopic GBM models

Strong anti tumor activity even at the lowest dose tested in orthotopic GBM xenograft model



Nude mice inoculated with U87-MG-Luc cells, N=8/group



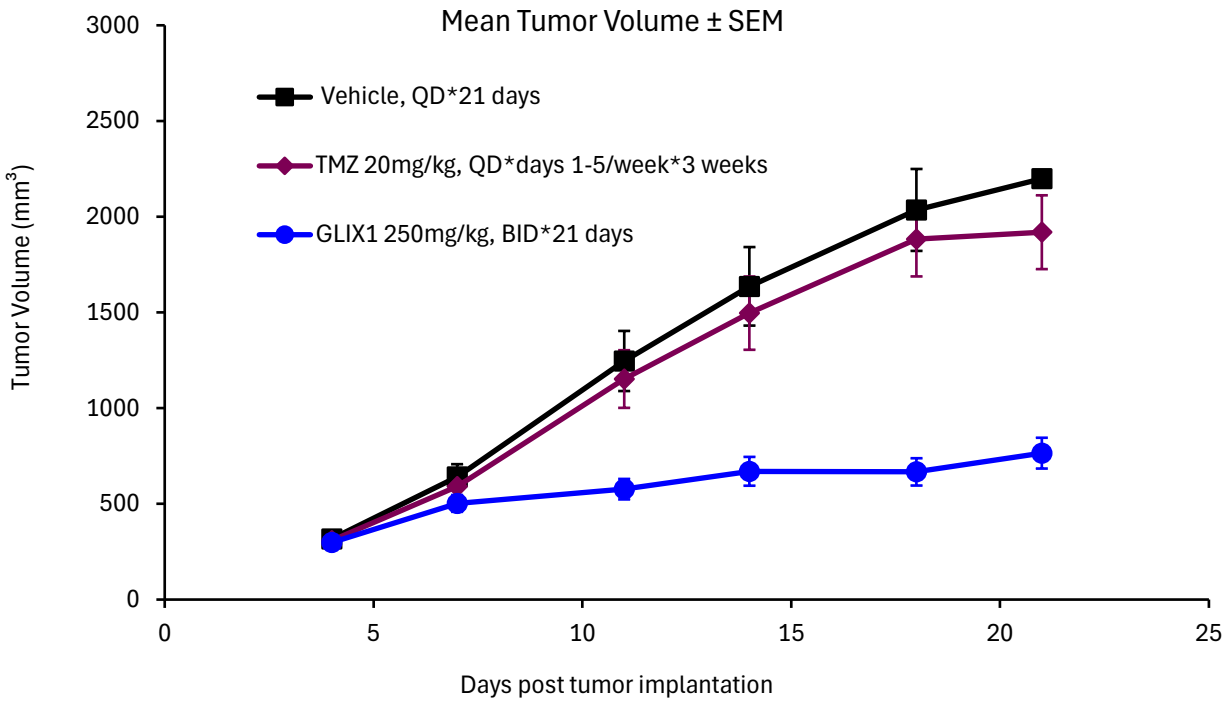
Nude mice inoculated with U87-MG-Luc cells, N=8/group

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GLIX1 also demonstrated potent anti-tumor activity in TMZ-resistant patient-derived xenograft (PDX) model

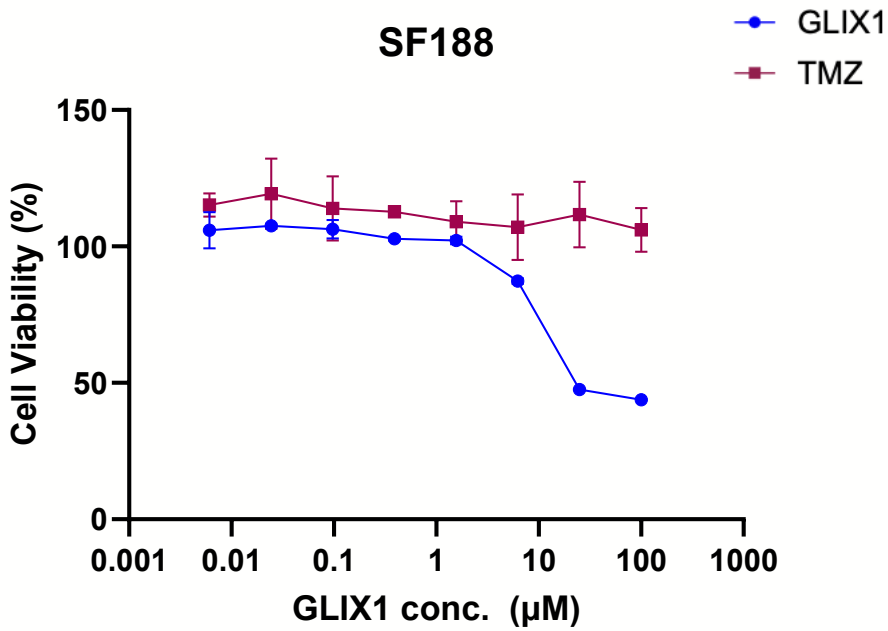
Efficacy in TMZ-resistant model supports GLIX1's potential to treat a broad range of GBM patients, including patients with unmethylated MGMT promoter status (more than half of all GBM patients)

