

KAZIA
THERAPEUTICS



Beyond Inhibition. Reprogramming Cancer Control.

2026 Corporate Overview

H.C. Wainwright 28th Annual Global Investment Conference
September 15, 2026

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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 enrollment of patients in Kazia's clinical and preclinical programs, the potential benefits of paxalisib, NDL2, MSETC, and EVT801, the potential results of combination studies of paxalisib and other collaborations, the preliminary nature of clinical data from Kazia's ongoing Phase 1b trial of paxalisib in advanced triple-negative breast cancer, including the observed reductions in exhausted T-cells, which are based on a small number of patients and may not be predictive of results in a larger patient population or in later-stage clinical trials, the anticipated timing of topline data from the Phase 1b trial in advanced triple-negative breast cancer and the expected enrollment of patients in such trial, Kazia's plans to expand clinical development of paxalisib into hormone receptor-positive, HER2-negative breast cancer and colorectal cancer, and the timing, cost and results of any such expansion, the potential of paxalisib to act as an immunotherapy sensitizer, remodel the tumor microenvironment, reinvigorate anti-tumor immunity or enable checkpoint inhibitor activity in historically refractory settings, timing for any regulatory submissions or discussions with regulatory agencies, the potential market opportunity for paxalisib, NDL2, MSETC, 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. 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.

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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 nuclear 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 at 12/31/25 (not including August 2026 public offering)
- No outstanding debt
- American Depositary Shares Outstanding (Fully Diluted): Approximately 14.1 million

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 <i>per se</i> (granted), synthesis of paxalisib (granted), and secondary medical uses / combination treatments (pending)

Paxalisib Pipeline

STUDY / INDICATION	TRIAL ID	PRECLINICAL	PHASE 1	PHASE 2	PHASE 3	MARKET	SPONSOR
Breast Cancer							
PaxPlus-ABC TNBC: Triple-Negative Breast Cancer & HER2-	ACTRN12624001340527						Kazia Therapeutics
Neuro-Oncology							
GBM AGILE Glioblastoma	NCT03970447						Global Coalition for Adaptive Research (GCAR)
5G-PEARL Glioblastoma	NCT07391215						Institute of Cancer Research (UK)
Paxalisib & Diet Combination Glioblastoma	NCT05183204						Weill Medical College of Cornell University
LUMOS2 Gliomas	ACTRN12623000096651						University of Sydney
OPTIMISE Pediatric Brain Cancer	NCT06208657						University of Sydney
PNOC022 DIPG: Diffuse Intrinsic Pontine Glioma	NCT05009992						Pediatric Neuro-Oncology Consortium (PNOC)
PNOC035 AT/RT: Atypical Teratoid Rhabdoid Tumor	NCT07447076						Pediatric Neuro-Oncology Consortium (PNOC)
Genomically-Guided Brain Metastases Brain Metastases	NCT03994796						Alliance for Clinical Trials in Oncology

Expansion Opportunity

Targeting difficult-to-treat cancers where therapeutic options remain limited

Glioblastoma (GBM)

- PI3K/mTOR signaling pathway is a central regulator of cell growth and is hyperactivated in approximately ~85% to 90% of glioblastoma cases.
- Unlike other PI3K inhibitors approved for peripheral cancers, paxalisib crosses the blood-brain barrier overcoming a major obstacle in CNS drug delivery.



Childhood Brain Cancer / Metastasis

- 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).
- 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.



Advanced Breast Cancer (ABC)

- PI3K/mTOR signaling pathway 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.



Expanding Indications

- Broad PI3K/mTOR signaling across solid tumors
- Tumor and tumor microenvironment (TME); emerging data suggests paxalisib can reshape both and potentially overcome resistance and recurrence
- Clear opportunity to expand into HR+/HER2- breast cancer, MSS/pMMR CRC, and early-stage, high-risk TNBC

Data on file, market research performed 2021, [Glioblastoma Multiforme Treatment Market Size Report, 2034](#) [NCI: Childhood Brain and Other Nervous System Cancer](#) [NIH: PI3K Pathway in Pediatric Brain Tumors](#) [Strategic Market Research: Pediatric Brain Tumor Market Report](#) [Cancer.org 2024](#) [Data Bridge Market Research](#)

Kazia-Sponsored Clinical Phase 1b Study Overview

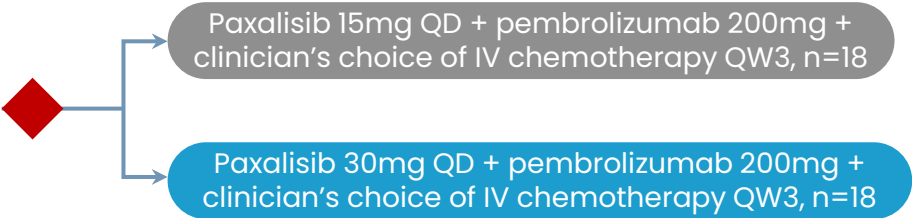
Multi-center, open-label, randomized trial

