KAZIA THERAPEUTICS

Beyond Inhibition. Reprogramming Cancer Control.

Kazia Therapeutics Overview

BIO International Convention
June 23, 2026

NASDAQ: KZIA | X: @KaziaTx

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Our mission is to redefine cancer treatment by moving beyond conventional pathway inhibition toward therapeutic reprogramming of cancer biology by targeting regulatory drivers of growth, immune escape, and relapse to deliver more durable outcomes for patients with the greatest unmet need.

Company Highlights

01 Clinical-stage oncology company advancing next-generation differentiated therapies designed to overcome resistance in aggressive and immunotherapy-refractory cancers

02 Lead asset **paxalisib** is an oral, brain-penetrant dual PI3K/mTOR inhibitor for solid tumor indications, including breast cancer, evaluated to date in >550 adult and pediatric patients across Phase 1-3 clinical trials and expanded access programs

03 Advancing **NDL2**, a potentially first-in-class protein degrader designed to target intracellular PD-L1 to overcome immune resistance beyond checkpoint blockade

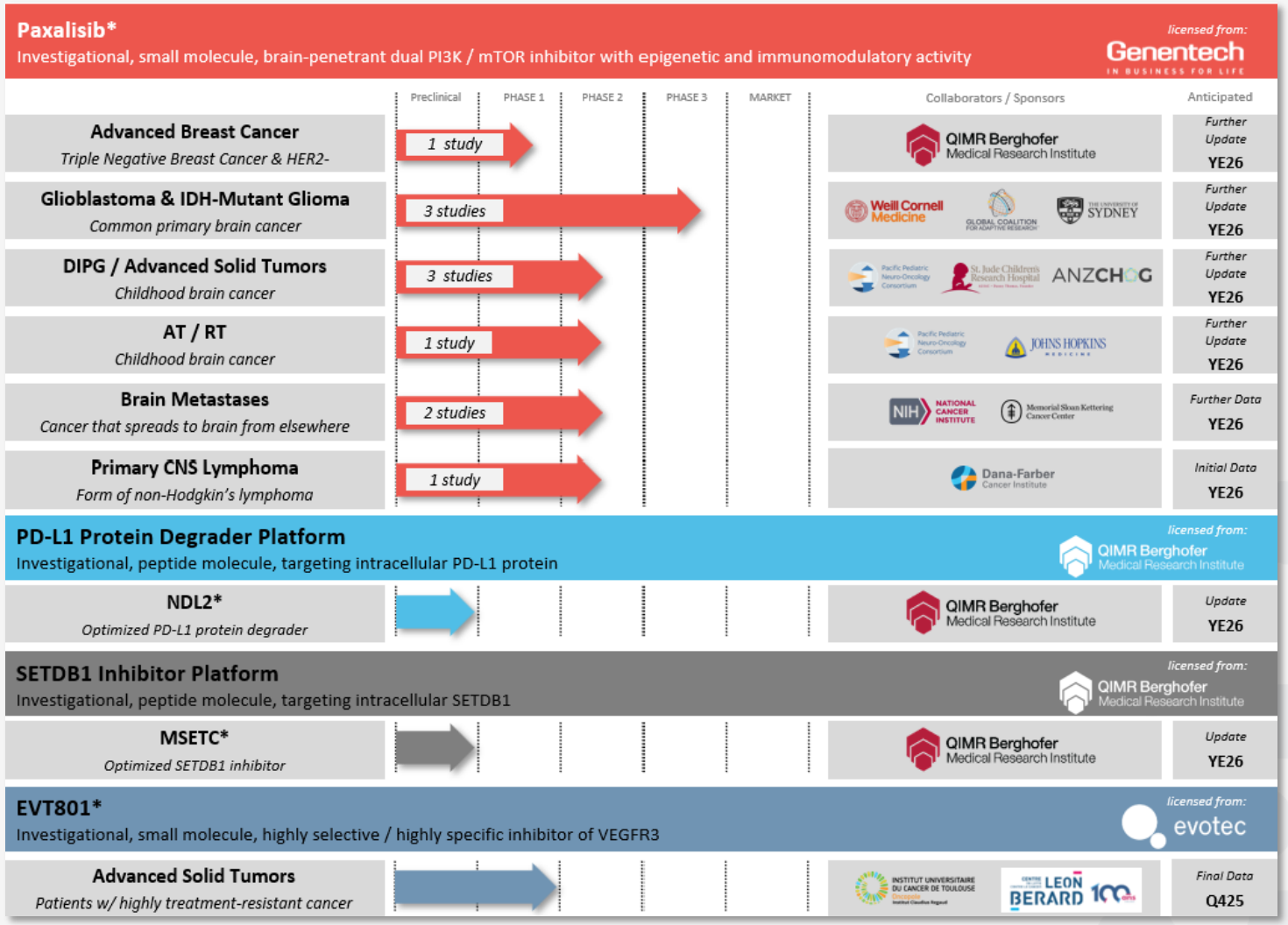
04 Advancing **MSETC**, a potentially first-in-class SETDB1 inhibitor designed to target SETDB1, a key epigenetic regulator of immune evasion

