



K A I D A
B i o P h a r m a

**Seeking Victory in the Fight
Against Ovarian Cancer**

**June 2025
Corporate Presentation**



Forward-Looking Statements

These slides and the accompanying oral presentation contain forward-looking statements and information. Forward-looking statements are subject to known and unknown risks, uncertainties, and other factors that may cause our or our industry's actual results, levels or activity, performance or achievements to be materially different from those anticipated by such statements. The use of words such as “may”, “might”, “will”, “should”, “could”, “expect”, “plan”, “anticipate”, “believe”, “estimate”, “project”, “intend”, “future”, “potential” or “continue”, and other similar expressions are intended to identify forward looking statements. For example, all statements we make regarding (i) the initiation, timing, cost, progress and results of our preclinical and clinical studies and our research and development programs, (ii) our ability to advance product candidates into, and successfully complete, clinical studies, (iii) the timing or likelihood of regulatory filings and approvals, (iv) our ability to develop, manufacture and commercialize our product candidates and to improve the manufacturing process, (v) the rate and degree of market acceptance of our product candidates, (vi) the size and growth potential of the markets for our product candidates and our ability to serve those markets, and (vii) our expectations regarding our ability to obtain and maintain intellectual property protection for our product candidates, are forward looking. All forward-looking statements are based on current estimates, assumptions and expectations by our management that, although we believe to be reasonable, are inherently uncertain. All forward-looking statements are subject to risks and uncertainties that may cause actual results to differ materially from those that we expected. Any forward-looking statement speaks only as of the date on which it was made. We undertake no obligation to publicly update or revise any forward-looking statement, whether as a result of new information, future events or otherwise, except as required by law. This presentation is not, and nothing in it should be construed as, an offer, invitation or recommendation in respect of our securities, or an offer, invitation or recommendation to sell, or a solicitation of an offer to buy, any of our securities in any jurisdiction. Neither this presentation nor anything in it shall form the basis of any contract or commitment. This presentation is not intended to be relied upon as advice to investors or potential investors and does not take into account the investment objectives, financial situation or needs of any investor.

Our Mission

We are advancing targeted anti-cancer therapies to address hormone-driven cancers affecting women





Highlights

Lead product candidate (KAD101, best-in-class next generation prolactin antagonist) indication: platinum resistant ovarian cancer (PROC)

90% of patients are in need for an effective treatment

Targeting Primary Cause of Hormone-Driven Cancers: Prolactin

- KAD101 demonstrated promising initial human clinical data
- Leadership has extensive preclinical, clinical, and drug development experience/expertise to execute clinical development plan
- Targeting prolactin; over expressed in ~80% of patients with ovarian cancer¹
- Kaida SAB lead, Anil K. Sood, MD – MDACC², is internationally recognized PROC KOL and leading expert on prolactin (PRL) biology
- KAD101 Phase 1 study initiation expected Q4 2026/Q1 2027
- Pipeline expansion opportunity into Breast and Uterine cancer. Prolactin over expressed in ~90% of patients³

1. V. Levina et al. Biological Significance of Prolactin in Gynecologic Cancers. Cancer Res 15 June 2009; 69 (12): 5226-5233. <https://doi.org/10.1158/0008-5472.CAN-08-4652>; 2. MDACC: The University of Texas MD Anderson Cancer Center 3. Faupel-Badger et al. Prolactin receptor expression and breast cancer: relationships with tumor characteristics among pre- and post-menopausal women in a population-based case-control study from Poland. Horm Cancer. 2014 Feb;5(1):42-50. doi: 10.1007/s12672-013-0165-7. Epub 2013 Nov 19. PMID: 24249584; PMCID PMC3906637.

Kaida Management Team and Advisors

Proven leadership with drug development and business experience...

Dr. Stella Vnook Co-Founder

Major Biopharma Executive, Transformational Leader with Extensive Pharma Background, Doctorate in Economics of PH and Pharmacy and MBA



Craig Pierson Chairman, Co-Founder

LifeTech Capital, Founder of AiM Medical Robotics MSE/CE Life Science Banker for 26 Years



John Langenheim, PhD CSO, Co-Founder

Prolactin Receptor Antagonist Subject Matter Expert, Assistant Professor of Cancer Biology for Sidney Kimmel Medical College at Thomas Jefferson University



Pam Swiggard Regulatory Affairs

Accomplished pharmaceutical executive in global regulatory affairs and quality assurance



Jay Campbell CFO/COO

Experienced life sciences executive and leader in financial, business/ corporate development, and operational roles



Scientific and Regulatory Expert Advisors

Anil K. Sood, M.D.

