KAZIA THERAPEUTICS

Beyond Inhibition.
Reprogramming Cancer Control.

2026 Corporate Overview

NASDAQ: KZIA | X: @KaziaTx

March 2026

Forward-Looking Statements

This presentation contains forward-looking statements, which can generally be identified as such by the use of words such as “may,” “will,” “estimate,” “future,” “forward,” “anticipate,” “plan,” “expect,” “explore,” “potential” or other similar words. Any statement describing Kazia's future plans, strategies, intentions, expectations, objectives, goals or prospects, and other statements that are not historical facts, are also forward-looking statements, including, but not limited to, statements regarding: the timing for interim or final results and data related to Kazia’s clinical and preclinical trials, or third-party trials evaluating Kazia’s product candidates, timing and plans with respect to enrolment of patients in Kazia’s clinical and preclinical programs, the potential benefits of paxalisib, NDL2, and EVT801, the potential results of combination studies of paxalisib and other collaborations, timing for any regulatory submissions or discussions with regulatory agencies, the potential market opportunity for paxalisib, NDL2, and EVT801, and Kazia’s strategy and plans with respect to its business and programs. Such statements are based on Kazia’s expectations and projections about future events and future trends affecting its business and are subject to certain risks and uncertainties that could cause actual results to differ materially from those anticipated in the forward-looking statements, including risks and uncertainties: associated with clinical and preclinical trials and product development, the risk that interim and preclinical data may not be reflective of final data, related to regulatory approvals, and related to the impact of global economic conditions, including disruptions in the banking industry. These and other risks and uncertainties are described more fully in Kazia’s Annual Report, filed on Form 20-F with the SEC, and in subsequent filings with the SEC. Kazia undertakes no obligation to publicly update any forward-looking statement, whether as a result of new information, future events, or otherwise, except as required under applicable law. You should not place undue reliance on these forward-looking statements, which apply only as of the date of this presentation.

Certain information contained in this presentation and statements made orally during this presentation relate to or are based on studies, publications, surveys, and other data obtained from third-party sources and our own internal estimates and research. While we believe these third-party studies, publications, surveys and other data to be reliable as of the date of this presentation, it has not independently verified, and makes no representations as to the adequacy, fairness, accuracy or completeness of, any information obtained from third-party sources. In addition, no independent source has evaluated the reasonableness or accuracy of our internal estimates or research, and no reliance should be made on any information or statements made in this presentation relating to or based on such internal estimates and research.

Kazia Therapeutics Limited is a Sydney-based clinical-stage oncology company focused on high-need cancers of the central nervous system and select aggressive solid tumors. Founded in 2016, the company is dedicated to advancing transformative therapies by harnessing next-generation science, disciplined development, and a commitment to clinical impact.

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

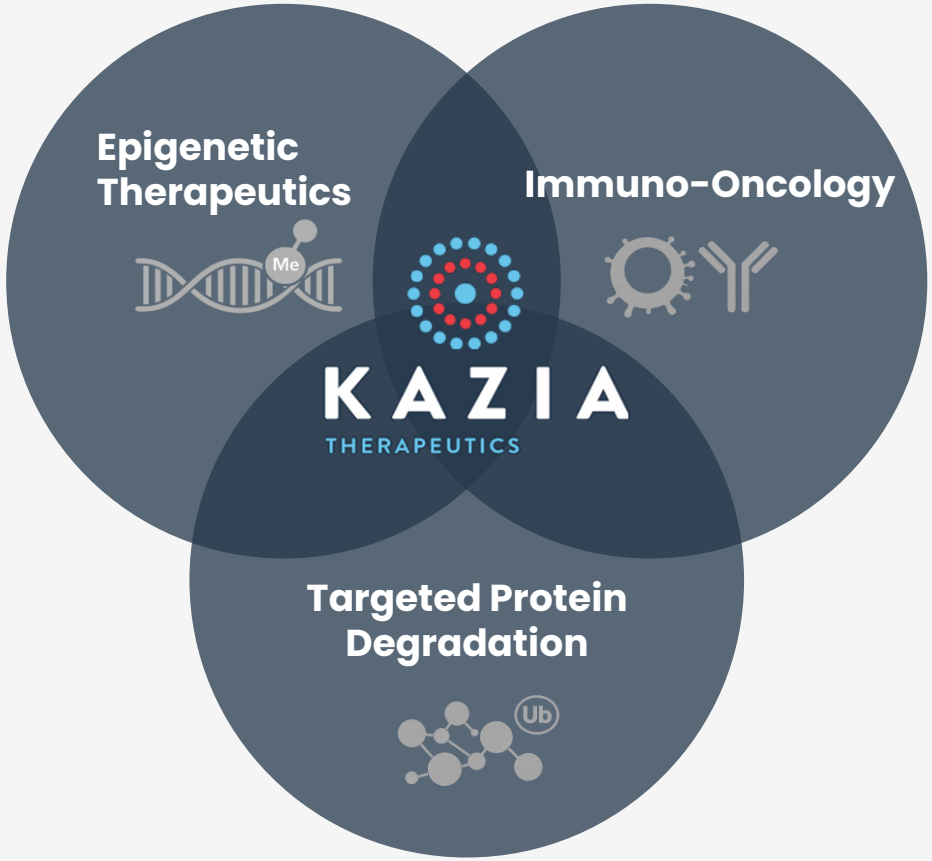
05
- Cash & Cash Equivalents: Approximately US$46 million (Runway expected to fund planned operations into 2029, excluding additional or expanded trials)
- No outstanding debt
- American Depository Shares Outstanding: Approximately 11.4 million

Our Approach

We believe Kazia is positioned at the intersection of three of the fastest-growing and strategically important areas in oncology.

A New Approach to Cancer

- Cancer progression, metastasis, and treatment resistance are increasingly understood not as single-gene problems, but as failures of regulatory control.
- Transcriptional programs, chromatin states, and immune checkpoints adapt dynamically to evade therapy.
- The next wave of oncology innovation is shifting beyond simple inhibition toward therapies that reprogram cancer biology.



Complementary Layers of Cancer Control

Kazia is building a differentiated oncology portfolio aligned with the current shift in oncology, designed to overcome resistance in aggressive and immunotherapy-refractory cancers.

Paxalisib

Brain-penetrant dual PI3K / mTOR inhibitor with epigenetic and immunomodulatory activity

Epigenetic Pathway Modulation

- Inhibits PI3K-driven epigenetic regulators
- Reprograms metastatic and immune-suppressive tumor states

Enhances Immune Responsiveness

- Reduces tumor-driven immune suppression
- Improves sensitivity to immunotherapy and other targeted therapeutics

Clinically Validated Asset

- Evidence of clinical activity across CNS, breast cancer, and other solid tumors
- Fast Track, Orphan Drug, and Rare Pediatric Disease designations from US FDA

Strategic Backbone Across CNS & Breast Cancer

- Demonstrated relevance in high-unmet-need CNS tumors, including glioblastoma
- Expanding applicability into large breast cancer populations beyond TNBC
- Shared biological drivers enable multi-indication development and commercial scale

Safety and tolerability evaluated in >550 patients to date
Phase 1b clinical study in TNBC ongoing

PD-L1 Protein Degrader Platform

First-in-class optimized PD-L1 protein degrader

NDL2 (Lead Compound) is First-in-Class PD-L1 Protein Degrader

- Target intracellular PD-L1 protein rather than transiently block receptor–ligand interaction
- Designed to overcome mechanisms of resistance to antibody-based checkpoint inhibitors

Epigenetic & Immune Reprograming of Resistant Tumors

- Targets tumor-intrinsic immune evasion not reached by existing immunotherapies
- Potential to restore immune sensitivity in refractory tumors

Platform & Combination Potential

- Potential broad applicability across solid tumors and rationale for combination with pathway inhibitors and cytotoxics
- Potential backbone for next-generation immuno-oncology regimens

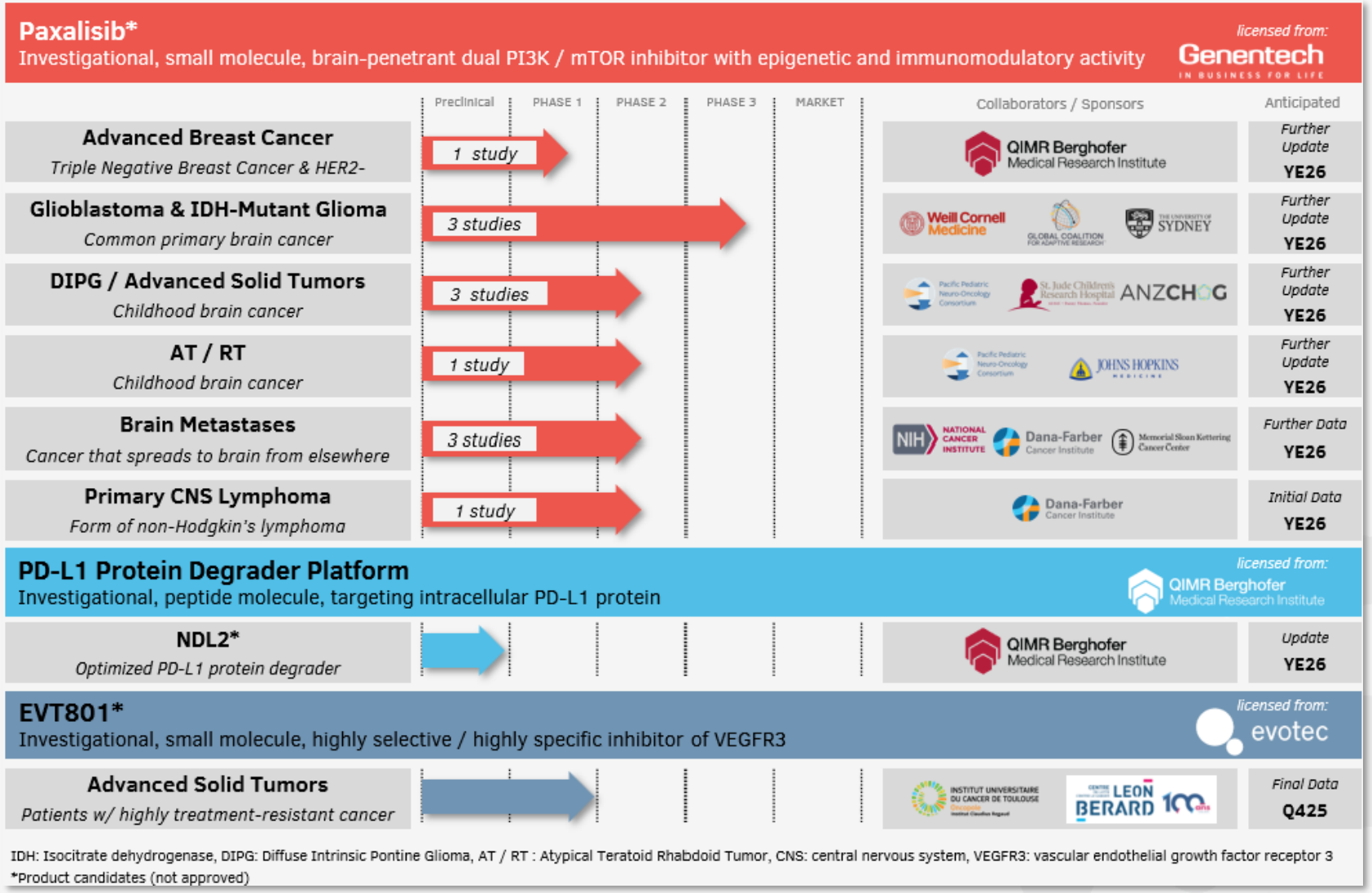
Clear Development & Value Inflection Path