Significant efficacy in TMZ resistant PDX GBM model



Nude mice implanted subcutaneously with (BN2276) Patient Derived tissue, N=8/group

Cytotoxicity demonstrated in several TMZ-resistant cell lines



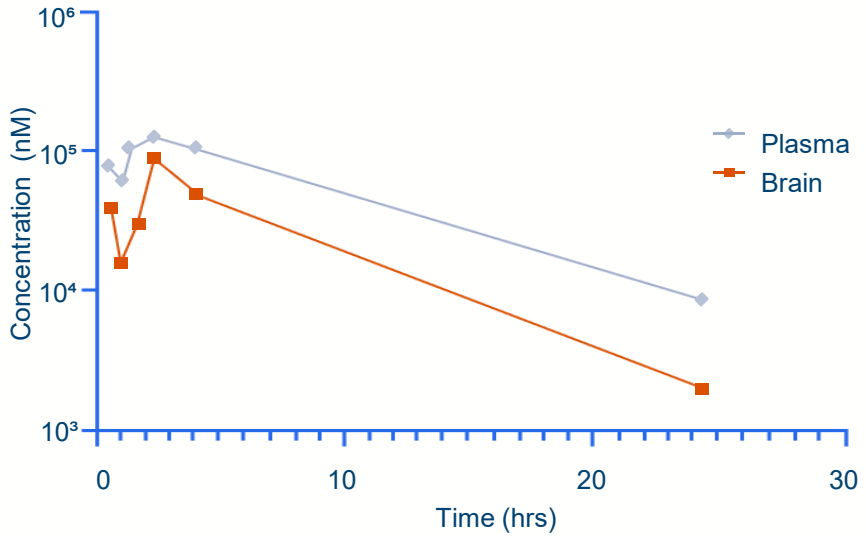
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GLIX1 addresses key challenges in GBM drug development

GLIX1 demonstrates BBB penetration

BBB penetration is the first feature to impair efficacy and a major challenge to chemotherapies and immunotherapies

Strong **blood-brain-barrier penetration** in healthy mice following 1000 mg/kg PO



Group	AUC (nM*Hours)	Cmax (nM)	Tmax (Hours)	T1/2 (Hours)
Plasma	1200000	132000	2	5
Brain	500000	90200	2	4

GLIX1 enters the brain via **nucleoside transporters**

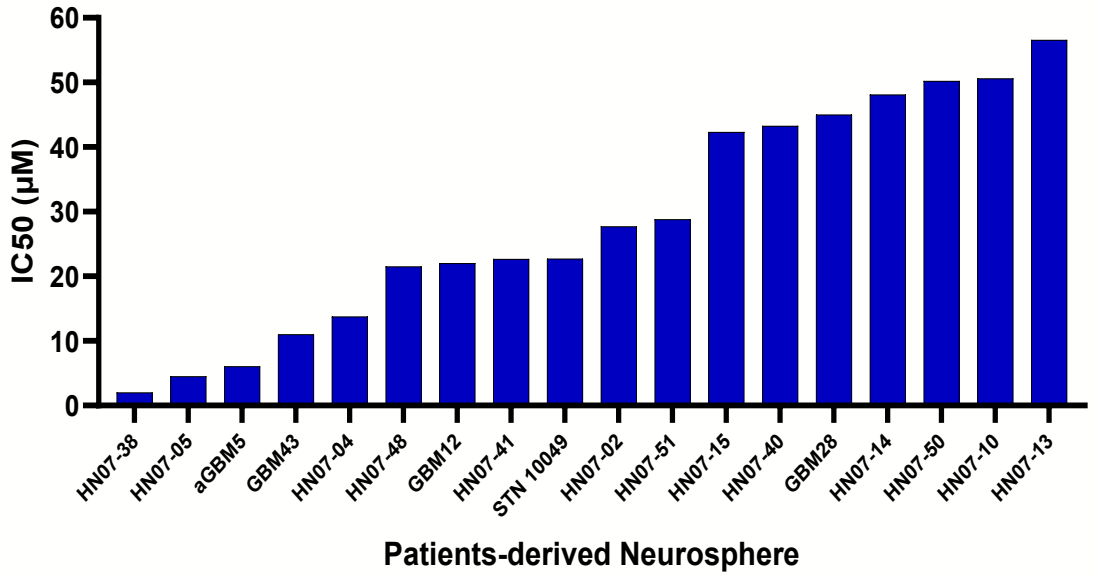
- GLIX1 is a substrate for CNT1, CNT3, ENT1 and ENT2 transporters
- GLIX1 is not a substrate for the efflux transporters P-gp and BCRP, which are expressed abundantly at the blood–brain interface, function as gatekeepers to actively pump out xenobiotics

