December 14, 2017
Global Equity Research led by
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Overweight

Kazia Therapeutics: Targeting Unmet Oncological Needs Arising from Today's Standard of Care

Recommendation Summary

- We are initiating coverage of Kazia Therapeutics Limited ("Kazia") with an **Overweight** rating and a June 2018 **price target of AUD0.81**, representing a potential upside of approximately 131% from the closing price of AUD0.35 as of 13 December 2017.

- Kazia is a clinical-stage biotechnology firm with two leading drug candidates currently in human-stage clinical studies. The company is headquartered in Sydney, Australia, and is dual-listed on the **Australian Securities Exchange** under the ticker: **KZA**, and on the American **NASDAQ** Stock Market under the ticker: **KZIA**.

- The company is **oncology-focused**, targeting cancers with significant unmet medical needs that today's standard of care has failed to address. Kazia's current drug pipeline houses novel therapies, sourced from internal discovery engines as well as through in-license from external partners.

- **Cantrixil**, the first drug candidate, is derived from the superbenzopyran (SBP) drug platform developed by Kazia in conjunction with scientists at Yale University, and it **targets chemotherapy-resistant cancer stem cells**. The drug's mode of action involves uniquely the tackling of two individual cellular pathways – activating caspase-mediated apoptosis, while inhibiting a critical cell survival pathway. Kazia has **initially** positioned Cantrixil to **target ovarian cancer** stem cells, which are responsible for over 70% of cancer recurrence post-treatment with today's standard of care. Cantrixil is currently recruiting patients for phase I clinical trials in ovarian cancer. Kazia is expected to announce maximum tolerated dose (MTD) in 1Q 2018 and exploratory efficacy data later in 2018.

- **GDC-0084**, the second drug candidate, is a **dual PI3K-mTOR inhibitor**, being prepared to commence phase II clinical trials by year-end 2017. In-licensed from Genentech, a subsidiary of the Switzerland-based Roche, GDC-0084 is positioned to **target glioblastoma multiforme (GBM)**, the most common and aggressive cancer originating from the brain. GDC-0084 addresses a significant downfall experienced by patients receiving today's standard of care with the intake of the chemotherapeutic agent temozolomide (TMZ). GDC-0084 is capable of penetrating the blood-brain barrier to exert its effect and **target a pathway that is dysregulated in approximately 88% of GBM patients**, compared to the alkylating agent temozolomide of which fewer than 50% of GBM patients are responders.

- **We believe both Cantrixil and GDC-0084 show promise in being used as the first-line therapy**, targeting their respective diseases by addressing notable unmet medical needs due to the deficiency of today's standard therapy.

- We value Kazia using a **sum-of-parts valuation**, which includes the **discounted cash flow (DCF)** valuation model after adjusting for the respective marketing risk associated with its two key drug candidates. This valuation methodology allows us to take into account the various stages of development of drugs in the pipeline, while also incorporating the value of Kazia's other commitments and projects.

KAZIA THERAPEUTICS

Price (13 Dec'17): AUD0.35
Target Price (Jun'18): AUD0.81
Fiscal Year Ends on June 30th

Market Data (13 Dec'17):

52-Wk High: AUD1.05 (16 Feb'17)
52-Wk Low: AUD0.335 (20 Nov'17)
52-Wk Range: AUD0.715
Market Cap: AUD16.9M
Shares Outstanding: 48.4M
Shares Floating: 35.9M
Annual Dividend: N/A
Dividend Yield: N/A

Table of Contents

I. COMPANY BACKGROUND

Kazia is an oncology drug development company with two leading anti-cancer drug candidates currently in clinical trials. The company is headquartered in Sydney, Australia, and dual-listed on the Australian Securities Exchange (ticker: **KZA**) and NASDAQ (ticker: **KZIA**, in the form of American depository receipt or ADR)**.** It has three distinguishable technologies, namely PI3K inhibitor (phosphoinositide 3-kinase), superbenzopyran (SBP) and anti-tropomyosin (ATM), each of which is capable of developing its own portfolio of products.

Kazia is currently headed by CEO Dr. James Garner, a seasoned life sciences executive who has held several senior managerial positions at large multinational firms such as Biogen (BIIB), Takeda (4502 JP) and Sanofi (SNY). He has positioned the company to mainly focus on clinical-stage assets for oncology indications with highly unmet medical needs, which currently include two drugs in clinical development, namely GDC-0084 and Cantrixil, targeting glioblastoma[1] and ovarian cancer respectively.

The drug targeting glioblastoma, **GDC-0084**, is in-licensed from Genentech, a subsidiary of Roche (ROG VX), which initiated development of the drug through a successful phase I clinical study. Meanwhile, **Cantrixil**, the drug targeting ovarian cancer, was developed with the support from researchers at Yale University, and its early-stage of development and pre-clinical studies were performed by CanTx Inc., the joint venture established by both parties. CanTx was then dissolved in May 2016, and Kazia has since retained all the rights with regards to Cantrixil.

Company's Recent Development

Kazia has undergone major transformations in the past years. Along with bringing in new management in February 2016, Kazia terminated the clinical development of one of their drugs, ATM-3507 (Anisina), in April 2017 noting the toxicology findings outweighed its clinical benefits and prompting the company to initiate separate drug discovery programs. Anisina was originally slated to begin phase I clinical trials earlier in 2017.

Exhibit 1: Recent Development of Kazia

Company Development	Year	Drug Development
	2014	• **November 2014** – Anisina identified as the lead ATM drug candidate
	2015	• **April 2015** – Pre-clinical studies demonstrated that Cantrixil is active in rodent model of human ovarian cancer • **April 2015** – Cantrixil received Orphan Drug Designation in the U.S. for ovarian cancer • **July 2015** – Anisina was granted the Orphan Drug status by the U.S. Food and Drug Administration (FDA) for the potential treatment of neuroblastoma[2]

[1] It is the most common type of malignant brain tumor among adults, also known as glioblastoma multiforme (GBM)
[2] It is a type of cancer that forms in certain types of nerve tissue. It most frequently starts from one of the adrenal glands, but can also develop in the neck, chest, abdomen, or spine

		• **October 2015** – Kazia and Yale University disclosed key pre-clinical data generated in an animal model of recurrent ovarian cancer for Cantrixil • **November 2015** – Pre-clinical studies confirmed the activity of the lead ATM compound Anisina • **November 2015** – patent application, covering superbenzopyran (SBP) derived drugs, including Cantrixil, accepted in Australia
• **February 2016** – Dr. James Garner joined Kazia as Chief Executive Officer and the company's Board of Directors • **August 2016** – Key management personnel appointed to drive transition to a development focused organization, with Dr. Gordon Hirsch appointed Chief Medical Officer and Dr. Peng Leong appointed as Chief Business Officer • **September 2016** – Scientific advisory board established to guide the development of the oncology pipeline • **October 2016** – Kazia acquired Glioblast Pty Ltd., a privately-held neuro-oncology company based in Australia, for AUD2.1 million in cash and ordinary shares plus performance-related milestones, with the intention that Glioblast would assist in the development of GDC-0084 in glioblastoma multiforme (GBM). Ms. Leslie Chong, formerly Clinical Program Lead for GDC-0084 at Genentech, became consultant to Kazia, advising on clinical development	**2016**	• **February 2016** – patent for the superbenzopyran (SBP) derived drugs, including Cantrixil, was granted in Australia, providing intellectual property (IP) protection until 2035 • **June 2016** – Patent covering Anisina granted in Australia, providing full protection of Kazia's IP until 2035 • **August 2016** – Investigational New Drug (IND) application submitted for Cantrixil in ovarian cancer to the U.S. FDA • **September 2016** – U.S. FDA approved Investigational New Drug application for Cantrixil in ovarian cancer with first-in-human (FIH) phase I study initiated in 4Q 2016 • **October 2016** – Kazia in-licensed phase II-ready molecule (GDC-0084) from Genentech for development in glioblastoma • **December 2016** – Kazia enrolled first patient in its U.S. and Australia phase I study of Cantrixil
• **February 2017** – Kazia was awarded grant of up to AUD3 million over the course of three years by the Australian Federal Government under the Cooperative Research Centre Project (CRC-P) scheme for novel drug discovery program within Kazia's ATM technology platform • **November 2017** – Kazia announced it will establish a Wholly Foreign Owned Enterprise (WFOE), headquartered in Shanghai, to facilitate cooperation with the Chinese Food and Drug Administration (CFDA) and the development of its drug portfolio in China as well as its expansion into the Greater China region	**2017**	• **April 2017** – Kazia terminated the preclinical development program of Anisina due to unfavorable balance of preclinical activity relative to emerging toxicology findings, and unlikely to gain regulatory approval and achieve business success, while maintaining the Cooperative Research Centre Project (CRC-P) grant • **September 2017** – Kazia completed consultation with the FDA regarding the upcoming initiation of the phase II study of GDC-0084 • **November 2017** – Kazia out-licensed a suite of early-stage preclinical assets to the start-up Heaton-Brown Life Sciences, LLC (HBLS), in return for equity in HBLS, performance milestones and royalties

Source: Kazia, Cedrus Research

II. OVERVIEW OF TECHNOLOGY AND DRUG PORTFOLIO

Kazia's portfolio of drugs currently in development are derived from two distinct technology platforms with proven anti-cancer properties, while a preclinical product based on a third platform is currently under development.

Exhibit 2: Kazia's Technology Portfolio and Derived Products

Technology Platform	Phosphoinositide 3-Kinase (PI3K) Inhibitor	Superbenzopyran (SBP)	Anti-tropomyosin (ATM)
Brief Description	The PI3K pathway is a key regulator in cell growth, proliferation, motility, survival and apoptosis. Over-activation of this pathway leads to survival and proliferation of tumor cells in human cancers. PI3K inhibitors are small molecule inhibitors of the PI3K pathway, ultimately inducing cell death	Benzopyran-derived compounds can have a wide range of therapeutic applications. It is capable of inhibiting cancer cells through distinct cellular pathways, including modulation of genomic and non-genomic mechanisms	Tropomyosin is a protein responsible for giving cells their cytoskeleton structure. ATM drugs can be targeted to disable tropomyosin that is specific to certain cancer cells
Derived Product(s)	**GDC-0084**	**Cantrixil**	**Next-generation ATM**
Therapeutic Indication	Glioblastoma	Ovarian cancer	Solid tumors
Current Status	Phase II ready (on track to begin by year-end 2017)	Recruiting for phase I	Pre-clinical development

Source: Cedrus Research, Kazia

Exhibit 3: Kazia's Product Portfolio Status

Development Status	Indication	Pre-Clinical Proof-of-Concept	Investigational New Drug Toxicology & Chemistry	Clinical Trials			Filing	Marketing
				Phase I	Phase II	Phase III		
Clinical Development								
GDC-0084	Glioblastoma				Preparing to initiate phase II			
TRXE-002-1 (Cantrixil)	Ovarian Cancer			Maximum Tolerated Dose Expected in 1Q 2018				
Pre Clinical Development								
Next-generation ATM	Solid Tumours							

Source: Cedrus Research, Kazia

Drug in Clinical Development: GDC-0084

Overview of GDC-0084

Kazia in-licensed GDC-0084 from Genentech in October 2016. GDC-0084 is an orally administered small molecule phosphoinositide 3-kinase – mammalian target of rapamycin (PI3K-mTOR[3]) inhibitor that targets a frequently dysregulated cancer cell growth signaling pathway. Genentech originally designed the drug to effectively cross the blood-brain barrier[4] (BBB), allowing it to target glioblastoma multiforme (GBM), the most prevalent and aggressive brain cancer. The drug has completed a successful phase I clinical trial in patients with advanced glioma[5], and quantitative data have indicated that GDC-0084 is capable of crossing the blood-brain barrier. GDC-0084 is patent protected until at least 2032 (with the likelihood of being extended) in majority of the major markets, including the U.S., Australia and China.

Glioblastoma Multiforme (GBM) Overview

GBM is the most common and aggressive primary central nervous system (CNS) cancer with median survival duration of 15 months post-diagnosis. GBM accounts for over 45% of malignant primary brain and CNS tumors, 54% of all gliomas (tumor arising from glial cell[6]), and 16% of all primary brain and CNS tumors[7]. According to American Association of Neurological Surgeons, the incidence rate of GBM stands at approximately 3 per 100,000 people a year. Although GBM can occur at any age, the peak incidence rate is between the age of 45 and 70.

The risk factors of GBM are still unclear. There is little evidence of correlation of the disease with genetic anomalies and environmental factors, rendering any sort of preventative measures useless. However, GBM is slightly more likely to occur in males, suggesting a correlation with the sex-specific hormones.

Magnetic resonance imaging (MRI) is usually performed on the patient to confirm the presence of GBM and determine its severity. On a MRI scan, the area of the brain affected will appear structure-less with noticeable edema (swelling).

[3] mTOR stands for mammalian or mechanistic target of rapamycin. It functions as a serine/threonine protein kinase that regulates cell growth, cell proliferation, cell motility, cell survival, protein synthesis, autophagy, and transcription. Blocking mTOR may cause cancer cells to die

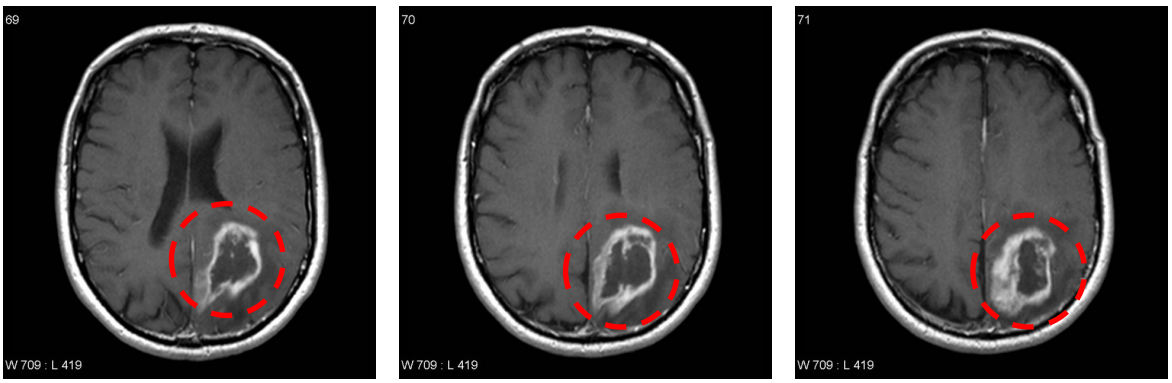
[4] A filtering mechanism of the capillaries that carry blood to the brain and spinal cord tissue, blocking the passage of certain substances

[5] A malignant tumor of the glial tissue of the nervous system

[6] The glial cell surrounds neurons and provides support for and insulation between them. Glial cells are the most abundant cell types in the central nervous system. There are several types of glial cells

[7] Epidemiologic and Molecular Prognostic Review of Glioblastoma (2014) Jigisha P. Thakkar, et al.

Exhibit 4: MRI Cross-Sections of a Patient with GBM



Source: Radiopaedia – Dr. Henry Knipe and Prof. Frank Gaillard et al

GBM is classified as a Grade IV (most serious) glioma by the World Health Organization, being highly malignant and invasive. The cancer cells can spread rapidly to nearby brain tissue, making surgical removal of all tumor cells nearly impossible without significantly impairing the patient.

There are also remarkable challenges for drug-based therapies because the tumors are in the brain. The brain also has limited capacity of repairing itself; therefore, introducing traditional chemotherapeutic substances could induce irreversible neurotoxicity. More importantly, the brain and the nervous system are protected by the blood-brain barrier (BBB), a specialized barrier that is much more selective than other blood barriers in the body, restricting the passage of the majority of drugs and foreign substances.

The BBB prevents almost 100% of all large molecule drugs and approximately 98% of all small molecules[8] from entering the brain via blood. The BBB is made up of highly compact cerebral endothelial cells that lack many of the paracellular[9] and transcellular channels commonly seen in non-central nervous system endothelial membranes, restricting only lipid-mediated free diffusion through the BBB, or carrier/receptor mediated transport through the BBB. Inadvertently, the BBB plays a major obstacle in drug discovery for medical conditions originating in the brain.