Arm B

- Recurrent, unresectable or metastatic TNBC
- Confirmed that tumors express PD-L1 with a combined positive score (CPS) ≥ 1
- Meet all current prescribing criteria for commencing pembrolizumab therapy

Total enrollment: 36

Paxalisib + Pembrolizumab / Chemotherapy (21-Day Cycle)



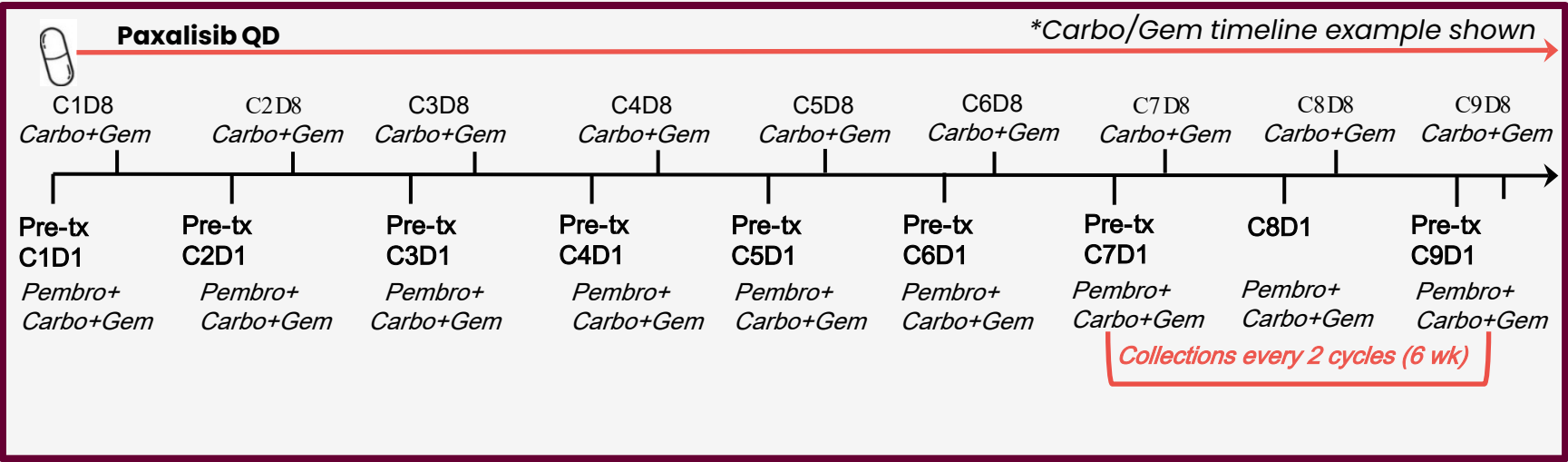
Endpoints

Primary

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

Secondary

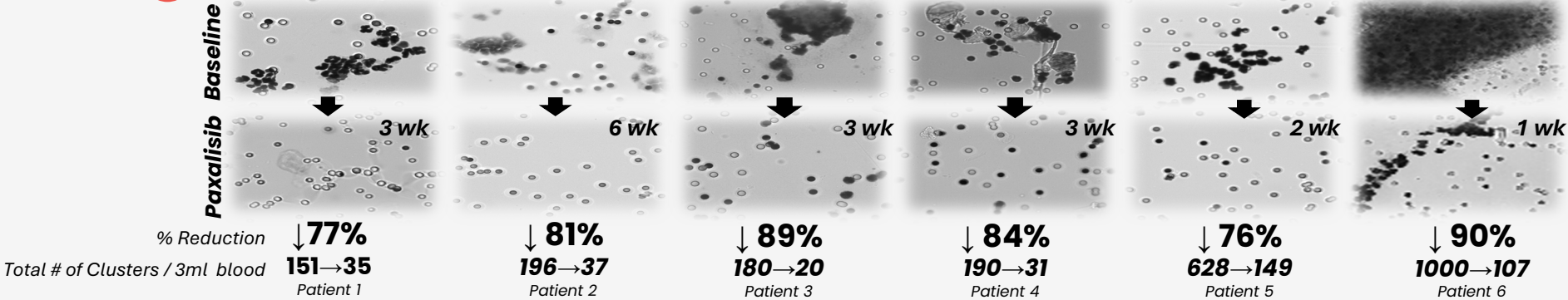
- Progression free survival and overall response rates
- To assess the utility of novel liquid biopsy assessments