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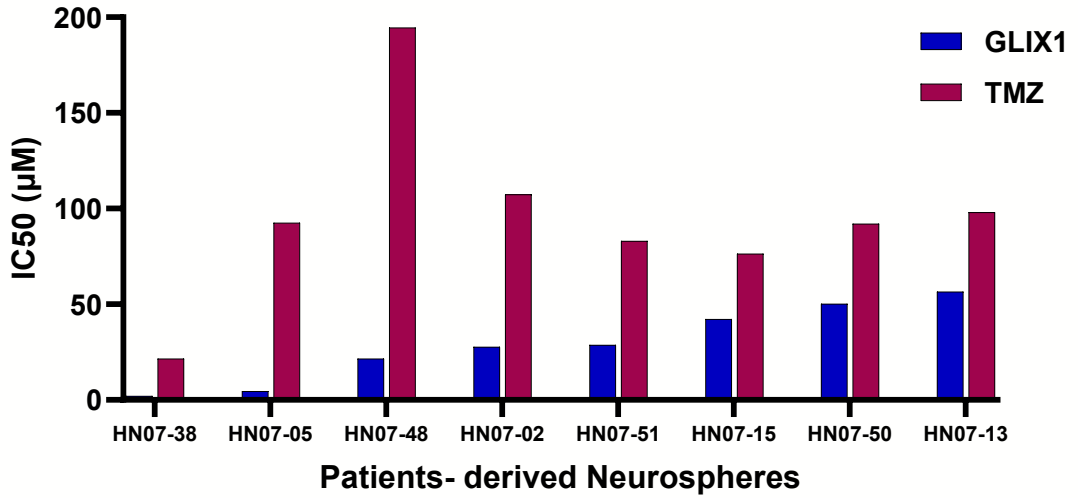
GLIX1 shows efficacy in patient-derived neurospheres (glioma stem cells)

GLIX1 has potential to target cancer stem cells that contribute to tumor development, heterogeneity, drug resistance and cancer recurrence

Potency of GLIX1 (reflected by IC50) demonstrated in patient-derived neurospheres; at concentrations applicable to humans



Head-to-head comparison of GLIX1 and TMZ in patient-derived neurospheres suggests that GLIX1 may have a superior ability to target and eliminate GSCs



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GLIX1's potential to address common reasons for failure in GBM trials

Common reason for failure	GLIX1 potential to overcome failure
Lack of BBB penetration	✓ Demonstrated BBB penetration ✓ Crosses the BBB via nucleoside transporters
Complex tumor biology: – Cancer stem cells contribute to tumor development, drug resistance and recurrence – Heterogenous nature	✓ Cytotoxic to patient-derived neurospheres (glioma stem cells) ✓ Efficacious in TMZ-resistant PDX model ✓ Does not target specific mutation
Failures with immunotherapy	✓ Does not rely on the immune system
Clinical trial design: – Overly restrictive eligibility criteria hinder participation and generalizability – Single center phase 1 studies	✓ Protocol is not restrictive (e.g., it allows variation of prior treatment regimens used in clinical practice) ✓ Clinical trial led by Prof. Stupp and carried out at 3 selected world-class sites
Toxicities restrict treatment duration and combination treatment	✓ No toxicities in animal tox studies

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GLIX1 clinical development plan

GLIX1 Phase 1/2 clinical trial design

Phase 1

Recurrent and progressive GBM

Open label, dose escalation (BOIN)

Maximum tolerated dose (MTD) and **recommended dose** based on safety, PK/PD and preliminary efficacy

N = up to 30

Timeline: ~1.5 years

3 top US centers participating in Phase 1 of the trial

Study Principal Investigators



Ditte Primdahl, MD
Assistant Professor of Neuro-Oncology
Northwestern Medicine



Roger Stupp, MD
Chief of Neuro-Oncology
Northwestern Medicine

Phase 2

Potential population cohorts:

- GBM – **newly diagnosed and/or recurrent**
- Additional cancers – with/without PARPi combination