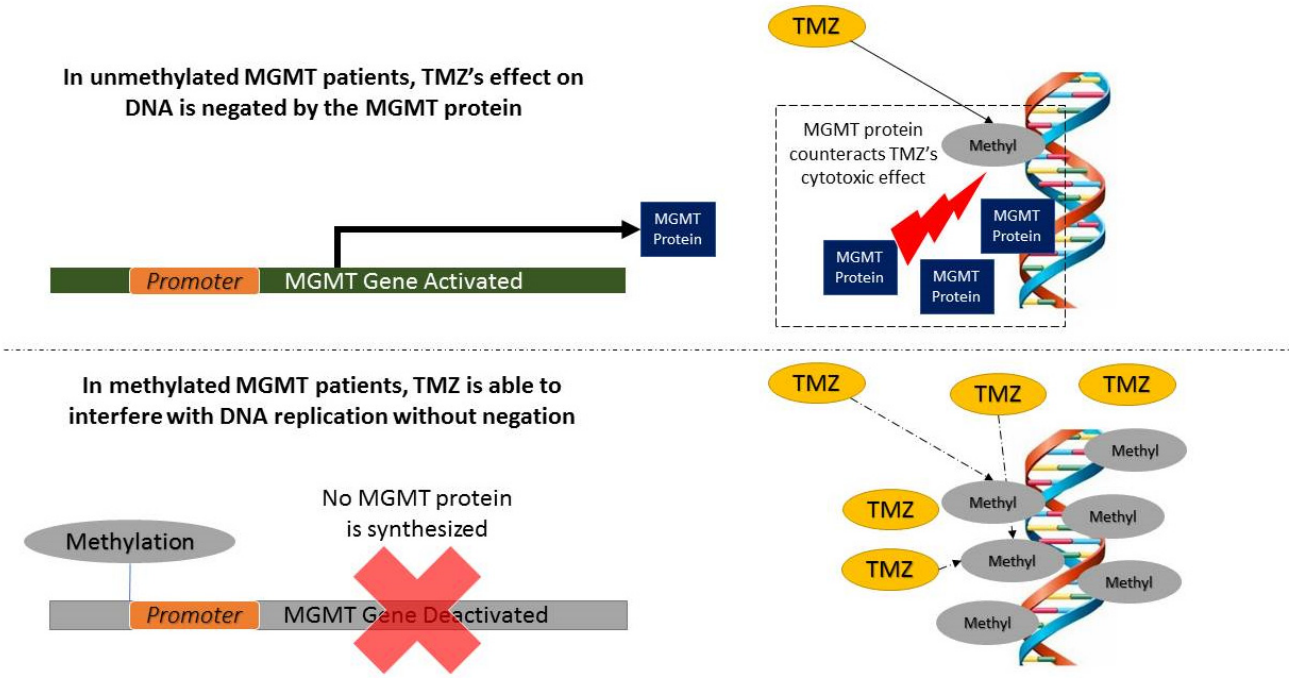
A Significant Unmet Medical Need in Over 50% of GBM Patients

The current standard of care for GBM patients involves maximal tumor resection (if possible), followed by radiotherapy and concomitant with adjuvant[10] chemotherapy, most commonly temozolomide (TMZ). TMZ is an oral chemotherapeutic drug originated by Merck (MRK) and is capable of crossing the BBB. The drug is an alkylating agent[11] that methylates DNA adenine and guanine residues, thus interfering with the regular DNA replication process, ultimately leading to cell cycle arrest and death. However, fewer than 50% of GBM patients respond to TMZ, with the response rate being positively correlated to the presence of the methylation status of the O^6-methylguanine-DNA methyltransferase (MGMT) gene. The average survival

[8] Drug transport across the blood-brain barrier (2012) William M Pardridge
[9] Passing or situated beside or between cells
[10] A therapy applied after initial treatment for cancer, especially to suppress secondary tumor formation
[11] A substance that causes replacement of hydrogen by an alkyl group that inhibits cell division and growth and is used to treat some cancers

duration for patients treated concomitantly with radiotherapy and TMZ that had methylated MGMT was 21.7 months compared to 12.7 months for patients with unmethylated MGMT[12]. Methylation of the MGMT promoter is only found in 35%-45%[3] of GBM patients, leaving a significant remainder of the patients medically unattended.

Exhibit 5: Mechanism of TMZ and Role of MGMT Methylation



Source: Cedrus Research, Slideshare

Discovery of GDC-0084

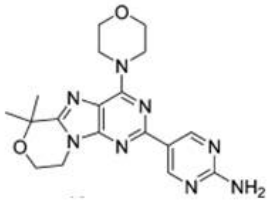
The discovery of GDC-0084 was based on existing thienopyrimidine compounds Genentech had first identified to be clinical inhibitors for PI3K suited for the treatment of peripheral diseases. These compounds innately had PI3K-inhibiting properties, but had poor projected clearance in the human body based on quantitative data of human liver microsomal stability (indicator of drug clearance), which represented foreseeable high toxicological side effects in clinical trials by Genentech.

Gradual chemical modification of the early stage thienopyrimidine analogues led to the formulation of GDC-0084, a compound that is a potent inhibitor of PI3K and mTOR and capable of penetrating the blood-brain barrier in sufficient quantities to exhibit anti-cancer effects in mice and rat clinical subjects, as well as exhibiting the required human metabolic stability in microsomal and hepatocyte incubations *ex vivo*.

[12] Personalized treatment strategies in glioblastoma MGMT promoter methylation status (2013) Niklas Thon, et al.

Exhibit 6: The discovery of GDC-0084 – Structure (Left), BBB Penetration (Middle), Efficacy (Right)

Molecular Structure of GDC-0084



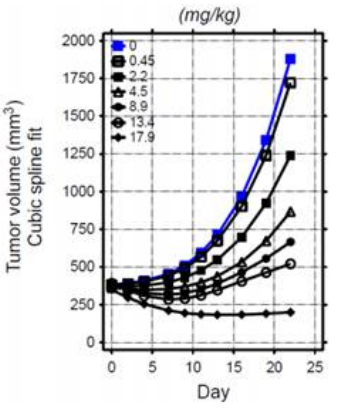
GDC-0084 retains the thienopyrimidine structure often seen in PI3K inhibitors

Relative Concentration of GDC-0084 in Pre-clinical Subjects

Species	[Brain]/ [Plasma]	$[Brain]_u$/ $[Plasma]_u$*	[CSF]/ $[Plasma]_u$**
Mouse	1.4[a]	0.4[a]	--
Rat	1.9-3.3[b]	--	0.7 - 1.0[c]

GDC-0084 was found to concentrate in the brain, with sufficient unbound concentration to remain pharmacologically active in pre-clinical subjects

Human Xenograft Efficacy Test of GDC-0084 by Dosage



Optimization of GDC-0084 dosage vs tumor reduction in human xenografts

Source: Genentech, Cedrus Research

The PI3K-mTOR Pathway as an Attractive Therapeutic Target in GBM

The **p**hosphatidyl**i**nositol **3**-**k**inase-**m**ammalian **t**arget **o**f **r**apamycin pathway (PI3K-mTOR) is a widely acknowledged target for anti-cancer drugs. The PI3K-mTOR pathway is deregulated in over one-third of human cancers[13], with some cancers displaying higher or lower levels of aberrancy. In GBM, the PI3K-mTOR pathway, being a key target to exploit for therapeutics, is altered in around 88% of GBM patients[14]. While a number of PI3K inhibitor drugs are in various stages of development, there are only two marketed products of this class. More importantly, almost none of these agents are able to cross the blood-brain barrier, and few have meaningful activity against mTOR, making GDC-0084 a key drug candidate in the GBM pipeline.

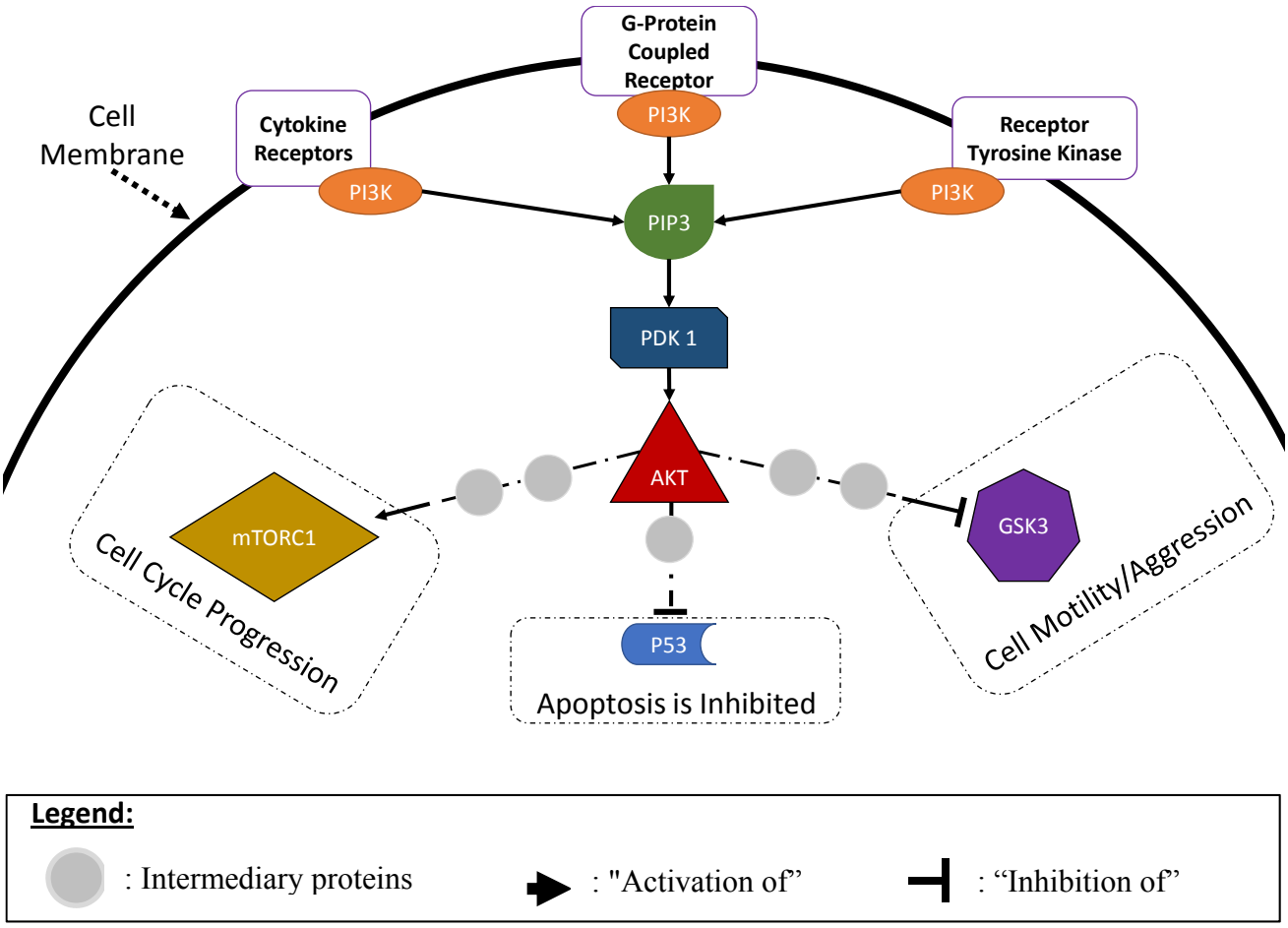
PI3Ks represent a family of lipid kinases[15] that are responsible for **upstream** intracellular signaling pathways. There are three classes of PI3Ks, with class I being responsible for cell growth, survival and autophagy[16]. Within class I PI3K, there are 4 isoforms, PI3Kα, PI3Kβ, PI3Kδ, and PI3Kγ. They transduce signals received from transmembrane receptors such as cytokine receptors[17], receptor tyrosine kinases[18] (RTKs) and G-protein coupled receptors[19] (GPCRs). When transmembrane receptors are activated through coupling with extra-cellular substrates, they activate PI3Ks, which then phosphorylate the 3'-hydroxyl group of PIP3

[13] Phosphatidylinositol 3-Kinase (PI3K) inhibitors as cancer therapeutics (2013) Akintunde Akinleye, et al.
[14] Glioblastoma Multiforme: A Review of its Epidemiology and Pathogenesis through Clinical Presentation and Treatment (2017) Farina Hanif, et al.
[15] Kinase – an enzyme that catalyzes the transfer of phosphate groups to specific substrates
[16] Consumption of the body's own tissue as a metabolic process occurring in starvation and certain diseases
[17] Cell surface proteins which recognize specific cytokines as ligands
[18] High-affinity cell surface receptors for many polypeptide growth factors, cytokines, and hormones
[19] It is the largest and most diverse group of membrane receptors in eukaryotes. These cell surface receptors act like an inbox for messages in the form of light energy, peptides, lipids, sugars, and proteins

(phosphatidylinositol 3). PIP3 then activates PDK1, which in turn activates AKT (also known as Protein Kinase B or PKB), the key effector molecule responsible for maintaining cell growth, cell proliferation and apoptosis (programmed cell death). The AKT protein is often found to be over-expressed in cancer patients.

The mammalian Target of Rapamycin, **mTOR**, also known as mTOR-complex when activated, is the key regulator of protein translation, a critical component for cell cycle progression. Belonging to the PI3K pathway, it is the **downstream** culprit that is often over-activated in cancers. Besides being activated through the PI3K pathway, mTOR has also found to be activated by alternative mechanisms without upstream signaling by PI3K. Therefore, it is optimal for drugs that target the PI3K-mTOR pathway to have characteristics in inhibiting both PI3K and mTOR.

Exhibit 7: PI3K-mTOR Pathway and its Role in Tumorigenesis



Note: AKT is a downstream effector of many cell growth mechanisms. In this exhibit, only three tumorigenesis pathways are shown. mTORC1 activation by a series of intermediary proteins initiated by AKT induces cell cycle progression. P53 inhibition by a series of intermediary proteins initiated by AKT leads to the inhibition of apoptosis. GSK3 inhibition by a series of intermediary proteins initiated by AKT results in cell growth and motility – an indicator of cancer metastasis.

Source: Cedrus Research

The PI3K-mTOR pathway offers an alternative pathway in tackling GBM as opposed to the standard of care using TMZ, which is highly sensitive to MGMT methylation. The **higher consistency of deregulation** across GBM patients should validate the first-line use of PI3K-mTOR inhibitors over TMZ.

While **GDC-0084** shows activity of inhibiting all class I PI3Ks, it is more selective in targeting the α and β isoforms. It has also shown to be an optimized inhibitor of the mTOR protein. Pre-clinical studies on human cell incubations have demonstrated inhibition of the key effector molecule AKT. Being a **dual inhibitor of PI3K and mTOR**, GDC-0084 presents some potential advantages over other PI3K pathway inhibitors under development.

Overview of PI3K-mTOR Inhibitors for GBM

GDC-0084 is among the 50 or more PI3K-mTOR inhibitors designed and produced for treating cancer. However, the development of a large number of these PI3K-mTOR inhibitors by large pharmaceutical companies such as Gilead (GILD), Sanofi (SNY) and Novartis (NVS) were discontinued in recent years due to their side effects on cancer patients. The list of drug candidates for GBM narrows even further, as the majority of PI3K-mTOR inhibitors fail to cross the blood-brain barrier.

Other than GDC-0084, the only other PI3K-mTOR pathway inhibitor being developed for GBM is Novartis' BKM120. Novartis has brought BKM120 through clinical trials for various indications, with advanced breast cancer (NCT01633060) leading at phase III, while BKM120 has recently completed phase II clinical trial in GBM (NCT01339052) with evaluation underway.

BKM120 is a pan-isoform class I PI3K inhibitor, meaning it inhibits all four isoforms of class I PI3K, but it has no direct effect on mTOR. Early phase III clinical trial results of BKM120 for advanced breast cancer have indicated high toxicity because of the drug's PI3K non-specificity. Some patients in the study showed high blood-levels of aspartate transaminase and alanine transaminase (which indicates liver damage), as well as psychiatric issues, including depression, anxiety, and suicidal tendencies. BKM120 was found to have various off-target binding, including inhibiting tubulin[20] function. Although the phase III study in breast cancer has reached its primary endpoint, it raised questions of clinical applicability. Evaluation is currently underway.

GDC-0084 is a dual inhibitor of PI3K and mTOR. PI3K and mTOR share similar binding domains, making it possible for a single molecule to effectively bind and inhibit both. In terms of binding with PI3K, GDC-0084 is more selective in inhibiting the PI3K α-isoform and β-isoform. More importantly, it is also an optimized inhibitor of mTOR. In terms of mTOR binding, GDC-0084 was designed to mildly inhibit mTOR rather than complete inhibition because strong inhibition of this protein was found to induce a positive feedback mechanism, which would over-activate the PI3K pathway. The **superior specificity in PI3K-isoform targeting** of GDC-0084 compared to pan-isoform class I PI3K inhibitors as well as the **optimal inhibition of mTOR** by GDC-0084, should yield a product with **higher efficacy and less toxicity**.

[20] A protein that is the main constituent of the microtubules of living cells

GDC-0084 Phase I Clinical Trials Completed with Maximum Tolerated Dose Established

Genentech completed GDC-0084 phase I clinical trials for GBM in January 2015. The study included 47 patients diagnosed with progressive or recurrent high-grade gliomas, including GBM and malignant astrocytoma[21].

The safety profile demonstrated general PI3K/mTOR-inhibitor related adverse effects, suggesting acceptable tolerability. The recommended dose was established at 45 mg daily based on half-life analysis.