05 Developing **EVT801**, an oral, highly selective VEGFR3 inhibitor targeting tumor lymphangiogenesis and metastatic spread, with strong partnering potential

06
- Cash & Cash Equivalents: Approximately US$46 million (Runway expected to fund planned operations into 2029, excluding additional or expanded trials)
- No outstanding debt
- American Depositary Shares Outstanding: Approximately 11.4 million

Pipeline Overview

High-value indications with strong partnering potential



*Product candidates (not approved); IDH: Isocitrate dehydrogenase, DIPG: Diffuse Intrinsic Pontine Glioma, AT / RT: Atypical Teratoid Rhabdoid Tumor, CNS: central nervous system, TNBC: triple negative breast cancer, VEGFR3: vascular endothelial growth factor receptor 3

KAZIA THERAPEUTICS

Overview

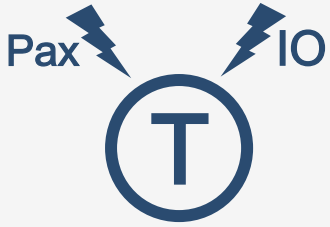
Asset Overview	Brain-penetrant, oral, once daily small molecule dual PI3K/mTOR inhibitor with epigenetic and immunomodulatory activity
Origin	Discovered by Genentech (GDC-0084); In-licensed by Kazia Therapeutics
Development Stage	Clinical
Lead Indications	Advanced Breast Cancer (ABC, Phase 1), Glioblastoma (GBM, Phase 3), Childhood Brain Cancer (Phase 2 / Preclinical)
Formulation	Oral, capsule
Key Differentiators	Oral once-per-day dual PI3K/mTOR inhibitor in clinical development for ABC; addresses key mechanisms of immunotherapy resistance; as well as ability to cross the blood brain barrier (BBB) as a potential therapy for primary and secondary brain cancers
Mechanism of Action	PI3K/mTOR kinase inhibitor that disrupts core oncogenic signalling driving tumor growth and survival, while also affecting tumor epigenetic regulation, transcriptional state, and the immune microenvironment beyond its core kinase inhibition
FDA Designations	Fast Track and Orphan Drug for GBM; Fast Track for PI3K-altered brain metastases with radiation; and Rare Pediatric Disease and Orphan Drug for select pediatric gliomas, including diffuse intrinsic pontine glioma (DIPG) and atypical teratoid/rhabdoid tumor (AT/RT)
IP	Patent protection projected until 2043; coverage in over 40 jurisdictions, including U.S., Europe, Japan, China, and India; three patent families: paxalisib molecule *per se* (granted), synthesis of paxalisib (granted), and secondary medical uses / combination treatments (pending)

Clinical & Translational Manifestations

In clinical settings, there is evidence that paxalisib's downstream epigenetic effects manifest as:

Reduction of Aggressive Circulating Tumor Cell (CTC) and CTC-Cluster Phenotypes

Observable impact on metastatic biology



Enhanced Activity When Combined with Immune Checkpoint Inhibition

Combination -enabling potential

Modulation of Immune Exhaustion Markers

Evidence of tumor microenvironment (TME) reprogramming

Paxalisib is positioned as a backbone therapy within the emerging category of functional epigenetic reprogramming therapeutics.