Head of Scientific Advisory Board



- *Internationally Recognized KOL in Gynecologic Oncology and Cancer Biology*
- *Professor and Vice Chair for Trans. Research Depart. Gynecologic Oncology*
- *Director of the multi-disciplinary Blanton-Davis Ovarian Cancer Research Program and Co-leads the Ovarian Cancer Moon Shot Program at MD Anderson*

David Rosen, J.D.

FDA and Reg Affairs Expert



- *Partners and public policy lawyer with Foley & Lardner LLP*
- *Background in pharmacy and law, and 14 years of regulatory experience at the Food and Drug Administration (FDA) and expert on strategic guidance on FDA submissions*
- *FDA Council, Former FDA Panel Member, Author of Orange Book*

Internationally Recognized KOL in Gynecologic Oncology and Cancer Biology



Department of Gynecologic Oncology and Reproductive
Medicine, Division of Surgery

University of Texas MD Anderson Cancer Center

Antagonism of Tumoral Prolactin Receptor Promotes Autophagy-Related Cell Death

Nestor Avila¹, Behrang Zand², Bailett Ozpolat³, Mirrezaei J. Kazemian⁴, Churusha Liu⁵, Erhan Yang⁶, Amy Carroll⁷, Jennifer Ayler⁸, Chandra Balaraman⁹, Ibrahim Tawfik¹⁰, Yoon Seung Park¹¹, Giuseppe Ragozzino¹², Chen Yi Peng¹³, Richard P. Mason¹⁴, Robert A. Neufeld¹⁵, Michael H. Staudenmann¹⁶, David M. Croteau¹⁷, Joseph S. Binkley¹⁸, Yanyan Wei¹⁹, Yanyan Chen²⁰, Gabriel Legido-Quinones²¹, Marta Szepietz²², Naoto U. Uno²³, Robert L. Coleman²⁴, and Anil K. Boob²⁵

¹Department of Biomedical Sciences, University of Illinois at Chicago, Chicago, IL 60607, USA
²Department of Hematology/Oncology, The University of Texas MD Anderson Cancer Center, Houston, TX 77030, USA
³Department of Internal Medicine, Health and Needs Oncology, The University of Texas MD Anderson Cancer Center, Houston, TX 77030, USA
⁴Department of Hematology/Oncology, The University of Texas MD Anderson Cancer Center, Houston, TX 77030, USA
⁵Department of Pathology, The University of Texas MD Anderson Cancer Center, Houston, TX 77030, USA
⁶Department of Hematology/Oncology, The University of Texas MD Anderson Cancer Center, Houston, TX 77030, USA
⁷Winn-Dixie Cancer Center, University of Pittsburgh Medical Center, Pittsburgh, PA 15213, USA
⁸Departments of Psychology and Neuroscience, University of North Carolina at Chapel Hill, NC 27599, USA
⁹Department of Biological Sciences, Clemson University, Clemson, SC 29634, USA
¹⁰Department of Otorhinolaryngology, Medical University of Vienna, Vienna, Austria
¹¹Department of Radiological Science, Pusan National University of Medicine, Pusan 605-030, Korea
¹²Department of Physiology and Biochemistry, Faculty of Medicine, Hacettepe University, Ankara, Turkey
¹³Correspondence: nestor.avila@utoronto.ca
doi.org/10.1002/jbm.b.30140
This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>)

SUMMARY

Therapeutic upregulation of macroautophagy in cancer cells provides an alternative mechanism for cell death. Prolactin (PRL) and its receptor (PRLR) are considered attractive therapeutic targets because of their role in tumor growth and progression [1]. We utilized G129R, an antagomist PRLR peptide, to block activity of the tumor PRL/PRLR axis, which resulted in inhibition of tumor growth in orthotopic human breast tumor xenografts. In combination treatment with G129R induced the accumulation of redundant autolysosomes in 3D cancer spheroids, leading to a type II programmed cell death. This induction of autophagy was dependent on the PI3K/Akt/mTOR-dependent pathway and was sustained by an acetylated phosphoprotein (PEA-15) and protein kinase C ζ (PKC ζ) [2]. The induction of autophagy in clinical samples were associated with longer patient survival. Our findings provide an understanding of the mechanisms of tumor growth inhibition through blocking PRL/PRLR may have clinical implications.