In the phase I trials, GDC-0084 was rapidly absorbed by the subjects, and a linear- and dose-proportional increase in exposure was established. At 45 mg of GDC-0084 daily, the drug exhibited consistent anti-tumor activity in the subjects as it did in the pre-clinical models. FDG-PET scans and anti-tumor activity supported that GDC-0084 was able to cross the BBB to exert its anti-cancer effect on the affected areas. This was further confirmed later by the analysis of tissue samples from a surgical brain biopsy from a patient.

Single-agent anti-tumor activity was minimal. 55% of the patients demonstrated a best response of progressive disease, and 40% displayed stable disease, suggesting that GDC-0084 to be used in a combination therapy, similarly to TMZ being used in combination with radiotherapy.

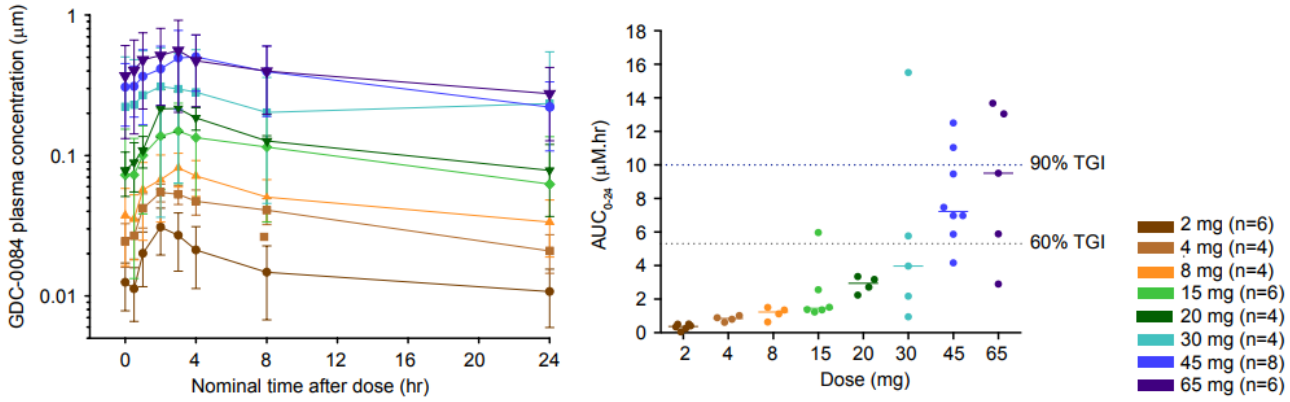
Exhibit 8: GDC-0084 Clinical Trial Phase I Overview

Clinical Trial Phase I - Official Title	Drug	Indication	Number of Patients (N)	NCT Identifier	Completion Date
An open-lable, phase I, dose-escalation study evaluating the safety and tolerability of GDC-0084 administered to patients with progressive or recurrent high-grade glioma	GDC-0084	Glioblastoma Multiforme	47	NCT01547546	January 2015
Primary Outcome Measures		**Secondary Outcome Measures**			
Saftey: Incidence of adverse events (up to 2 years)		Pharmacokinetics: Area under the concentration-time curve			
Maximum tolerated dose (MTD) up to 1 year		Best overall response rate, tumor assessments according to Response Assessment in Neuro-Oncology			
		Duration of response			
		Progression-free survival			
Results					
Primary Outcome:		**Secondary Outcome:**			
- Most frequent adverse events were fatigue, hyperglycemia, nausea, rash, hypertriglyceridemia, mucositis, hypophosphatemia, decreased appete and diarrhea		- GDC-0084 displayed an approximately linear and dose proportional increase in concentration and area under the curve in all cohorts (2-65 mg once daily)			
		- Rapidly absorbed, peaking at 2 hours after a single dose			
- The Maximum Tolerated Dose was determined to be 45 mg GDC-0084 given once daily and taken orally in 28-day cycles		- Half-life of approximately 18.7 hours			
		- GDC-0084 detected at similar levels in brain tumor and brain tissue			

Source: Genentech, Kazia, Cedrus Research

[21] It is a type of cancer of the brain, originating in a particular kind of glial cells

Exhibit 9: Phase I Clinical Trial Results – Concentration-Time Plot (Top Left), Steady-State Area under the Curve in Patients (Top Right) and GDC-0084 Concentration in Surgical Brain Specimen (Bottom)



GDC-0084 Concentration in a Surgical Brain Specimen		
Sample Area	**Free + Bound GDC-0084**	**Free GDC-0084**
Plasma	0.56uM	0.11uM
Brain Tissue	0.86uM	0.058uM
Brain Tumor	0.80uM	0.054uM

Notes:

Concentration-time plot shows steady plasma concentration over time at all dosages, suitable for daily dosing **Steady-state area under the curve** shows that at 45mg GDC-0084 dosage, the total growth inhibition (TGI) of the tumor was on average above 60%

GDC-0084 concentration in human brain specimen confirms that GDC-0084 is capable of penetrating the BBB, with effective concentrations of free and bound GDC-0084 found in a surgical biopsy of the brain at the Maximum Tolerated Dose of 45mg per day
Source: Genentech, Kazia, Cedrus Research

GDC-0084 Phase II Clinical Trials are Expected to commence by Year-End 2017

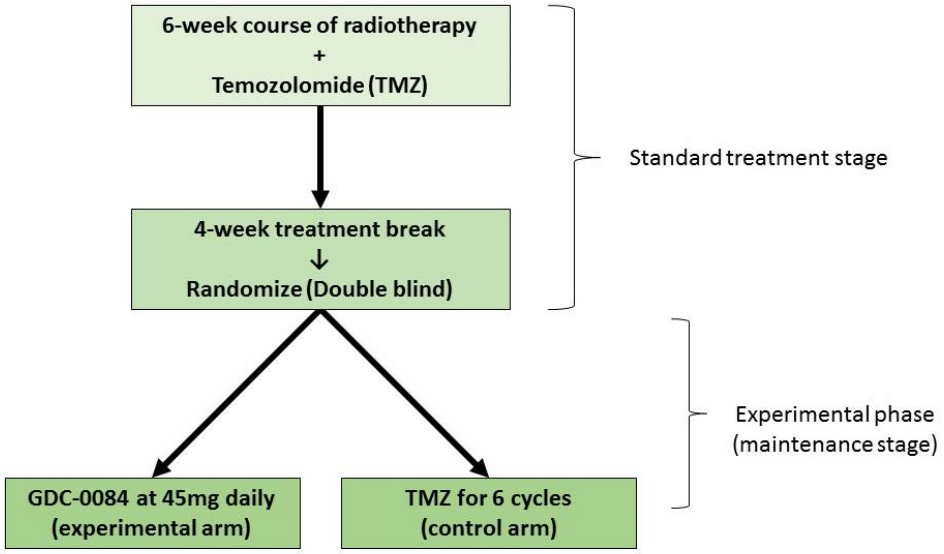
Kazia took the reins in the worldwide development and usage rights for GDC-0084 in October 2016. Kazia agreed to pay Genentech a total of USD5 million upfront together with modest regulatory and commercial milestones as well as industry-average royalties on net global sales.

To further commit to the development of GDC-0084, Kazia fully acquired the privately-held Glioblast Pty Ltd. Kazia believes that the experienced executives of Glioblast, Mr. Paul Hopper and Ms. Leslie Chong, the latter being the clinical program principal for GDC-0084 at Genentech, could provide instrumental direction and advice to Kazia on an ongoing basis. Kazia acquired Glioblast for AUD2.1 million, with AUD0.6 million in cash, and AUD1.5 million in equity. As part of the transaction, Glioblast's executives will also be entitled to four further milestone payments in cash or equity based on the performance of the phase II clinical trials. The first two of these milestones are valued at AUD1.25 million each and will be paid only in equity, while the remaining two in the amount of approximately AUD3.4 million and AUD3.8 million will be settled in either cash or Kazia's shares at Kazia's discretion.

Kazia is on track to commence the phase II clinical trials of GDC-0084 before the end of 2017. The trial will target the recruitment of patients with unmethylated MGMT promoter who have undergone surgical

resection of GBM tumors. Patients will first undergo the standard procedure of treatment – a six-week cycle of radiotherapy in combination with TMZ. The study participants will then be put on a treatment break to minimize residual effects of drugs. The subjects will then be randomly enrolled to either one of the following two options: 1) a daily dose of 45 mg of GDC-0084 (experimental arm), or 2) the standard 6 cycles of TMZ (control arm). The primary endpoints will include progression-free survival, as well as overall survival.

Exhibit 10: Proposed Phase II Clinical Trial Design for GDC-0084



Source: Kazia, Cedrus Research

We believe the proposed study design is advantageous to GDC-0084, given that the targeted unmethylated MGMT patient population is knowingly likely to be non-responders to TMZ. Any anti-tumor activity triggered by GDC-0084 should be apparent in the study, proving GDC-0084 is capable of treating an unmet medical need observed in a large proportion of GBM patients. We believe this could potentially warrant an "Accelerated Approval" designation by the U.S. FDA, and similarly, the "Priority Review" status by Australia's Therapeutic Goods Administration (TGA).

If such an accelerated designation is granted, it could benefit Kazia financially, as marketing of the drug occurs alongside its confirmatory clinical studies. While we have explored this scenario, we believe it is too early to include it in our forecast for valuation purposes.

We note that Kazia will most likely need to conduct a secondary study to prove safety and efficacy for GDC-0084 to be administered concomitantly with radiotherapy to position the drug to replace TMZ. Satisfactory results from this secondary study could also potentially pave way for a more straightforward execution of its phase III clinical trials.

On 21st September 2017, Kazia met with the U.S. FDA in relation to the proposed phase II clinical study. The study design will include a lead-in component, which attempts to optimize dosing in the patient population,

potentially generating preliminary data as early as 12-15 months after commencement of the study. This could provide substantial visibility to the drug going forward and warrant a revised marketing-probability risk adjustment. Kazia has contracted Chiltern Oncology, a leading international Contract Research Organization (CRO), to prepare the set-up activities of the upcoming study.

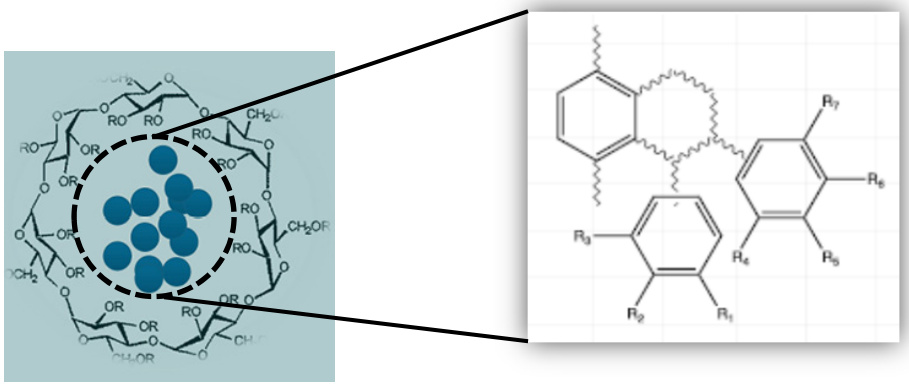
Drug in Clinical Development: Cantrixil

Overview of Cantrixil

Cantrixil is a low-molecular-weight molecule derived from the superbenzopyran technology platform. Superbenzopyran adds another layer of complexity and potentially creates a new family of drugs based on the basic benzopyran structure. Cantrixil, the active ingredient, is encapsulated in a cyclodextrin, a common "carrier" molecule. The drug will revive the use of intra-peritoneal (IP) administration, a means of direct delivery of the drug to the peritoneal[22] cavity, which will be administered immediately post-debulking surgery. The unique hallmark of Cantrixil is its ability to **target cancer stem cells**, **the main source of recurrent ovarian cancer**.

Kazia had applied for patent protection for Cantrixil in 25 jurisdictions worldwide and was granted protection in major pharmaceutical markets, including the U.S., the European Union, Australia and China.

Exhibit 11: Molecular Structure of Cantrixil – Active Ingredient is Enclosed by a Cyclodextrin or "Carrier" Molecule (Left), Cantrixil (TRXE-002-1), the Active Ingredient (Right)



Source: Kazia, Cedrus Research

Cantrixil was first identified from a library of analogues as being able to induce apoptosis in both cancer and cancer stem cells (CSC) across a range of cancers. Pre-clinical studies of the drug also showed that it was capable of disrupting the hallmark spheroid cultures[23] that arise from cancer stem cells *in vitro*[24].

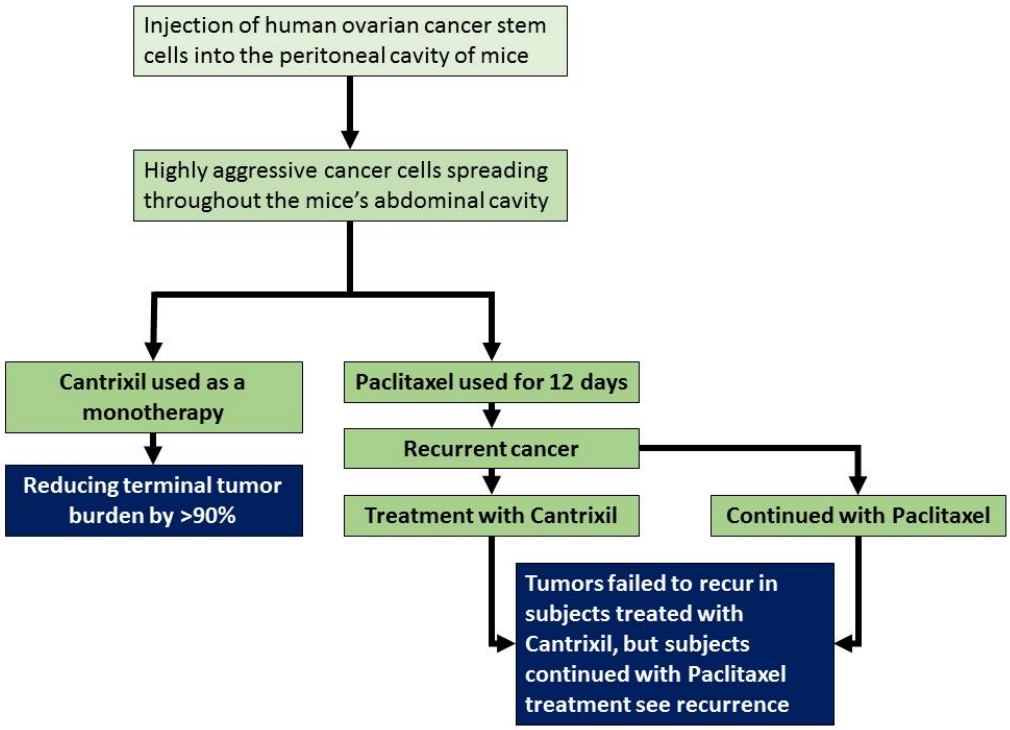
[22] The membrane that lines the walls of the abdomen and the pelvis (called the parietal peritoneum) and encloses the abdominal and pelvic organs (called the visceral peritoneum.)

[23] A 3D cell culture is an artificially-created environment in which biological cells are permitted to grow or interact with their surroundings in all three dimensions

The study later moved into *in vivo* rodent models that represented intra-peritoneal human ovarian cancer. Human ovarian cancer stem cells were injected directly into the peritoneal cavity of mice, which developed into highly aggressive cancer cells spreading throughout the abdominal cavity. Mice were first treated with paclitaxel (Taxol®), the standard first-line treatment for ovarian cancer, and an initial partial response was observed. Some of the mice's tumors quickly regained rapid growth even with ongoing treatment with paclitaxel.

In the scenario with **Cantrixil** being administered intraperitoneally as a monotherapy, **tumor growth was inhibited, significantly reducing tumor burden[25] by over 90%**. In the second scenario, Cantrixil was even able to **reduce tumor recurrence in animal subjects** despite being treated with paclitaxel first.

Exhibit 12: Pre-clinical Studies of Cantrixil in Mice Models



Source: Kazia, Cedrus Research

In April 2015, Cantrixil was granted the "Orphan Drug Designation" by the U.S. FDA for ovarian cancer, which entails financial subsidies during clinical research and development stages, tax incentives, and extension of patent protection when marketing begins. Cantrixil's Investigational New Drug (IND) application, a prerequisite for initiating clinical trials in the U.S., in ovarian cancer was approved by the FDA in September 2016.

[24] A process performed or taking place in a test tube
[25] Refers to the number of cancer cells, the size of a tumor, or the amount of cancer in the body

Today's Standard of Care Leaves a Significant Unmet Therapeutic Gap

Ovarian cancer prognosis is based on the stage at which it is diagnosed. Unfortunately, in the early stages (I and II) ovarian cancer is usually asymptomatic[26], resulting in low diagnostic rates. In these early stages, the cancer is usually benign and surgical removal of the tumor area is usually sufficient, and chemotherapy is also given if necessary. At the later stages of ovarian cancer (III and IV), metastasis[27] usually occurs, leading to a variety of symptoms across the abdominal area. Debulking surgery (removing as much of the tumor as possible) is following by 3-6 cycles of chemotherapy. The usual chemotherapy regimen used is a combination of platinum-based agents coupled with a non-platinum-based drug, usually paclitaxel, either through intravenous (IV) injection or intraperitoneal (IP) injection.