Open label and/or **controlled**

Dose optimization

Efficacy based on response, PFS and OS

PD markers

N: up to 20 per arm

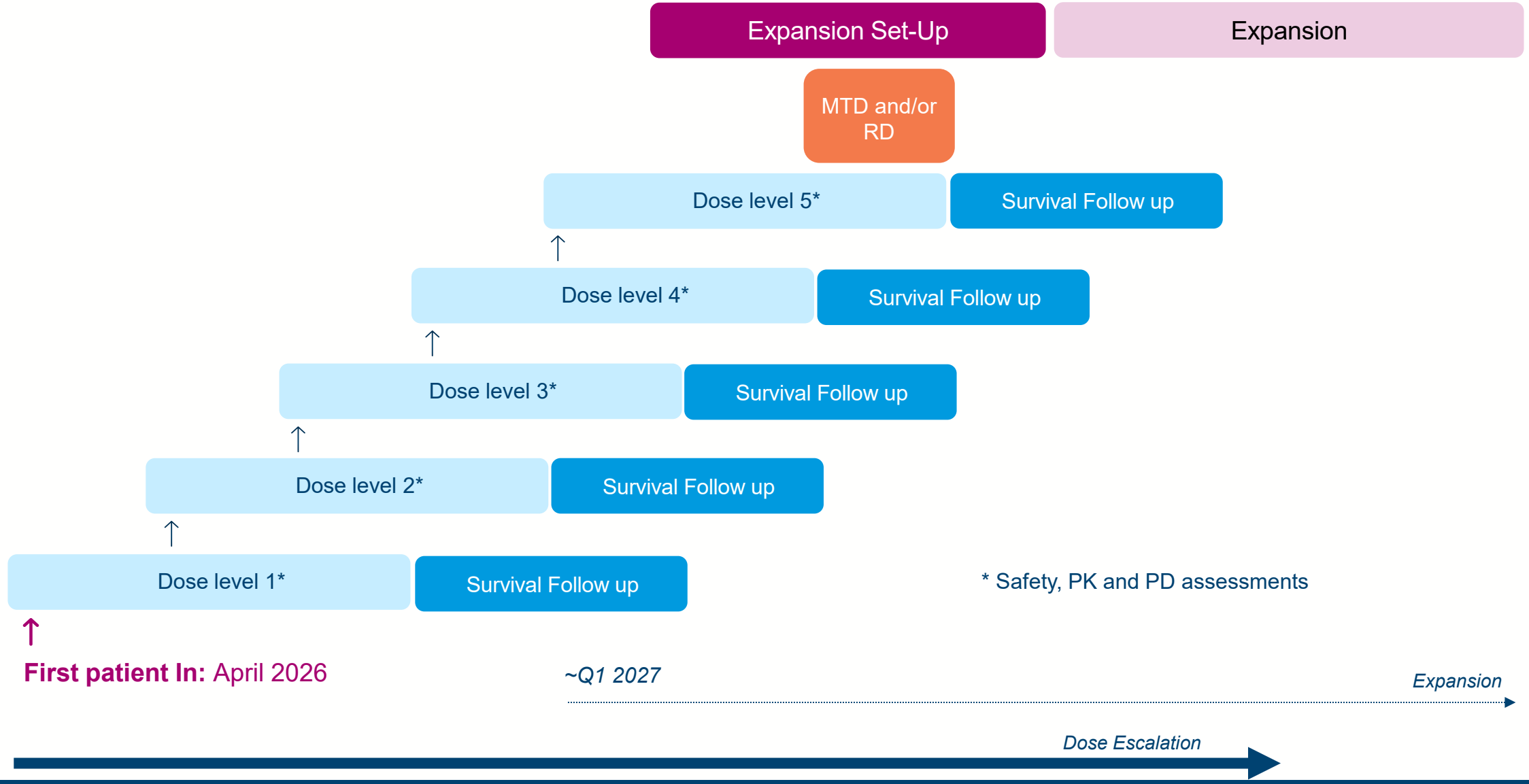
Planned timeline: ~2.5 years

Additional top centers in US and Europe likely to be added

Phase 1/2 clinical trial initiated in Q1 2026 (NCT07464925)

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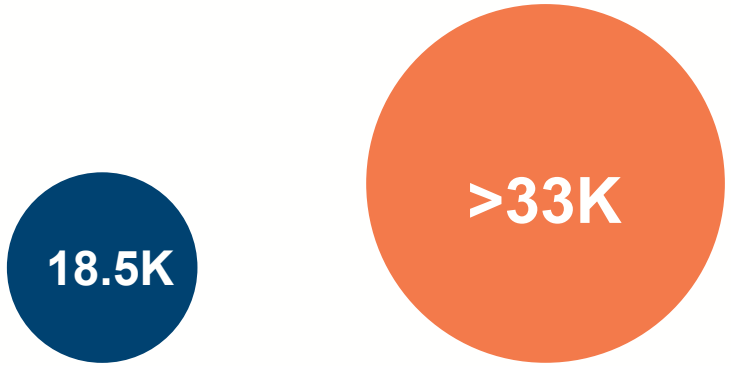
Phase 1/2 dose escalation plan



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GLIX1 represents a multi-billion-dollar market opportunity in GBM

Estimated diagnosed annual incidence in 2030:



in US

in US and Europe

Estimated total addressable market in 2030 for US + 4EU major markets + UK:



Data on Temozolomide
(current standard of care)