INTRODUCTION

The multifunctional hormone, prolactin (PRL), is not only essential for normal reproduction and maintenance of pregnancy but also contributes to pathogenesis of gynecologic malignancies.

488 Cell Reports 7, 488–500, April 24, 2014 ©2014 The Authors

including ovarian and endometrial cancers [Liu *et al.*, 2009; Mao *et al.*, 2009; Tan *et al.*, 2011]. Human PRL has proproliferative effects on ovarian and endometrial cancer cells [Aaga-Sato *et al.*, 2009]. Recent studies support a robust role for PRL in ovarian cancer cell survival and invasion, which implicates it as a therapeutic target [Tan *et al.*, 2011]. PRL binding to its membrane-associated prolactin receptor (PRLR) is followed by activation of oncogenic signaling pathways such as JAK2 and STAT3, stimulating proliferation of cancer cells and tumor growth [Blaz *et al.*, 1994; Xie *et al.*, 2003]. Despite the importance of the PRL/PRLR-signaling complex in tumor growth, the underlying mechanisms are not well understood, and the ability to target this pathway is limited by the lack of a PRLR with its activity. G129P, a variant of normal human PRL that differs by a single amino acid substitution mutation, inhibited PRL-induced oncogenic signaling responsible for cancer cell proliferation [Liu *et al.*, 2009].

Autophagy is a lysosome-dependent cellular degradation pathway that can be triggered by many stimuli, including metabolic stress, hypoxia, or treatment with chemotherapy agents, or radiation (Rubinstein et al., 2007). Key proteins regulate the autophagy and expansion of vesicular structures known as autophagosomes, which fuse with lysosomes to form autolysosomes. Under normal conditions, basal levels of autophagy in proliferative cells function as a survival mechanism (Matthew et al., 2007). Prolonged exposure to therapeutic agents, however, can lead to progression of destructive autophagy and eventual programmed cell death (Bailly et al., 2007; Kimmelman et al., 2008). The study of autophagy can induce sustained autophagy, offer new therapeutic opportunities (Santoro et al., 2006), particularly in breast.

MOLECULAR CANCER
THERAPEUTICS

Blockade of the Short Form of Prolactin Receptor Induces FOXO3a/EIF-4EBP1-Mediated Cell Death in Uterine Cancer

Yunfei Wen¹, Ying Wang², Anca Chelariu-Raicu¹, Elaine Stur¹, Yuan Liu^{1,3}, Sara Corvigno¹, Faith Bartsch⁴, Lauren Redfern¹, Behrouz Zand¹, Yu Kang¹, Jinsong Liu⁵, Keith Baggerly², and Anil K. Sood¹

ABSTRACT

Abnormal activity of human prolactin receptor (PRLR) and its membrane-associated receptor (PRLR) contributes to the progression of uterine carcinoma. However, the underlying mechanisms are not well understood, and current means of targeting the PRLR/PRLR axis in uterine cancer are limited. Our integrated analyses using the Cancer Genome Atlas and Genotype-Tissue Expression (GTEx) datasets revealed that the PRLR isoform PRLR (PRLR_SF) is the isoform predominantly expressed in human uterine cancers, expression of this PRLR_SF was elevated in uterine cancers in comparison with cancer-free uterine tissues. We hypothesized that the overexpression of PRLR_SF in uterine cancer cells contributes, in part, to the oncogenic activity of the PRLR/PRLR axis. Next, we generated a PRLR_SF-specific siRNA and human PRLR, to block the PRLR/PRLR axis in both PTEN^{+/+} and

Introduction

Human prolactin (PRL) acts primarily to regulate the normal functions of the female reproductive system (1), but it is also involved in multiple processes during tumor pathogenesis, including angiogenesis and regulation of the immune system (2). Levels of circulating PRL are elevated in many types of cancer, including breast cancer (3). PRL plays key regulatory roles during the development and progression of endometriosis (1), as the production of PRL by the endometrium is elevated during the normal menstrual cycle (5). Substantially elevated levels of PRL and its receptor (PRLR) have been reported in serum samples from patients with uterine cancer (2), suggesting that PRL signaling may play a potentially important role in the development of cancer (3) as well as a possible marker for uterine cancer (4).

