Driving cancer innovation forward

Corporate Presentation

April 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.

BioLineRx investor highlights

Leveraging successful track record of clinical and regulatory development to advance targeted therapies for cancers with high unmet needs

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

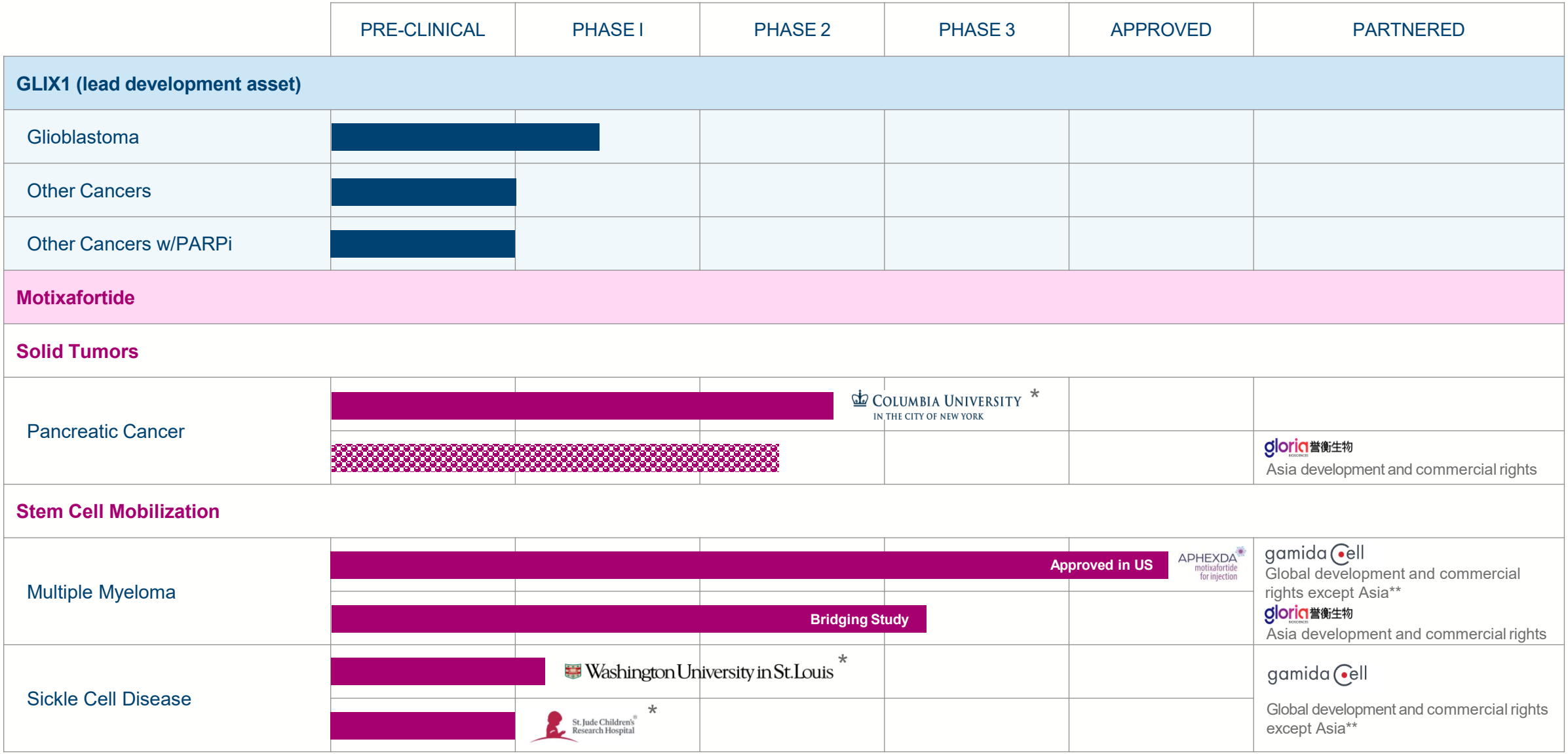
- Novel MOA focused on DNA damage repair
- Potential for strong synergy demonstrated in combination with PARP inhibitors
- Excellent blood-brain barrier penetration
- Initial development in glioblastoma (GBM), followed by other cancers
- Phase 1/2a study in GBM initiated, with part 1 dose escalation 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

Pipeline assets

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



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

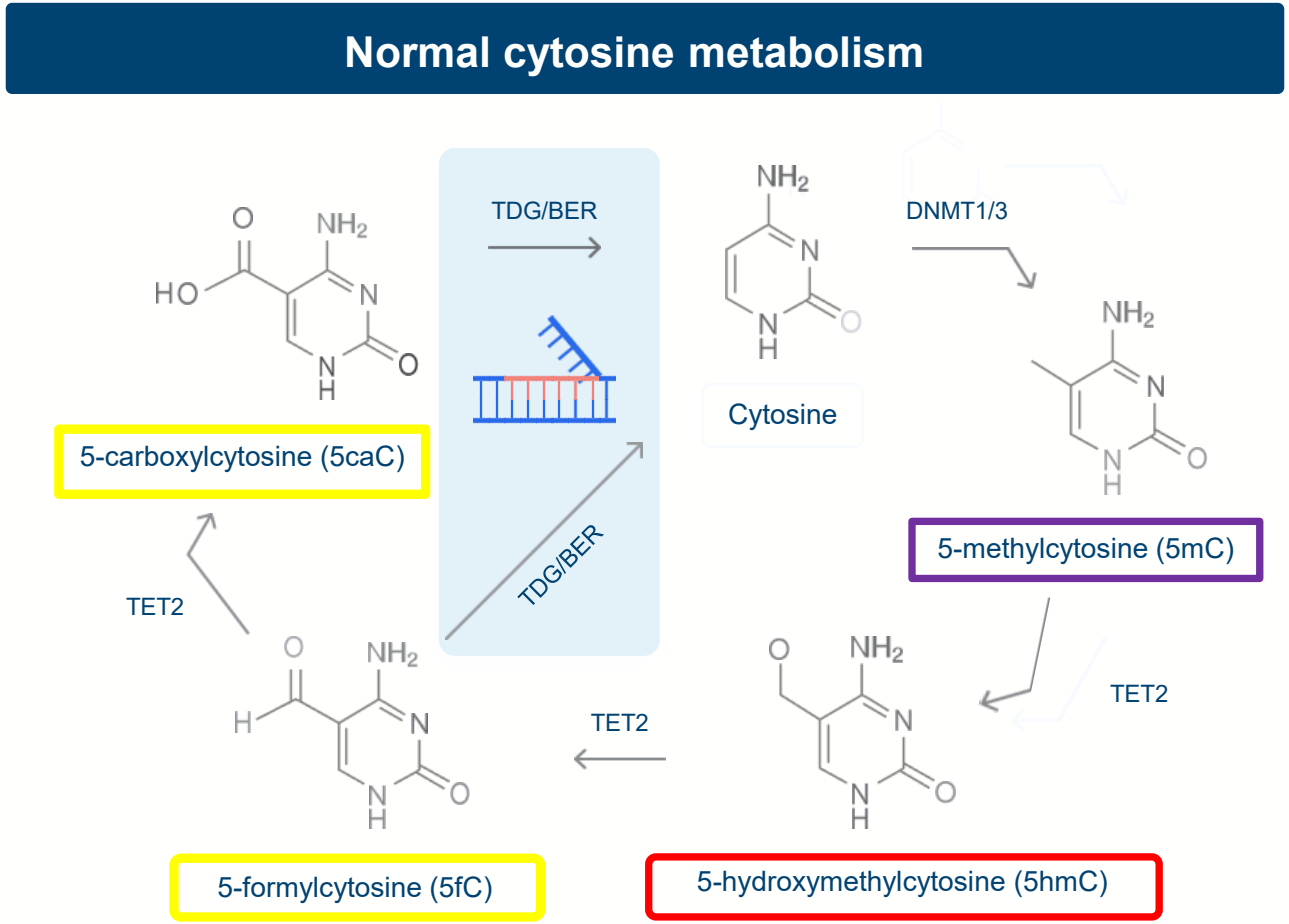
Glioblastoma, the first target indication, expected to provide data to support development in CNS and additional cancers