- Designed for rapid transition to first-in-human trials
- Multiple preclinical and translational milestones prior to clinical proof-of-concept

IND initiating studies commencing 2026

Pipeline Overview

High-value indications with strong partnering potential



IDH: Isocitrate dehydrogenase, DIPG: Diffuse Intrinsic Pontine Glioma, AT / RT : Atypical Teratoid Rhabdoid Tumor, CNS: central nervous system, VEGFR3: vascular endothelial growth factor receptor 3

*Product candidates (not approved)

KAZIA THERAPEUTICS

Development Plan At a Glance

Trial ID	Indication	Sponsor	1H-26	2H-26	1H-27	2H-27	1H-28	2H-28	1H-29	2H-29	1H-30	2H-30
Paxalisib												
ACTRN12624001340527	TNBC	Kazia Therapeutics	Phase 1b				Phase 2 / 3					
NCT03970447	Glioblastoma	Global Coalition for Adaptive Research (GCAR)	CSR Pending									
NCT07391215	Glioblastoma Multiforme Malignant Primary Gliomas	Institute of Cancer Research (UK)	Phase 2									
NCT05183204	Glioblastoma	Weill Medical College of Cornell University	Phase 2									
ACTRN12623000096651	Gliomas	The University of Sydney	Phase 2									
NCT06208657	Childhood Brain Cancer	Australian & New Zealand Children's Haematology / Oncology Group	Phase 2									
NCT05009992	Diffuse Midline Gliomas	Pediatric Neuro-Oncology Consortium (PNOC)	Phase 2									
NCT07447076	AT / RT	Pediatric Neuro-Oncology Consortium (PNOC)		Phase 2								
NCT04192981	Brain Metastases	Memorial Sloan Kettering Cancer Center	Phase 2									
NCT03994796	Brain Metastases	Alliance for Clinical Trials in Oncology	Phase 2									
NCT04906096	Primary CNS Lymphoma	Dana-Farber Cancer Institute	Phase 2									
NDL2	Advanced Solid Tumors	QIMR Berghofer Medical Reseach Institute	IND-Enabling Studies		Phase 1							
EVT801	Advanced Solid Tumors	Kazia Therapeutics	Regional / global patnership search									

KAZIA THERAPEUTICS

Market Landscape: Growth & Opportunity

- We believe oncology innovation will drive exponential market growth.
- New modalities are expanding therapeutic options for previously resistant cancers.
- We believe Kazia's pipeline is strategically aligned with these high-growth areas.

Epigenetic Drugs

- **Current Market:** $15–20B
- **Projected 2034:** $80B+
- **Growth Driver:** Oncology applications

Immuno-Oncology

- **Current Market:** $83.34B
- **Projected 2035**: $409.44B
- **Growth Driver**: Increasing prevalence of cancer and need for innovative therapies, rising success of immunotherapies

Targeted Protein Degradation

- **2025 Market:** $699.3m
- **Projected 2033:** $3.26B
- **Growth Driver:** Prevalence of chronic disease conditions such as cancer, demand for novel treatments

1. Epigenetic Drugs Market 2. Immuno-Oncology Market 3. Targeted Protein Degradation Market

KAZIA THERAPEUTICS

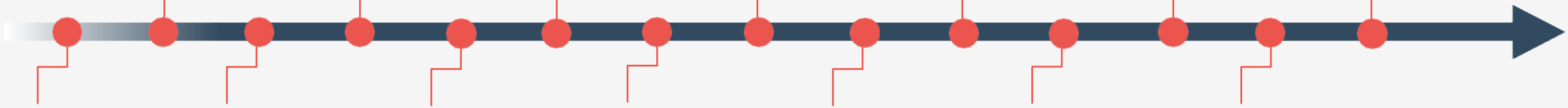
Paxalisib

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	Full IP portfolio comprising three families of patents: compound, process, and secondary with major jurisdictions including U.S., Europe, Japan, China, and India

Developmental History & Milestones

Expanding the clinical footprint of paxalisib into solid tumors beyond CNS



Early 2000's
Genentech develops **GDC-0084** as a potential new therapeutic for glioblastoma

2012–2015
Genentech **Phase 1** clinical study in 47 patients with advanced, **high-grade glioma**. Study demonstrated a favorable safety profile and provided efficacy signals

2016
Kazia in-licenses GDC-0084 from Genentech following deep due diligence which included Phase 1 study and animal data

February 2018
GDC-0084 awarded **Orphan Drug Designation** by the US FDA in glioblastoma

March 2018
Kazia commences company-sponsored **Phase 2** clinical study of GDC-0084 as a first line therapy in patients with **glioblastoma**

August 2019
GDC-0084 becomes '**paxalisib**' with the granting of an International Non-Proprietary Name (INN) by the World Health Organization

August 2020
Paxalisib granted **Breakthrough Designation** by the US FDA for glioblastoma

January 2021
GBM AGILE pivotal study commences recruiting paxalisib arm

July 2022
Paxalisib granted **Rare Pediatric Disease Designation** by the US FDA for **rare childhood brain cancer, AT / RT**

July 2023
Paxalisib received **Fast Track Designation** by the US FDA for solid tumor **brain metastases** with PI3K pathway mutations in combination with radiation therapy

July 2024
GBM AGILE Phase 2/3 trial data showed clinically meaningful improvement in a prespecified secondary analysis for OS in paxalisib-treated, newly diagnosed unmethylated patients with glioblastoma

June 2025
Kazia commences **Phase 1b ABC-Pax study** of paxalisib in combination with immunotherapy in patients with **triple negative breast cancer (TNBC)**

July 2025
ABC-Pax Phase 1b preliminary results from the first TNBC patient achieved >50% reduction in circulating tumor cells

January 2026
Complete metabolic response in a stage IV TNBC patient treated under expanded access, and **two partial responses** reported in the ongoing Phase 1b study in metastatic TNBC

KAZIA THERAPEUTICS

Paxalisib (GDC-0084)

Designed to be best-in-class PI3K pathway inhibitor with modest mTOR activity

Relevant marketed or active clinical P13K inhibitor programs

	Paxalisib (Kazia)	**PIKTOR** (two drugs, Faeth, acquired by Sensei Bio)	**Zovegalisib (RLY-2608)** (Relay Therapeutics)	**Alpelisib** (Novartis)	**Gedatolisib** (Celcuity)	**WXFL-10030390** (Jiatan)2
Targets	PI3K / mTOR	PI3Kα + mTOR	PI3Kα	PI3Kα	PI3K / AKT / mTOR	PI3K / mTOR
Development Stage	P3 (GBM) P1 (TNBC & HER2- BC)	P2 (EC, OC) P1(HR+ / HER2-BC)	P3 (HR+ / HER2- BC)	Marketed (HER2- BC)	P3 (HR+ / HER2- BC)	P2 in China (solid tumors)
Safety	Limited toxicities	Grade 3 AEs with paclitaxel	Grade 3 AEs with fulvestrant	Severe Hyper-sensitivity warning[3]	Grade 4 neutropenia reported in combo with palbociclib[1]	Unknown
ROA	Oral (QD)	Oral	Oral	IV	IV	Oral
Brain Penetration	Yes	Unknown	Unknown	Poor	Partial	Unknown

Five PI3K inhibitors have been approved by the US FDA.

Zydelig (idelalisib) 150 mg 100 mg tablets	Chronic lymphocytic leukemia Follicular lymphoma
Aliqopa (copanlisib) 60 mg vial for injection	Follicular lymphoma
Copiktra (duvelisib) 15mg | 25mg capsules	Chronic lymphocytic leukemia Follicular lymphoma
PIQRAY (alpelisib) tablets 50 mg · 150 mg · 200 mg	Breast cancer
UKONIQ umbralisib 200 mg tablets	Follicular lymphoma

EC: endometrial, OC: ovarian cancer, ROA: route of administration 1. Layman et al 2024 2. GlobalData 3. Piqray

Unlocking the Potential of PI3K / mTOR Pathway Inhibition

Brain penetrant PI3K / mTOR inhibitor with epigenetic and immunomodulatory activity

While traditionally classified as a kinase inhibitor, emerging clinical and translational data support a broader and more differentiated mechanism of action.