Although some antibodies targeting PRLR have been shown to reduce tumor malignancy by blocking autocrine/endorcine PRL activity

ities [6,7], the full-length PRLR gene product was not detected in endometrial tissues [8]. Our results from screening an array of human uterine cancer cells indicated that transcriptionally spliced isoforms of PRLR products might be responsible for mediating activities of the tumoral PRL/PRLR axis. However, the expression and functional mechanisms of different PRLR isoforms remain uncharacterized. PRLR-P1L1 is reportedly involved in multiple signalling pathways, such as activating PI3K/Akt pathway through its C-terminal members and JAK2, refs. 12–14; GRB2 signaling cascade, and regulation of transcription factors such as c-Myc, Jun, and T-cell factors, refs. 15–17. This diversity is partly due to the wide variety of PRLR isoforms, which in turn leads to the regulation of different downstream signalling cascades. RefSeq data from the USC Genome Browser predicted nine isoforms among the transcripts encoded by the PRLR gene, while the other two are alternative splicing variants of PRLR isoforms, while the other is transcribed, transcribed, alternatively.

STRUCTURE, while the other is a noncoding transcript variant. Primarily, the extracellular ligand-binding domains are highly conserved and retain PRL-binding activity, while the membrane-proximal region, including the transmembrane domains and intracellular domain, varies between isoforms; this variation contributes to the diversity in PRL signaling activities (18). High expression of variable PRL isoforms has been reported to be involved in cancer cell survival in gynecologic (19) malignancies (20).

Among the PRLR transcribed isoforms, three have been characterized in animals and humans as transmembrane receptors: the long form (LF, ~100 kDa), intermediate form (IM, 65–70 kDa), and short form (SF, 45–50 kDa). PRLR_LF is transcribed from exons 3–10 (21) and PRLR_IM (65–70 kDa) from an alternative splicing deletion of

produced via alternative splicing of exons 10 and 11 during transcription of the PRLR gene (23, 24). The PRLR_SF isoform is functionally different from PRLR_LF because of their involvement with distinct downstream factors in mediating PRL signaling in cancer cells (25).

301. VASCULAR WALL BIOLOGY, ENDOTHELIAL PROGENITOR CELLS, AND
PLATELET ADHESION, ACTIVATION, AND BIOCHEMISTRY | NOVEMBER 29, 2018

Platelets Promote Activation of the Complement System in Ovarian Cancer

Omayra Gonzalez Pagan, MD,¹ Min Soon Cho, PhD,² Vahid Afshar-Kharghan, MD³

¹UT Houston - McGovern Medical School, Houston, TX

²MD Anderson Cancer Center, Houston, TX

³M.D. Anderson Cancer Center, Houston, TX

Blood (2018) 132 (Suppl_1) : 4970.

<http://doi.org/10.1182/blood-2018-99-116752>

Abstract

Platelets promote metastasis and growth of ovarian cancer. We have shown that platelets extravasate into the tumor microenvironment (TME) and increase proliferation and epithelial-mesenchymal transition (EMT) in ovarian cancer cells. We have also shown that activation of the complement system in TME of ovarian cancer enhances tumor growth. Ovarian cancer cells secrete complement proteins that upon activation in the TME increase proliferation of cancer cells and promote EMT via an autocrine pathway. The activators of the complement system in the TME have not been identified. We have demonstrated that upon activation platelets activate the complement system on their surface. In the current study, we examined whether extravasated platelets inside tumors contribute to the complement activation in the TME.