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

- 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

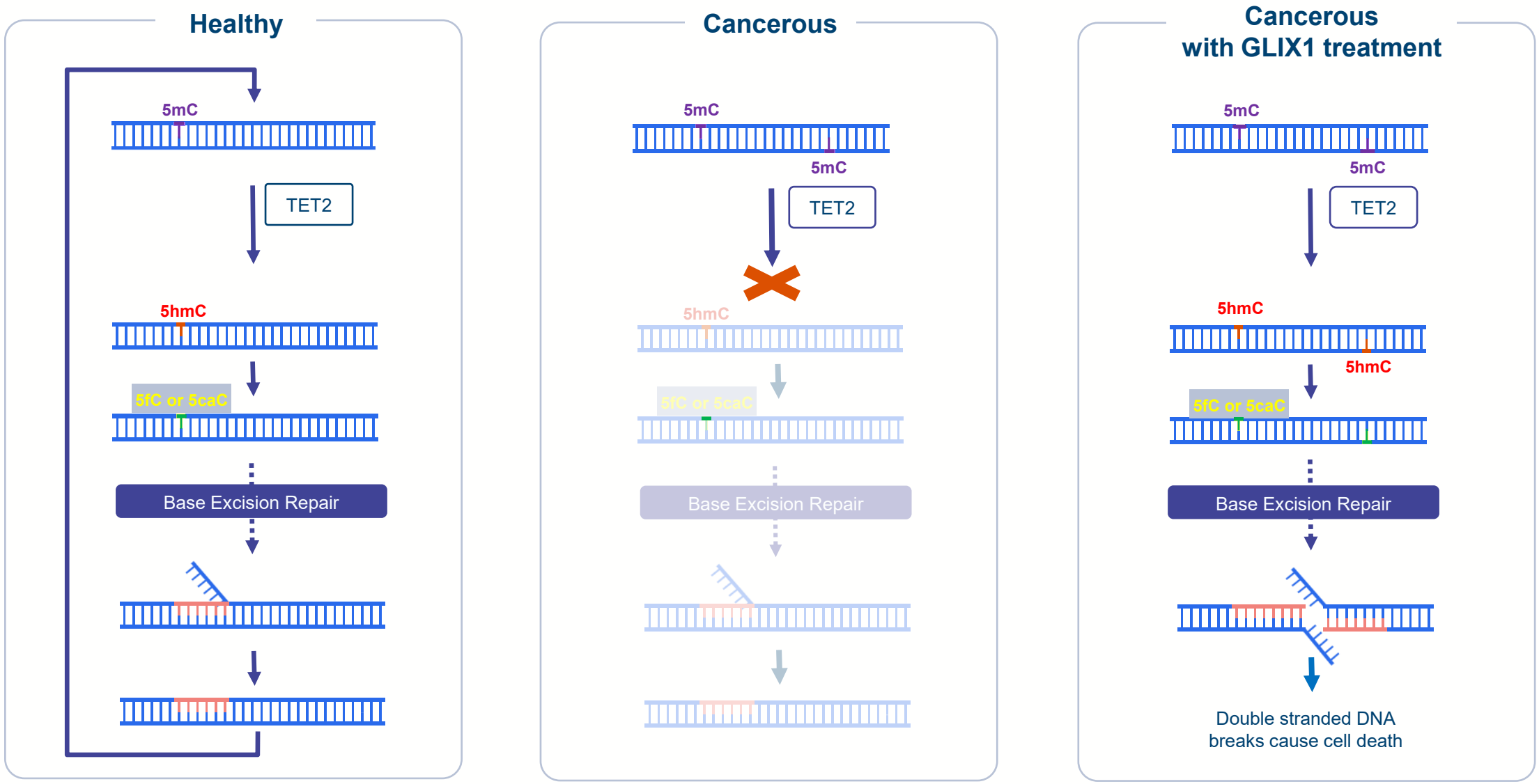
- In cancer cells, hypermethylated regions are common and **TET2 is inhibited by oncometabolites**, leading to increased DNA methylation (5mc) in close genomic proximity[3]

- 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]



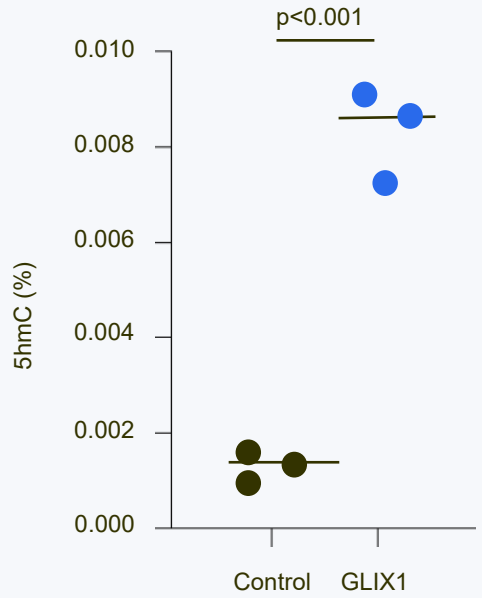
1.Jinrong Zhu et al, Cancer Bio Med, 2023, 21(2):111–116; 2. Wenxin Da et al, Clinical and Translational Oncology, 2024, 26:2156–2165; 3. Johnson KC et al, Nature Communications, 2016, 7, 13177; 4. Luisa Cimmino et al, Cell, 2017, 170(6):1079-1095

Illustration of TET2 activity in healthy versus cancer cells



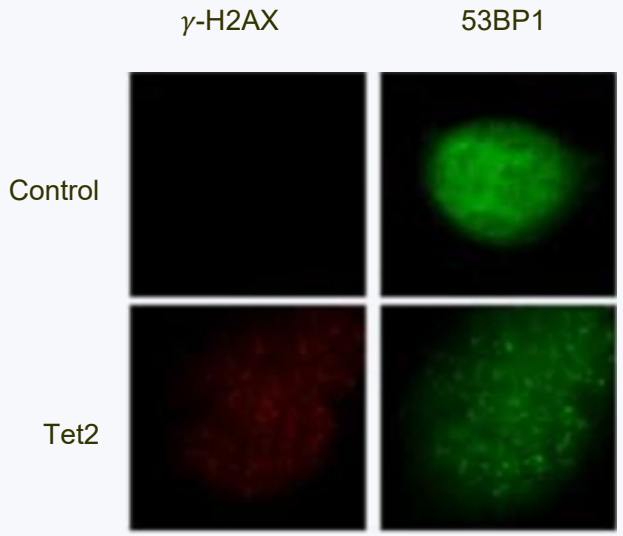
Pre-clinical studies confirm increase in 5hmC levels after GLIX1 treatment – leading to double-stranded DNA breaks and cancer cell death

GLIX 1 enhances TET2 activity resulting in increased TET2 product (5hmC)



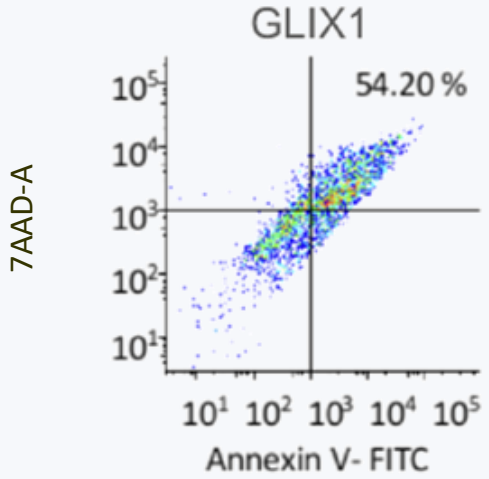
Resected tumor tissue confirms increased **5hmC** levels after GLIX treatment

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



Restoring the **5hmC** levels in cancer cells leads to double stranded DNA breaks