Traditional View

Core Signalling Inhibition – PI3K / mTOR Axis

- Disrupts key oncogenic networks controlling tumor growth, survival, metabolism, and protein translation

New Evidence

Downstream Epigenetic Modulation (EZH2 Axis)

- Alters epigenetic regulators including EZH2
- Reshapes chromatin accessibility and transcriptional programs linked to metastasis and therapeutic resistance

Transcriptional Reprogramming & Cellular State Shifts

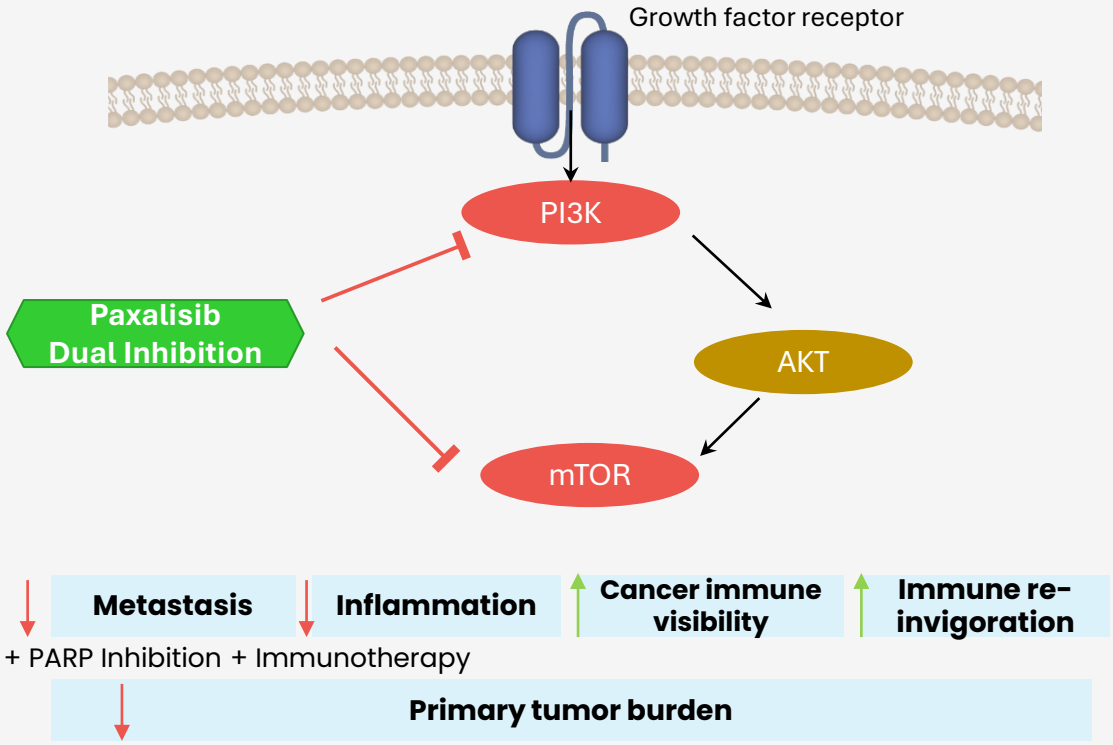
- Suppresses metastatic and resistance markers
- Shifts tumor biology away from mesenchymal, invasive states toward less aggressive phenotypes

Tumor Immune Microenvironment Modulation

- Reduces inflammatory and immune exhaustion signatures
- Enhances immune visibility through viral mimicry pathways and supports synergy with immune checkpoint inhibition

Paxalisib Mechanism of Action

PI3K oral inhibitor clinically proven to cross the blood-brain barrier



Comparative Potency vs. Other PI3K Inhibitors

	IC_{50} (nM)				
	p110 α	p110 β	p110 γ	p110 δ	mTORC 1/2
Paxalisib	**2**	**46**	**10**	**3**	**70**
Idelalisib	820	565	89	2.5	>1,000
Alpelisib	5	1200	250	290	>9,100
Buparlisib	52	166	262	116	4,600
Pilaralisib	39	383	23	36	>15,000
Taselisib	0.3	9.1	1.0	0.1	1,200
Pictilisib	3	33	75	3	580

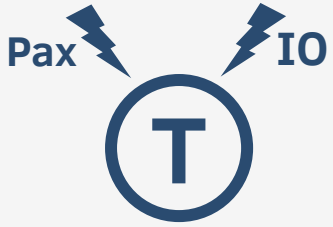
Note: lower IC_{50} implies more potent activity
Source: HF Zhao et al. (2017) *Molecular Cancer*. 16:100

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.

Licensing & Collaborations

Opportunistic partnering and strategic collaborations continue to add value

Licensing

Summary	Simcere	Sovargen
Territories and Responsibilities	To develop and commercialize paxalisib in Greater China, Hong Kong, Macau, and Taiwan	To develop, manufacture, and commercialize paxalisib as a potential treatment for intractable epilepsy in focal cortical dysplasia type 2 (FCD T2) and tuberous sclerosis complex (TSC) disease
Upfront Payment	US$11m, comprising US$7m in cash and a US$4m equity investment	US$1.5m
Milestone Payments	Contingent milestone payments of up to US$281m in GBM + further milestones payable in indications beyond GBM	Potential milestone payments of up to US$19m upon the achievement of development and regulatory milestones
Royalties on Net Sales	Mid-teen percentage royalties on commercial sales	A percentage of sub-licensing revenues and royalties on net sales of products incorporating paxalisib

Key Collaborations

QIMR Berghofer Medical Research Institute

Cutting-edge preclinical program to evaluate paxalisib in combination with immuno-therapies for advanced breast cancer

Pacific Pediatric Neuro-Oncology Consortium | **Johns Hopkins University**

- Paxalisib alone and in combination with other targeted agents is active in preclinical models of DIPG and AT / RT.
- US FDA has awarded Orphan Drug Disease and Rare Pediatric Disease Designations in DIPG and AT / RT.
- If paxalisib were to be approved, Kazia could be entitled to receive a pediatric priority review voucher which are tradeable and have historically commanded prices in excess of USD $100 million.

Paxalisib Potential in Breast Cancer

The most commonly diagnosed cancer and leading cause of death cancer in women

Breast Cancer

- >300,000 new cases of invasive BC in the US

Triple Negative Breast Cancer

- Rare, fast-growing, and aggressive form of breast cancer
- Accounts for approximately 10-15% of all breast cancer
- 5-year survival rate of 3-5%
- Predicted market growth of $1.5B by 2030

P13K / mTOR in Breast Cancer

P13K / mTOR signaling is frequently dysregulated in breast cancer and associated with tumor proliferation, disease progression, and therapeutic resistance.

Brain metastases develop in ~25–46% of patients with metastatic TNBC over the disease course, with ~10–15% present at initial metastatic diagnosis.

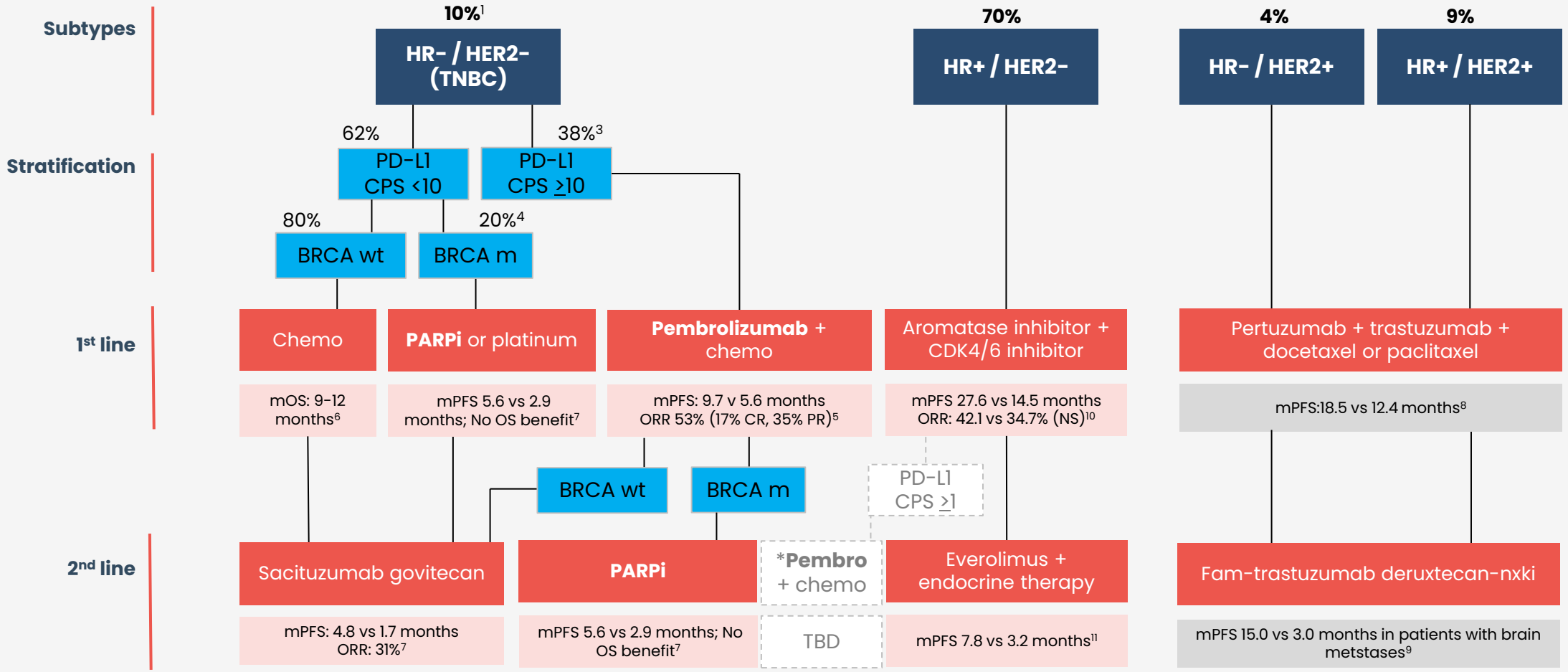
Paxalisib Opportunity

Paxalisib inhibits PI3K / mTOR, a central oncogenic pathway driving cell survival and proliferation, resistance to endocrine, HER2-targeted, and cytotoxic therapies, and immune suppression within the tumor microenvironment (TME).