1) We examined the effect of antiplatelet reagents on platelet extravasation into TME, using murine models of ovarian cancer. Tumors induced by injection of ovarian cancer cells into the peritoneum of Nu/Nu mice were resected after 6-8 weeks and the number of extravasated platelets was determined by immunostaining tumor sections and counting the number CD42 (GPIb) positive cells that were outside the blood vessels (CD31 positive). We found that platelet extravasation is an active process and platelet inhibition by aspirin or ticagrelor reduces the number of extravasated platelets. Furthermore, P2Y12 deficient platelets extravasate less than normal platelets. In all of these experiments, the number of

MD Anderson
~~Cancer~~ Network®

AACR American Association
for Cancer Research



Development Pipeline

Program	Indication	Discovery	Preclinical	Phase 1	Phase 2	Highlights
KAD101	Ovarian Cancer					New formulation complete PK analyses + manufacturing development underway Targeting Ph1 initiation in Q4 2026/Q1 2027
KAD102	Uterine Cancer					New molecular entity – prolactin antagonist with improved half-life and bioavailability SRA with MDACC (2026)

Ovarian Cancer

Patient Journey is Grim & Needs a Solution to Improve Outcomes

Typically identified when already late stage making treatment difficult and costly. Our focus is to target the cause that created the cancer.

Market Opportunity¹

\$3.7B Current therapies have limited efficacy but represent large market

\$6.4B Expected to grow at a 14.4% CAGR in 2024

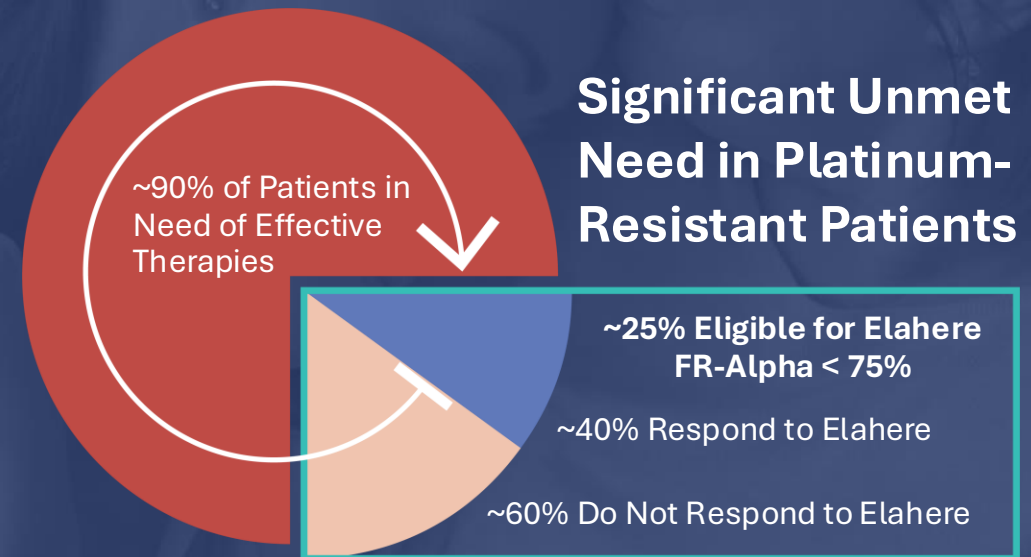
0 Therapeutics that offer a durable response