2.5 months
improvement in overall survival

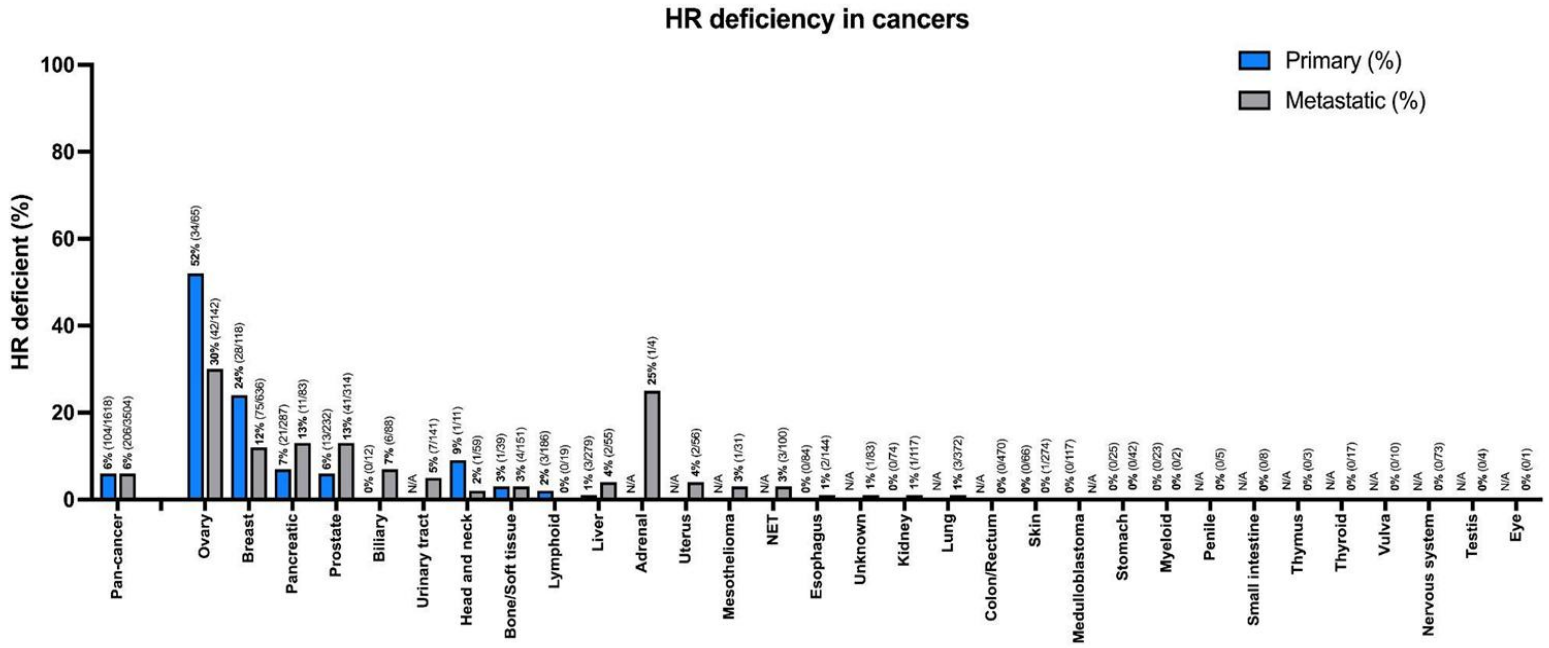
50-75%
of patients do not respond

$1B/year
peak sales while on patent

GLIX1 and PARP inhibitors (PARPi) – potential for strong synergy

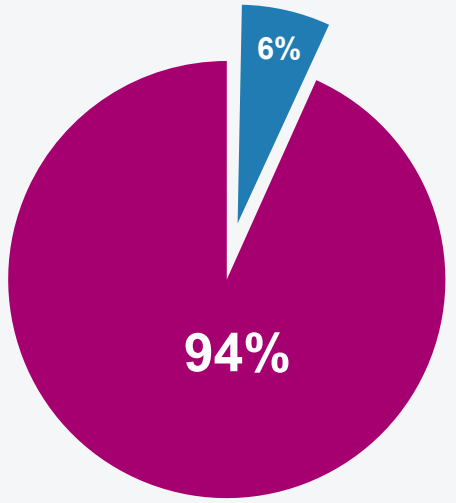
GLIX1 substantially expands the range of cancers and patient populations that can be treated with PARP inhibitors by facilitating the formation of single-stranded DNA breaks

Homologous recombination (HR) Deficiency in Cancers



PARP inhibitors are efficacious in HR-deficient cancers[1, 2]

HR-deficient cancers
(treatable with PARPi)