1. Cancer.org 2024 2. Data Bridge Market Research

Current Treatment Landscape

Modest results for HER2- subtypes, despite checkpoint and PARP inhibitors



* Potential future regimen, depending on Keynote B49 readout (NCT04895358); CPS = Combined Positive Score; wt = wildtype; NS = not statistically significant

1. BC subtypes and distribution: (7% unknown subtype) Cancer.org; 2. Treatment: NCCN Guidelines Version 4.2025, simplified and with focus on preferred regimens; 3. https://www.pdl1portal.com/tnbc/; 4. Pavese et al. 2022; 5. Keytruda.com; 6. Khosravi-Shani et al 2017; 7. Gacia-Saenz et al 2025; Bardia et al 2024; 8. Baselga et al 2012; 9. Jacobson 2022; 10. Wu et al 2020; 11. Yardley et al 2013

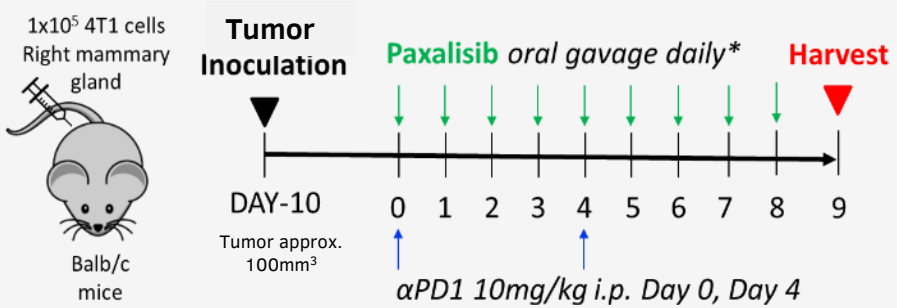
KAZIA THERAPEUTICS

Consistent & Statistically Significant Signals

Preclinical studies combining paxalisib with either checkpoint inhibitor or PARP inhibitor resulted in highly consistent and statistically significant signals of efficacy.

4T1 Mouse Model

- Standard model for TNBC
- Immunotherapy resistant model
- Highly tumorigenic and invasive



Checkpoint Inhibitor

- Reduced tumor volume
- ↓ Lung metastases
- ↓ Lymph node metastases
- ↓ Liver inflammation
- ↓ Lung inflammation
- ↓ Liver and spleen EMH
- No observed toxicity

PARP Inhibitor

- Reduced tumor volume
- ↓ Lung metastases
- ↓ Lymph node metastases
- ↓ Liver inflammation
- ↓ Lung inflammation
- ↓ Liver and spleen EMH
- No observed toxicity

* αPD1: pembrolizumab or “pembro”

Paxalisib in Triple Negative Breast Cancer

"*In treatment-resistant preclinical models of breast cancer, paxalisib (4T1 mouse model, TNBC) has shown encouraging results in inhibiting both the primary tumor burden and metastasis by reinvigorating the immune system within the tumor microenvironment*" – Professor Sudha Rao, Group Leader, QIMR Berghofer

- Leading transcriptional biology and epigenetics expert, Professor Rao identified an entirely novel effect of PI3K inhibition:
  - Immune modulator of the tumor and the surrounding microenvironment
  - Administration of PI3K inhibitors such as paxalisib, at doses and frequencies different to those conventionally used, appears to activate or reinvigorate the immune system in the tumor, making it more susceptive to immunotherapy
- Preliminary data from our collaboration was presented at San Antonio Breast Cancer Symposium in 2024 and 2025

Combination Paxalisib + KEYTRUDA® (pembrolizumab) data in TNBC preclinical models

Combination Paxalisib + LYNPARZA® (olaparib) data in advanced breast cancer preclinical models

Paxalisib influence on immune system (example, T cells, B cells, NK cells) and within the tumor and its micro-environment

Intellectual Property (IP)

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 15mg QD + olaparib 300 mg BID, n=6
- Paxalisib 30mg QD + olaparib 300 mg BID, n=6

Paxalisib plus Pembrolizumab / Chemotherapy (21-Day Cycle)

- Paxalisib 15mg QD + pembrolizumab 200mg + clinician's choice of IV chemotherapy QW3, n=6
- Paxalisib 30mg QD + pembrolizumab 200mg + clinician's choice of IV chemotherapy QW3, n=6

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 patient achieved a confirmed complete metabolic response following re-treatment with pembrolizumab / chemotherapy plus paxalisib (under an expanded access protocol)
- 2/2 evaluable 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

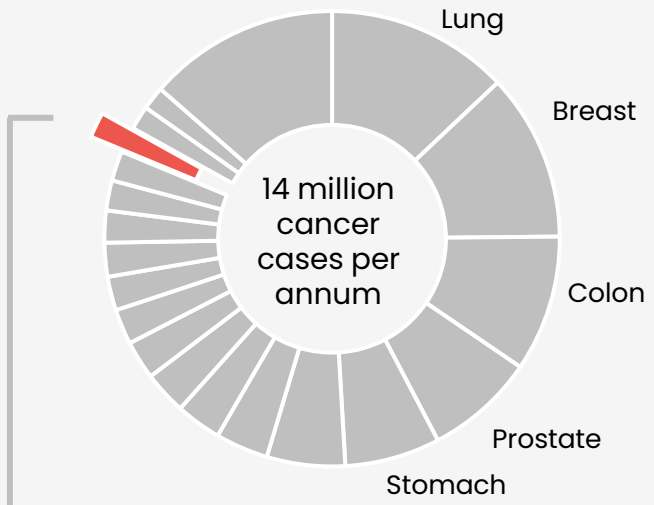
Safety

- 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

Paxalisib Potential in Glioblastoma

The most aggressive malignant brain cancer



Glioblastoma Multiforme

- 133,000 cases per annum worldwide
- 5-year survival of 3-5%
- $3.6B market size (2024)

P13K / mTOR in Glioblastoma

PI3K / mTOR signaling pathway is a central regulator of cell growth and is hyperactivated in approximately ~85% to 90% of glioblastoma cases.

Paxalisib Opportunity

Unlike other PI3K inhibitors approved for peripheral cancers, paxalisib crosses the blood-brain barrier overcoming a major obstacle in CNS drug delivery.

Data on file. Market research performed 2021. Glioblastoma Multiforme Treatment Market Size Report, 2034

Paxalisib in Newly Diagnosed Unmethylated GBM Patients

Consistent median overall survival data

	Phase 2 (NCT03522298)	GBM-AGILE (Phase 2/3) (NCT03970447)	
Study Type	Open-label, single arm	Adaptive, multi-arm randomized	
Patient Population	Newly diagnosed unmethylated GBM	Newly diagnosed unmethylated GBM and recurrent GBM	
MGMT Status	Enriched for unmethylated	Mixed: subgroup analyses	
Comparator	Historical SOC	Concurrent SOC control	
Primary Endpoint	**OS**	**OS Paxalisib**	**OS SoC**
mOS	**15.7 months (n=30)**	**15.54 months* (n=54)** in prespecified secondary analysis in NDU GBM	**11.9 months* (n=46)** in prespecified secondary analysis in NDU GBM

*GBM-AGILE; Prespecified secondary analysis of median Overall Survival

Overall Summary

Paxalisib demonstrated a ~3.8 month improvement in median Overall Survival

- 15.5 months vs. 11.9 (SoC) in a prespecified secondary analysis in newly diagnosed unmethylated GMB (GBM-AGILE) and 15.7 months in the Phase 2 trial

Represents a strong OS signal reported in this GBM population

Designations granted:

- Orphan Drug, Fast Track, and Rare Pediatric Disease

NDU: Newly Diagnosed Unmethylated, OS: Overall Survival

US FDA Type C Meeting

Paxalisib in GBM commercial and development path forward

December 2024

FDA position:

- Data on OS would generally not be appropriate for accelerated approval but could be considered to support a traditional / standard approval
- Secondary endpoint OS data from the GBM-AGILE study may be supportive and informative for designing and executing a pivotal registrational study in pursuit of a standard approval

Kazia position & next steps:

- Aligned with the FDA on key aspects of the design of a proposed registrational / pivotal Phase 3 study in NDU GBM patients
- To finalize the protocol for the pivotal Phase 3 study and discuss with a number of global contract research organizations (CROs) with experience in the neuro-oncology drug development space

Kazia Press Release 31 December 2024

October 2025

Kazia plans to request follow-up meeting with the FDA to:

- Discuss overall survival (OS) findings in NDU GBM patients treated with paxalisib
- Seek agency feedback on a potential commercial and development pathway aligned with the FDA Oncology Center of Excellence's Project FrontRunner initiative

Kazia Press Release 27 October 2025

2026

FDA follow-up next steps:

- Meeting anticipated for 1H 2026
- Anticipated outcome: Update from FDA discussion regarding paxalisib commercial and development path forward in GBM

Paxalisib Potential in Childhood Brain Cancer

High unmet need especially in patients with diffuse midline gliomas (DMGs)

Childhood Brain Cancer

- The most common malignancy of childhood
- Represents about one third of childhood cancer deaths
- 5-year survival rates below 20% in pediatric high-grade gliomas (particularly DMGs)
- Predicted market growth of $3.5B by 2030

P13K / mTOR in Childhood Brain Cancer

PI3K / mTOR signaling pathway is a central regulator of cell growth and is hyperactivated in approximately ~65% to 70% of pediatric high-grade brain tumors, including DMGs and atypical teratoid / rhabdoid tumors (AT / RT).

Paxalisib Opportunity

Given the frequent dysregulation of the PI3K / mTOR pathway in pediatric brain tumors and limited effective therapies, paxalisib's ability to cross the blood–brain barrier and combine with multiple treatment modalities presents a compelling therapeutic opportunity.