Exhibit 13: Ovarian Cancer Statistics Overview by Stage

Ovarian Cancer	% of Case Diagnosed at this Stage	Risk of Recurrence	Relative 5-Year Survival Rate
Stage I	15%	10%	90%
Stage II	19%	30%	46%
Stage III	60%	70-90%	39%
Stage IV	6%	90-95%	17%

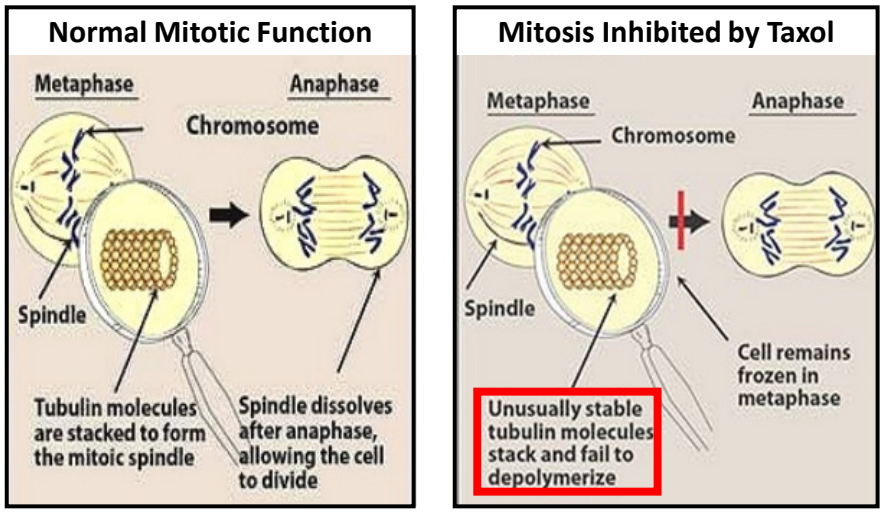
Source: American Cancer Society, Ovarian Cancer Research Alliance

Platinum-based agents, such as carboplatin or cisplatin, are capable of damaging the DNA in cells. They show high rates of complete tumor response, compared to non-platinum-based drugs, but exhibit more adverse effects, including nausea, vomiting, and could lead to a lower platelet count (immunocompromised). Physicians have been debating the risk-benefit profile of platinum-based agents, and some physicians may even skip administering a platinum-based agent and only prescribe paclitaxel.

Paclitaxel is a microtubule-stabilizing drug that is often used for the treatment of ovarian, breast and lung cancer. Microtubules are responsible for cell growth and reproduction. When paclitaxel is introduced in the cellular environment, it induces cell mitotic arrest by preventing chromosome mis-segregation, a critical step for cells to divide and grow. When the cancer cell has detected that mitosis has been stalled, further growth is inhibited, and apoptosis occurs.

[26] Producing or showing no symptoms
[27] The development of secondary malignant growths at a distance from a primary site of cancer

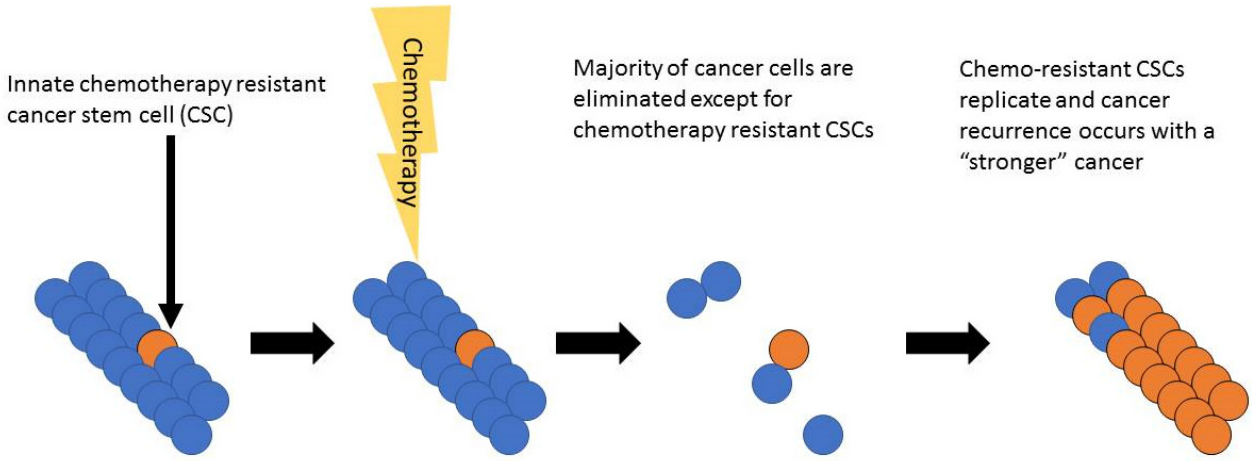
Exhibit 14: Paclitaxel (Taxol®) Mechanism of Action



Source: Dr. K. Saminathan. M. Pharm, M.B.A, Ph.D., Cedrus Research

The chemotherapy regimen used in today's standard of care exploits cellular pathways that are often not as prominent in cancer stem cells (CSC). Various solid tumor CSCs have certain innate *chemo-resistant mechanisms* capable of defending itself from modern chemotherapeutic agents, for example, through physical blockage or removal of the drug from entering the cell, and cellular repair mechanisms. These chemo-resistant cells are usually present in small quantities initially, but when modern chemotherapy is used to flush away non-chemo-resistant cells, it is these ***chemo-resistant CSCs*** that begin to grow, differentiate and eventually **cause the onset of an increasingly *chemo-resistant cancer***.

Exhibit 15: Innate Chemo-Resistant Cancer Cells Cause Cancer Recurrence



Source: Kazia, Cedrus Research

These chemo-resistant CSCs are characterized by the capacity to grow, ability to give rise to heterogeneous progeny cells, and able to modulate its own differentiation and growth based on its cellular environment. In many solid tumors, it has been established that chemo-resistant CSCs share common biomolecular markers, $CD44^+$ and $MyD88^+$ [28], markers that are either lacking or in remarkably lower quantities in chemo-sensitive cells (denoted as $CD44^-$ and $MyD88^-$ respectively).

CD44 is a cell surface antigen used for cell-to-cell interactions, and is overexpressed in virtually all chemo-resistant CSCs of many solid malignancies. $CD44^+$ cell culture doubles every 36 hours whereas $CD44^-$ cells double every 16 hours[29]. It is most likely that paclitaxel is more effective in these non-chemo-resistant $CD44^-$ cells because the drug is able to interfere with their relatively quicker replicating abilities.

$MyD88^+$ is the other revealing sign of chemotherapy resistant ovarian cancer. MyD88 is an adaptor protein responsible for activating the inflammatory response pathway. Although the pathway is not fully understood, MyD88 has been found to play a critical role in the process of repair and differentiation in CSCs. It could be that, when chemotherapeutic drugs damage $MyD88^+$ cells, overexpression of MyD88 induces cellular repair pathways that overwhelm any chemotherapeutic effects.

Cantrixil's Mechanism of Action – Promoting Pro-Death Pathway and Inhibiting Pro-Survival Pathway

Cancer recurrence post-chemotherapy is due to the expansion of surviving innately drug-resistant cancer stem cells that remain in tumor niches during and post-chemotherapy. The expansion of these cells, coupled with the expression of these pro-survival pathways, is responsible for the development of increasingly chemo-resistant recurrent cancers.

Cantrixil has shown *in vitro* and *in vivo*[30] that it has anti-cancer activity in a range of cancer types, and Kazia has initially positioned the drug to target ovarian cancer because of the large percentage of cases of recurrent cancer post-chemotherapy. According to the Ovarian Cancer Research Fund Alliance, over 70% of patients diagnosed at stage III or IV and treated with chemotherapy are at risk of experiencing tumor recurrence (see exhibit 13).

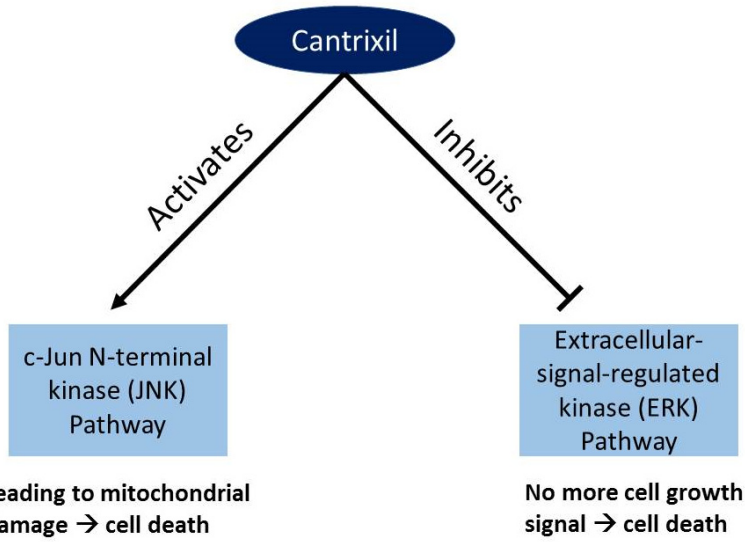
Cantrixil's novel anti-cancer mechanism is associated with the **activation of two apoptotic pathways** – the *pro-death* c-Jun N-terminal kinase (JNK) pathway and concurrent inhibition of the extracellular-signal-regulated kinase (ERK), one of the four mitogen-activated protein kinase (MAPK) *pro-survival* pathways. This dual effect on pro-cell-death and anti-survival mechanisms makes Cantrixil an **efficient killer of both ovarian cancer cells and chemo-resistant ovarian cancer stem cells**. These mechanisms bypass the cell cycle inhibition effect exhibited by paclitaxel and have shown to be equally effective in both $CD44^+$/$MyD88^+$ cells, suggesting cancer stem cells do not have any innate ability to counteract Cantrixil.

[28] The + superscript indicates that the cell expresses the phenotype, while a – superscript indicates that the cell does not express the phenotype

[29] Molecular phenotyping of human ovarian cancer stem cells unravel the mechanisms for repair and chemo-resistance (2009) Ayesha B. Alvero, Rui Chen, et al.

[30] A process performed or taking place in a living organism

Exhibit 16: Cantrixil's Dual-Mechanism Approach

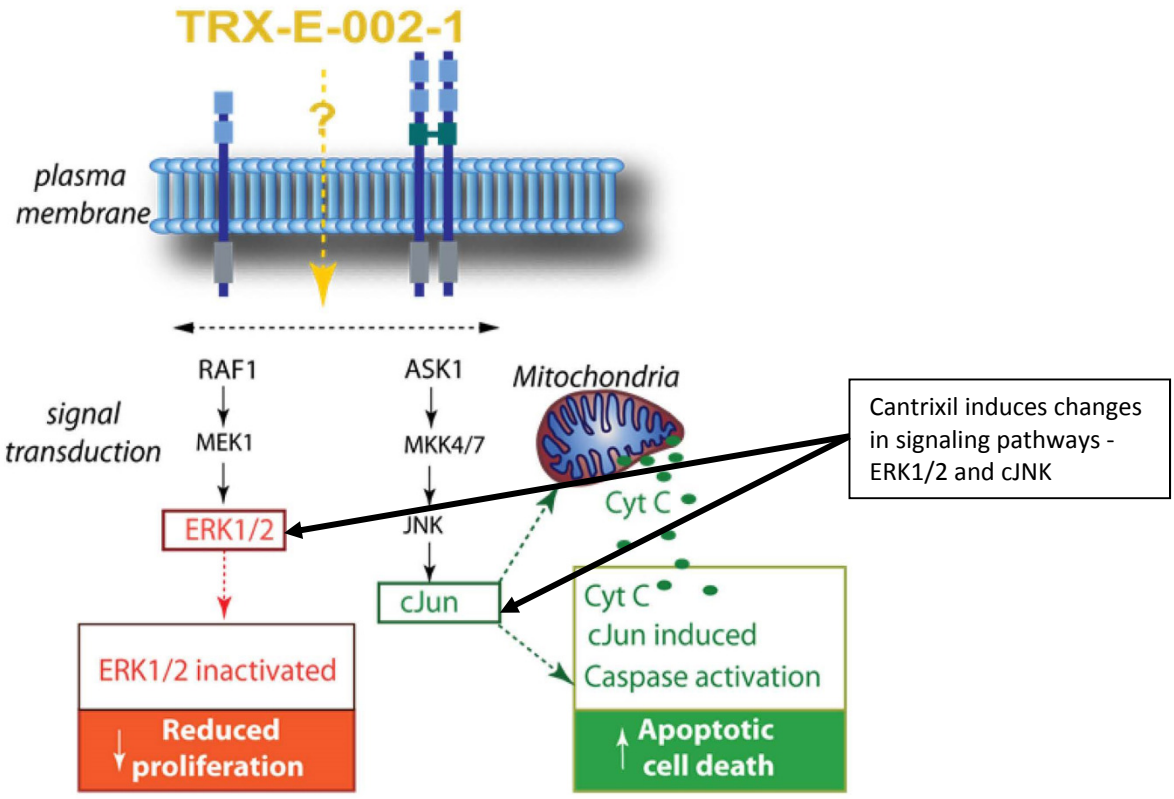


Source: Kazia, Cedrus Research

Both the JNK and ERK pathways belong to the family of mitogen-activated protein kinase (MAPK). MAPK is responsible for the regulation of a series of substrates accountable for cell growth. The ERK pathway is directly involved with the regulation of mitosis. The JNK pathway is stress-activated, and upon activation, ultimately restrains protein synthesis. Despite the JNK complex involves many downstream regulatory targets, Cantrixil was found to mainly activate the JNK-c-Jun pathway, which recruits proapoptotic[31] proteins resulting in activation of caspases[32].

[31] Promoting or causing apoptosis
[32] A family of protease enzymes playing essential roles in programmed cell death

Exhibit 17: Cantrixil (TRXE-002-1) Mechanism of Action



Note: It is still unclear how Cantrixil crosses the cell membrane; however, its impact on the intracellular ERK and JNK pathways has been confirmed in pre-clinical tests

Source: Kazia

Cantrixil Clinical Trial Phase I Overview

Kazia commenced phase I clinical trials of Cantrixil in ovarian cancer (NCT02903771) in December 2016. The primary objectives are to determine the maximum tolerated dose (MTD), its safety profile in human subjects, and the drug's pharmacokinetic[33] profile when administered as a monotherapy as well as in combination with standard chemotherapy agents. With recruitment ongoing, the study will include up to 60 ovarian cancer patients across three hospitals in Australia and two hospitals in the U.S. Kazia expects to announce MTD in 1Q 2018, complete the phase 1 study by 1H 2018, and obtain exploratory efficacy data later in 2018.

[33] The bodily absorption, distribution, metabolism, and excretion of drugs

Pre-Clinical Drugs in Development: Next-Gen ATM Drug

Kazia is also developing a **Next-Generation ATM** drug, which aims to exploit the cell's actin[34] cytoskeleton[35] as an anti-cancer drug. Such drugs have been extremely difficult to formulate due to their high toxicity. However, the cytoskeleton remains a desirable anti-cancer target because it controls many fundamental processes of a cancer cell, such as its growth, movement, and intra-cellular interactions.

Kazia is currently formulating a new Next-Generation ATM product after the development of the company's previous ATM product, Anisina (ATM-3507), was terminated earlier in 2017 due to the toxicology levels of the product. The company was awarded in February 2017 a AUD3 million grant, receivable over a three-year period, under the Cooperative Research Centre Project (CRC-P) funded by the Australian government to develop this Next-Generation ATM drug. The development will be led by Kazia, in collaboration with the University of New South Wales (UNSW) and ICP Firefly Pty Ltd., a private contract research organization headquartered in Sydney, Australia. Kazia has committed AUD1 million of its own funds to the program over the three-year term of the grant.

III. MARKET OVERVIEW AND ANALYSIS

Glioblastoma Multiforme Treatment Overview and Drug Pipeline Analysis

Current Standard of Treatment Overview

At present, there are no curative options for glioblastoma multiforme. Upon diagnosis, the average duration of survival is three months without treatment, and up to an average of two years with treatment of currently approved drugs. The standard of care usually entails a combination of radiation and the chemotherapy drug, temozolomide (TMZ), and sometimes debulking surgery if possible, followed by a maintenance phase with just temozolomide. If the disease continues to show progression after initial treatment, physicians have no clear guideline with respect to recurrence or second-line therapies. Some patients are given a single-agent alternative treatment, a combination of such treatments, or undergo the standard of care procedure once again.