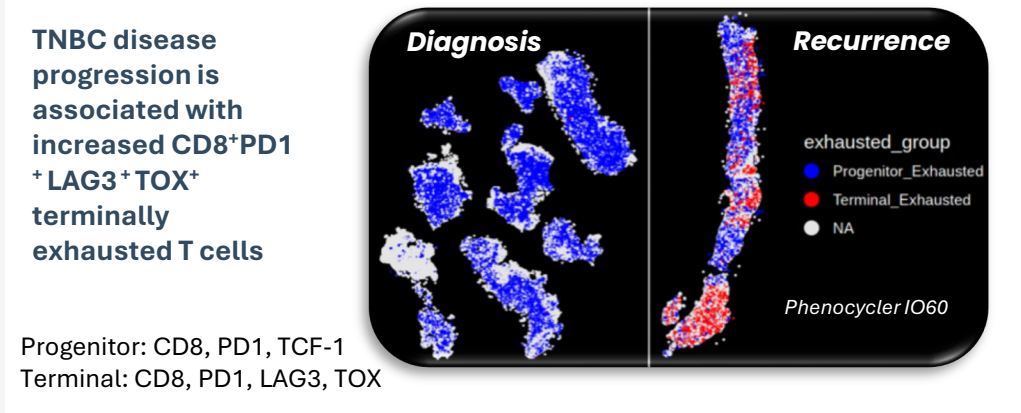
Stage IV TNBC Highlights

Translational evidence in 6/6 evaluable patients: paxalisib targeting CTC clusters and terminally exhausted T cells

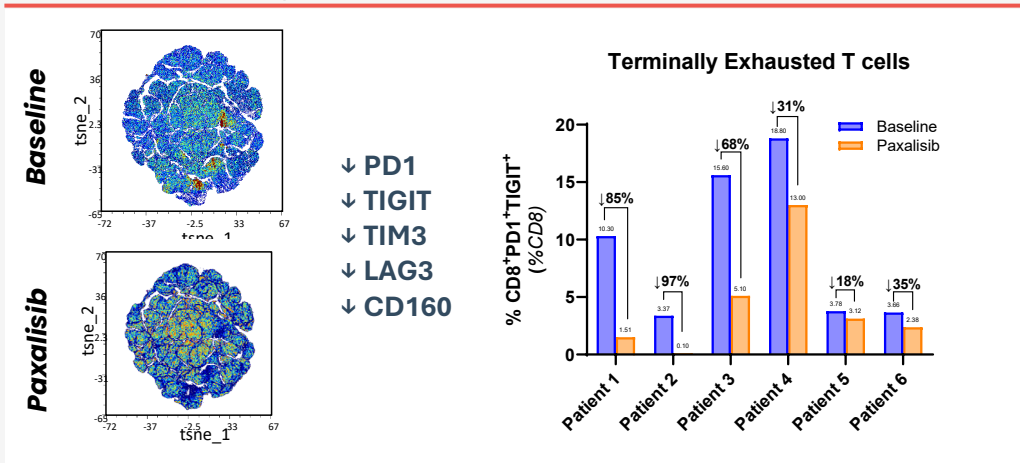
1 Early disruption of CTC clusters by paxalisib combination treatment in Stage IV TNBC



2 Increased terminally exhausted CD8+ T cells in progression of TNBC



3 Paxalisib rapidly reduces terminally exhausted CD8+ T cells in Stage IV TNBC after 1-3 week treatment

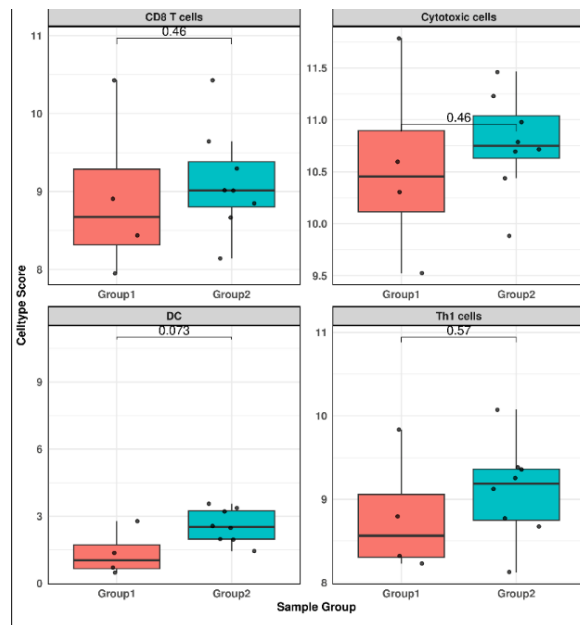


Stage IV TNBC Highlights (Cont.)

Translational evidence: paxalisib combination reinvigorating the immune landscape

Nanostring RNA profiling of PBMCs isolated from longitudinal patient bloods pre- and post-paxalisib treatment