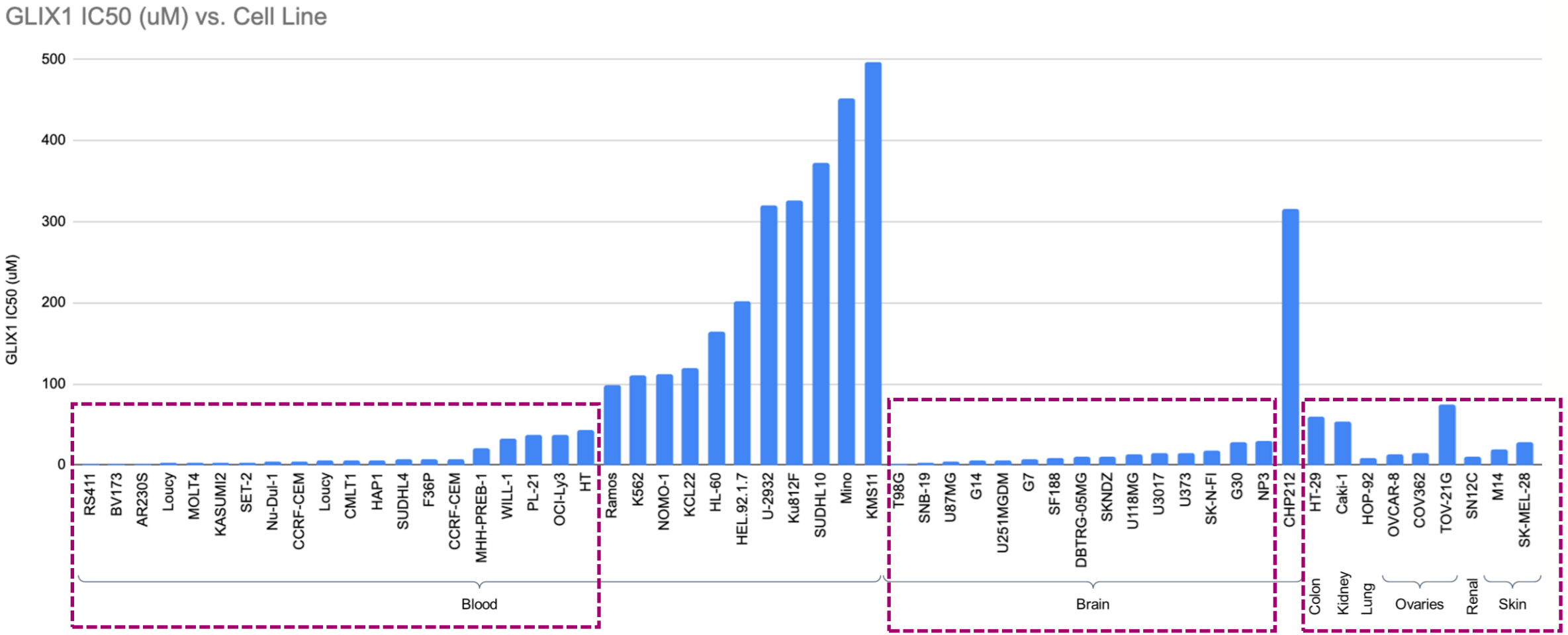
Double stranded DNA breaks cause cell death



Treatment with GLIX1 results in apoptosis

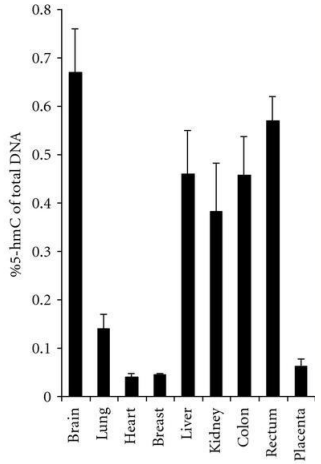
GLIX1 shows efficacy in numerous different cancer cell lines

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

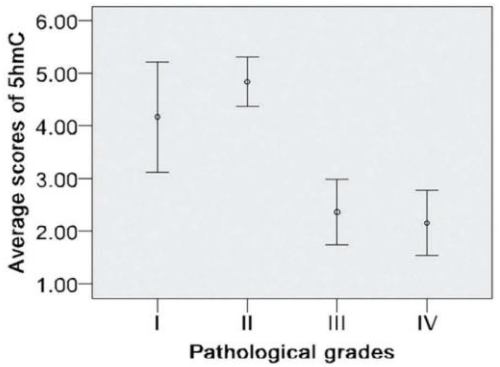


Glioblastoma (GBM) chosen as initial indication supported by strong rationale

5hmC levels are significantly reduced in GBM

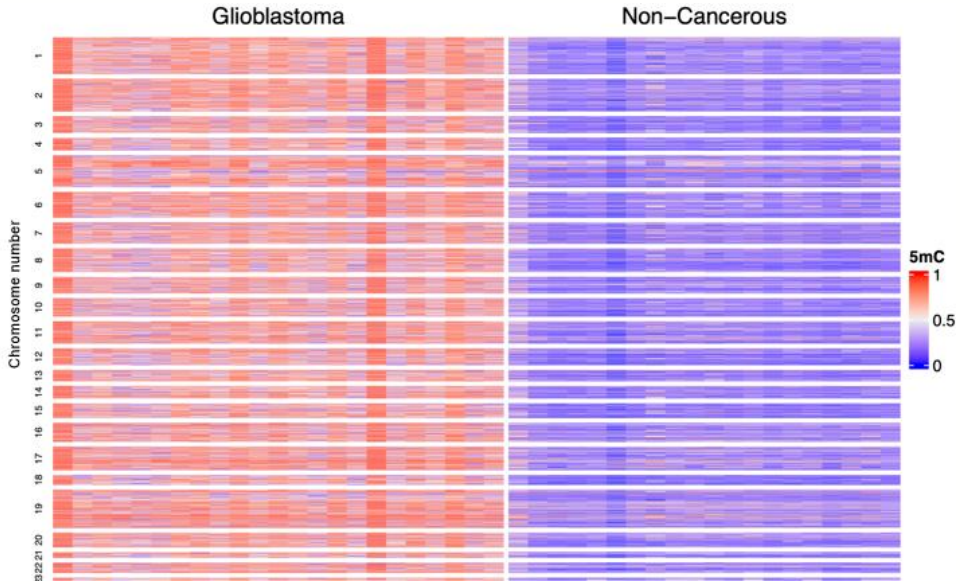


Healthy human brain tissue has high levels of 5hmC.[1]