Alternative treatments for GBM are extremely limited and can only delay the progression of the disease. Some of these alternative therapies include Genentech's Avastin®, a monoclonal antibody targeting the VEGF-cytokine that inhibits new blood vessels from forming, thus choking off the blood supply to the tumor – a mechanism known as "tumor starving". Bristol-Myers-Squibb's (BMY) Carmustine wafer procedure, which involves physically implanting wafers loaded with Carmustine at the cancerous region. Carmustine, a cytotoxic[36] agent, will be released gradually over the affected area, delaying cancer progression. Despite the wide disparity of data related to the survival benefits of these alternative therapies, the Carmustine wafer procedure has been approved and used as a first-line therapy usually administrated alongside radiotherapy. Using modern approved treatments, the estimated overall survival of recurrent GBM is about 4-7 months.

[34] A family of globular multi-functional proteins that form microfilaments
[35] It consists mainly of actin filaments and microtubules and plays an important role in cell movement, shape, growth, division, and differentiation, as well as in the movement of organelles within the cell
[36] Refers to a substance or process which results in cell damage or cell death

Glioblastoma Multiforme Treatment Pipeline

There has been limited approval of new drugs in treating GBM, as many developing drugs have failed to show improved clinical benefits as compared to the standard of care, while some even have shown noteworthy adverse side effects. The blood-brain-barrier still represents a major hurdle for drugs in development.

The newest class of drugs in the development pipeline for GBM includes immunotherapeutic biologics. These are vaccines that activate the immune system to attack cancer cells. However, the recent immunotherapeutic candidates have demonstrated limited survival benefits over existing treatments. These vaccines include Celldex Therapeutics' (CLDX) Rintega, the development of which has been discontinued after poor phase III clinical results, and ImmunoCellular's (IMUC) ICT-107, which has also reported a poor efficacy profile, and its development has been suspended due to lack of financial resources.

GDC-0084's Unique Therapeutic Position

GDC-0084 is among one of the very few PI3K-pathway inhibitors capable of penetrating the blood-brain barrier. Despite the PI3K-pathway being widely dysregulated across GBM patients, majority of PI3K-pathway inhibitors fail to penetrate the blood-brain barrier, constituting GDC-0084 a unique and promising GBM drug candidate.

GDC-0084 targets a much more common pathway compared to today's standard of care. GDC-0084 targets the PI3K/mTOR pathway that is observed to be deregulated in approximately 88% of GBM patients, as opposed to TMZ and other GBM drug candidates, such as Val-083 developed by DelMar Pharmaceuticals (DMPI), that target and damage DNA synthesis (humans have many innate repair mechanisms to deal with damaged DNA). We believe GDC-0084 has the potential to replace TMZ as the standard of care.

GDC-0084 provides dual-PI3K and mTOR inhibition. The PI3K-mTOR pathway is a well-established target in cancer therapeutics. As compared to other PI3K inhibitors, GDC-0084 is capable of inhibiting both the upstream signaling molecule PI3K and the key downstream cell growth regulator mTOR. The only other PI3K-inhibitor being developed for GBM is Novartis' BKM120. It is a pan-class I PI3K-inhibitor, which failed to exhibit notable anti-cancer properties because it allows the existence of alternative pathways capable of activating mTOR. In addition, the non-specificity of BKM120 across PI3K isoforms has displayed adverse side effects not observed in GDC-0084, as it targets specifically the alpha-isoform and beta-isoform of the PI3K.

Exhibit 18: Overview of Currently Commonly Used Drugs for Glioblastoma Multiforme

Drug/Procedure	Originator	Indication	Administration Regimen	Mode of Action	Efficacy Measures	Procedure Pricing
Temozolomide (Temodar)	Merck	First-line treatment for GBM	Oral capsule taken initially in combination with radiotherapy, and then taken as monotherapy for maintenance treatment	DNA alkylating agent (cytotoxic), interferes with DNA replication, leading to cell cycle arrest	Extends survival duration of approximately 12.1-14.6 months. Less than 50% response rate in patients	USD30,000 pre-patent expiry → USD20,000 post-patent expiry
Bevacizumab (Avastin®)	Genentech (Roche)	Recurrent GBM or second-line therapy	Monoclonal antibody through intravenous infusion	Bind and neutralize all subtypes of **VEGF** – the cytokine responsible for angiogenesis[37] – a method known as tumor starving	Mixed/Unestablished treatment benefits	USD53,000
Lomustine (CCNU, CeeNU)	Corden Pharma	Recurrent GBM or second-line therapy	Oral capsuled nitrosourea-based drug	DNA and RNA alkylating agent (cytotoxic) causing inter- and intra-strand cross-linking of DNA, leading to cell cycle arrest	Mixed/Unestablished treatment benefits; more side effects than TMZ	Not available
Carmustine wafer (BCNU, Gliadel® wafer)	Bristol-Myers-Squibb	First-line or recurrent GBM	Carmustine wafers implanted at the cancerous region during surgery	Gradual release of Carmustine over the cancerous area. The drug is a DNA alkylating agent (cytotoxic), leading to cell cycle arrest	Extends survival duration of approximately 13.9 months. 30% response rate in patients	USD30,000

Source: Merck, Genentech, Corden Pharma, Bristol-Myers-Squibb, Cedrus Research

[37] The development of new blood vessels

Exhibit 19: Leading Investigational New Drugs in the Pipeline for Glioblastoma Multiforme

Drug	Originator	Drug Type	Mode of Action	Preliminary Efficacy Measures	Current Status	NCT Identifier
Phase III Clinical Trials						
DCVax®-L	Northwest Biotherapeutics (NWBO)	Immuno-therapeutic vaccine	Utilizes autologous activated dendritic cells to activate the immune system to attack **all** cancer-specific antigens	Estimates that 25% of GBM patients live four years or more	Study completed. Evaluation ongoing	NCT00045968
ICT-107	ImmunoCellular (IMUC)	Immuno-therapeutic vaccine	Utilizes autologous activated dendritic cells to activate the immune system to attack **six** cancer-specific antigens	Phase II results: Patients live 2 months longer (not statistically significant) compared to those receiving *standard of care*	Suspended due to lack of financial resources	NCT02546102
Nivolumab (Opdivo)	Bristol-Myers-Squibb (BMY)	Monoclonal antibody	Nivolumab binds and inhibits PD-1, a cell surface receptor, enabling the immune system to attack the cell	Showed no benefit over *standard of care* in recurrent GBM patients	Recruiting two phase III trials as the first-line therapy for new GBM patients	NCT02667587 NCT02617589
Toca 511 and Toca FC	Tocagen (TOCA)	Retroviral Replicating Vector (RRV)	Toca 511 is administered to the tumor, which produces cytosine deaminase (CD). Toca FC is then orally administered, and converted into an anti-cancer drug by CD	Phase I results: median overall survival of 14.3 months in recurrent GBM	Phase II/III Data due: 1H 2018	NCT02414165
Phase II Clinical Trials						
AdV-tk	Advantagene	Gene-directed therapy using therapeutic viruses	Bioengineered virus delivered locally to the tumor + oral prodrug causes tumor cytotoxicity	Phase II results: median 3.6 months longer in overall survival than control	Completed in April 2017. Evaluation ongoing	NCT00589875
GDC-0084	Genentech (licensed to Kazia Therapeutics [KZA AU, KZIA])	Dual PI3K/mTOR inhibitor	Inhibits the class I PI3K-**α and β isoforms** and mTOR, the respective upstream and downstream regulator for cell growth. Such inhibition prevents cell growth and induces apoptosis	Not yet established	Preparing to initiate phase II (tentatively by year-end 2017)	N/A
Val-083	DelMar Pharmaceuticals (DMPI)	Alkylating agent	Readily crosses the blood-brain barrier, inducing interstrand cross-links at guanine-N7, causing DNA double-strand breaks, a pathway independent of MGMT pathway	Overall survival benefit of 8.4 months when used in combination with radiotherapy vs. radiotherapy alone (control)	Recruiting for phase II. Completion is expected in June 2021. (Already marketed for leukemia and lung cancer in China)	NCT02717962
BKM120 (Buparlisib)	Novartis (NVS)	Pan-class 1 PI3K inhibitor	Inhibits **all** isoforms of class I PI3K, the upstream regulator for cell growth. Such inhibition prevents cell growth and induces apoptosis	Not yet established, but high incidence of adverse events were reported in phase III breast cancer study	Three phase I/II clinical trials are ongoing	NCT01339052 NCT01349660 NCT01473901

Source: Clinictrials.gov, Cedrus Research

Ovarian Cancer Treatment Overview and Drug Pipeline Analysis

Since the early 2000s, the standard of care for ovarian cancer has been established. Patients undergo debulking surgery, if possible, and are given carboplatin in combination with paclitaxel in the adjuvant and first-line treatment, while sometimes opting for paclitaxel alone.

Attempts have been made to improve survival and response rates by a wide variety of means, but they failed to demonstrate superiority. More targeted therapies, new routes of administration, different dosing times, and different combinations of approved treatments were explored, but all of them have failed to produce any compelling results such that they can supplant the standard chemotherapeutic procedures that use paclitaxel and platinum-based chemotherapeutic drugs. Regardless, due to the variations of the disease across ovarian cancer patients, many different treatment regimens based on alterations of the standard of care are pursued today.

For early stage (I and II) ovarian cancer patients (approximately 34% of patients are diagnosed at these stages), the tumorous area is usually benign, and a debulking surgical procedure is usually sufficient to treat the condition. Such surgical treatment in the U.S. costs in the USD10,000-20,000 range. For patients with late-stage (III and IV) ovarian cancer (approximately 66% of patients diagnosed at these stages), they will undergo a debulking surgery if feasible; however, the cancer has usually metastasized across the abdominal area, and chemotherapy is sometimes the only viable option. Chemotherapy treatment alone, with physician supervision and follow-ups, costs approximately USD66,000.

Recently, a new class of drugs known as poly (ADP-ribose) polymerase (PARP) inhibitors has steadily been granted marketing approval by the U.S. FDA. PARP inhibitors are meant to be used in combination with chemotherapy by augmenting their cytotoxic capabilities without increasing side effects, and they have minimal therapeutic effect when used alone. PARP inhibitors prevent cancer cells from repairing damage in their DNA. Avastin®, the monoclonal antibody inhibiting VEGF, was also approved to be used for ovarian cancer in combination with TMZ and platinum-based chemotherapeutic agents.

The Niche Position of Cantrixil

Cantrixil is a niche product in the ovarian cancer drug pipeline. Cantrixil was produced specifically for eliminating cancer stem cells, while the competition aims to kill matured cancer cells. Targeting dual cellular pathways, ERK and JNK, by Cantrixil is also innovative in terms of treating cancer cells. Cantrixil addresses the large number of patients experiencing cancer recurrence arising from the deficiency of today's standard of care. Because Cantrixil is more potent in killing cancer stem cells than mature cancer cells, it will most likely be administered in combination with paclitaxel and a platinum-based chemotherapeutic agent to obtain the optimal therapeutic effect. If Cantrixil achieves marketing approval, it could potentially eliminate the recurrence of ovarian cancer.

Cantrixil could revive intraperitoneal (IP) administration. By administrating Cantrixil through IP injection, a physician will be able to deliver the drug directly to the affected site. Strong clinical benefits for IP administration of chemotherapy in ovarian cancer patients have been observed, with various ongoing studies underway for confirmation (NCT00951496, NCT00993655 and NCT01506856). However, IP administration has had limited adoption most likely due to the need to insert and manage the IP catheter.

Exhibit 20: Current Drug Approved for Treatment of Ovarian Cancer

Drug/ Procedure	Drug Type	Indication	Administration Regimen	Mode of Action	Efficacy Measures
Paclitaxel (Taxol)	Alkylating agent	Used in combination therapy for first-line treatment of ovarian cancer	Intravenous Injection usually in combination with either Carboplatin or Cisplatin	Paclitaxel binds selectively and is reversible to tubulin; over-promoting polymerization causes a cell cycle halt	Progression-free survival of 14.7 months when used in combination with carboplatin
Carboplatin	Platinum-based alkylating agent	Used in combination therapy for first-line treatment of ovarian cancer	Intravenous injection usually in combination with Paclitaxel	Forms platinum complexes that cause intra- and inter-strand cross-linkage of DNA, inhibiting DNA synthesis	Progression-free survival of 14.7 months when used in combination with Paclitaxel
Cisplatin (Platinol)	Platinum-based alkylating agent	Used in combination therapy for first-line treatment of ovarian cancer	Intravenous Injection usually in combination with Paclitaxel	Forms platinum complexes that cause intra- and inter-strand cross-linkage of DNA, inhibiting DNA synthesis	Similar efficacy measures as Carboplatin when used in combination with Paclitaxel
Bevacizumab (Avastin®)	Monoclonal antibody (through intravenous infusion)	More commonly used to treat recurrent ovarian cancer	Intravenous injection	Binds and neutralizes all sub-types of **VEGF** – the cytokine responsible for angiogenesis – a method known as tumor starving	When used in combination with Paclitaxel: Overall survival of 42.2 months vs 37.3 months with Paclitaxel alone

Source: Cedrus Research

Exhibit 21: Leading Investigational New Drugs in the Pipeline for Ovarian Cancer

Drug	Originator	Drug Type	Mode of Action	Preliminary Efficacy Measures	Current Status	NCT Identifier
Phase III Clinical Trials						
Mirvetuximab Soravtansine (IMGN853)	ImmunoGen (IMGN)	Folate receptor alpha (FRA)-targeting antibody-drug conjugate	IMGN853 is specific to FRA-expressing cancer cells to deliver the anti-tumor agent DM4 to kill the cancer cell	From Phase I/II study: Objective response rate (ORR) of 26% and progression-free survival (PFS) of 4.8 months vs. control arm's ORR of 15-20% and PFS of 3-4 months	Recruiting for phase III; estimated completion date: Feb 2019	NCT02631876
Phase II Clinical Trials						
Kevetrin	Innovation Pharmaceuticals (IPIX)	Tumor suppressor activator	Kevetrin activates p53, a key tumor suppressor protein. P53 is mutated in a large number of cancers	Unestablished	Recuiting for phase IIa in platinum-resistant/refractory ovarian cancer; estimated to be completed in Dec 2017	NCT03042702
TPIV-200	TapImmune (TPIV)	Multi-epitope peptide vaccine, targeting folate receptor alpha	The vaccine will activate T-cells to develop antibodies against over-expressed folate receptor alpha	Unestablished	Recruiting for two phase II studies to be used as a stand-alone product and in combination	NCT02764333 NCT02978222
Farletuzumab	Morphotek	Monoclonal antibody, targeting folate receptor alpha	Attacks FRA-expressing cells by introducing antibody-mediated cytotoxicity	Early studies used in combindation with standard of care have not shown any clinical benefits	Recruiting for phase II to be used in combination with anti-PD-L1 drug; estimated to be completed in Dec 2020	NCT02289950
PTX-200	Prescient Therapeutics (PTX AU)	AKT-inhibitor	Prevents AKT from anchoring to the cell membrane, inhibiting its activation and subsequent cancer cell division and growth	Unestablished	Recruiting for phase I/II to be used in combindation with Carboplatin; estimated to be completed in Dec 2017	NCT01690468
Phase I Clinical Trials						
Ribociclib (LEE011)	Novartis (NVS)	CDK4/6 inhibitor	Inhibits CDK4/6 to help slow the progression of cancer. The drug phosphorylates the retinoblastoma protein (pRb)	Unestablished	Marketing for breast cancer. Currently recruiting for phase I to be used with platinum-based chemotherapy in recurrent ovarian cancer; estimated completion date: Feb 2019	NCT03056833
NUC-1031	Imperial College Healthcare NHS Trust and NuCana plc (NCNA)	Pyrimidine analogue	NUC-1031 is converted into the active metabolites gemcitabine diphosphate (dFdCDP) and gemcitabine triphosphate (dFdCTP). These reduce ribonucleotide reductase, decreasing the deoxynucleotide pool for DNA replication and inducing apoptosis	Unestablished	Recruiting for phase IB to be used in combination with Carboplatin to treat recurrent ovarian cancer; estimated completion date: Mar 2017	NCT02303912
Cantrixil	Kazia Therapeutics (KZA AU, KZIA)	Superbenzopyran	Novel dual-mechanism pathway. 1) Activates JNK-c-Jun pathway for apoptosis signaling and 2) Inhibits the ERK pathway, a cell growth signaling pathway	Unestablished	Recruiting for phase I to be used through intra-peritoneal administration; estimated completion date: Dec 2018	NCT02903771

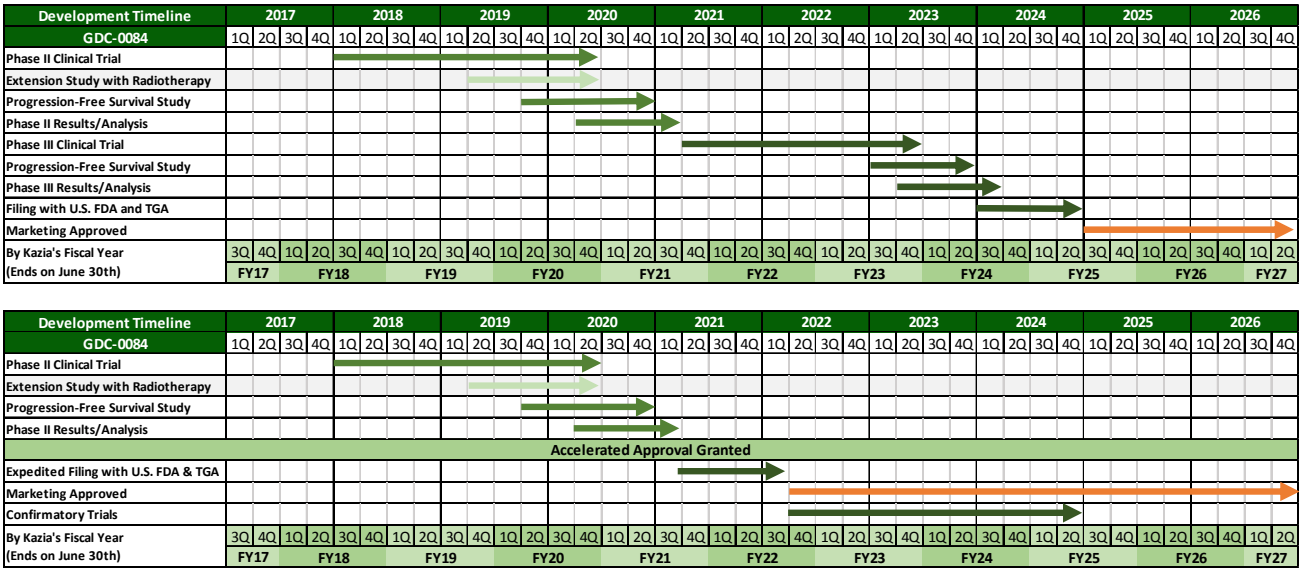
Source: Cedrus Research, ClinicalTrials.gov

IV. DEVELOPMENT TIMELINE OVERVIEW

Timeline of Kazia's Drug Development

GDC-0084 is on track to commence its phase II clinical trial by year-end 2017. Our anticipated timeline puts GDC-0084 in the marketing stage as early as the beginning of 2025. In the event GDC-0084 obtains the *Accelerated Approval Designation,* marketing could be brought forward to early 2022.