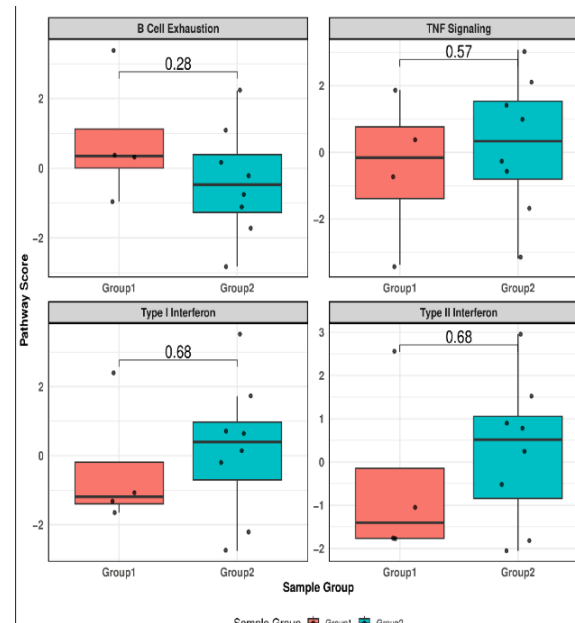
Increased cytotoxicity, IFN γ , DCs



Group 1: Pre-treatment Group 2: Post-treatment

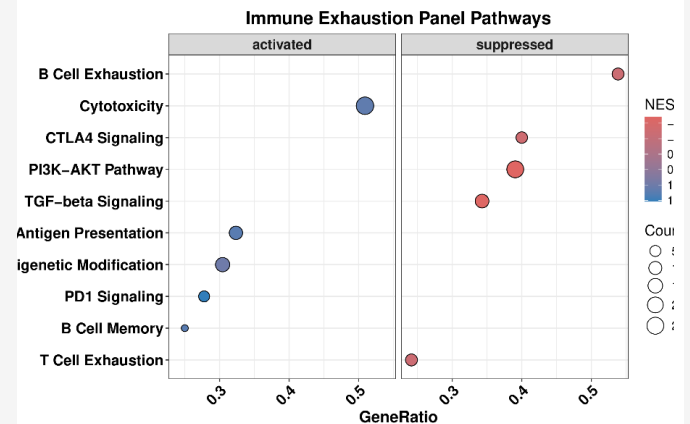
- Increased anti-tumor immune cells
 - ↑ Cytotoxic T cells
 - ↑ Th1 IFN γ producing cells
 - ↑ Antigen presenting DCs

Increased antigen presentation, reduced B cell exhaustion



- ↑ Activation of Type I/ II IFN
- ↑ TNF signalling indicative of enhanced cytotoxicity
- ↑ Antigen presentation
- ↓ B cell exhaustion

Inhibition of T cell exhaustion, increased adaptive immune activation

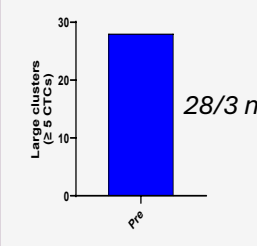
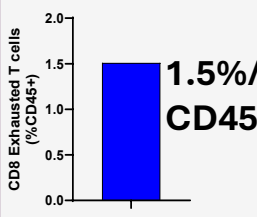
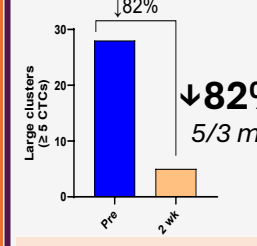
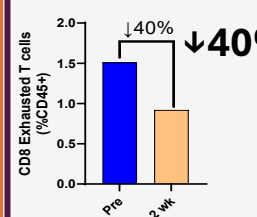
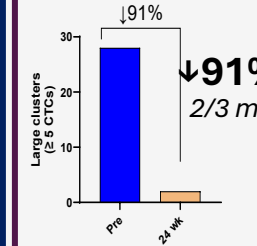
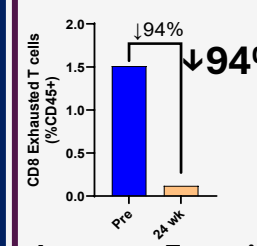
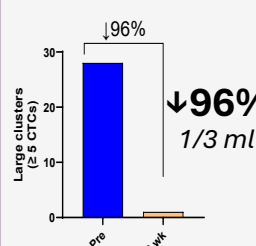
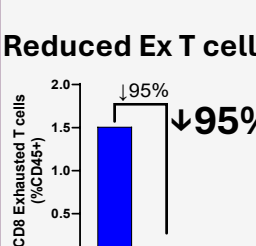
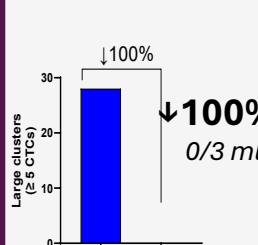


- Global activation of adaptive immune responses
- Inhibition of immunosuppressive signalling (B and T cell exhaustion)
- Confirmation of paxalisib induced inhibition of its target, the PI3K-AKT pathway

Stage IV TNBC Highlights (Cont.)