1. NCI: Childhood Brain and Other Nervous System Cancer 2. NIH: PI3K Pathway in Pediatric Brain Tumors 3. Strategic Market Research: Pediatric Brain Tumor Market Report

Paxalisib in Childhood Brain Cancer

	Kazia is actively pursuing three forms of childhood brain cancer:		
	Diffuse Midline Gliomas (DMG, DIPG)	**Atypical Teratoid / Rhabdoid Tumors (AT / RT)**	**Advanced Childhood Cancer (PI3K / mTOR activated)**
Preclinical Research	Positive preclinical data in combination with ONC201	Positive preclinical data as monotherapy and in combination (AACR 2022, 2023, 2024)	Research proposals under discussion
Clinical Trials	Phase 1 monotherapy clinical trial at St. Jude Children's Research Hospital completed (NCT03696355) PNOC022, Phase 2 clinical trial in combination with ONC201, ongoing (NCT05009992)	PNOC035, Phase 2 clinical trial evaluating paxalisib safety and efficacy Awaiting site initiation and first patient enrolled (NCT07447076)	Phase 2 clinical trial in combination with chemotherapy for treatment of high-risk malignancies commenced 2024 (ACTRN12623000096651)
Regulatory Interaction	Orphan Drug Designation (ODD) and Rare Pediatric Disease Designation (RPDD) granted by FDA in Aug 2020	ODD and RPDD granted by FDA in June and July 2022, respectively	Regulatory strategy under discussion

PNOC: Pediatric Neuro-Oncology Consortium

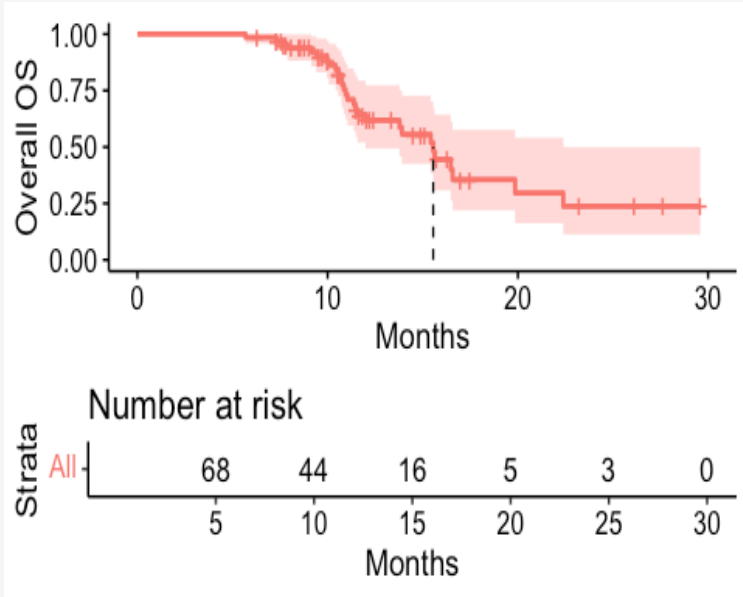
Paxalisib in Diffuse Midline Gliomas (DMG)

PNOC-sponsored Phase 2 trial in combination with ONC201

In spite of research that has helped improve treatment for DIPG patients, the prognosis remains poor with the median survival range being from 8-11 months.[1]

- 68 patients with biopsy-proven DMG were enrolled in the PNOC Phase 2 study between November 2021 and June 2023 (median age 9 years [range 3-37], n=41, female [60%])
- Updated Median OS from time of diagnosis was 15.6 months (Confidence interval (CI) 12.0, 22.4)
- Cohort 3 enrolled 30 recurrent patients (in conjunction with radiation therapy) had median OS 8.7 months [CI 95% 8.5, NA]
- Most common grade 3 and above treatment-related adverse events were decreased neutrophil count (n=4); mucositis (n=3); and colitis, drug reaction with eosinophilia and systemic symptoms, decreased lymphocyte count, hyperglycemia, and hypokalemia (n=2)
- Next Steps: Further PK and biomarker analyses ongoing for subsequent cohorts; anticipate further update 2026

Overall Survival - Cohort 2 (post RT)
Median OS 15.6 months [CI 95%; 12.0, 22.4]



Central imaging review analysis of PFS ongoing

Trial ID: NCT05009992 1. Hargrave, D., Bartels, U. & Bouffet, E. Diffuse brainstem glioma in children: critical review of clinical trials. Lancet Oncol 7, 241-8 (2006)

KAZIA THERAPEUTICS

Paxalisib in Brain Metastasis

MSKCC-sponsored Phase 1 trial

Interim analysis showed encouraging clinical activity of paxalisib in combination with radiation therapy.

August 2022

Data from first stage presented at **2022 Annual Conference on CNS Clinical Trials and Brain Metastases**, Toronto, Canada

All 9 patients evaluated for efficacy exhibited a clinical response, according to RANO-BM criteria, with breast cancer representing the most common primary tumor

July 2023

Fast Track Designation granted by US FDA for paxalisib in combination with radiation therapy in patients with solid tumor brain metastases and PI3K pathway mutations

Preliminary data presented at two 2024 scientific congresses: American Society for Radiation Oncology Annual Meeting (ASCO) & Society for Neuro-Oncology Annual Meeting (SNO)

February 2024

Announced early conclusion, based on Stage 2 positive safety data and **promising clinical response** findings observed to date.

Full clinical study report pending

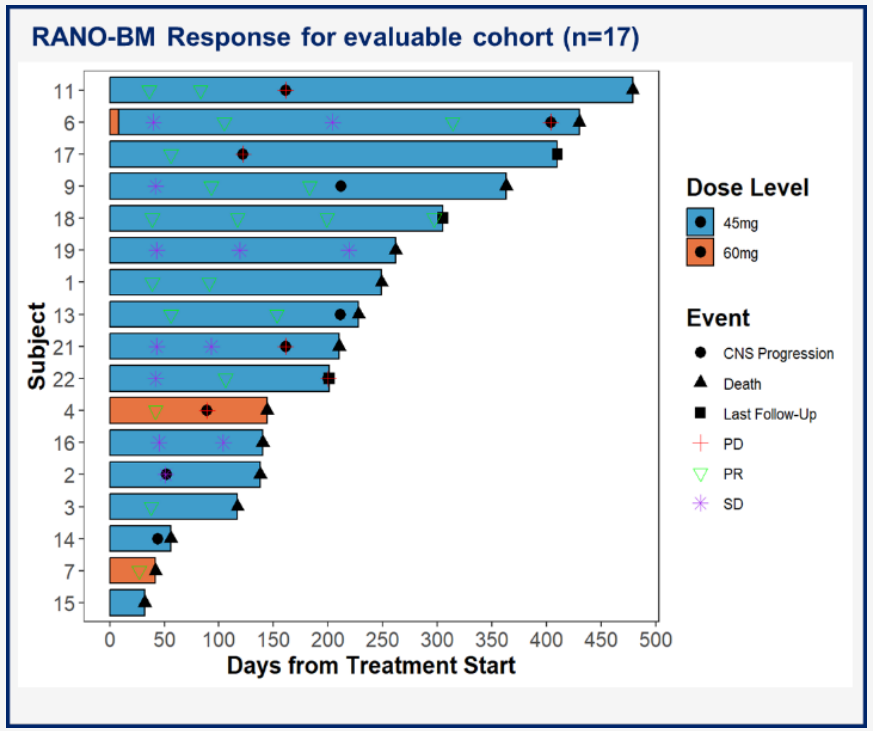
Trial ID: NCT04192981

Paxalisib in Brain Metastasis

MSKCC-sponsored Phase 1 trial

Interim analysis showed encouraging clinical activity of paxalisib in combination with radiation therapy.

Response signal seen for concurrent paxalisib and brain RT



Overall Summary

Primary objective of identifying the maximum tolerated dose (MTD) was met:

- Concurrent daily administration of paxalisib with brain radiotherapy was generally well-tolerated at a maximum dose of 45mg per day in advanced solid tumor patients with brain metastases and PI3K pathway mutations

Over two-thirds of the patients at MTD achieved intracranial response compared to historical response rates (20-40%)[1] for WBRT alone

Future goals include:

- Extending the duration of PI3K inhibition, neoadjuvant, adjuvant, and maintenance (ideally with complementary systemic therapy options)
- Integrating PI3K inhibition with CNS tumor types with relevant pathway driver mutations and potentially SRS

Trial ID: NCT04192981 RANO-BM: Response assessment in neuro-oncology brain metastases, WBRT: whole-brain radiation therapy, SRS: stereotactic radiosurgery 1. Zhou et al. 2021, Kim et al. 2020

Summary & Next Steps

Summary

- Brain-penetrant dual pan-PI3K / mTOR inhibitor in development (Only 2% of small-molecule drugs are brain-penetrant)
- Designed to be best-in-class pan-PI3K pathway inhibitor with modest mTOR activity (dual targeting required to inhibit cancer cell proliferation & migration in TNBC cell model)
- Overcoming metastasis and drug resistance in combination with immunotherapy (epigenetic reprogramming of dormant cancer cells and improved cancer immune visibility)
- Generally well tolerated (evaluated in 550 adult and pediatric patients across Phase 1-3 clinical trials and expanded access programs)

Clinical and Translational Epigenetic Data Demonstrate That Paxalisib:

- Reduces CTC and CTC-clusters as well as aggressive mesenchymal phenotypes (observable impact on metastatic biology)
- Enhances activity when combined with immune checkpoint inhibition (combination-enabling potential)
- Modulates immune exhaustion markers (evidence of tumor microenvironment reprogramming)

On Strategy

- Positioned as a backbone therapy within the emerging category of functional epigenetic reprogramming therapeutics

Next Steps

- **Advanced Breast Cancer**: 1) Provide additional preclinical data and updates from the QIMR collaboration throughout the year and 2) ongoing updates from Phase 1b advanced breast cancer clinical study (ACTRN12624001340527) throughout 2026
- **Glioblastoma**: 1) Anticipated follow-up FDA Type C meeting to discuss commercial and development path forward, 2) finalize Phase 3 approval protocol, assess costs / timelines, and select strategic CRO partner, and 3) updates from ongoing GBM trials
- **Pediatric & Brain Metastasis Programs**: 1) PNOC team to complete PK / biomarker data analysis and provide update, 2) complete analysis and close out MSKCC Phase 2 clinical brain metastasis study, 3) data release / updates from other brain met trials, and 4) initiate enrollment for PNOC035 AT / RT study

PD-L1 Protein Degrader Platform

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 Professor Sudha Rao (QIMR-Berghofer) in 2020
Development Stage	Preclinical; IND expected in 12-18 months
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

Current Anti-PD-1 / L1 Therapy Landscape

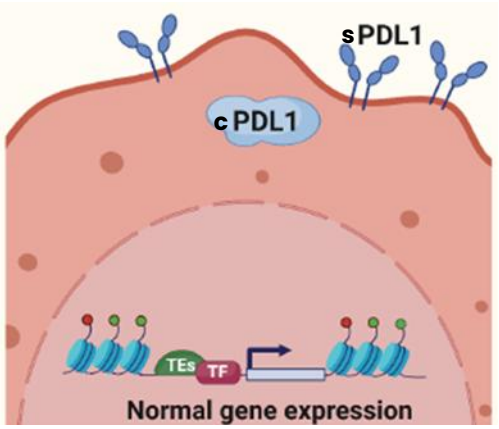
Limited by intracellular PD-L1

Distinct **distributions of PD-L1** (cell surface, cytosol, nucleus) have **different functions** on tumor progress.