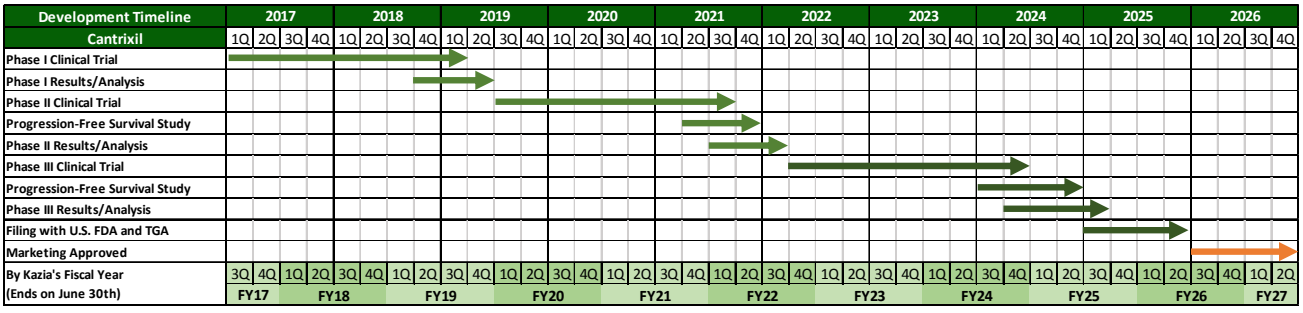
Exhibit 22: Development Timeline of GDC-0084 – Base Case (Top) and *Accelerated Approval* Case (Bottom)



Source: Cedrus Research

Cantrixil commenced its phase I clinical trial in December 2016, with an estimated study completion date around the year-end 2018 or early 2019. As of now, our anticipated timeline for the development of Cantrixil puts the drug on the market in 2026.

Exhibit 23: Development Timeline of Cantrixil



Source: Cedrus Research

V. FINANCIALS

Estimating Addressable Market

Kazia's current clinical drug development is being carried out mainly in Australia and the U.S., putting Cantrixil and GDC-0084 on a course to obtain marketing approval in these two countries initially. However, with the harmonization of medical goods between Australia's Therapeutic Goods Administration (TGA) and EU's European Medicine Agency (EMA), we believe it is reasonable to include the European market in our forecast too.

The company plans to establish an entity in China to facilitate the development of its drug portfolio and pursue partnerships and commercialization opportunities in the country. Having closer proximity and direct interaction with the CFDA are likely to accelerate Kazia's marketing initiatives; however, we believe it would be too early to justify a valuation of Kazia's drug portfolio in the Chinese market due to the lack of visibility currently.

Population Forecast

We use the World Bank's population forecast for the U.S., Australia and the EU in our projection.

Cantrixil

We believe Cantrixil will be used as a first-line therapy after approval, most likely in combination with the chemotherapeutic agent paclitaxel. We assume the crude annual incidence rate of 14.5 cases/100,000 females. In addition, we assume Cantrixil will be administered to approximately 85% of all ovarian cancer patients – those diagnosed between stage II and stage IV of the cancer and representing the patient population that requires chemotherapy. We exclude the 15% of the cases that will be diagnosed at stage I because these patients will likely only undergo a debulking procedure.

GDC-0084

GDC-0084 will most likely be positioned similarly to TMZ in treating GBM. We believe the drug has the potential to substitute TMZ as the first-line therapy because of its greater expected number of treatment-responders compared to TMZ based on the dual pathways GDC-0084 targets. We assume the average annual incidence rate of GBM at 3.19 cases/100,000 people. We estimate the addressable market for GDC-0084 will include 100% of the GBM patient population, positioning it to rival TMZ.

Exhibit 24: Addressable Population Estimate

Drug	Cantrixil	GDC-0084
Incidence Rate	**14.5/100,000 females**	**3.19/100,000 people**
Addressable Cases as Share of Total	**85%**	**100%**
Addressable Population Estimate (2017)		
Australia	**1,508**	**780**
U.S.	**20,225**	**10,382**
European Union	**32,251**	**16,333**
Japan	9,396	4,040
China	82,704	44,165

Note: We show the forecast for Japan because we believe the Japanese market is not out of reach for the two drugs. Historically, a bridging study under the supervision by the Pharmaceuticals and Medical Devices Agency (PMDA) in Japan would usually suffice for market approval, with an approximate time lag, compared to U.S. FDA approval, of 2-3 years.

We also show the estimated addressable population of China because we believe the Chinese market is a very attractive one due to its sheer size, increasing healthcare awareness of the Chinese, and stepping-up adoption and harmonization by China with international Food and Drug agencies. Kazia is also in the process of establishing a Chinese entity to further its commercialization and partnerships efforts in the Chinese market.

However, as of currently, we have excluded the Japanese and the Chinese markets from our valuation model due to the lack of visibility of the timeline.

Source: Cedrus Research

Expenses Forecast

Drug Development Costs

Majority of cash expenses in the short to medium-term will be related to completing the Cantrixil phase I clinical study program as well as funding the GDC-0084 phase II clinical trials.

We estimate that for Cantrixil, the whole clinical development cycle (from phase I to marketing) will cost around USD40 million (~AUD50 million). With respect to GDC-0084, we forecast the cost of bringing it to market to be approximately USD51 million (~AUD64 million).

Exhibit 25: Development Cost Forecast of Cantrixil and GDC-0084 (in AUD)

Cost Breakdown By Drug (in AUD) (Fiscal year ends June 30th)	FY18	FY19	FY20	FY21	FY22	FY23	FY24	FY25	FY26
Development of Cantrixil:									
Phase I	3,187,500	1,912,500							
Phase II			6,375,000	5,100,000	1,275,000				
Phase III					2,812,500	19,687,500	5,625,000		
Filing with Health Regulatory Authorities								625,000	625,000
Development of GDC-0084:									
Phase II	1,500,000	9,000,000	4,500,000						
Milestones due to Glioblast Pty Ltd.	2,500,000*	3,352,500	3,800,500						
Phase IIB		6,000,000	1,500,000						
Phase III				3,375,000	20,250,000	10,125,000			
Filing with Health Regulatory Authorities							625,000	625,000	
Milestone due to Genentech									1,394,000

Note: *The milestone due to Glioblast Pty Ltd. in FY18 will be paid in Kazia's shares, not cash, and are excluded in our DCF valuation. For the milestones to be paid in FY19 and FY20, we assume cash payments for valuation purposes, and they are included in our DCF valuation

Source: Cedrus Research

Overview of Payables to Third Parties and Commitment to Developing New Product

Kazia has committed to paying a total of four milestones to Glioblast's executives if GDC-0084 successfully passes through the upcoming phase II clinical trials. According to Kazia's fiscal year 2017 (FY17 ended 30 June 2017) financial statements, the first two milestone payments will be payable in Kazia's shares, while the remaining two milestone payments, valued at approximately AUD3.4 million and AUD3.8 million, are to be settled either in cash or Kazia's shares at the discretion of Kazia. To be conservative, we assume these milestones will be paid in cash. These four milestone payments are payable based on the progression of the phase II GDC-0084 trial.

Kazia will pay royalties on net sales of GDC-0084 to Genentech as well as pre-determined commercial milestones. We estimate the royalty percentage to be approximately 11% of net sales, which is on par with other licensing deals struck at phase II clinical trials. According to Kazia's FY17 financial statements, the milestone due to Genentech includes one payment of USD1.12 million (~AUD1.39 million) payable on the first product sale, which we anticipate to happen in FY26 in the base-case scenario.

Kazia has also **committed USD800,000 (~AUD1 million) of its own funds** to the development of its **Next-Gen ATM product** over the course of three years.

Cash and Liquidity

Kazia reported a cash balance of AUD14.5 million as of 30 June 2017. Incorporating Australia's research and development tax incentive scheme, which entails a cash refund of 43.5% of R&D expenditure for companies generating revenue less than AUD20 million per annum, we believe Kazia has a cash runway of approximately 12-18 months according to our projections. The company will require further funding for the development of both its clinical-stage drugs in the short- to medium-term.

Revenue Forecast

Due to the early stage of development of Kazia's two pipeline drugs, we have made the following assumptions:

Our anticipated timing for GDC-0084 and Cantrixil to obtain marketing approval is 3Q FY25 (base-case scenario) and 3Q FY26 respectively at the earliest. We note that Kazia could seek and be granted accelerated approval designation by the U.S. FDA and priority review by Australia's TGA for GDC-0084, potentially allowing marketing of the drug to start as early as in 4Q FY22, while concomitantly conducting confirmatory trials. If that materializes, it will warrant an upward revision to our current valuation.

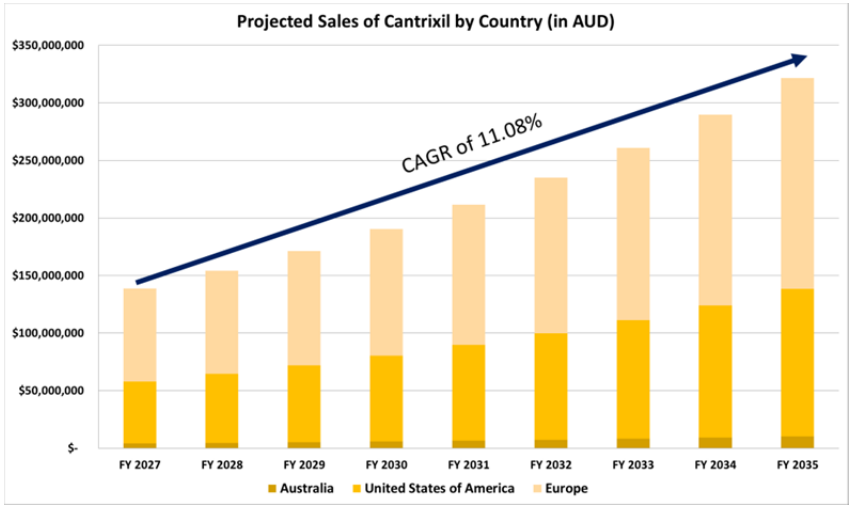
We assume the treatment cycle with Cantrixil will be priced at approximately USD4,000 (~AUD5,000) at launch. This is on par with the price of a treatment cycle of paclitaxel before the introduction of its generics. Currently, a treatment cycle with paclitaxel costs around USD1,500 (~AUD1,880).

For all the geographical markets that we project Cantrixil to enter, we assume an initial market adoption rate of 50%, peaking at 90% in FY35, the year of patent expiry (expiration in February 2035). We view that Cantrixil, as a potent anti-cancer stem cell agent, would readily be accepted by patients and physicians alike. Because of its potential ability to prevent cancer recurrence, we are positive regarding the likelihood of administrating Cantrixil as part of the first-line therapy for ovarian cancer.

We assume the treatment cycle with GDC-0084 will be priced at approximately USD35,000 (~AUD44,000) at launch. This is on par with the price of temozolomide before generic pricing available and the Carmustine wafer treatment.

For the geographical markets that we expect GDC-0084 to enter, we assume an initial market adoption rate of 5%, peaking at 40% in 2032, when the patent expires. Although GDC-0084 may be fairly well received by many GBM patients, we are assigning lower adoption rates (relative to those for Cantrixil), as GDC-0084 does not appear to offer patients a cure, likely dampening its market adoption.

Exhibit 26: Breakdown of Projected Sales of Cantrixil (top), and GDC-0084 (bottom)

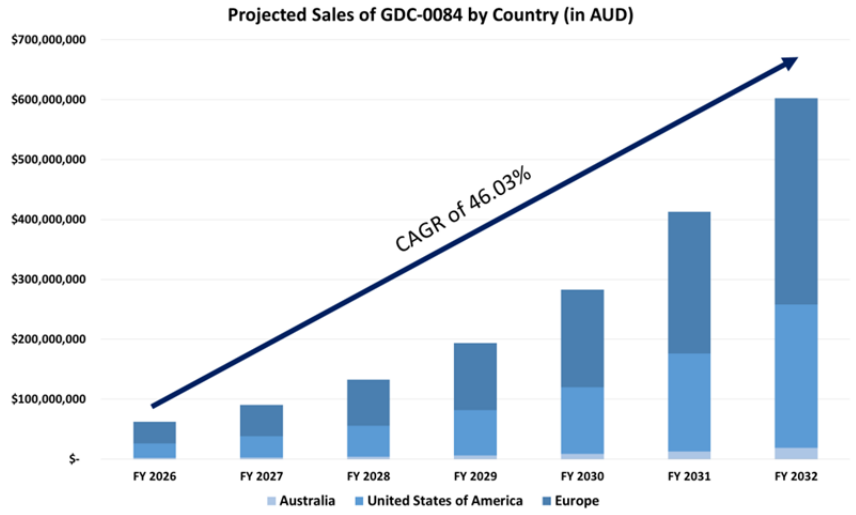


Overview of Cantrixil Revenue Projection

- We forecast sales in Australia, the U.S, and the European Union countries in the projected period.
- We estimate the market adoption rate to be at 50% in FY27, the year of its expected launch and peak at 90% in FY35, the patent expiry year.
- Our projected cumulative sales for Cantrixil up to its patent expiry in February 2035 would be AUD1,974 million.

Overview of GDC-0084 Revenue Projection

- We forecast sales in Australia, the U.S., and the European Union countries.
- We assign a market adoption rate of 5% in FY26, the year of its projected launch (without the accelerated approval designation) and peak at 40% in the patent expiry year of FY32.
- Our projected cumulative sales for GDC-0084 up to its patent expiry at year-end 2032 to be AUD1,777 million.



Source: Cedrus Research

VI. VALUATION

We value Cantrixil and GDC-0084 as two separate assets, using the discounted cash flow (DCF) methodology. We then deduct the market value of all the outstanding dilutive securities, including the market value of Kazia's shares payable to Glioblast Pty Ltd., from the combined value of these two drugs in development. We also include the discounted value of Kazia's financial commitments to the Next-Gen ATM project and the CRC-P grant from the Australian government, as well as the company's cash and cash equivalent balance as at 30 June 2017. Furthermore, we adjust the number of shares outstanding by the amount of company's shares to be issued to Triaxial Pharmaceutical Pty. Ltd.[38] to determine our eventual estimated fair value per share of Kazia.

Terminal Value (TV)

For Cantrixil, we assume a long-term growth rate of 3% to calculate the terminal value of the drug. The life of the drug could potentially be extended by (i) new formulation of the drug, which could warrant new patents, and (ii) extension into other cancer indications for which a high proportion of patients experiencing cancer recurrence. To justify this potential drug-life extension, we have included an appropriate amount of research and development (R&D) costs, as well as selling, general & administrative (SG&A) expenses.