Early efficacy readout predicts complete metabolic response

MAR 25, 2025 JUL 23, 2025 **AUG 6, 2025** AUG 20, 2025 AUG 21, 2025 NOV 10, 2025 JAN 21, 2026* FEB 26, 2026 APR 15, 2026 AUG 4, 2026

PET Scan Multiple bilateral pulmonary nodules New FDG avid skeletal metastatic disease	BLOOD High CTC Clusters <i>(Large clusters ≥5 CTCs)</i>  High % Ex T cells  ↓Immune Function Low Granzyme A,B High IL-6 (CTC inducer) (Plasma)	Paxalisib + Pembrolizumab + Chemotherapy Treatment Started	BLOOD Reduced Clusters  Mesenchymal ↓92% Vim+NRF2+Snail+ Reduced Ex T cells  ↑Immune Function ↑ Granzyme A,B ↓IL-6 (CTC inducer) (Plasma)	PET Scan Reduction in pulmonary nodules Resolution of the FDG activity in the left ilium No new FDG activity within the axial or appendicular skeleton	PET Scan COMPLETE RESPONSE No evidence of metastatic malignancy	BLOOD Reduced Clusters  Reduced Ex T cells  ↑Immune Function ↑ Granzyme A,B ↓IL-6 (CTC inducer) (Plasma) *PET Scan confirmed CR	BLOOD Reduced Clusters  Reduced Ex T cells  ↑Immune Function ↑ Granzyme A,B ↓IL-6 (CTC inducer) (Plasma)	BLOOD Reduced Clusters  ↑Immune Function ↑ Granzyme A,B ↓IL-6 (CTC inducer) (Plasma)	PET Scan COMPLETE RESPONSE No FDG active locally recurrent, nodal metastatic, or avid distant metastatic disease.
 SCAN Matched	 SCAN Predicted		 SCAN Predicted			 SCAN Predicted	 SCAN Predicted		Data on file

Stage IV TNBC Individual Patient Responses

Patient	Age	Dose	Disease Sites	Response	Time to Response
1	61	30mg	1 target lesion, left upper lobe of lung	Partial	~5 months
2	47	30mg	Lung, liver, bone, lymph node lesions	Partial	~3 months
3	45	15mg	5 target lesions	Partial	~3 months
4	70	15mg	2 target lesions	Stable disease	~5 months
5	43	30mg	CNS target lesions	Partial (Near complete resolution of dural metastases)	~6 months
6	44	30mg	Lung and skeletal target lesions	Complete	~3 months

Key Takeaway:
Responses were observed across visceral, skeletal and CNS metastases.

Stage IV TNBC Safety Profile Supports Continued Clinical Development

0

Paxalisib-Related
Serious Adverse Events (SAEs)

0

Suspected Unexpected Serious
Adverse Reactions (SUSARs) or
Significant Safety Issues (SSIs)
Reported

13

Total SAEs Reported

- 1 patient had 1 SAE
- 1 patient had 3 SAEs
- 1 patient had 9 SAEs
- 3 patients had 0 SAEs

Reported SAEs:

Unrelated (10)

- Port-related infection (polymicrobial port colonization)
- Chest wall pain (non-cardiac)
- Parainfluenza
- Lung infection
- Nausea
- Fatigue
- Cather-related infection
- Diarrhea
- Sepsis (2 events)

Unlikely Related (3)

- Infectious colitis / diarrhea
- Diarrhea
- Low-grade fevers (Temps: 37.5° - 37.9° C / 99.5° - 100.22° F)

No SAEs were considered treatment-related and no suspected serious safety signals were reported.

Stage IV TNBC Highlights Summary

Clinical evidence: first-in-human signals from six evaluable patients

100% of evaluable patients (6/6) showed clinical benefit.

Selective immune reinvigoration

- Terminally exhausted CD8⁺ T cells reduced in **100% of evaluable patients (6/6)**
- **Median 51% reduction** (range 18–97%) within ~3 weeks
- **Total CD8⁺ T-cell numbers unchanged**, indicating selective reduction of dysfunctional T cells

Robust disruption of metastatic CTC clusters

- **100% of evaluable patients (6/6)** showed reductions in CTC clusters
- **Median 83% reduction** (range 76–90%) by 6–7 weeks of treatment

Clinical implication

- Early, consistent pharmacodynamic responses suggest **paxalisib extends beyond PI3K/mTOR inhibition**
- Supports our hypothesis of paxalisib's ability to target **immune dysfunction** and **metastatic dissemination**, two key drivers of TNBC
- One expanded access patient demonstrated durable complete metabolic response and robust reductions in CTC clusters and exhausted terminally exhausted CD8⁺ T cells