19,710¹

Estimated new cases diagnosed in the US in 2023

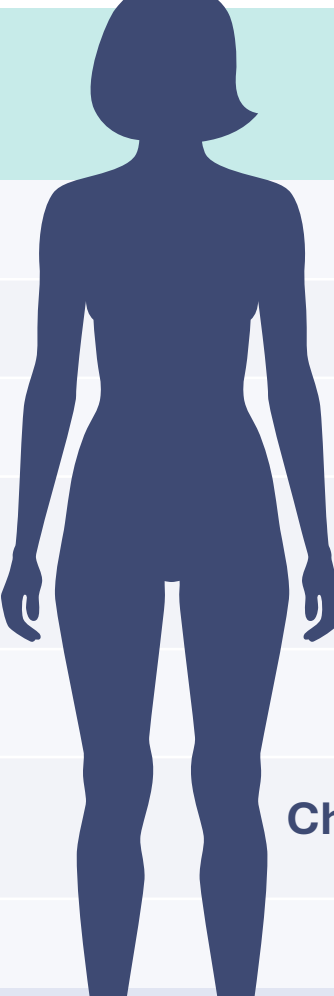
13,270¹

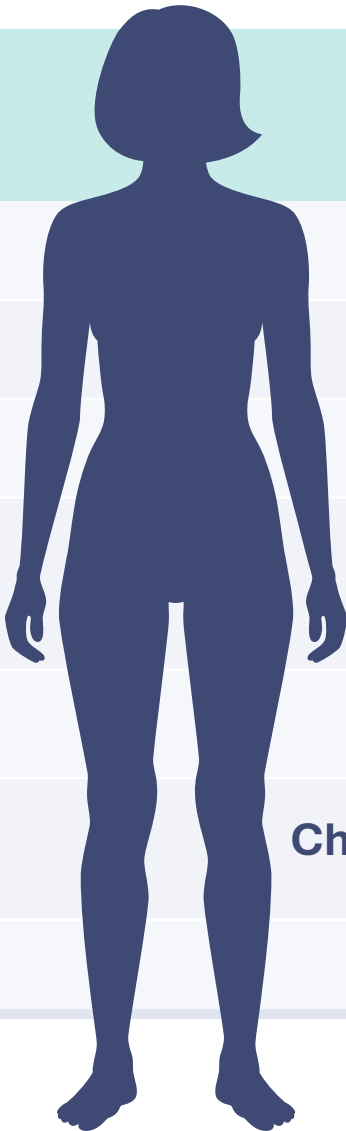
Approximate deaths resulting from ovarian cancer in the US in 2023



Consistently Poor Results Across Therapies

Underpins Need for New Innovative Approach

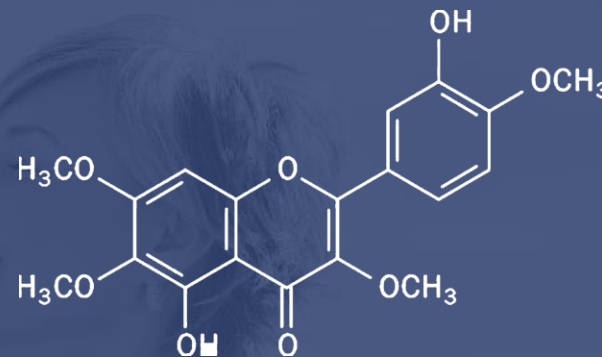
Drug	Target	% of Patients Expression		ORR	mPFS (mo)	mOS (mo)	
<i>KAD101 KAD102</i>	<i>Prolactin</i>	<i>~80%</i>		<i>KAIDA Opportunity</i>			
Abbvie: Elahere	FR-Alpha ≥75%	~25-30%		42%	5.6	16.5	
Sutro: Luvelta	FR-Alpha ≥25%	~60-80%		~38%	NA	NA	
Corcept: Relacorilant	Glucocorticoid Receptor (GR)	~40%		33%	5.6	13.9	
Checkpoint Inhibitors	PD-(L)1	~10-20%		~5-15%	2.1-3.5	11.8-18.7	
				Chemo	~15-20%	~3.5 Average	~13.4 Average
Mersana: Upfitamab	NaPi2b	~50%		13%	Study Failed		



KAD101 Initially Targeting Ovarian Cancer

Novel Biologic that Blocks the Prolactin Receptor to Prevent Cancer Cell Growth Signals, Incite Autophagy, and Re-sensitization to Chemotherapies

- Novel encapsulated formulation of de-risked asset, KAD101, which has seen promising initial human clinical data
- Multiple expansion opportunities into endometrial, uterine and breast cancers
- Opportunity as maintenance therapy



Prolactin

Higher Expression Correlates with Reduced Survival Contributing to Tumor Growth and the Development of Malignancies

Targeting Prolactin

- Potential to disrupt tumor growth and reverse the process through autophagy
- KAD 101 prevents prolactin receptor dimerization

Impact on Cell Signaling

- Involved in pathways like JAK/STAT5 and PI3K/Akt, essential for cell proliferation

Chemotherapy Resistance

- The down-regulation of GST is directly linked to chemotherapy resistance, making patients receptive again, a major treatment hurdle

Differentiated Mechanism of Action

KAD101 represents a longer half life molecule that blocks the prolactin receptor to prevent cancer cell growth signals and initiate autophagy