For GDC-0084, we assume a long-term growth rate of 0% to calculate the terminal value of the drug. We view GDC-0084 as highly specific to GBM, with limited opportunities for extension into other indications. However, in view of the vast majority of PI3K-inhibitors under development is for a wide-range of cancer indications, we do explore these possibilities for GDC-0084. We have incorporated relatively moderate levels of R&D and SG&A expenses compared to Cantrixil to justify our assigned long-term growth rate of 0%.

Weighted Average Cost of Capital (WACC)

For calculating the appropriate discount rate, we use Australia's risk-free rate of 2.80% and a market risk premium of 6.25%. Moreover, we assign a beta of 1.2 to reach at a discount rate of 10.30%. To account for the inherent risk of Kazia's early-stage drugs, we further add five percentage points to the discount rate before arriving at our weighted average cost of capital of 15.30%. We apply this discount rate for GDC-0084, Cantrixil and the Next-Gen ATM project.

Marketing Probability Adjustment

We apply a marketing-probability-adjusted discount to our calculated valuation of the two assets (GDC-0084 and Cantrixil) based on their respective stage of clinical development. These probability rates are based on the reported rates specific to oncology drugs, determined in a research collaboration between Bio, Biomedtracker and Amplion[39]. The marketing probability rate we use for Cantrixil and GDX-0084 is 5.10% and 8.10% respectively.

[38] In acquiring Triaxial in 2012 for its superbenzopyran technology platform, a loan to the value of AUD1,885,000 in the form of a convertible note was extended by Triaxial's shareholders to Kazia to complete the transaction. In return, Kazia is obliged to issue shares to Triaxial's shareholders

[39] Clinical Development Success Rates 2006-2015 (2016). Biotechnology Innovation Organization, Biomedtracker, Amplion

Other Committed Projects – Next-Gen ATM

We incorporate Kazia's cash grants and cash commitments related to its Next-Gen ATM project, and the discounted value of which has been accounted for in our valuation of Kazia.

Cash and Cash Equivalents

Our valuation includes Kazia's reported cash and cash equivalent position of AUD14.5 million as of June 30th 2017.

Dilutive Financial Instruments

The company has 12 tranches of options outstanding, convertible into 7,746,833 shares in the event they are all executed. To value these dilutive securities, we use the Black-Scholes option pricing model. Our total valuation of Kazia's outstanding options is AUD1,246,626.

Exhibit 27: Valuation of Kazia's Outstanding Options

Tranche	Grant Date	Expiry Date	Exercise Price	Balance	Market Value
1	16-Dec-14	16-Dec-19	$1.500	46,647	$11,501
2	18-Dec-14	18-Dec-19	$1.500	19,952	$4,946
3	4-Jun-15	4-Jun-20	$4.000	2,948,400	$219,766
4	30-Jun-15	4-Jun-20	$4.000	200,000	$14,907
5	30-Jun-15	30-Jun-20	$4.000	2,906,500	$238,318
6	16-Nov-15	16-Nov-20	$2.200	363,333	$110,954
7	18-Mar-16	1-Feb-21	$1.990	500,000	$198,578
8	18-Mar-16	1-Feb-21	$2.610	250,000	$70,916
9	5-Sep-16	5-Sep-21	$1.630	200,000	$130,389
10	31-Oct-16	1-Nov-21	$1.380	50,000	$39,853
11	12-Oct-16	17-Oct-21	$1.560	62,000	$43,916
12	21-Nov-16	23-Nov-21	$1.380	200,000	$162,583
Total:				7,746,833	$1,246,626

Source: Kazia, Cedrus Research

Kazia also has an outstanding convertible note with Triaxial, a company founded by the previous owners of the company. While two tranches have been executed, the third tranche remains outstanding. The triggers for this third tranche, for the issuance of 2,400,000 new shares of Kazia, are either a completion of the phase II clinical trials of any Kazia's drug in development, or achieving the U.S. FDA's *Breakthrough Therapy Designation*. Based on our understanding of Kazia's drug positions, a *Breakthrough Therapy Designation* is unlikely; therefore, we adjust these 2,400,000 new shares by the additive probability of both GDC-0084 and Cantrixil passing phase II clinical trials.

Using the average oncology drug clinical success rates, the probability of success for Cantrixil, which is in phase I clinical trials, to proceed past phase II is 15.4%. Meanwhile, GDC-0084, which is planned to

commence phase II clinical trials by year-end 2017, has a success rate of 24.6%. Therefore, we adjust our outstanding shares by 960,000 [(15.4%$_{Cantrixil}$ + 24.6%$_{GDC-0084}$) X 2,400,000$_{shares}$) to calculate our estimated fair value per share for Kazia.

Exhibit 28: Valuation of Cantrixil (in AUD)

Fiscal Year Ending June 30th	FY 2027	FY 2028	FY 2029	FY 2030	FY 2031	FY 2032	FY 2033	FY 2034	FY 2035
Total Revenue	138,797,011	154,259,933	171,417,781	190,454,515	211,572,680	235,000,879	260,987,782	289,814,632	321,795,968
Cost of Goods Sold	13,879,701	15,425,993	17,141,778	19,045,452	21,157,268	23,500,088	26,098,778	28,981,463	32,179,597
As a % of Revenue	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*
Gross Margin	124,917,310	138,833,939	154,276,003	171,409,064	190,415,412	211,500,791	234,889,004	260,833,169	289,616,371
as a % of Revenue	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*
SG&A	31,923,313	35,479,785	39,426,090	43,804,538	48,661,716	54,050,202	60,027,190	66,657,365	74,013,073
As a % of Revenue	*23.00%*	*23.00%*	*23.00%*	*23.00%*	*23.00%*	*23.00%*	*23.00%*	*23.00%*	*23.00%*
Research & Development	6,939,851	7,712,997	8,570,889	9,522,726	10,578,634	11,750,044	13,049,389	14,490,732	16,089,798
As a % of Revenue	*5.00%*	*5.00%*	*5.00%*	*5.00%*	*5.00%*	*5.00%*	*5.00%*	*5.00%*	*5.00%*
Depreciation Costs:	1,475,954	1,537,452	1,601,513	1,668,242	1,737,752	1,810,159	1,885,582	1,964,148	2,045,987
Earnings Before Tax	84,578,193	94,103,706	104,677,512	116,413,557	129,437,309	143,890,386	159,926,843	177,720,924	197,467,513
As a % of Revenue	*61%*	*61%*	*61%*	*61%*	*61%*	*61%*	*61%*	*61%*	*61%*
Taxes @30%	25,373,458	28,231,112	31,403,253	34,924,067	38,831,193	43,167,116	47,978,053	53,316,277	59,240,254
Earnings After Tax	59,204,735	65,872,594	73,274,258	81,489,490	90,606,116	100,723,270	111,948,790	124,404,647	138,227,259
As a % of Revenue	*43%*	*43%*	*43%*	*43%*	*43%*	*43%*	*43%*	*43%*	*43%*
Investments Into Capital:									
Working Capital	6,939,851	7,712,997	8,570,889	9,522,726	10,578,634	11,750,044	13,049,389	14,490,732	16,089,798
As a % of Revenue	5%	5%	5%	5%	5%	5%	5%	5%	5%
Δ in Working Capital	6,939,851	773,146	857,892	951,837	1,055,908	1,171,410	1,299,345	1,441,342	1,599,067
CAPEX:	1,475,954	1,537,452	1,601,513	1,668,242	1,737,752	1,810,159	1,885,582	1,964,148	2,045,987
As a % of Revenue	1.06%	1.00%	0.93%	0.88%	0.82%	0.77%	0.72%	0.68%	0.64%
Free Cash Flow	52,264,885	65,099,448	72,416,366	80,537,653	89,550,208	99,551,860	110,649,445	122,963,304	136,628,192
Year:	10	11	12	13	14	15	16	17	18
DCF Value:	12,586,846	13,597,370	13,118,526	12,653,713	12,202,710	11,765,483	11,341,757	10,931,438	10,534,472
Sum of NPV (FY18-FY35):	67,118,188								

Terminal Value Calculation	
Ending Cash Flow (FY2035)	136,628,192
Long-Term Growth Rate	3.00%
Terminal Value (TV)	1,144,122,258
NPV of Terminal Value	88,215,501

Sensitivity Analysis:		Terminal Growth Rate				
		1.00%	**2.00%**	**3.00%**	**4.00%**	**5.00%**
WACC	**11.30%**	323,457,075	346,567,203	375,246,037	411,782,085	459,916,879
	13.30%	211,872,520	223,639,047	237,690,336	254,763,409	275,950,474
	15.30%	141,522,504	147,908,879	155,333,689	164,072,625	174,508,441
	17.30%	95,332,107	98,967,866	103,112,123	107,879,576	113,422,225
	19.30%	64,087,104	66,235,704	68,647,936	71,375,492	74,484,525

DCF Valuation	
Sum of NPV	67,118,188
NPV of Terminal Value	88,215,501
Total Valuation	155,333,689
Marketable Probability	*5.10%*
Market Adjusted - NPV	7,922,018

Note: We only show our model for the years between the start of Cantrixil's anticipated marketing year and the year of patent expiry for simplicity. From FY18 to FY26, there will be costs associated with the development of Cantrixil. We forecast approximately AUD4 million in SG&A in FY18, increasing 10% each fiscal year thereafter until FY26. We also assume 43.5% R&D expenditure to be refunded to Kazia each year from FY18 through FY25 (We estimate GDC-0084's annual revenue to exceed the AUD20 million threshold in FY26)

Source: Cedrus Research

Exhibit 29: Valuation of GDC-0084 (in AUD)

Fiscal Year Ending June 30th	FY 2026	FY 2027	FY 2028	FY 2029	FY 2030	FY 2031	FY 2032
Total Revenue	62,130,284	90,761,214	132,567,066	193,597,031	282,678,576	412,685,514	602,402,590
Cost of Goods Sold	6,213,028	9,076,121	13,256,707	19,359,703	28,267,858	41,268,551	60,240,259
As a % of Revenue	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*
Royalty Payment to Genentech	*6,834,331*	*9,983,733*	*14,582,377*	*21,295,673*	*31,094,643*	*45,395,407*	*66,264,285*
as a % of Revenue	*11%*	*11%*	*11%*	*11%*	*11%*	*11%*	*11%*
Gross Margin	49,082,924	71,701,359	104,727,982	152,941,655	223,316,075	326,021,556	475,898,046
as a % of Revenue	*79%*	*79%*	*79%*	*79%*	*79%*	*79%*	*79%*
SG&A	11,183,451	16,337,018	23,862,072	34,847,466	50,882,144	74,283,393	108,432,466
As a % of Revenue	*18.00%*	*18.00%*	*18.00%*	*18.00%*	*18.00%*	*18.00%*	*18.00%*
Research & Development	621,303	907,612	1,325,671	1,935,970	2,826,786	4,126,855	6,024,026
As a % of Revenue	*1.00%*	*1.00%*	*1.00%*	*1.00%*	*1.00%*	*1.00%*	*1.00%*
Depreciation Costs:	2,602,786.58	2,711,236	2,824,204	2,941,879	3,064,458	3,192,143	3,325,149
Earnings Before Tax	33,281,384	51,745,492	76,716,036	113,216,339	166,542,688	244,419,165	358,116,405
As a % of Revenue	*54%*	*57%*	*58%*	*58%*	*59%*	*59%*	*59%*
Taxes @30%	9,984,415	15,523,648	23,014,811	33,964,902	49,962,806	73,325,750	107,434,921
Earnings After Tax	23,296,969	36,221,844	53,701,225	79,251,438	116,579,882	171,093,416	250,681,483
As a % of Revenue	*37%*	*40%*	*41%*	*41%*	*41%*	*41%*	*42%*
Investments Into Capital:							
Working Capital	3,106,514	4,538,061	6,628,353	9,679,852	14,133,929	20,634,276	30,120,129
As a % of Revenue	5%	5%	5%	5%	5%	5%	5%
Δ in Working Capital	3,106,514	1,431,546	2,090,293	3,051,498	4,454,077	6,500,347	9,485,854
CAPEX:	2,602,787	2,711,236	2,824,204	2,941,879	3,064,458	3,192,143	3,325,149
As a % of Revenue	4.19%	2.99%	2.13%	1.52%	1.08%	0.77%	0.55%
Free Cash Flow	20,190,454	34,790,298	51,610,932	76,199,939	112,125,804	164,593,069	241,195,629
Year	9	10	11	12	13	14	15
DCF Value	5,606,377	8,378,477	10,780,014	13,803,936	17,616,701	22,428,552	28,505,576
Sum of NPV (FY18-FY32):	55,823,277						

Terminal Value Calculation	
Ending Cash Flow (FY2032)	241,195,629
Long-Term Growth Rate	0.00%
Terminal Value (TV)	1,576,442,022
NPV of Terminal Value	186,310,956

Sensitivity Analysis		Terminal Growth Rate				
		-2.00%	-1.00%	0.00%	1.00%	2.00%
WACC	11.30%	467,157,625	500,094,348	538,860,579	585,154,233	641,403,512
	13.30%	316,813,753	336,005,885	358,084,052	383,752,165	413,963,307
	15.30%	217,299,951	228,955,304	242,134,232	257,156,368	274,437,470
	17.30%	149,659,957	156,974,528	165,134,714	174,296,150	184,655,160
	19.30%	102,725,977	107,441,476	112,645,627	118,418,537	124,858,836

DCF Valuation	
Sum of NPV	55,823,277
NPV of Terminal Value	186,310,956
Total Valuation	242,134,232
Marketable Probability	*8.10%*
Market Adjusted - NPV	19,612,873

Note: We only show our model for the years between the beginning of GDC-0084's anticipated marketing year and the year in which the patent will expire for simplicity. From FY18 to FY25, there will be costs associated with the development of GDC-0084. We forecast approximately AUD4 million in SG&A in FY18, increasing 10% each fiscal year thereafter until FY25. We also assume 43.5% R&D expenditure to be refunded to Kazia each year between FY18 to FY25 (We estimate GDC-0084's annual revenue to exceed the AUD20 million threshold in FY26) *Source: Cedrus Research*

Exhibit 30: Sum-of-Parts Valuation for Kazia

Total Valuation of Novogen (AUD)	
GDC-0084 Valuation	19,612,873
Cantrixil Valuation	7,922,018
Add: Cash and Cash Equivalents	14,454,784
Add: Value of Next-Gen ATM Commitment	1,514,610
Less: Value of Dilutive Securities	(1,246,626)
Less: Value of Shares for Glioblast	(2,500,000)
Total Valuation:	**39,757,659**
Current Number of Shares Outstanding	48,409,621
Adjustment for Shares due to Triaxial	960,000
Adjusted Shares Outstanding	**49,369,621**
Fair Value per Share:	**0.81**

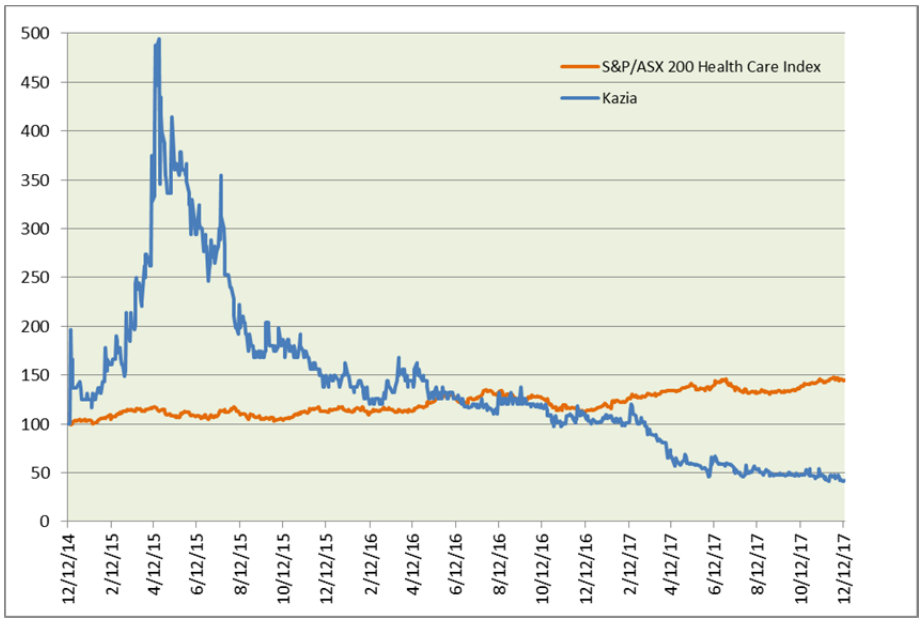
Source: Cedrus Research

Our estimated fair value per share for Kazia is AUD0.81, which presents around 131% upside potential compared to the closing price of AUD0.35 on 13 December 2017. We have not included a potential tax benefit carried forward of approximately AUD17.4 million as at the end of FY17, which will likely accumulate and be used to offset tax payable after the company's two drugs currently in development are marketed.