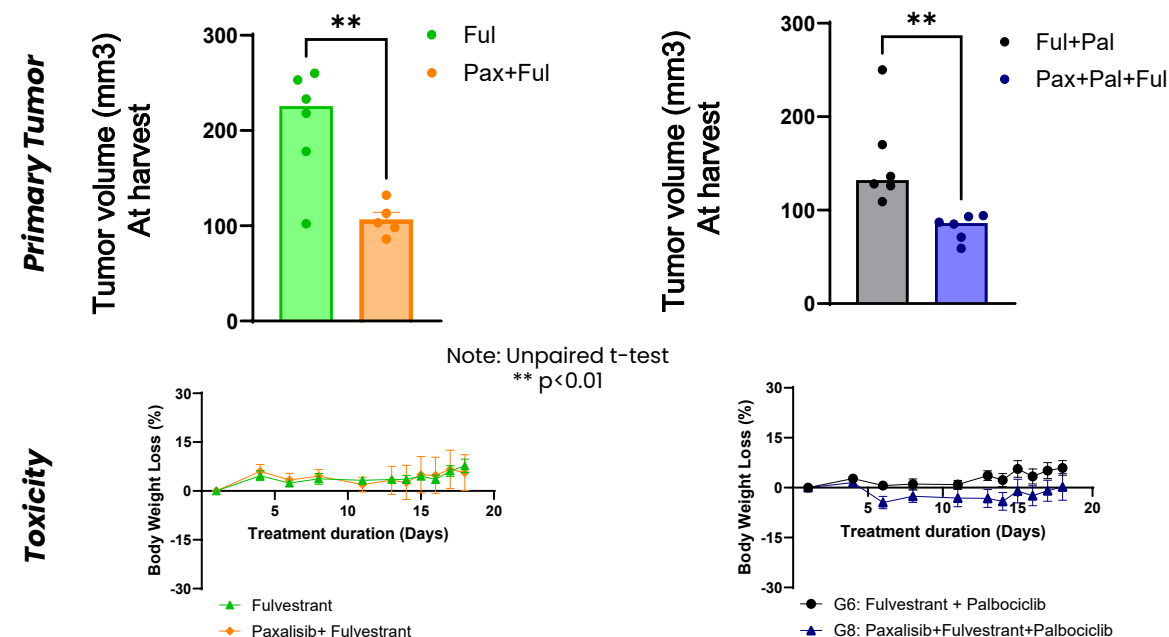
HR+/HER2- Innovation & Highlights

Key Highlights

- Patent protected
- Extensive preclinical safety and efficacy evidence supporting paxalisib in combination with standard of care (SOC) in HR+/HER2- breast cancer patients (both wild-type and mutant)
- Subpopulation with elevated novel PI3K/mTOR marker enriched in HR+/HER2- breast cancer that is tightly coupled with poor survival identified; axis uniquely targeted by paxalisib
 - Identified a novel epigenetic MOA that allows paxalisib (and not gedatolisib) to uniquely sensitize the CDK4/6 resistant subpopulation to overcome recurrence and metastases
 - Abstract submitted to upcoming conference
- Potential for paxalisib to treat HR+/HER2- patients with brain mets accounts for 10% HR+/HER2-
- Clinical-trial ready

Scientific Evidence

Paxalisib + fulvestrant +/- CDK4/6 inhibitor reduces tumor burden without toxicity

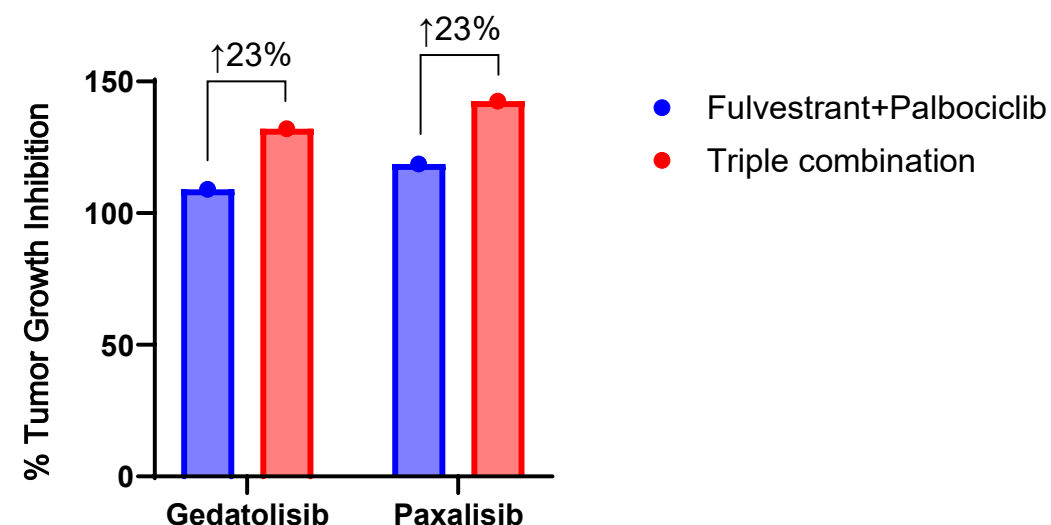


Data on file

HR+/HER2- Innovation & Highlights (Cont.)

Scientific Evidence

Comparison of tumor growth inhibition following treatment with Gedatolisib and Paxalisib in combination with Fulvestrant + Palbociclib in the MCF7 HR⁺ xenograft model



Paxalisib shows identical efficacy to gedatolisib in preclinical studies.