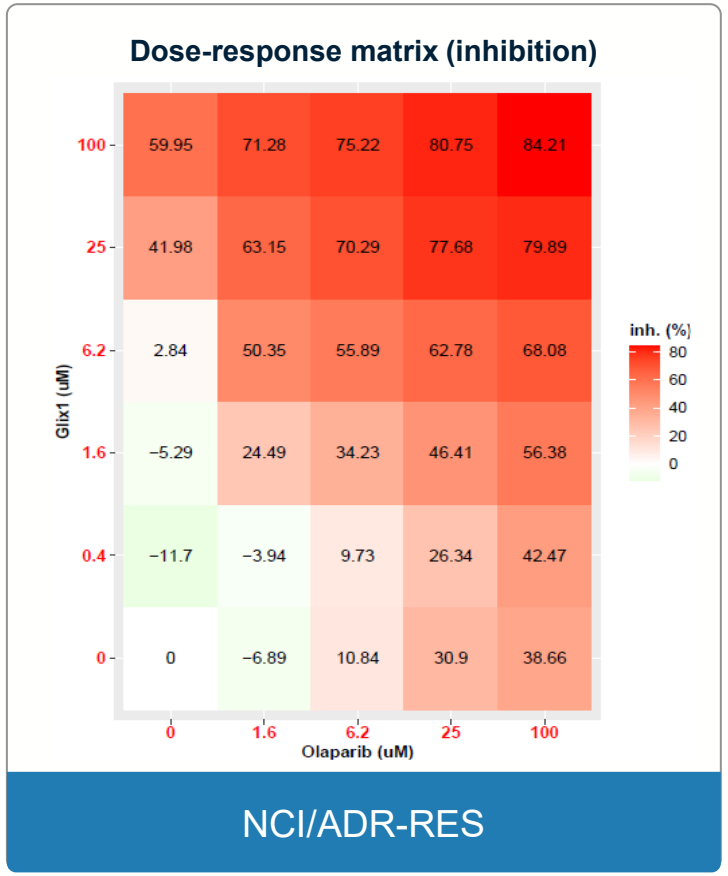
HR-proficient cancers
(potentially treatable with PARPi +GLIX1)

1- Luan Nguyen et al, Nature Communications (2020) 11:5584
2 - HR-deficient cancers are tumors whose cells cannot properly repair DNA double-strand breaks because the homologous recombination (HR) repair pathway is impaired.

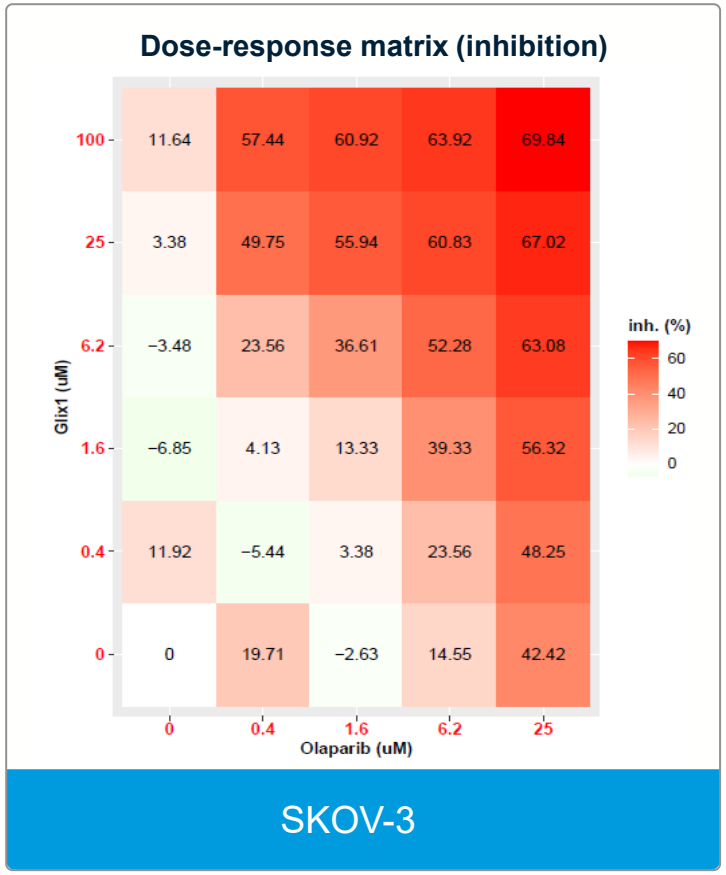
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GLIX1 shows strong synergy with PARPi in HR-proficient cancers