VII. RECOMMENDATION

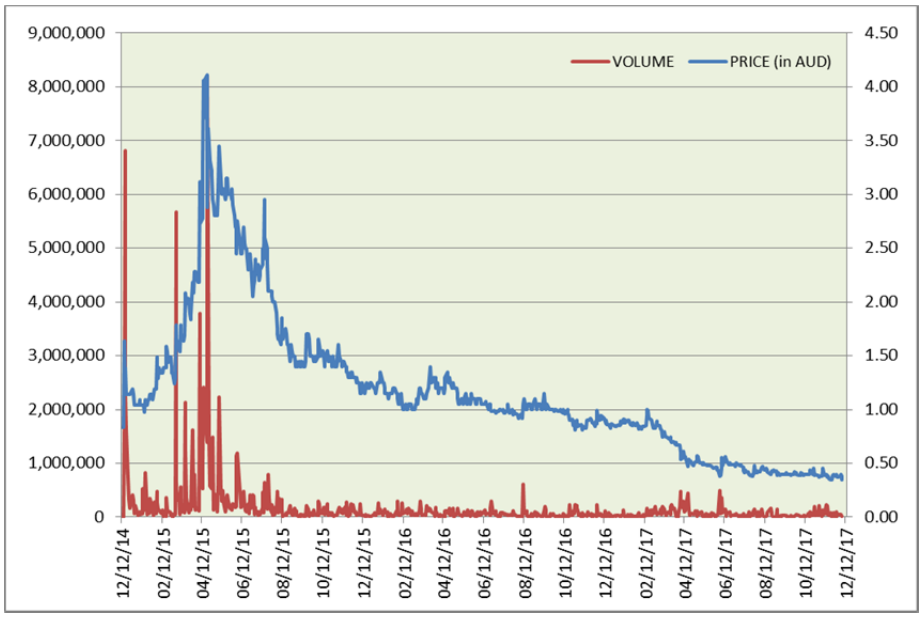
Share Performance

Exhibit 31: Kazia's Share Price Performance vs S&P/ASX 200 Health Care Index



Source: Bloomberg, Cedrus Research

Exhibit 32: Kazia's Share Price Performance and Trading Volume



Source: Bloomberg, Cedrus Research

Shareholder Analysis

As of 24th August 2017, there was a diverse pool of institutional and retail investors for Kazia, each of them with a less than 2% stake in the company besides the company's single substantial shareholder, Hishenk Pty Ltd., an Australia-based private company holding around 2.7 million shares and representing 5.57% of total shares outstanding.

Our Recommendation

Kazia is developing two drugs – Cantrixil and GDC-0084 – with the potential to address the downfall in today's standard of care in ovarian cancer and glioblastoma multiforme respectively.

Since the company's two drugs are early in their development cycle, there are inherent risks associated with their clinical progression and eventual marketing approval. However, we also note their **substantial potential** in view of their **unique mechanism of action and cellular targets** compared to approved existing treatments and those in the pipelines of the company's competition based on our analysis.

We believe **Cantrixil** will have wide applicability due to its novel **ability to kill cancer stem cells efficiently**, **potentially allowing its penetration into new indications**.

Despite many drug developers having attempted to develop PI3K inhibitors for GBM, in our view, **GDC-0084** is **unique** due to its **binding specificity** across class-I PI3K-isoforms (only the α and β isoforms instead of all isoforms), **optimized inhibition of mTOR**, and specific design to **penetrate the blood-brain barrier**.

Hence, we recommend investors to accumulate Kazia's share, as the company is on track to achieve notable progress in clinical development, providing added visibility and further de-risking the development programs of its two leading drug candidates.

Our estimated fair value per share for Kazia is AUD0.81, equivalent to an upside potential about 131% versus its 13 December 2017 closing price.

Key Catalysts

- Clinical trial results of Cantrixil and GDC-0084 could show better-than-expected therapeutic profiles.
- Potential for an *Accelerated Approval* designation by the U.S. FDA on Kazia's GBM drug candidate, GDC-0084.

Major Downside Risks

- Clinical trials could present weak therapeutic profiles.
- Termination of the drug programs.
- Delays in clinical trial progression, potentially arising from slower-than-anticipated patient recruitment, regulatory hurdles, among others.
- Potential dilution of existing shareholders' ownership through equity fundraising by the company.

News Flow in the Upcoming 12 -18 months

- Maximum tolerated dose (MTD) data for Cantrixil's phase I studies are expected in 1Q 2018.
- Exploratory efficacy data of Cantrixil is projected to be available in later 2018.
- Commencement of phase II clinical study of GDC-0084 is scheduled by year-end 2017.

APPENDIX: Company Management and Board of Directors

Company Management

Dr. James Garner MA, MBA, MBBS, BSc (Hons)
Chief Executive Officer and Executive Director

Dr. Garner is an experienced life sciences executive who has previously worked with companies ranging from small biotechs to multinational pharmaceutical companies such as Biogen and Takeda. His career has focused on regional and global development of new medicines from preclinical to commercialization.

Dr. Garner is a physician by training and holds an MBA from the University of Queensland. He began his career in hospital medicine and worked for a number of years as a corporate strategy consultant with Bain & Company before entering the pharmaceutical industry. Prior to joining Kazia in 2016, he led R&D strategy for Sanofi in Asia-Pacific and was based in Singapore.

Dr. Peng Leong MBA, PhD
Chief Business Officer

Dr. Leong has spent 18 years in the pharmaceutical industry, beginning as a scientist, and followed by eight years in healthcare investment banking and private equity, and eight years in business development. Dr. Leong started his career at Chiron Corporation, a pioneering biotechnology company based in California that was subsequently acquired by Novartis International AG (NVS). While working in healthcare investment banking at CIBC World Markets and Piper Jaffray, he was involved in raising more than USD1.4 billion for over 20 biotechnology companies.

In his various roles, including his most recent position at Merck Serono (FWB: MRK), Dr. Leong has had a leadership role in the acquisition or sale of over USD1 billion in pharmaceutical product rights. He holds a PhD in Biochemistry from the University of Toronto, Canada and an MBA from the University of California, Berkeley.

Dr. Gordon Hirsch MBA, MBBCh, FCP(SA), BSc (Hons)
Chief Medical Officer

Dr. Hirsch is a scientist, specialist physician and pharmaceutical executive with more than 20 years of experience in the pharmaceutical industry. He has held various scientific, medical and operational roles at country, regional and global levels with a range of responsibilities from development to commercialization in companies including Eli Lilly (LLY), Pfizer (PFE), Sanofi (SNY) and Takeda (4502 JP). He has held appointments in South Africa, Australia, France, Japan and the U.S.

He holds BSc (Hons) and MBBCh degrees from the University of the Witwatersrand, South Africa and an MBA from Henley Business School, U.K. Dr. Hirsch is also a Fellow of the South African College of Physicians.

Ms. Kate Hill ACA, GAICD, BSc (Hons)
Company Secretary

Ms. Hill has over 20 years of experience as an audit partner with Deloitte Touche Tohmatsu, working with ASX-listed and privately-owned clients. She has worked extensively in regulated environments, including assisting with Initial Public Offerings, capital raising and general compliance, as well as operating in an audit environment. She is also a Non-Executive Director of CountPlus Limited, an ASX-listed company, and Chairs their Audit and Risk Committee. Ms. Hill is also a Non-Executive Director of a small not-for-profit organization and a member of their Finance and Risk Committee.

Ms. Hill is a member of the Institute of Chartered Accountants in Australia and New Zealand, and a graduate of the Australian Institute of Company Directors.

Ms. Gabrielle Heaton BBUS (ACC), CPA
Director of Finance and Administration

Ms. Heaton has over 30 years of commercial experience in media, property services and healthcare for multinational, ASX-listed and overseas companies.

She has held a number of senior Finance positions, including CFO and Quality Auditor, and has also been responsible for Human Resources and IT.

Ms. Heaton has a Bachelor of Business degree from the University of Technology and is a member of CPA Australia.

Scientific Advisory Board

Professor Sir Murray Brennan GNZM, MD, FACS

Professor Sir Murray Brennan is a New Zealand-born surgeon, oncologist, cancer researcher, and academic, with over 50 years of experience in clinical practice.

He was Chair of the Department of Surgery at Memorial Sloan Kettering Cancer Center from 1985 to June 2006, and is currently Vice President of International Programs and Director of the Bobst International Center. He currently holds the Benno C Schmidt Chair in Clinical Oncology, and is Chairman Emeritus of the Department of Surgery. Prior to his career at Memorial Sloan Kettering, he was head of the surgical metabolism section at the National Cancer Institute.

Professor Brennan has lectured throughout the world and authored or co-authored more than 1,100 scientific papers and book chapters, and three books on soft tissue sarcoma. He has served as Director of the American Board of Surgery, Vice President of the American College of Surgeons, Chairman of the American College of Surgeons Commission on Cancer, and President of the Society of Surgical Oncology, the James IV Association of Surgeons, the Society of Clinical Surgery, the International Gastric Cancer Association, and the American Surgical Association.

He has been awarded Honorary Fellowships in the Royal College of Surgeons of Edinburgh, of England, and in Ireland, the Royal Australasian College of Surgeons, and the Royal College of Physicians and Surgeons of Glasgow and Canada. Professor Brennan has also received honorary doctorates from the Universities of Edinburgh, Otago, and Goteborg, and University College, London.

In 1995, Professor Brennan was honored with membership in the Institute of Medicine of the National Academy of Sciences, and in 2000 he received the American College of Surgeons' highest award, the Distinguished Service Award. In January 2015 he was appointed by Her Majesty The Queen as Knight Grand Companion of the New Zealand Order of Merit.

Dr. Karen Ferrante MD

Dr. Karen Ferrante is a haematologist-oncologist with over 20 years of experience in clinical oncology drug development, spanning both large pharmaceutical companies and fast-growing biotechs.

She received her MD degree from Georgetown University School of Medicine and then completed her internship and residency in internal medicine at the New England Deaconess Hospital in Boston (Beth Israel Deaconess), followed by a fellowship in haematology and oncology. While at Beth Israel Deaconess, she served as Instructor, Clinical Instructor, and Clinical Fellow in Medicine at Harvard Medical School.

Dr. Ferrante commenced her industry career with Bristol-Myers Squibb in 1995, and then spent more than eight years working at Pfizer, culminating in a role as Vice President of Oncology Development. She joined Millennium Pharmaceuticals in 2007 and rose to the position of Chief Medical Officer, with shared responsibility over Research and Development (R&D), where she supported the development activities for supplemental indications for Velcade® (bortezomib), the first proteasome inhibitor for multiple myeloma, as well as initial approval in Europe for Adcetris® (brentuximab vedotin), an antibody-drug conjugate directed to CD30, and the oral proteasome inhibitor, Ninlaro® (ixazomib). Following the acquisition and integration of Millennium by the Takeda Pharmaceutical Company, Dr. Ferrante became the Therapeutic Area Head for Oncology.

From 2014 until recently, she has served as Chief Medical Officer and Head of R&D for Tokai Pharmaceuticals, a previously NASDAQ-listed biotechnology company based in Boston, MA that is focused on the development of novel therapies for prostate cancer. She served on the Board of Baxalta, until its acquisition by Shire in 2016, and is on the Board of Progenics Pharmaceuticals.

Dr. Ferrante has also been an author of a number of papers in the oncology field. She is an active participant in academic and professional associations and symposia and holds several patents. She is a Board Member of the New England Women in Science Executives (NEWISE), and a member of the American Society of Clinical Oncology. In 2012, she was named by Pharma Voice as one of their 100 Most Inspiring People.

Professor Peter Gunning PhD

Professor Peter Gunning is the Head of the School of Medical Sciences at the University of New South Wales, and the co-inventor of Kazia's anti-tropomyosin technology, which includes Anisina (ATM-3507).

He completed his PhD at Monash University on gene expression in the nervous system, and then spent nine years at Stanford University, working first on neuronal differentiation and then on the regulation of muscle gene expression. Since returning to Australia, his research group has focused on studying the architecture of cells and tissues.

Professor Gunning's research has been principally concerned with diseases of childhood, primarily cancer and muscle damage. He is best known for his discovery of one of the key principles underlying the architecture of all cells and its application to childhood cancer. Professor Gunning has built a 20-year partnership with the Not-For-Profit organization, The Kids Cancer Project, to drive novel research into childhood cancers, and this was reported on the ABC's Australian Story program in March 2014.

Professor Gunning has published over 200 research papers and recently edited the book Tropomyosin. He is on the Board of the NSW Cancer Institute and served as the Chair of the NSW Cancer Institute Cancer Research Advisory Committee for six years. He was also the inaugural Chair of the Division of Research at The Children's Hospital at Westmead, Sydney, and the Founding Chair of Bio-Link Pty Ltd.

Professor Alex Matter MD

Professor Alex Matter is the Chairman and Chief Executive Officer of the Experimental Therapeutics Centre, and also Chief Executive Officer of the D3 Platform, both part of A*STAR, the Agency for Science, Technology, and Research, in Singapore. In addition, he is an Emeritus Professor of the Medical Faculty of the University of Basel, and an Honorary Adjunct Professor of the Department of Pharmacology in the Yong Loo Lin School of Medicine at the National University of Singapore.

Professor Matter received his medical degree from the University of Basel and undertook research in Switzerland, Britain, France, and the U.S. before entering the pharmaceutical industry. He worked in Hoffman-LaRoche, before joining CIBA-GEIGY, which became part of Novartis in 1996, where he rose to the position of Global Head of Oncology Research. At Novartis, he led a team of close to two hundred scientists to discover and develop Gleevec® (imatinib), one of the first targeted therapies for cancer, which was given a fast-track approval by FDA in 2001. He was also instrumental in the discovery of Tasigna® (nilotinib), and several therapies for infectious disease.

He was the founding director of the Novartis Institute for Tropical Diseases in 2003, where he attracted major grants from the Bill & Melinda Gates Foundation and the Wellcome Trust. He also co-founded the Esperanza Medicines Foundation, a Not-For-Profit organization that aims to develop affordable drugs for treatment and prevention of AIDS in developing countries.

Professor Matter has published more than 100 peer-reviewed scientific papers and several book chapters in the area of oncology and haematology. He is the recipient of the 13th Warren-Alpert Prize and the AACR-Bruce F Cain Memorial Award. He has held fellowships at the Swiss National Science Foundation and the

Swiss Academy for Medical Sciences, and was awarded the Szent-Györgyi Prize by the U.S. National Foundation for Cancer Research in 2013.

Board of Directors

Mr. Iain Ross BSc (Hons), C.DIR
Chairman and Non-Executive Director

Mr. Iain Ross is an experienced multinational pharmaceutical and biotechnology executive and is currently Chairman of e-Therapeutics plc (LSE: ETX), Redx Pharma plc (LON: REDX) and Biomer Technology Limited.

During his career, Mr. Ross has held senior positions at multinational companies, Sandoz AG, Hoffman La Roche, and Celltech Group plc and been a Chairman, CEO and Director of several biotech companies. He is a qualified Chartered Director and former Vice Chairman of the Council of Royal Holloway, London University

Mr. Bryce Carmine
Deputy Chairman and Non-Executive Director

Mr. Carmine spent 36 years working for Eli Lilly & Co. and retired as Executive Vice President for Eli Lilly & Co, and President, Lilly Bio-Medicines. Prior to this he led the Global Pharmaceutical Sales and Marketing and was a member of the company's executive committee.

Mr. Carmine previously held a series of product development portfolio leadership roles culminating when he was named President, Global Pharmaceutical Product Development, with responsibility for the entire late-phase pipeline development across all therapeutic areas for Eli Lilly.

During his career with Lilly, Mr. Carmine held several country leadership positions, including President Eli Lilly Japan, Managing Director Australia/New Zealand, and General Manager of a joint venture for Lilly in Seoul, Korea.

Mr. Steven Coffey CA
Non-Executive Director

Mr. Coffey is a Chartered Accountant, having spent his career in public practice since graduating from NSW University in 1983. He has been a partner in the chartered accounting firm Watkins Coffey Martin since 1993.

Mr. Coffey is a registered company auditor and audits a number of large private companies as well as not-for-profit entities. He previously served on the board of an Australian-listed public company and is currently a board member of a private family foundation.

Dr. James Garner MA, MBA, MBBS, BSc (Hons)
Chief Executive Officer and Executive Director

See above.

IMPORTANT DISCLOSURES

STOCK OWNERSHIP AND CONFLICT OF INTEREST DISCLOSURE

- Neither Randy Hice nor any member of the research team or their households is an owner of Kazia Therapeutics Limited common shares.

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Randy Hice hereby certifies that the views expressed in this research report accurately reflect his personal views about the subject companies and their securities. He also certifies that he has not been, and will not be, receiving direct or indirect compensation in exchange for expressing the specific recommendations in this report.

For additional information, please send an e-mail to information@cedrusinvestments.com