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

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



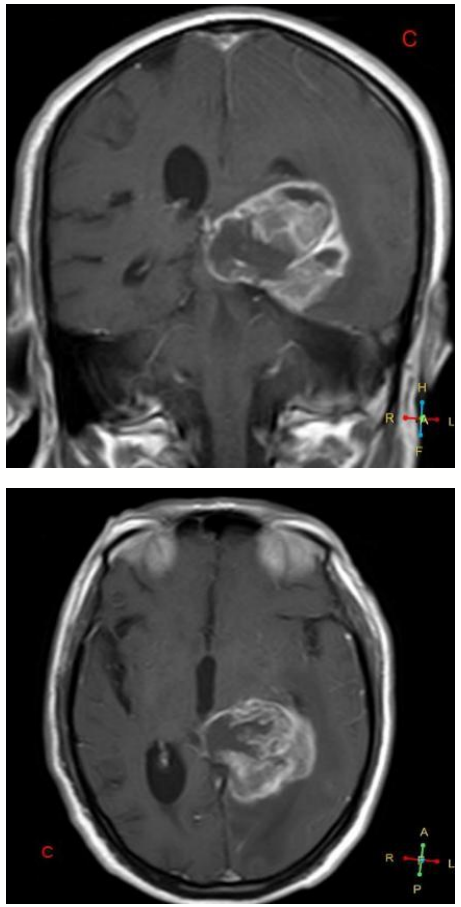
Healthy **Cancer**

5hmC Level → 5mC Level

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

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

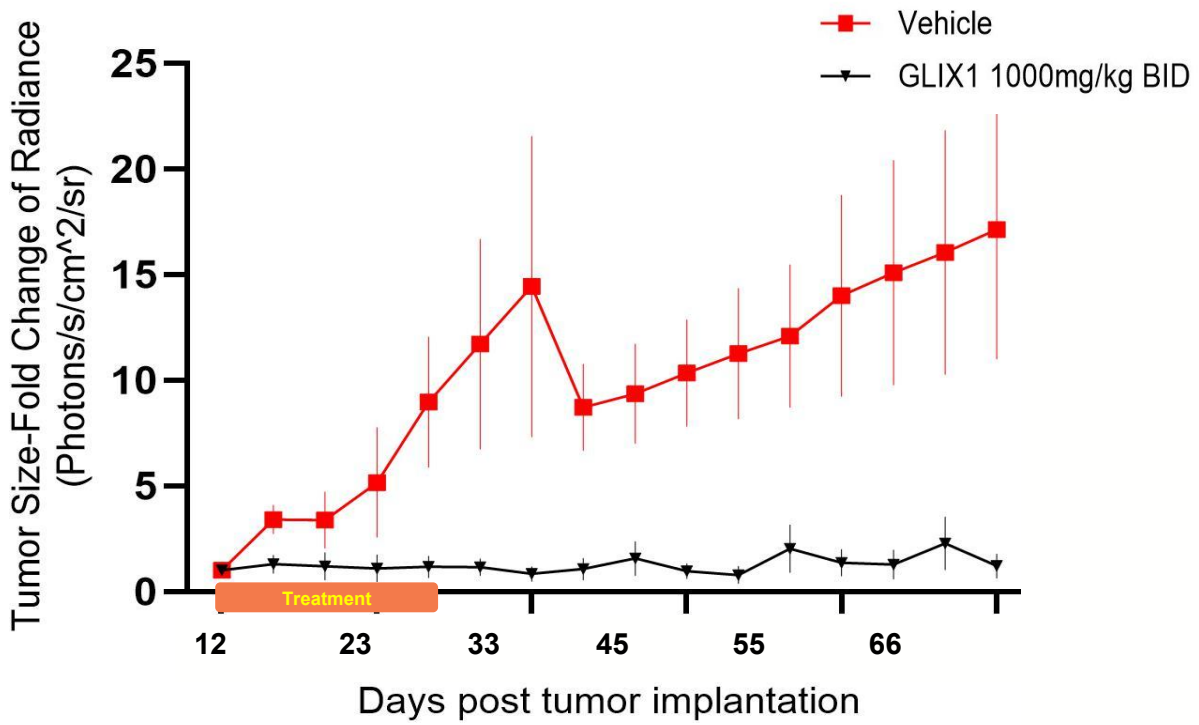
- GBM is the **most common** and most aggressive primary brain tumor
- Poor prognosis with **median survival of 12–18 months**[1,2]
- SoC for newly diagnosed GBM is surgery + radiotherapy + Temozolomide (TMZ)[3] established in 2005
  - Demonstrated marginal increase in OS:14.6 months with TMZ vs. 12.1 months without TMZ
  - **Mainly improves outcome for patients with methylated MGMT promoter** (only ~30% of GBM patients)
- **No established SoC for recurrent GBM**

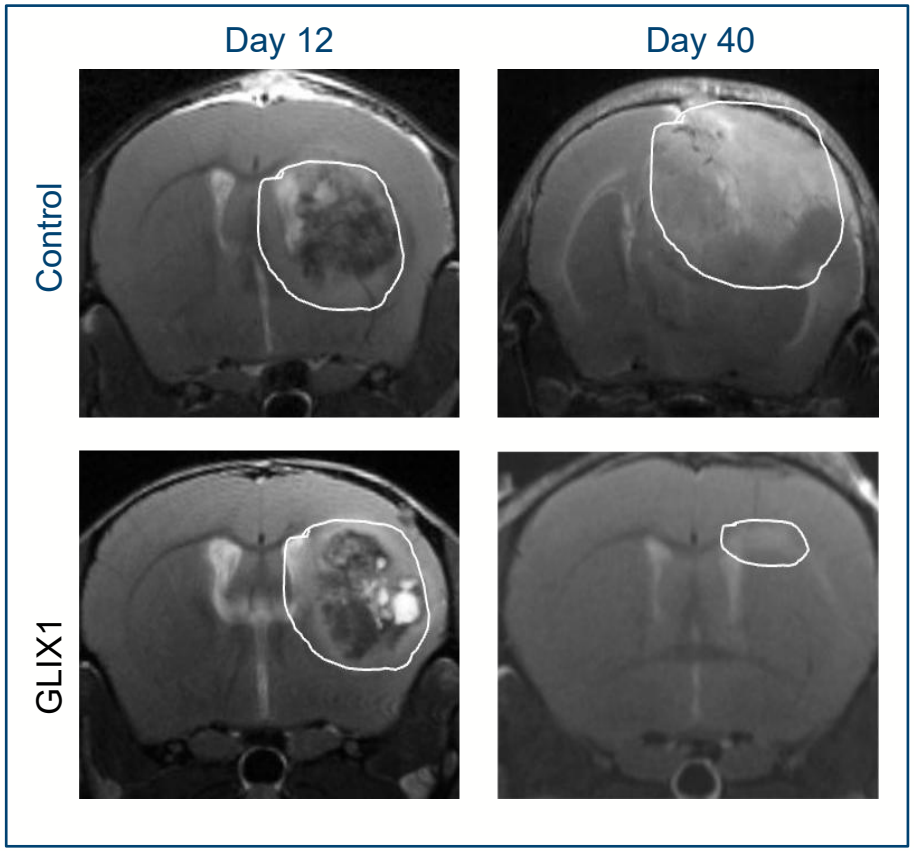


1. Biomedicines, 2021 Mar 22;9(3):324
2. SEER 2017
3. N Engl J Med 2005;352:987-996

GLIX1 demonstrated significant efficacy in multiple GBM models

Potent anti tumor activity in Orthotopic GBM xenograft model



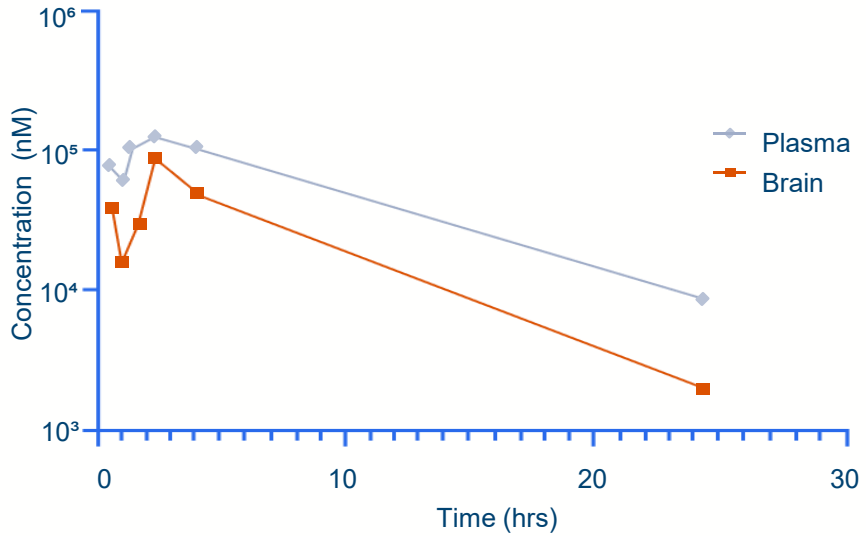


Nude mice inoculated with SNB-19-Luc cells; treatment started at day 12 for 2 weeks; n=7 per group

Stanford MEDICINE

GLIX1 addresses key challenges for GBM drug development

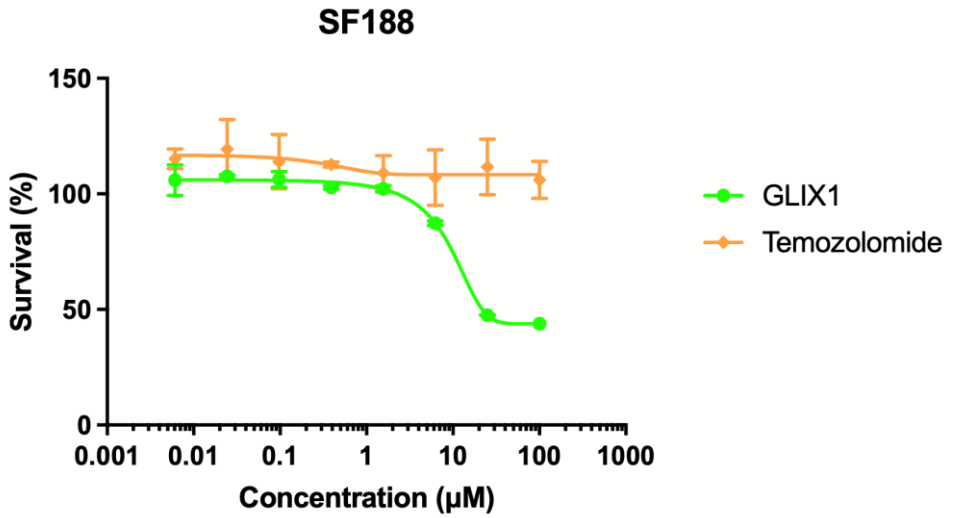
Good blood-brain-barrier penetration in healthy mice following 1000 mg/kg PO



Group	AUC (nM*Hours)	Cmax (nM)	Tmax (Hours)	T1/2 (Hours)
Plasma	1600000	132000	2	5
Brain	692000	90200	2	5

Efficacy in TMZ resistant cell lines

- More than half of all GBM patients are TMZ-resistant, due to unmethylated MGMT promoter status
- For these patients, there are **no other treatment options**
- GLIX1 showed efficacy in such TMZ-resistant cell lines