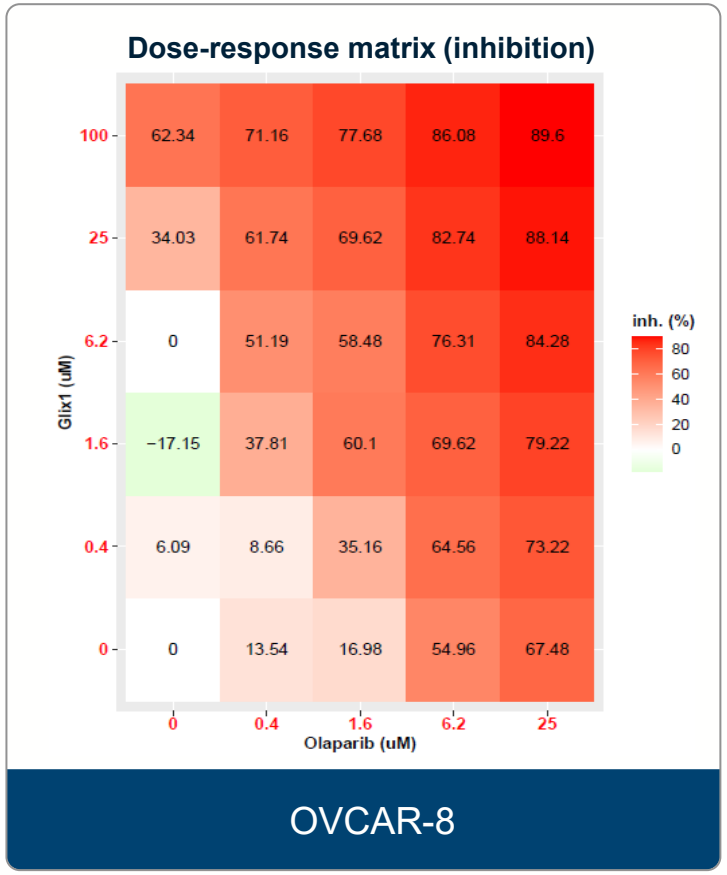
Synergy demonstrated in various HR proficient-ovarian cancer cell lines. Data support synthetic lethality between GLIX1 & PARPi indicating that GLIX1 removes the HRD requirement for PARPi activity



NCI/ADR-RES



SKOV-3



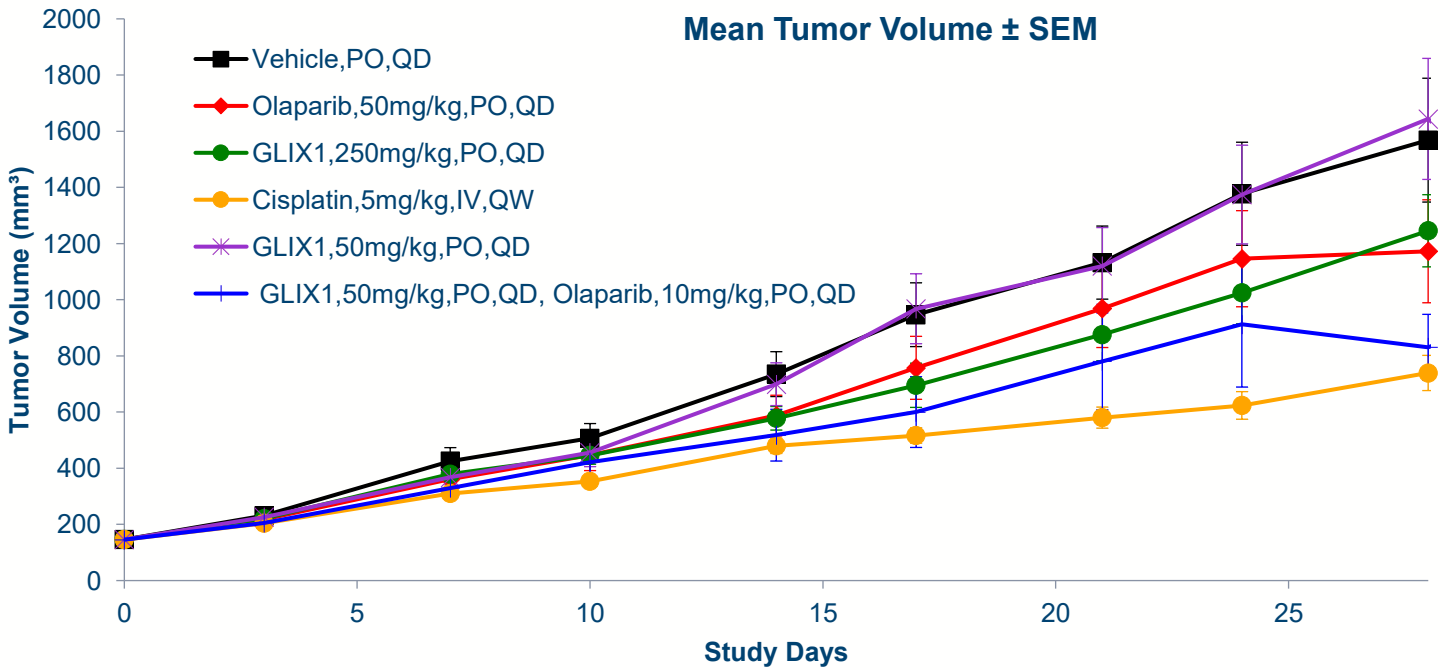
OVCAR-8

Synergy also demonstrated in different HR-proficient cancers and across PARPi

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Strong synergistic effect demonstrated between GLIX1 and PARPi in patient-derived xenograft (PDX) model in ovarian cancer

Results reinforce synthetic lethality between GLIX1 and PARPi, indicating that GLIX1 can broaden the use of PARPi in ovarian cancer



Nude mice implanted subcutaneously with (OV0250) Patient Derived tissue, N=8/group