MSS/pMMR CRC Innovation & Highlights

Key Highlights

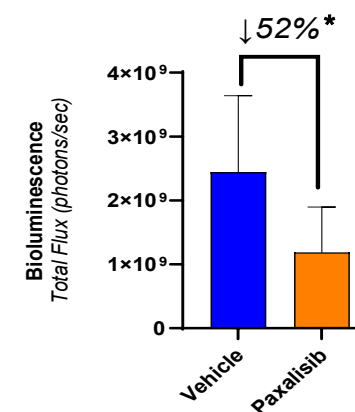
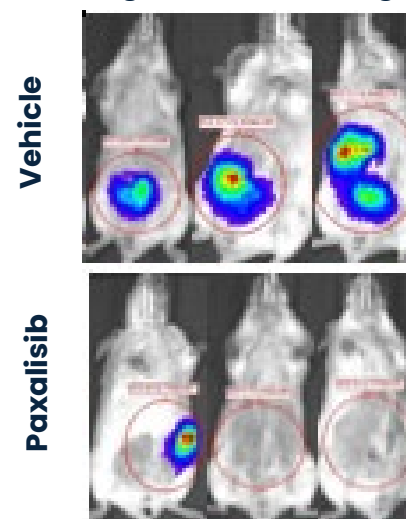
- Patent protected
- 52% reduction in tumor burden with paxalisib as a single agent ($p = 0.035$), well tolerated throughout
- A further 50% reduction in tumor volume when added to anti-PD-1, versus anti-PD-1 alone ($p = 0.022$)
- Novel liquid biomarkers to select patients and monitor response, where none exist today
- Targets the MSS/pMMR majority — the 85–90% of colorectal cancer patients who derive limited benefit from checkpoint inhibitors today
- Early access partners for spatial ATAC sequencing
- Clinical-trial ready — protocols in place

Scientific Evidence

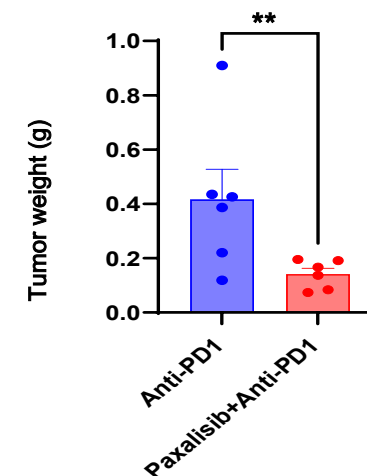
Paxalisib monotherapy and α PD1 combination suppresses primary tumor burden in MSS/pMMR+ CRC preclinical models

Monotherapy

Longitudinal colon imaging



Combination



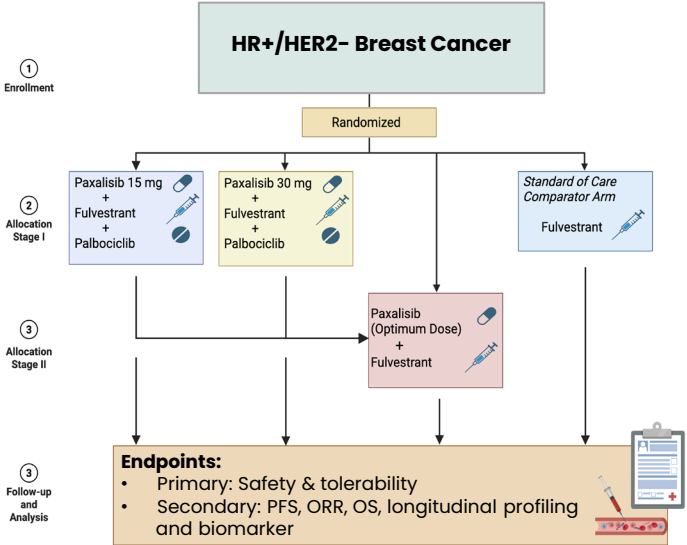
Note: Unpaired t-test
* $p < 0.05$ ** $p < 0.01$

Data on file

Clinical Trial Ready

HR+/HER2- Breast Cancer

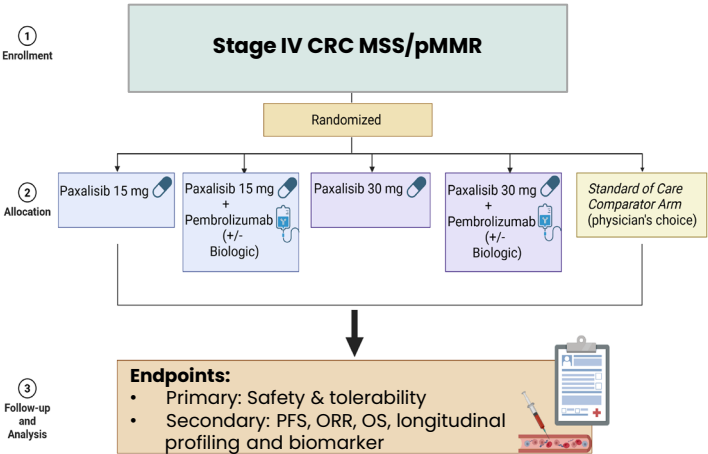
**Paxalisib + Fulvestrant + Palbociclib
for pre-treated HR+/HER2-
metastatic breast cancer
(Second line setting)**