Stanford MEDICINE

GLIX1 clinical trial design – Phase 1/2

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

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

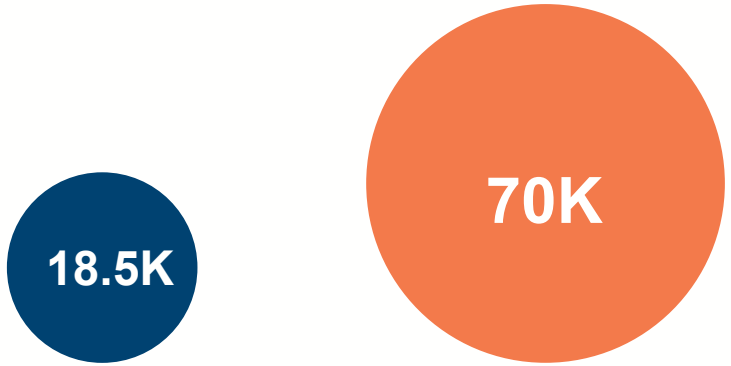
N: up to 20 per arm

Planned timeline: ~2.5 years

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

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

Estimated diagnosed annual incidence in 2030:



in US

in major markets

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

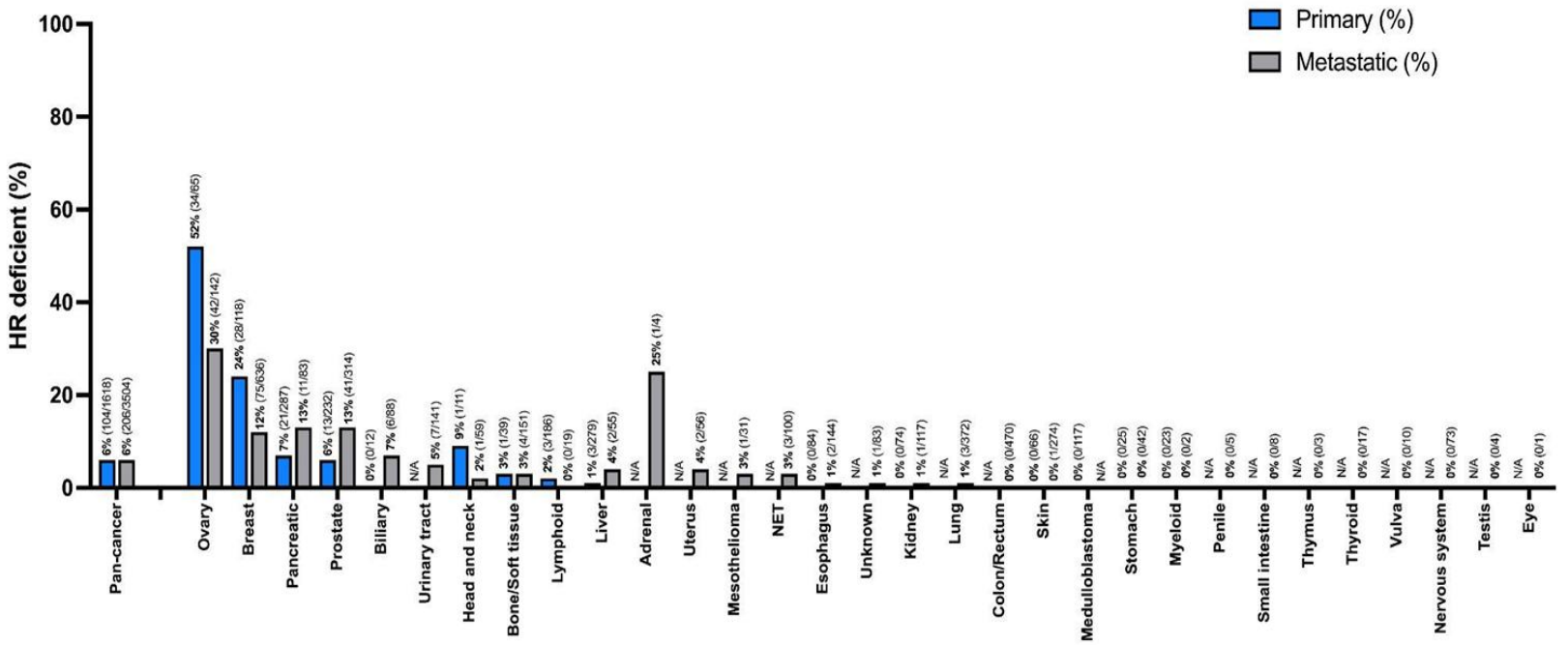
50-75%
of patients do not respond

$1B/year
peak sales while on patent

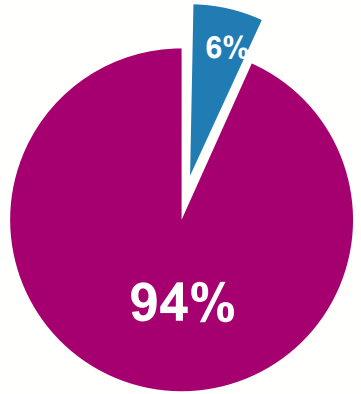
GLIX1 and PARP inhibitors (PARPi) – potential for strong synergy

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



HR-deficient cancers (treatable with PARPi)



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

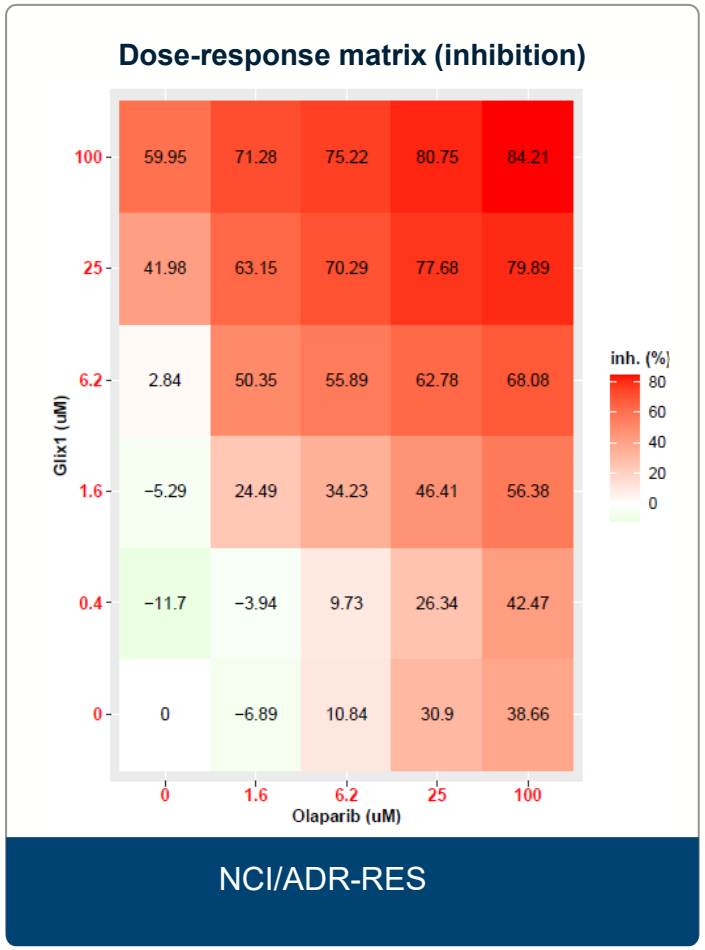
PARP inhibitors are efficacious in HR-deficient cancers[1, 2]