- Synergy demonstrated in the combination arm of GLIX1/olaparib at low doses, with substantially better efficacy vs. control and vs. each drug at its optimal dose
- GLIX1/olaparib combination showed comparable efficacy to that seen with cisplatin

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GLIX1 has potential upside in broadening market for PARPi

Approved Indication	Approved Assets	Market Size*
Ovarian Cancer	Olaparib, Rucaparib, Niraparib	~$4B
Breast Cancer	Olaparib, Talazoparib	~$33B
Prostate Cancer	Olaparib, Rucaparib, Talazoparib	~$11B
Pancreatic Cancer	Olaparib	~$2.5B



PARPi Annual Sales:

Olaparib ~$5B

Niraparib ~$500M

Rucaparib ~$150M

Talazoparib ~$110M

- PARPi have limited efficacy in HR-proficient cancers.
- Combining PARPi and GLIX1 may expand the patient population within currently approved indications
- Potential to also address other cancers that are highly HR-proficient

* Reflects market size for therapeutics, excludes surgery, diagnostics, supportive care, etc.; based on estimates and assumptions from Global Market Insights, Global Growth Insights, Grand View Research, and Precedence Research

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Overview of GLIX1 IP

Global portfolio of patents, both issued and pending, that secure GLIX1's value



GLIX1 for use in treating cancer of the central nervous system, such as glioblastoma

Granted in the US, Europe, and 13 other countries; valid until at least 2040*

GLIX1 for use in treating any cancer in which CDA is not over-expressed**

Granted in the US and pending in Europe and other countries; valid until at least 2040*

GLIX1, combined with a PARP inhibitor, for use in treating HR-proficient cancers**

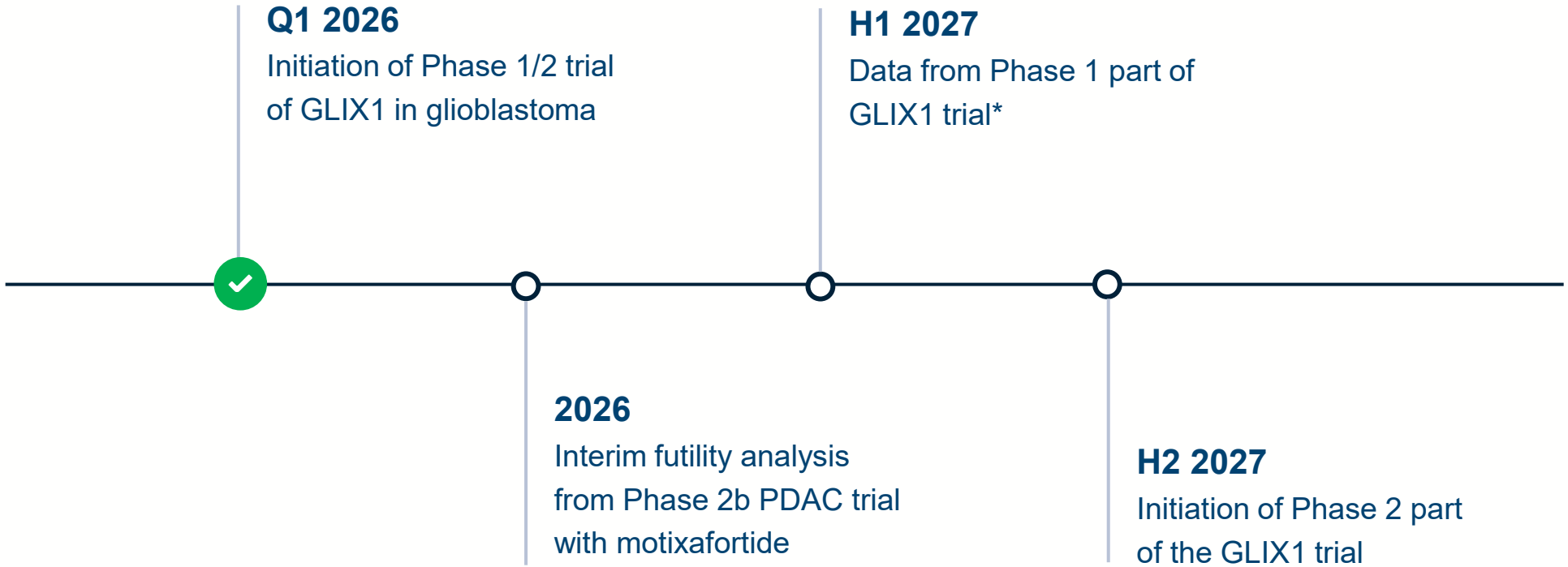
Pending int'l patent application; national-phase patents, if granted, will be valid until at least 2044*

* Not including possible patent term extension of up to the 5 years. ** Majority of cancers

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Upcoming milestones

Expected milestones in next 18-24 months



* In addition, as this trial is open-label, the data should be available on an ongoing basis; intention is to provide updates at medical conferences or other forums where possible

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