Kazia-Sponsored Clinical Phase 1b Study Overview

Multi-center, open-label, randomized trial

Inclusion Criteria

Arm A

- HER2-negative stage IV (metastatic) BC
- Confirmed gBRCAm (BRCA1, BRCA2 or both)
- Prior treatment with chemotherapy in the metastatic setting
- Meet all current prescribing criteria for commencing olaparib therapy

Total enrollment: 12

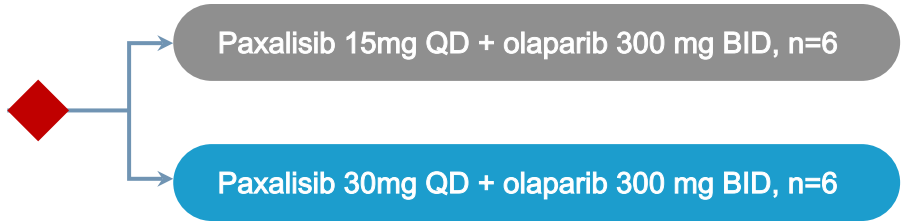
Arm B

- Recurrent, unresectable or metastatic TNBC
- Confirmed that tumors express PD-L1 with a combined positive score (CPS) ≥ 10
- Meet all current prescribing criteria for commencing pembrolizumab therapy

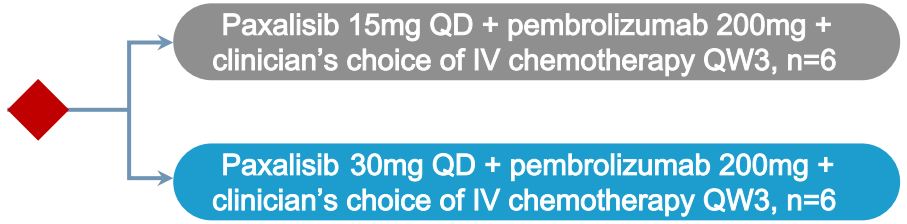
Total enrollment: 12

Treatment

Paxalisib plus Olaparib (28-Day Cycle)



Paxalisib plus Pembrolizumab / Chemotherapy (21 -Day Cycle)



Endpoints

Primary

- Safety and tolerability of paxalisib in combination with either olaparib or pembro / chemotherapy
- Determine recommended Phase 2 dose (RP2D)

Secondary

- Progression and response rates
- To assess the utility of novel liquid biopsy assessments

Trial ID: ACTRN12624001340527 SABCS 2025 poster presentation.

Early Clinical Trial Epigenetic Effects

Combination regimen results in >50% reduction in CTCs

First-in-human data reflects mechanistic synergy consistent with the preclinical data.

Circulating Tumor Cells (CTCs)

- Cancer cells that detach from a primary tumor or metastatic site and enter the bloodstream
- Cellular drivers of metastasis, capable of seeding new tumors in distant organs
- Clusters possess significantly higher metastatic potential than single CTCs (enhanced survival, immune evasion, and colonization capacity)
- CTCs are generally resistant to current therapies

Reductions in CTC burden indicate potential disruption to the metastatic process itself.

Patient One

Patient Profile

- 61-year-old woman with metastatic triple-negative breast cancer localized to the left upper lobe of the lung

Regimen

- Paxalisib + pembrolizumab + standard chemotherapy

Results at Day 21

End of Cycle 1

- >50% reduction in total circulating tumor cells count
- Comparable reduction in CTC clusters (these aggregates are associated with heightened metastatic potential)
- Reduction in the mesenchymal phenotype of the remaining CTCs; this phenotype is one of the hallmarks of aggressive metastatic seeding cancer cells
- Such results are not typically seen with chemotherapy or immunotherapy therapies

Trial ID: ACTRN12624001340527 Kazia Press Release 10 July 2025 SABCS 2025 poster presentation.

Early Evidence of Tumor Regression

1 CR in mTNBC patient treated under expanded access, and 2 PRs in ongoing study

Preliminary Clinical Responses

- 1 expanded access TNBC patient achieved a confirmed complete metabolic response following re-treatment with pembrolizumab / chemotherapy plus paxalisib (under an expanded access protocol)
- 2/2 evaluable TNBC trial patients achieved confirmed partial responses (iRECIST)
- Responses reported in patients with visceral and multi-organ metastases
- Median treatment duration at data cut-off: ~6.1 months

Safety

- Generally well tolerated at 30mg daily
- Majority of adverse events assessed as unlikely related to paxalisib
- All paxalisib-related events were mild to moderate to date