1- Luan Nguyen et al, Nature Communications (2020) 11:5584

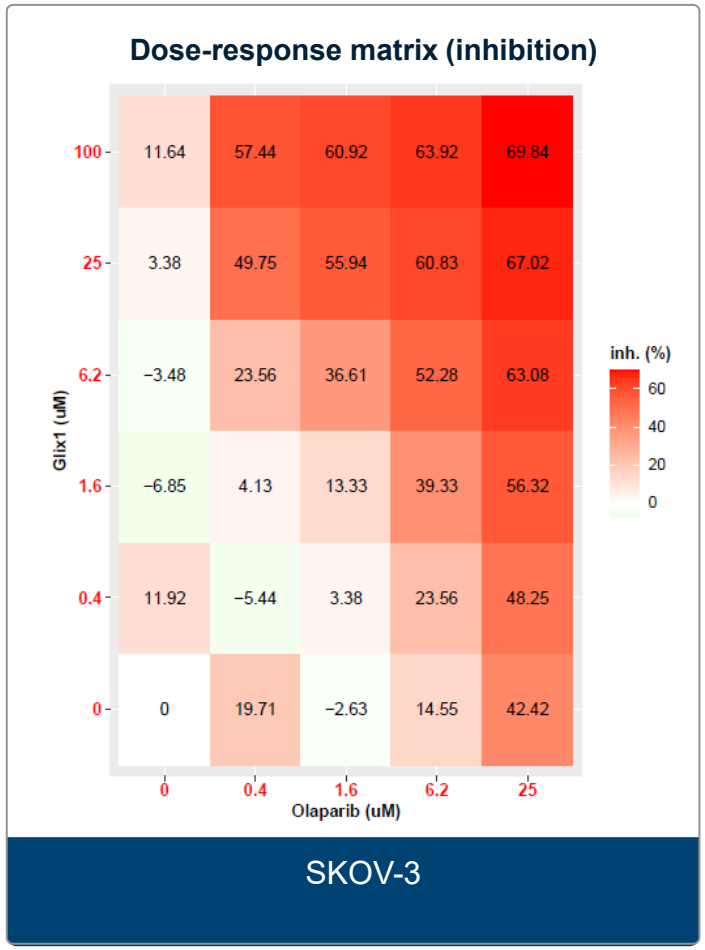
2 - HR-deficient cancers are tumors whose cells cannot properly repair DNA double-strand breaks because the homologous recombination (HR) repair pathway is impaired.

GLIX1 shows strong synergy with PARPi in HR-proficient cancers

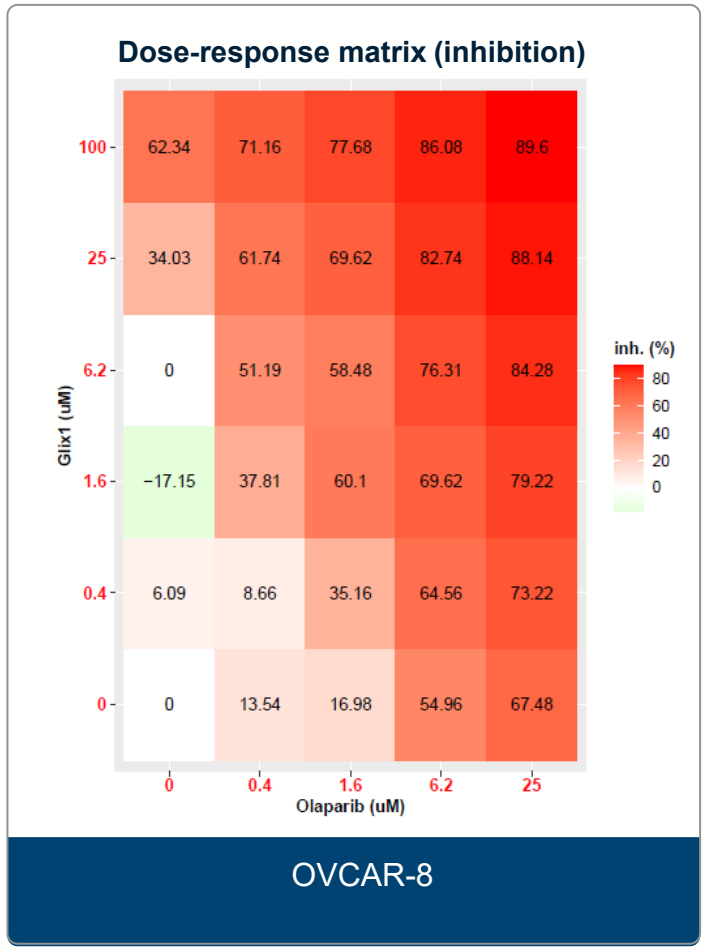
Synergy demonstrated in various HR proficient-ovarian cancer cell lines



NCI/ADR-RES



SKOV-3



OVCAR-8

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

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
- Can also potentially 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

Motixafortide in cancer immunotherapy for pancreatic ductal adenocarcinoma (PDAC) and other solid tumors

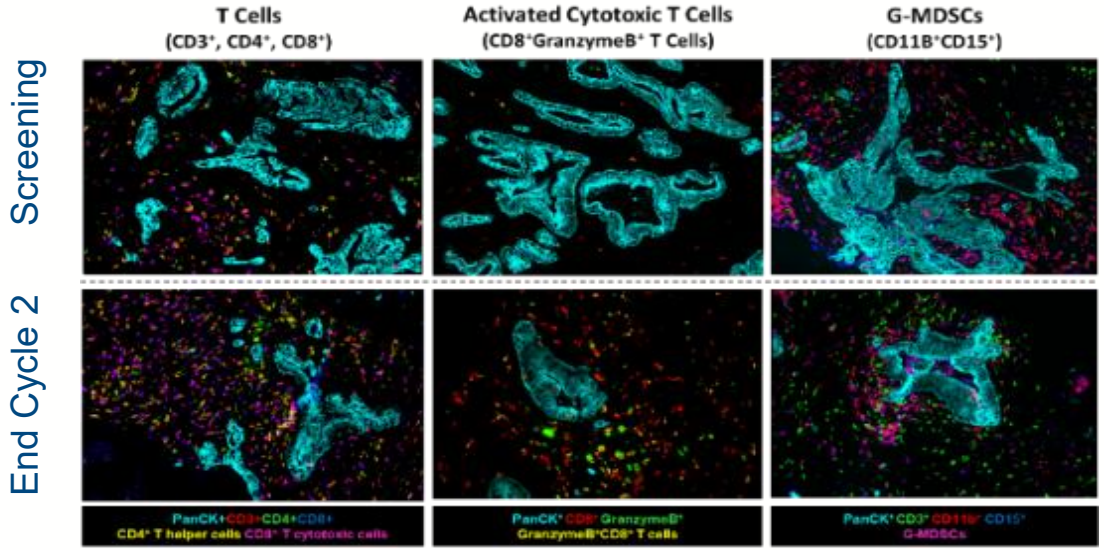
Motixafortide facilitates T cell trafficking and infiltration into tumor and modulates TME immune cell composition in PDAC patients

Motixafortide is a high affinity CXCR4 inhibitor that blocks the interaction between CXCR4 and its ligand SDF-1/CXCL12

Mechanism of action in solid tumors:

- Releasing immune cells to periphery by blocking their CXCR4-mediated retention in bone marrow stroma
- Enabling infiltration of effector T cells into the tumor, by blocking their CXCR4-mediated retention on CXCL12-secreting fibroblasts at the edge of the tumor
- Relieving immunosuppression by blocking CXCR4-mediated infiltration of immunosuppressor cells into the tumor

Biopsies from PDAC patients before and after treatment with combination of motixafortide + pembrolizumab



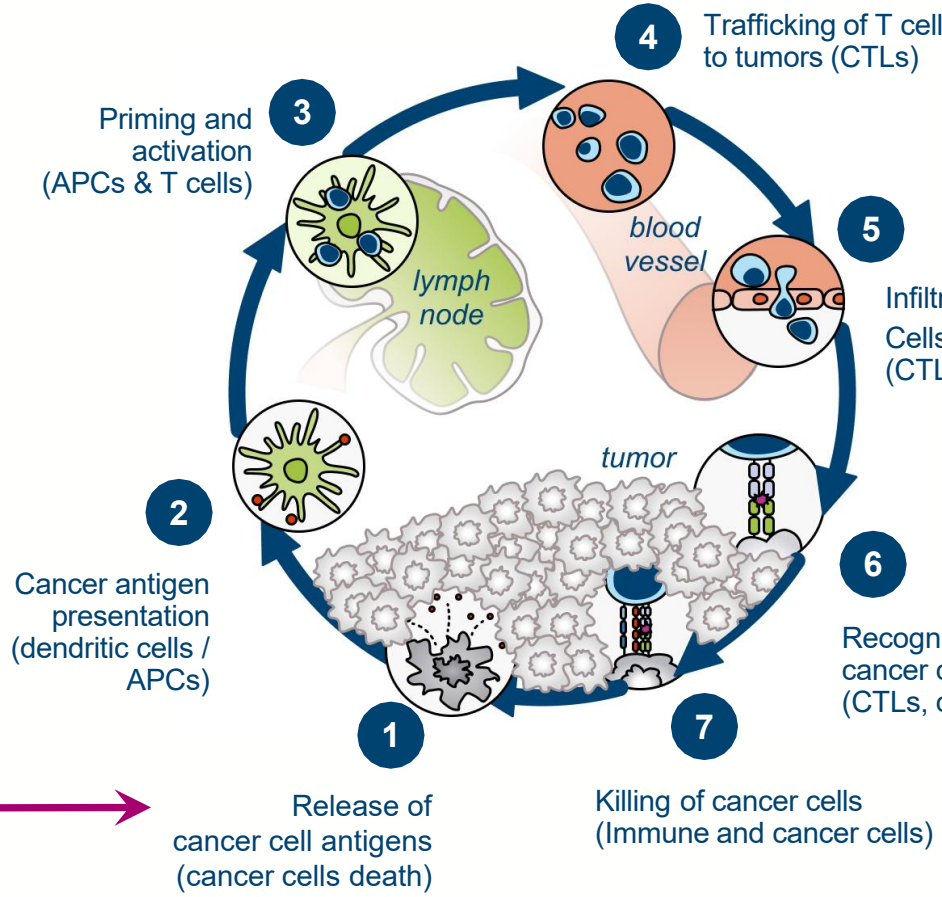
*Representative MultiOmyx™ data taken from SD patient with long treatment duration (11 combo cycles ~34 weeks). Data shown before treatment vs. after ~7w of treatment (end of cycle 2)[1]