Intracellular PD-L1 has been **implicated in immunotherapy resistance, metastatic progression, and immune suppression** independent of canonical PD-1 / PD-L1 extracellular signaling.

Targeting nPD-L1 could provide a **therapeutic benefit**, including in **patients who are resistant** to existing anti-PD-1 / L1 therapies.

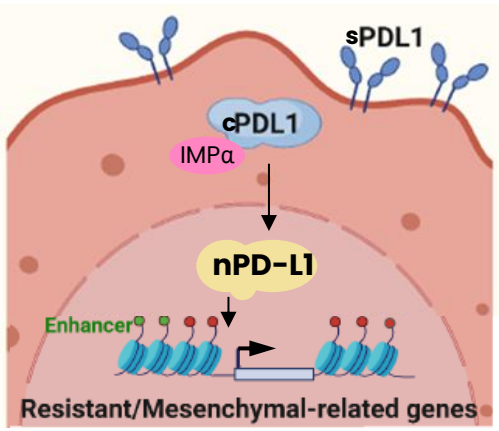
Healthy Cell



PD-L1 expressed on cell surface (sPD-L1)

- Binds to PD-1 on cell surface
- Prevents autoimmunity
- Limits tissue damage by suppressing overactive immune response

Cancer Cell



PD-L1 translocates into the nucleus (nPD-L1)

- nPD-L1 has been shown to induce transcription of genes leading to angio-genesis, tumori-genesis, drug resistance, and tumor recurrence
- Surface PD-L1 mediates immune evasion

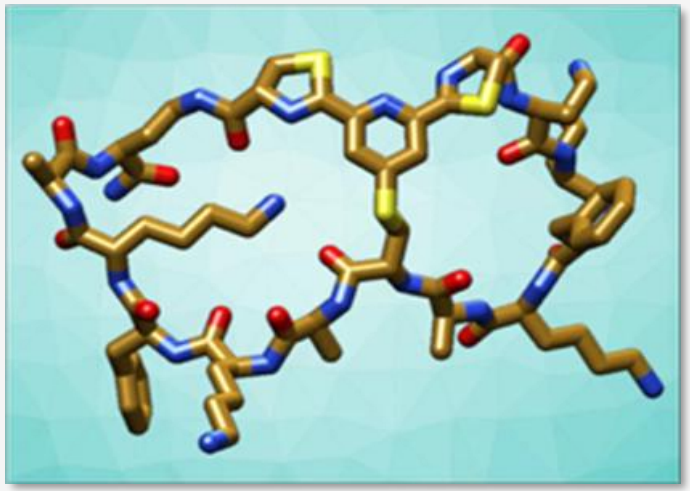
Asare-Werehene et al., Front. Immunol., 2025, DOI: 10.3389/fimmu.2025.1543529; Liu et al., Biochemical Pharmacology, 2024, DOI: 10.1016/j.bcp.2023.115984

KAZIA THERAPEUTICS

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

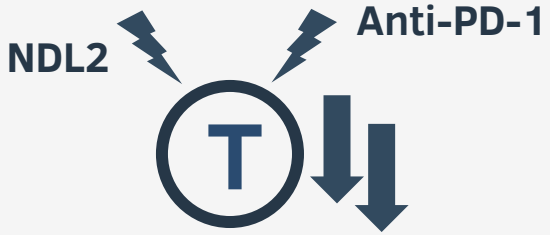
	NDL2 Peptide Degrader of PD-L1	PD-(L)1 mAbs
Primary Mechanism	Targeted protein degradation via intracellular E3 ubiquitin proteasome system	Block the PD-1:PD-L1 interaction at the cell surface; Fc effector function varies by isotype / engineering
Route of Administration (ROA)	IV	IV / SC
PD-L1 Pools Addressed	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

Preclinical & Translational Manifestations

Across multiple preclinical models and patient-derived samples, NDL2 demonstrated:

Reduced tumor growth as monotherapy



Enhanced activity of anti-PD-1 therapy in combination

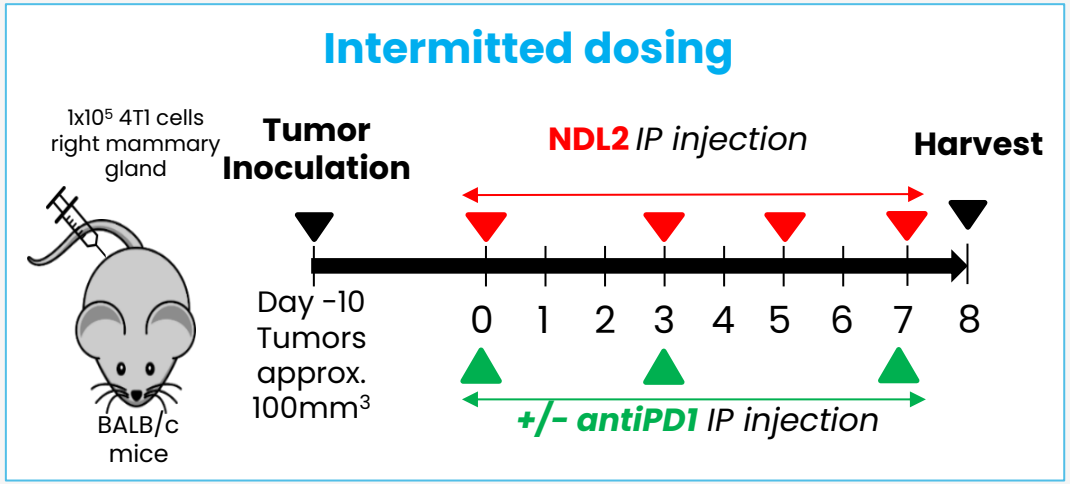
Suppressed metastatic spread in aggressive tumor models

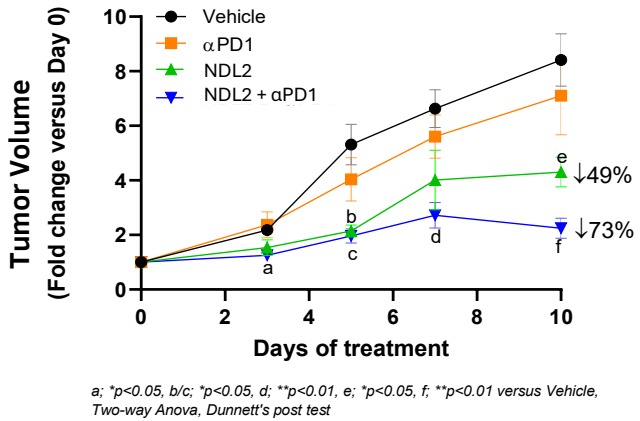
Targeted resistance mechanisms enriched in immunotherapy-refractory disease

Data generated collectively supports intracellular PD-L1 as a mechanistically distinct and therapeutically actionable driver of immune evasion, disease progression, and metastasis.

Efficacy in 4T1 Mouse Model

NDL2 monotherapy and anti-PD1 combination reduces primary tumor burden with intermittent dosing





*a; *p<0.05, b/c; *p<0.05, d; **p<0.01, e; *p<0.05, f; **p<0.01 versus Vehicle, Two-way Anova, Dunnett's post test*

- Gold Standard model for TNBC
- Immunocompetent BALB/c mice
- Highly tumorigenic and invasive

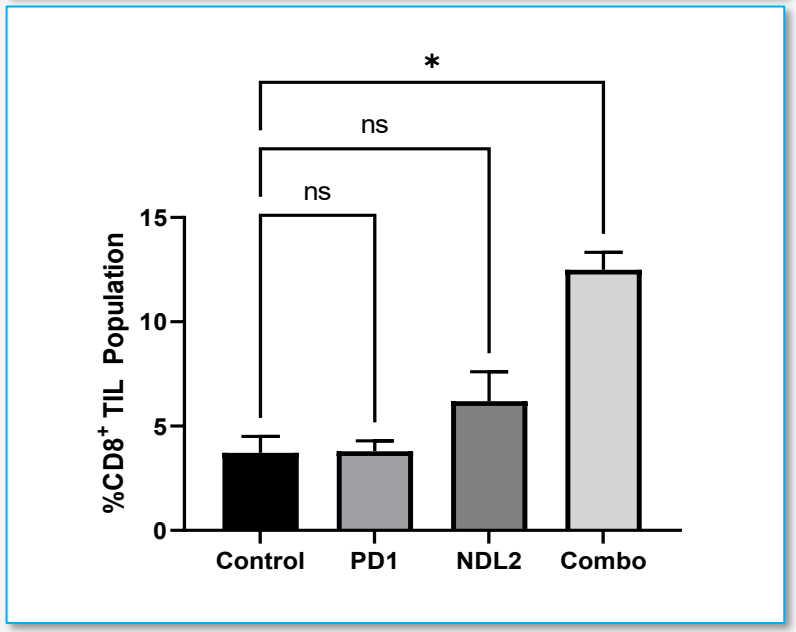
- NDL2 reduces primary tumor volume by 49% as a monotherapy
- NDL2 reduces primary tumor volume by 73% in combination with anti-PD1

KAZIA THERAPEUTICS

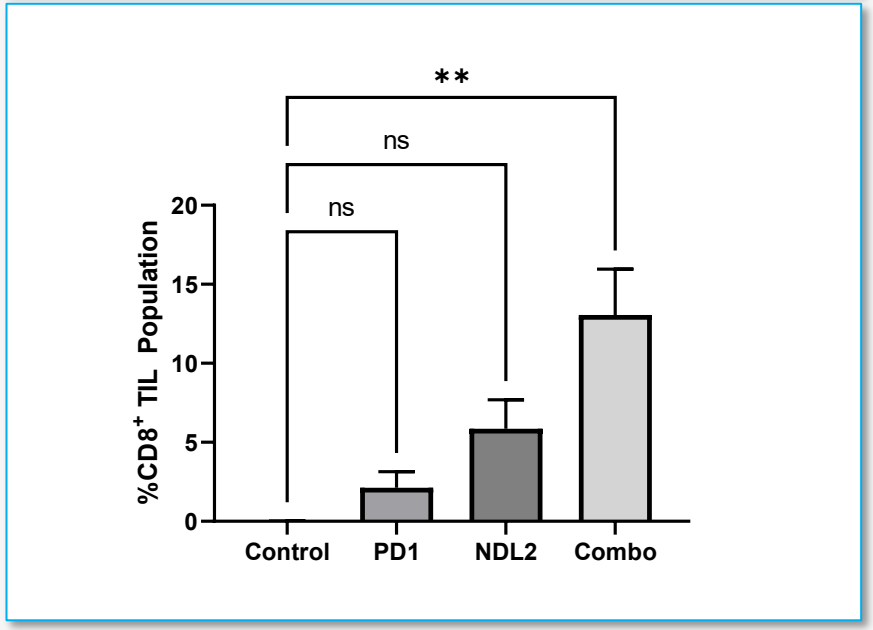
Efficacy in 4T1 Mouse Model (Cont.)