Trial ID: ACTRN12624001340527 Kazia Press Release 27 January 2026

Platform Overview

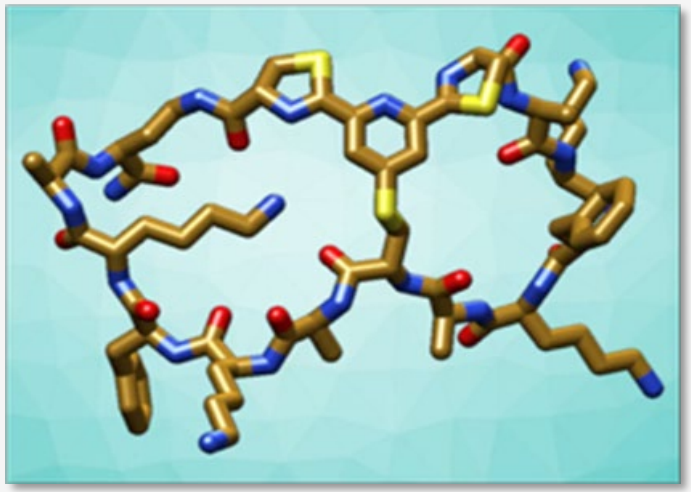
Complementing paxalisib's transcriptional and epigenetic effects, Kazia's NDL2 program addresses immune resistance at the post-translational regulatory level

Asset Overview	Lead candidate, NDL2, is an optimized bicyclic peptide PD-L1 degrader designed to target intracellular PD-L1, thereby restoring anti-tumor immune responses even in cases of immune evasion or checkpoint inhibitor resistance
Origin	Developed by QIMR Berghofer
Development Stage	Preclinical; IND expected in 2H 2027
Lead Indications	Breast cancer and non-small cell lung cancer (NSCLC) in early stage and metastatic settings
Formulation	Intravenous infusion
Key Differentiators	First in Class: As the science surrounding PD-L1 degraders is novel (<5yrs), there are no PD-L1 degraders in clinical studies at this time
Mechanism of Action	Target intracellular PD-L1 protein pools Recruits cellular degradation machinery Targets intracellular PD-L1 protein species that cannot be targeted by standard checkpoint inhibitors
FDA Designations	N / A
IP	Initial composition of matter IP filed in 2021

NDL2: First-In-Class PD-L1 Degrader

Targeting intracellular PD-L1

Targeted Protein Degradation (TPD) therapies opened a new frontier for treating cancer by exploiting naturally occurring protein degradation pathways to disrupt critical cellular processes.



NDL2 is an optimized bicyclic peptide PD-L1 degrader

- Designed to target intracellular PD-L1
- Formation of a ternary complex (intracellular PD-L1 : NDL2 : E3 ligase) which leads to proteasomal degradation
- Restoring anti-tumor immune responses
- Even in cases of immune evasion or checkpoint inhibitor resistance

Key Differentiator

NDL2 distinguishes itself as a modality facing minimal competitive challenges

	NDL2 Peptide Degrader of PD-L1	PD-(L)1 mAbs
Primary Mechanism	Targeted protein degradation via intracellular E3 ubiquitin proteasome system	Block the PD-1:PD-L1 interaction at the cell surface; Fc effector function varies by isotype / engineering
Route of Administration (ROA)	IV	IV / SC
PD-L1 Pools Addressed	Intracellular	Surface protein
Competitors / Development Stage	Academic programs in early preclinical development (no clinical programs nor industry programs identified)	Commonly cited global commercial checkpoint inhibitors include: Pembrolizumab, Nivolumab, Cemiplimab, Atezolizumab, Durvalumab, and Avelumab