- **Increased activated cytotoxic T cells**
- **Decreased suppressor cells in TME**
- **Reduction in tumor cells**

1. Bruno, Nature Medicine, 26, 878–885 (2020)

Rationale for motixafortide in combination with PD-1 inhibitors and chemotherapy

Solid tumors with low immune system visibility require a multi-pronged approach



TME modulation

Chemotherapy

- Chemotherapy induces tumor death, reducing tumor burden
- Chemotherapy induces immunogenic cell death, leading to activation & expansion of new tumor-reactive T-cell clones

Motixafortide

- Motixafortide facilitates T cell trafficking and infiltration into tumor core; TME modulation

Checkpoint Inhibitor

- PD-1 maintains & restores activity of T cells within tumor
- Effectiveness of ICIs in mPDAC remains limited[1,2]

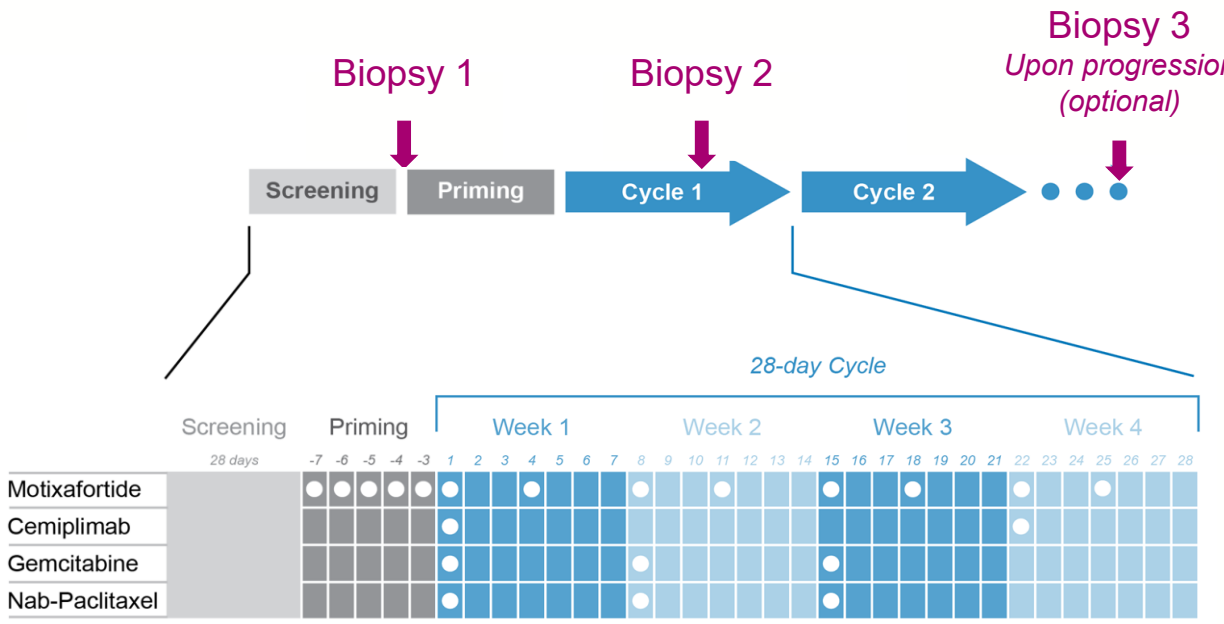
Adapted from Chen, D. et al. Immunity Review 2013

1. O'Reilly, EM JAMA Oncol. 1431-1438 (2019); Wainberg ZA Clin. Cancer Res. 4814–22 (2020) ; Renauf DJ Nat Commun. 13:5020 (2022)
2. Renauf DJ Nat Commun. 2022; 13: 5020

Chemo4MetPanc Phase 2 clinical trial in first-line PDAC: pilot phase

IIS under collaboration with Columbia University, equally funded by Regeneron and BioLineRx

PILOT TRIAL DESIGN



Motixafortide	1.25mg/kg SC	Gemcitabine	1000mg/m2
Cemiplimab	350mg IV qD21	Nab-paclitaxel	125mg/m2
			IV D1, 8, 15 q28D
			IV D1, 8, 15 q28D

Primary Endpoint:
Progression free survival at week 16

BASELINE CHARACTERISTICS

Characteristic	N = 11
Age	60 y (46–69)
Sex	
Female	2 (18%)
Male	9 (82%)
Race	
Black or African American	3 (27%)
White	8 (73%)
Site of Pancreas Primary	
Head	2 (18%)
Neck/Uncinate/Body	3 (27%)
Tail	4 (36%)
Metastatic Sites at Baseline	
1	9 (82%)
Site of Metastasis at Baseline	
Liver	10 (91%)
Peritoneum	1 (9%)
Lung	1 (9%)
Osseous	1 (9%)

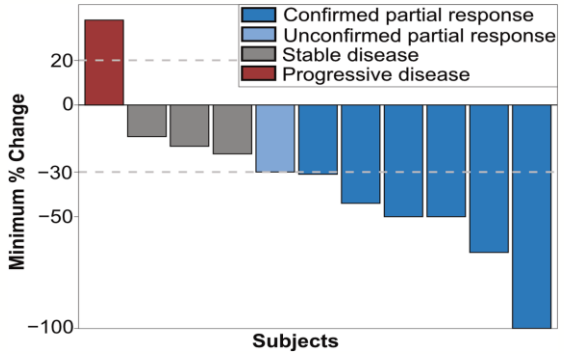
Comparable patient population to Van Hoff and Napoli3 trials
(trials that led to approvals of Gem/Abrax and Nalirifox, respectively)

Encouraging response and survival data from pilot phase

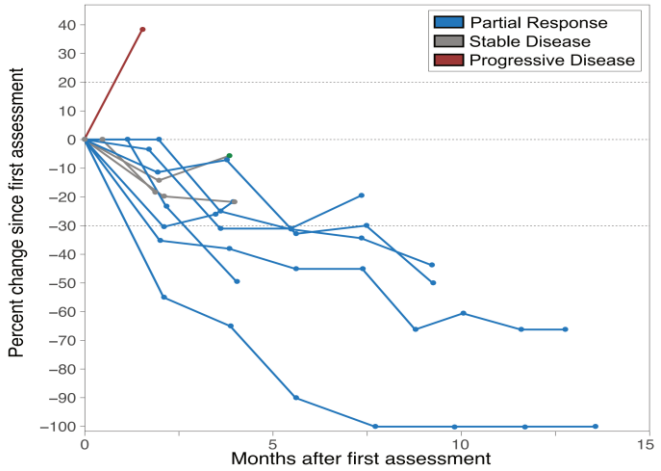
Radiological Response[1]

N = 11 (%)	
Disease Progression	1 (9%)
Stable Disease (SD)	3 (27%)
Partial Response (PR)	7 (64%)
Confirmed PR	6 (55%)
Disease Control Rate (DCR)	(91%)