NDL2 and anti-PD1 combination therapy reduces T cell exhaustion

Increase in CD8⁺ T Cell Infiltration



Increases in CD8⁺ T Cell Proliferation

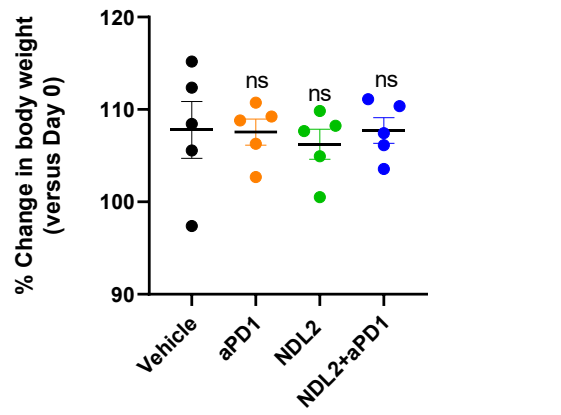


Method: FFPE sections from Control (vehicle), anti-PD1, NDL2 or NDL2 + anti-PD1 (10mg/kg) tumors were stained using the BondRX automatic FFPE staining platform and the change in expression characterized with high resolution digital pathology. Significant differences are calculated as one-way ANOVA Kruskal-Wallis test comparison to vehicle control (N = 3-5 mice per group).

Safety in 4T1 Mouse Model

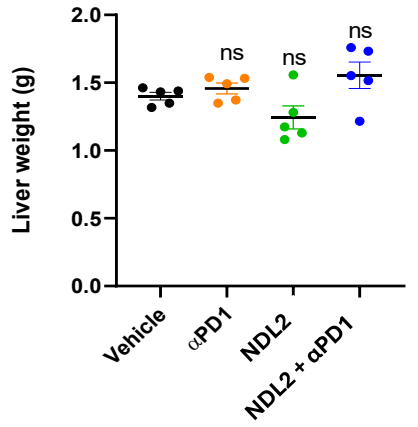
NDL2 shows no evidence of toxicity

Body Weight



* ns, not significant (verus Vehicle), One-way ANOVA, Dunnett's post test

Liver Weight



ns; not significant (versus Vehicle), One-way Anova, Dunnett's post test

- No toxicity observed for NDL2
- Normal body weight
- Normal liver weight for treatment duration

Summary & Next Steps

Innovation Breakthrough

- Selectively targets cancer intracellular enriched PD-L1 protein
- Amongst the first therapeutics to address metastasis-seeding cancer cells resistant to traditional immunotherapy
- Precisely degrades the resistance-associated form of PD-L1, preserving PD-L1 essential for normal immune regulation

Recent Preclinical and Translational Data Demonstrate That NDL2:

- Reduces tumor growth as monotherapy
- Enhances the activity of anti-PD-1 therapy
- Targets resistance mechanisms enriched in immunotherapy-refractory disease
- Preserves normal immune checkpoint function at the cell surface and therefore avoids toxicity associated with anti-PD-L1 agents

On Strategy

- NDL2 is reshaping cancer cell behavior by altering the functional protein landscape rather than simply blocking extracellular signaling
- NDL2 represents a complementary modality to epigenetic and transcriptional reprogramming, extending Kazia's strategy into targeted protein degradation

Next Steps

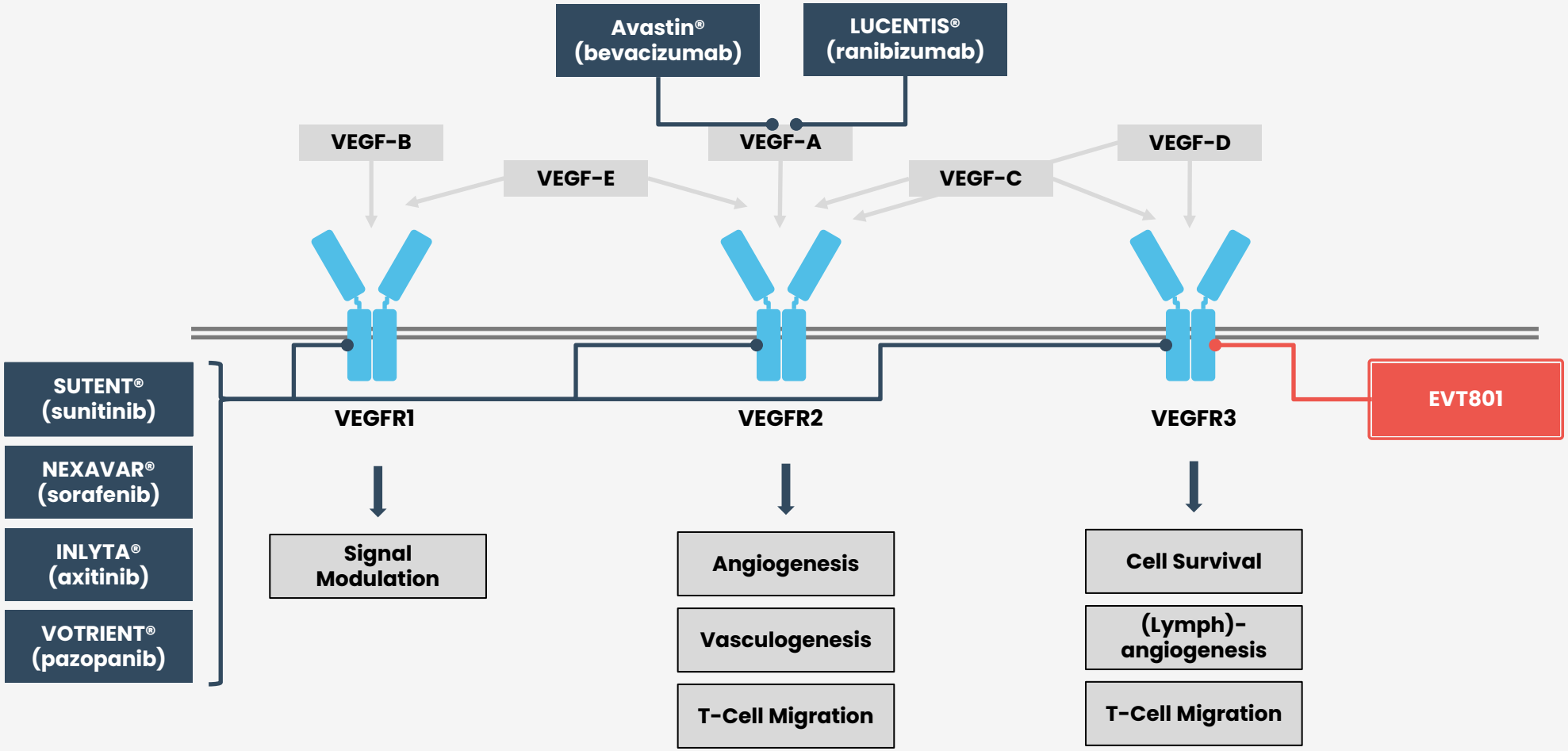
- Initiate IND enabling studies
- Integrate a clinical liquid biopsy to enable patient stratification, real-time monitoring, and personalized treatment optimization
- Launch PD-L1 degrader and NDL2 awareness campaign through abstracts, manuscripts, videos, and medical congress presentations