Target enrollment: 78
First patient enrolled: Q4 2026

MSS/pMMR CRC

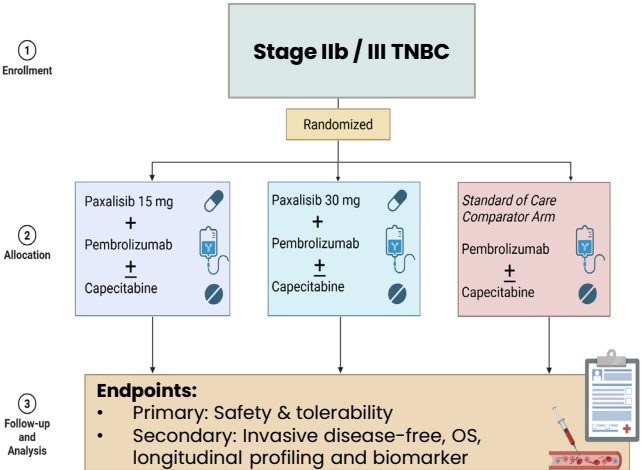
**Paxalisib Monotherapy and Paxalisib +
Pembrolizumab vs. Standard of Care
in pre-treated MSS/pMMR metastatic CRC
(Third line setting)**



Target enrollment: 90
First patient enrolled: Q1 2027

Stage IIb/III TNBC (in adjuvant, followed by neoadjuvant setting)

**Paxalisib + Pembrolizumab + Capecitabine
for high-risk Stage IIb/III TNBC
in the adjuvant setting
(First line setting)**



Target enrollment: 48
First patient enrolled: 2H 2027

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 throughout the year, 2) ongoing updates from Phase 1b advanced breast cancer clinical study (ACTRN12624001340527) throughout 2026, 3) expand into other clinical indications including HR+/HER2- breast cancer and early-stage high-risk TNBC and 4) participate in upcoming scientific conferences
- **Colorectal Cancer:** 1) Execute clinical development plan with Phase 2 clinical trial in MSS/pMMR CRC to evaluate paxalisib as monotherapy and in combination with pembrolizumab and 2) participate in upcoming scientific conferences
- **Glioblastoma:** 1) Anticipated follow-up FDA Type C meeting to discuss commercial and development path forward and 2) finalize clinical development plan based on FDA feedback
- **Pediatric & Brain Metastasis Programs:** 1) PNOC022 team to complete PK / biomarker data analysis and provide update, 2) data release / updates from other brain met trials, and 3) initiate enrollment for PNOC035 (AT/RT clinical study)

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 nuclear 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 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 nuclear PD-L1 protein pools Recruits cellular degradation machinery Targets nuclear 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

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 18 months
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

**Q3
2026**

**Q4
2026**

**Q1
2027**

**Q2
2027**

**→
2027**

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

Update of paxalisib preclinical development into additional breast cancer subtypes

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

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

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

PD-L1 degrader and SETDB1 inhibitor awareness, NDL2 and MSETC IND-enabling studies

Closed public offering of up to \$120 million

Launch expansion of ongoing clinical trial into HR+/HER2- breast cancer population

Launch expansion into MSS/pMMR CRC as monotherapy and in combination with pembrolizumab

Initiate Phase 1 trials for NDL2 and MSETC

 **Milestone**  **Anticipated Catalyst**

Investment Drivers

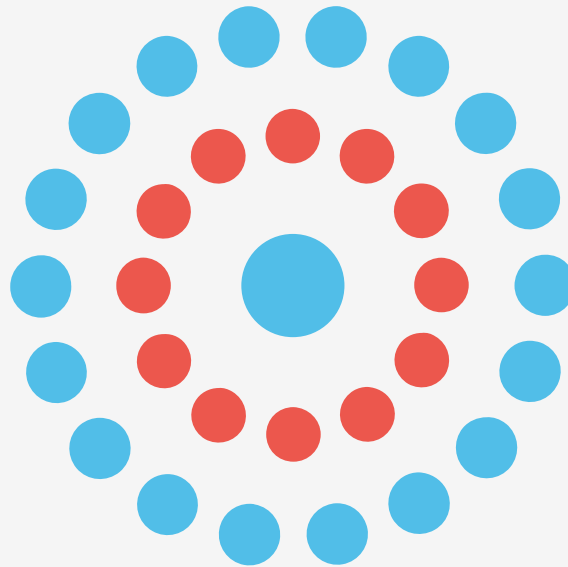
Kazia is helping to redefine oncology development by advancing a cohesive therapeutic approach aimed at reprogramming tumor biology.

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

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



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

Leadership

Management



Dr. John Friend
Chief Executive Officer



Dr. Sudha Rao
Chief Scientific Officer



James Levine
Chief Financial Officer



Jeffrey J. Kraws
Head of Corporate
Strategy & Development



Elissa Hansen
Corporate Secretary



Board of Directors



Robert Apple
Chairman

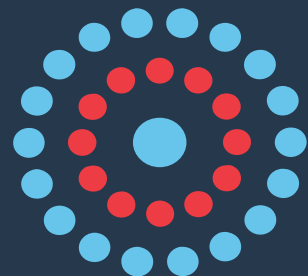


Steven Coffey
Non-Executive Director



Ebru Davidson
Non-Executive Director





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THERAPEUTICS

www.kaziatx.com

ir@kaziatx.com