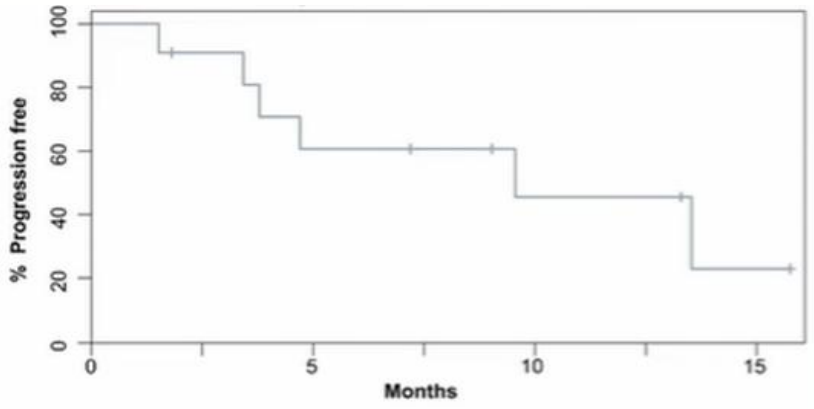
Compares favorably to historical PR (23%) and DCR (48%) with gemcitabine and nab-paclitaxel alone



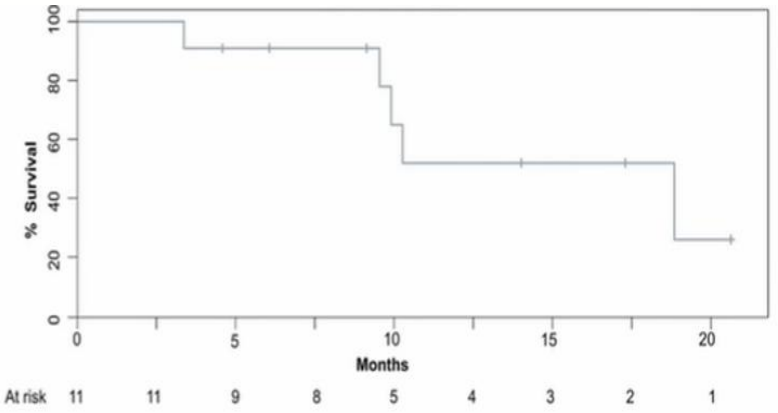
Radiological Response Duration[2]



Progression-Free Survival



Overall Survival



1. Manji, GA ASCO 2025; 2. Manji, GA AACR Pancreatic Cancer 2023 (Censor Date – July 20, 2023)

Motixafortide combination treatment resulted in 64% ORR and 9.6-month median PFS with a favorable tolerability profile in pilot phase

Efficacy data:

Gemcitabine, nab-paclitaxel, motixafortide and cemiplimab resulted in

- **Overall Response Rate 64%**
- **Median PFS 9.6 months**

2 patients underwent definitive treatment:

- One had **complete resolution of all liver lesions** and underwent definitive radiation to the primary pancreatic tumor
- One had a sustained partial response and **underwent Whipple procedure with pathology demonstrating a complete response**

Safety data:

The combination demonstrated a **tolerable safety profile.** No unexpected Grade 4 or 5 treatment related adverse events

MOA findings:

Correlative analyses on paired tumor and PBMCs reveal that **CD8+ T-cell tumor infiltration increased** across all 11 patients

Next steps:

The encouraging preliminary efficacy prompted a change in clinical trial design to a **randomized Phase 2 trial** *(Chemo4MetPanc; NCT NCT04543071)*

Updated data presented at ASCO 2025

Manji, GA ASCO 2025
Manji, GA AACR Pancreatic Cancer 2023 (Censor Date – July 20, 2023)

Chemo4MetPanc Phase 2b clinical trial in first-line PDAC - randomized phase

IIS under collaboration with Columbia University, equally funded by Regeneron and BioLineRx – currently enrolling

Randomized Trial Recruiting

Primary Endpoint
median PFS

Secondary Endpoints
OS, RR

Correlates
mIF, snRNAseq, cytokine

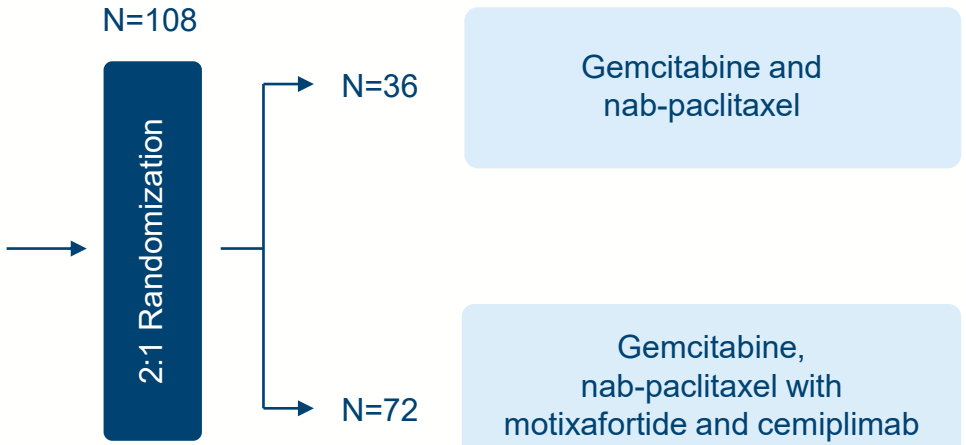
Trial sites

- Columbia University (PI Dr. Manji)
- Brown University
- Medical College of Wisconsin
- Beth Israel Deaconess Medical Center

Study Design

Key Eligibility

- Stage IV PDAC
- No prior systemic therapy
- KPS ≥ 70
- RECIST measurable



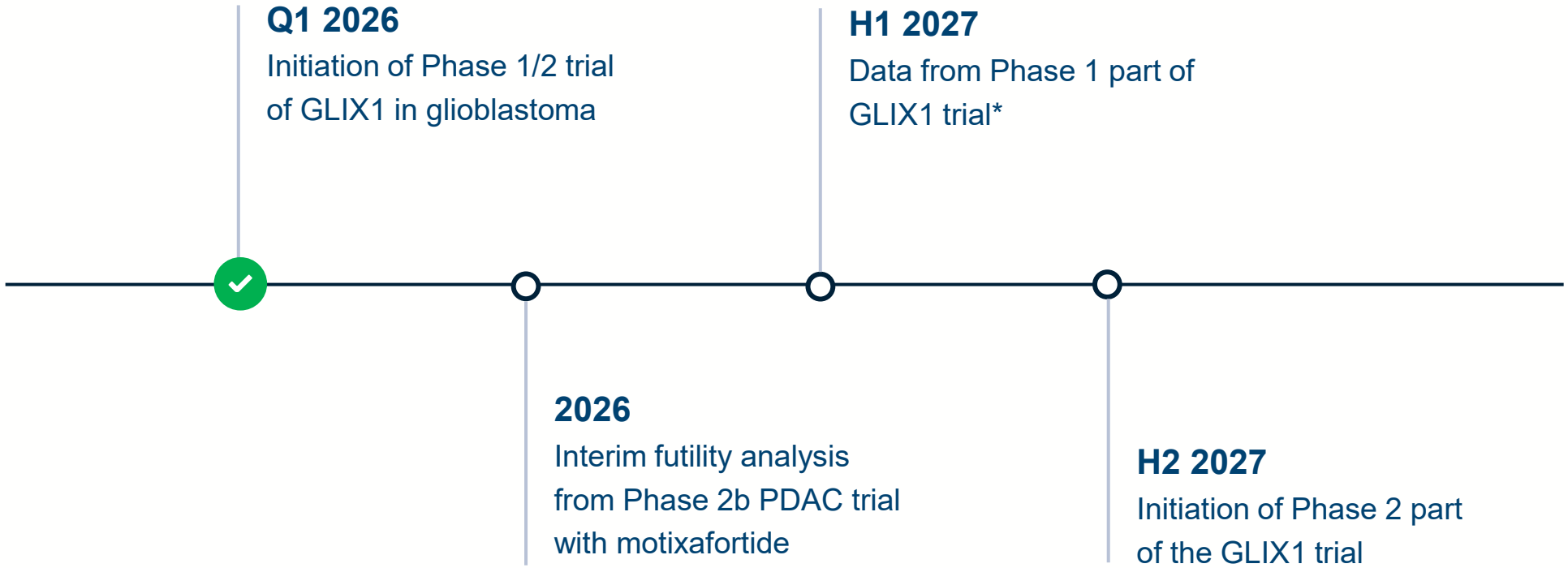
Key Milestones*

- **Prespecified interim futility analysis** (when 40% of PFS events observed) planned in 2026
- Full enrollment planned in 2027

*Independent Investigator Study (IIS) timelines, as well as other study related decisions, are ultimately controlled by the independent investigator-sponsor and are, therefore, subject to change.

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