Sullivan et al., PLoS ONE 2023, doi.org/10.1371/journal.pone.0286724

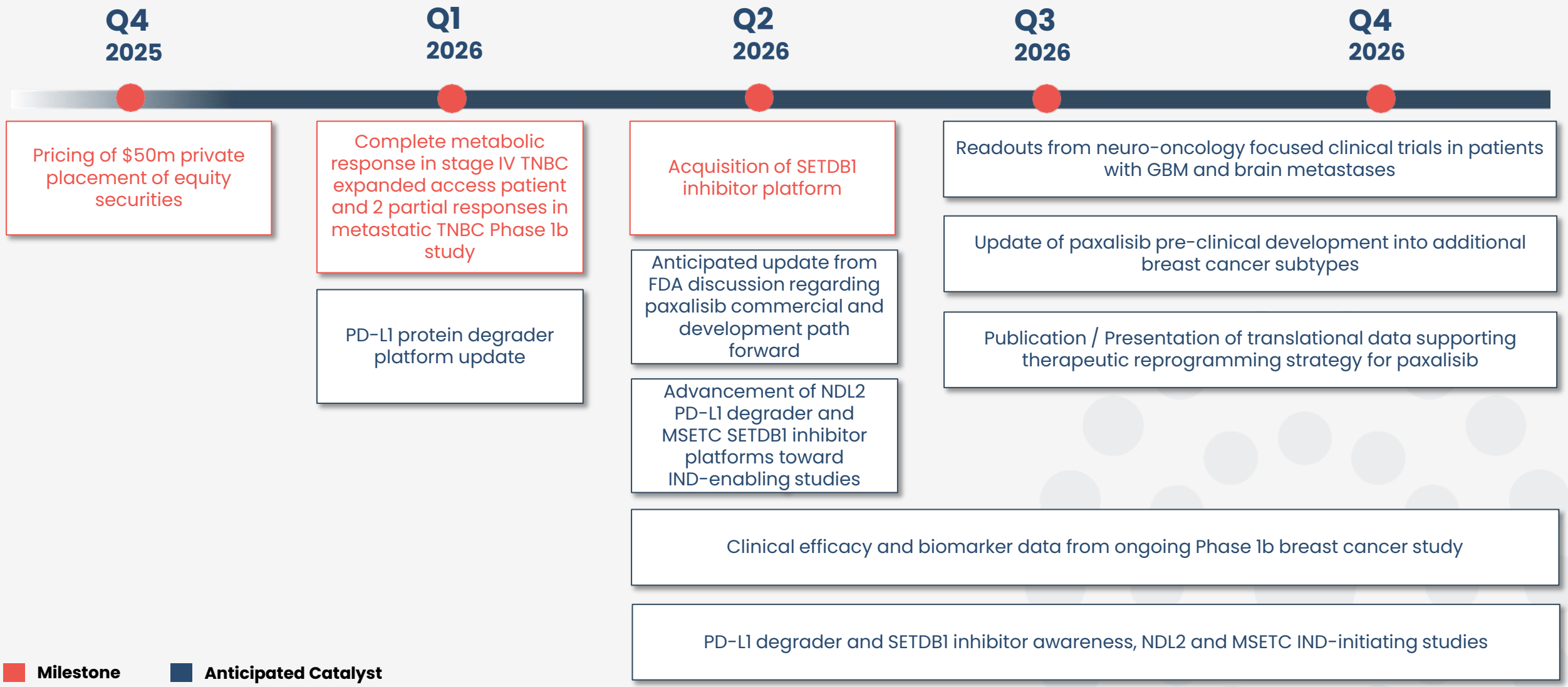
Platform Overview

Complementing paxalisib's transcriptional modulation and NDL2's protein degradation

Asset Overview	Lead candidate, MSETC is an optimized, bicyclic peptide designed to target SETDB1, a key epigenetic regulator of immune evasion. By inhibiting SETDB1, MSETC could restore anti-tumor immune responses, including in tumors resistant to checkpoint inhibitors.
Origin	Developed by QIMR Berghofer
Development Stage	Preclinical; IND expected in 2H 2027
Lead Indications	Solid tumors with low immunogenicity ("immune cold", e.g. TNBC, NSCLC, CRC)
Formulation	Intravenous infusion
Key Differentiators	First-in-class SETDB1 inhibitor (chromatin-level immune regulator); targets immune evasion; potential to convert "immune cold" tumors into responsive tumors; combination-ready with immunotherapy and targeted agents; intracellular / nuclear mechanism not addressed by antibody therapies
Mechanism of Action	SETDB1 is a histone methyltransferase implicated in immune resistance and immune evasion that silences genes involved in immune recognition and interferon signaling through chromatin modification; designed to suppress immune signaling pathways, increase tumor visibility to the immune system, and potentially reverse resistance to checkpoint inhibitors
FDA Designations	N / A
IP	Initial composition of matter IP filed in 2022

Corporate Highlights

Near-Term Milestones & Anticipated Catalysts



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Leadership

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HELSINN
ABBOTT

James Levine
Chief Financial Officer
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Dr. Sudha Rao
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Head of Corporate Strategy & Development
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ALEX. BROWN

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Corporate Secretary
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Robert Apple
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