EVT801

Overview

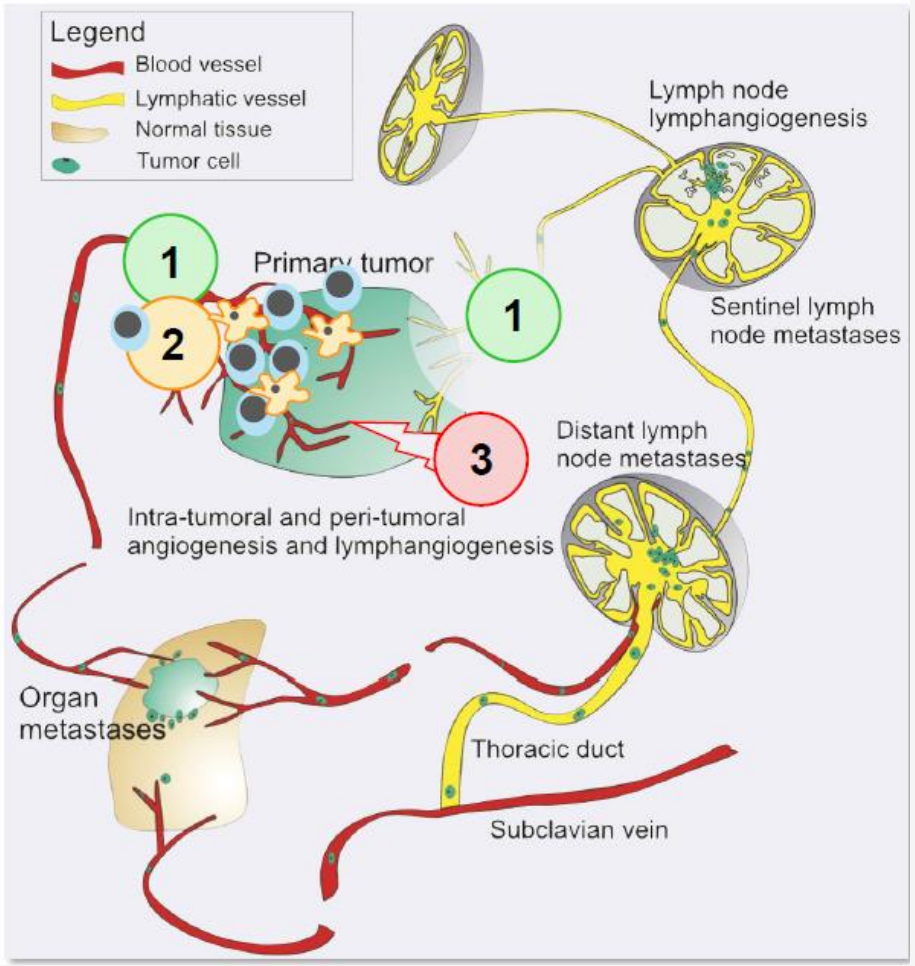
Asset Overview	A highly selective, potential first-in-class small molecule Vascular Endothelial Growth Factor Receptor-3 (VEGFR3) inhibitor, primarily inhibiting lymphangiogenesis
Origin	Discovered by Sanofi; Early development by Evotec SE; In-licensed by Kazia in 2021
Development Stage	Phase 1 completed
Lead Indications	Potentially high-grade serous ovarian cancer (HGSOC)
Formulation	Oral, capsule
Key Differentiators	Selective VEGFR3 inhibitor in clinical development; distinct from broader VEGF / VEGFR inhibitors that target multiple VEGF receptors, with the potential for a more favorable safety profile and reduced resistance due to hypoxia
Mechanism of Action	By inhibiting VEGFR-3, EVT801 restricts lymphangiogenesis (the formation of new lymphatic vessels), stabilizes tumor vasculature, reduces hypoxia, and thereby limits tumor escape and metastatic spread
FDA Designations	N / A
IP	Composition-of-matter to 2032 / 2033 in most jurisdictions (excluding PTE)

EVT801 Selectively Inhibits VEGFR3



KAZIA THERAPEUTICS

A Differentiated Anti-Tumor Approach



1 **Inhibition of tumor escape and metastasis**

- Stabilization of tumor vasculature
- Inhibition of (lymph)-angiogenesis
- Avoidance of hypoxia decreases potential for metastatic spread

2 **Increase in anti-tumor immune activity**

- No impact on T-cells viability
- Increased infiltration of effector T-cells
- Reduction in immunosuppressive myeloid cells

3 **Tumor Killing**

- Direct effect on VEGFR3-expressing tumor cells (typically from endothelial origin, e.g. sarcoma)

Phase 1 Dose-Finding Trial Completed

Study Background

Target population:
Histologically-confirmed advanced or metastatic solid tumors, unresponsive to standard treatment, or for whom no standard treatment is available or appropriate

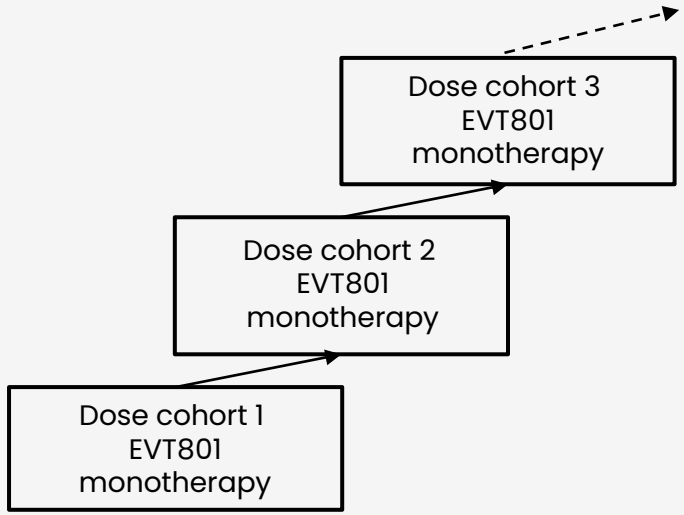
Sponsor: Kazia Therapeutics Limited

Product: EVT801

EudraCT Number: 2021-002483-47

Clinical Sites (France only):
- IUCT-Oncopole, Toulouse
  PI : Dr Gomez-Roca
- Centre Léon Bérard, Lyon
  PI : Dr Philippe Cassier

Design
Monotherapy dose escalation
N=26



- 6 cohorts
- Single-patient cohorts initially; expand to 3+3 when toxicity is encountered
- Mixed population of advanced solid tumors
- Doses from 50mg QD to 500mg BID

Overall Summary

Patients with 11 different cancer types were enrolled, with heavily pretreated advanced ovarian cancer being the most prevalent indication (11 patients)

MTD has been reached at 500mg BID

The recommended dose for Phase 2 is 400mg BID* in continuous monotherapy administration

Trial ID: KZA 0801-101, NCT05114668

MTD: Maximum Tolerated Dose; RP2D: Recommended Phase 2 Dose *Human active dose prediction based on predicted human clearance of 2.5 mL / min / kg: 375 mg BID*

Summary & Next Steps

Kazia has initiated a regional and / or global partnership search for EVT801.

1. Well-understood mechanism (anti-angiogenesis) with unique differentiating feature (high VEGFR3 selectivity)

2. Strong preclinical data package, with observed activity in multiple tumors and favourable toxicology

3. Potential for combination use with immuno-oncology therapies

4. Phase 1 completed demonstrating encouraging safety and tolerability profile to date
   - Clinical and biomarker data presented at AACR Ovarian Cancer Research Symposium September 2024
   - Primary and secondary objectives successfully met, with MTD and RP2D identified
   - Encouraging signal of activity observed in high-grade serous ovarian cancer (HGSOC) as well as strong VEGFR3 biomarker expression

5. Next clinical trial under discussion with scientific thought leaders
   - Consolidate safety data at RP2D and our hypotheses on EVT801 mode of action
   - Validate HGSOC as indication of choice for clinical trial Phase 2 as monotherapy or in combination with standard-of-care (ex. PARPi)

Corporate Highlights

Near-Term Milestones & Anticipated Catalysts

Pricing of $50m private placement of equity securities

Complete metabolic response in stage IV TNBC expanded access patient and 2 partial responses in metastatic TNBC Phase 1b study

PD-L1 protein degrader platform update

Anticipated update from FDA discussion regarding paxalisib commercial and development path forward

Advancement of NDL2 PD-L1 degrader platform toward IND-enabling studies

Readouts from neuro-oncology focused clinical trials in patients with GBM and brain metastases

Update of paxalisib pre-clinical development into additional breast cancer subtypes

Publication / Presentation of translational data supporting therapeutic reprogramming strategy for paxalisib

Clinical efficacy and biomarker data from ongoing Phase 1b breast cancer study

PD-L1 degrader awareness, NDL2 IND-initiating studies

Milestone **Anticipated Catalyst**

KAZIA THERAPEUTICS

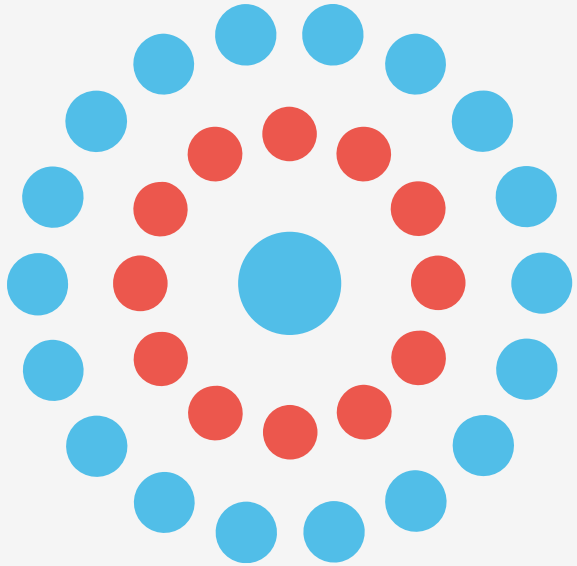
Investment Drivers

Kazia is helping to redefine oncology development by advancing a cohesive therapeutic approach aimed at reprogramming tumor biology.

Our **strategy is aligned with** where both **science and capital** are moving in oncology.

Our clinical and translational focus is advanced breast cancer and other immunotherapy-challenged solid tumors that represent **multi-billion-dollar markets with high unmet need**.

Resistance biology is well documented and combination strategies are actively sought by **strategic partners**.



Our **integrated approach** allows us to:

- **Participate meaningfully** in multiple large oncology markets **without overextending development resources**
  - Generate **early clinical and translational proof-of-concept data** that is highly attractive for strategic partnering
- Align with clear pharma appetite for **epigenetic modulation, immune-sensitization, and targeted degradation assets**

Our **assets are designed to remain relevant** across tumor heterogeneity and evolving treatment landscapes.

- Build **a portfolio that is greater than the sum of its parts**

Leadership

Management

Dr. John Friend
Chief Executive Officer
CELLECTAR BIOSCIENCES
HELSINN
ABBOTT

Jeffrey J. Kraws
Head of Corporate Strategy & Development
Bristol Myers Squibb
ABB
ALEX. BROWN

Jeffrey Bonacorda
VP Finance & Controller
HELSINN
VIVUS

Elissa Hansen
Corporate Secretary
Governance Institute of Australia
Australian Institute of Company Directors

Board of Directors

Robert Apple
Chairman
ANTARES THERAPEUTICS

Steven Coffey
Non-Executive Director
WSM CHARTERED ACCOUNTANTS

Ebru Davidson
Non-Executive Director
QBiotics Group
Thomson Geer
Lawyers

KAZIA THERAPEUTICS

KAZIA
THERAPEUTICS

www.kaziatherapeutics.com

ir@kaziatherapeutics.com