Activates Autophagy

- Triggers cell 'self-eating' process, leading to the death of cancer cells

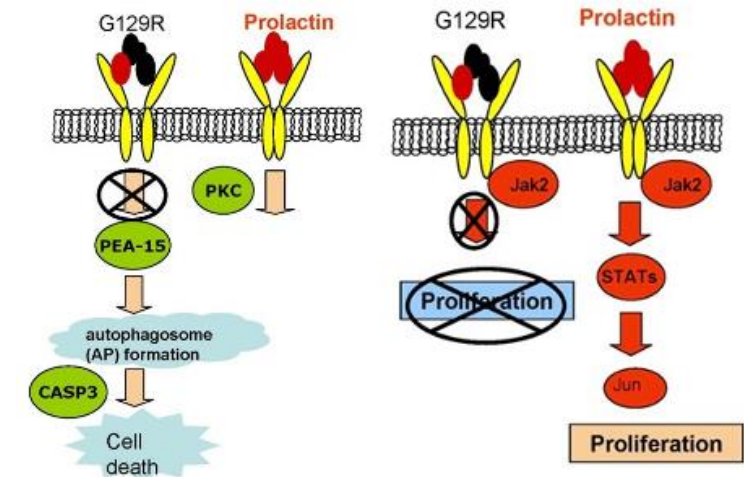
Cell Proliferation Halted

- Inhibits the Jak2 pathway, which is crucial for cancer cell multiplication

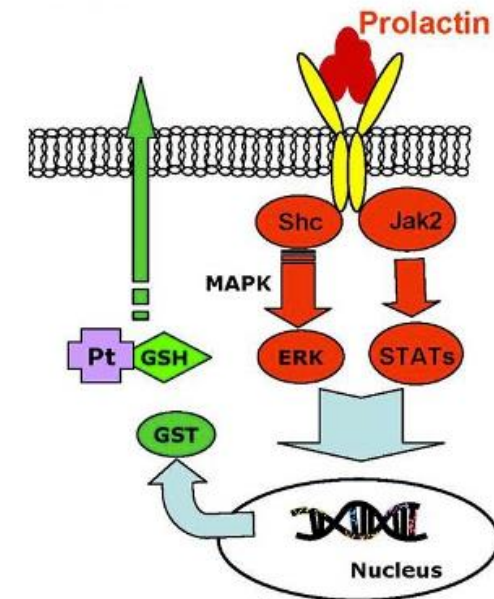
Chemoresistance Addressed

- Downregulates GST enzyme, which has shown to reactivate response to chemotherapy in chemo-resistant patients

Autophagy: Programmed Cell Death



Downregulates GST: Chemoresistance



G129R⁽¹⁾ First-In-Human Phase 1 Clinical Data

First cohort of subtherapeutic dosing resulted in therapeutic benefit/signal observed in all patients with clean safety profile/low toxicity

Patient 1

- Received Taxol with Neulasta
- Had 2 significant RECIST-measured tumors
- Stable disease achieved; no new cancer growths observed (day 29)
- Treatment with low-dose deemed effective

Patient 2

- Treatment history includes Taxol, Carboplatin, and Doxil
- Presented with one large RECIST-measured tumor
- Tumor shrank to non-measurable size post-KAD101 treatment (day 29)
- Demonstrated significant response to low-dose

Patient 3

- Prior treatments: Gemzar, Carboplatin, Doxil, Avastin
- Started the three RECIST-measured tumors
- Post-treatment, all three tumors reduced by 15-20% in size (day 29)
- Showed a marked reduction in tumor volume

KAD101 development addressing two key limitations of G129R, extremely short circulation half-life and necessity of daily injections

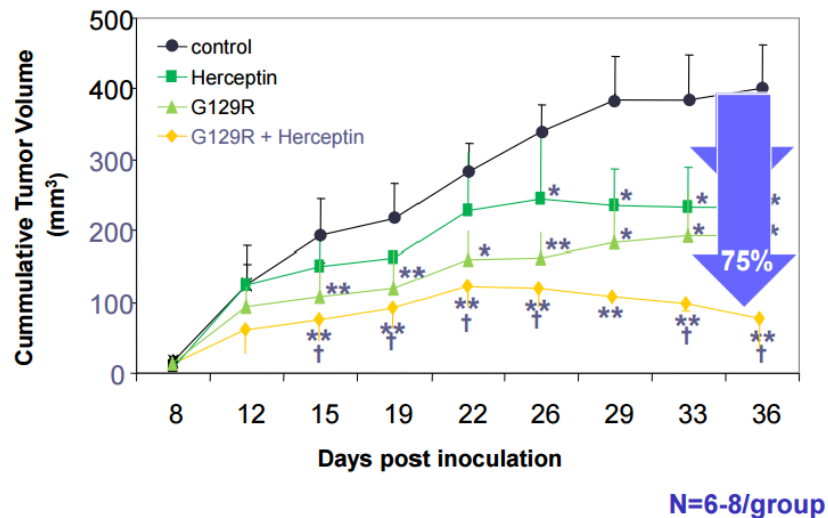
Notes:

(1) G129R (PRL antagonist) dosage administered to three enrolled patients in Phase 1 clinical trial, Oncolix, Inc. sponsor, was initial (lowest) dose intended as part of the three doses planned to be evaluated in the (dose escalation) study

G129R Preclinical Experiments

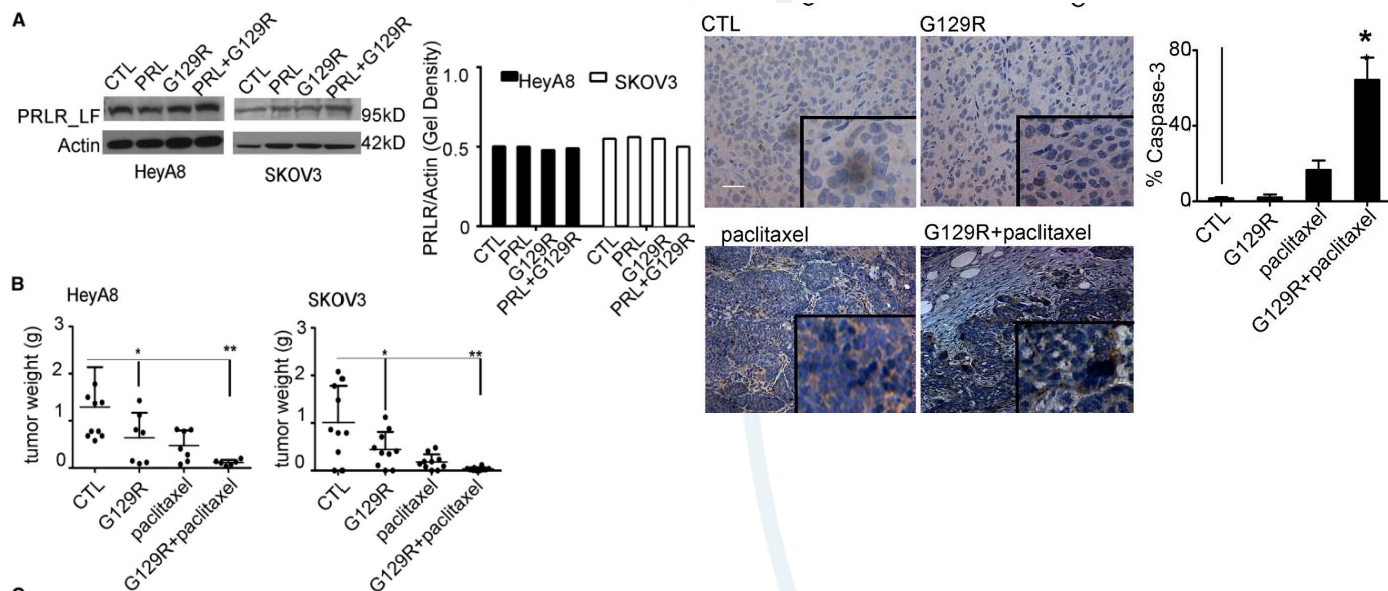
Preclinical data of KAD101 + standard of care therapies highlight therapeutic synergy

G129R Potentiates Herceptin Effects in T-47D Xenografts in Nude Mice⁽¹⁾



T-47D cells (8.5×10^6) were injected in the mammary fat pad of female nude mice. One week after tumor cell inoculation, G129R (4 mg/kg/daily) or Herceptin (4 mg/kg/bi-weekly), or a combination of both were injected i.p. for 4 weeks. Measurements of tumor growth were recorded once a week. Each value is a mean \pm SD of 6-8 mice/group. ** $P < 0.01$ and * $P < 0.05$ versus control; † $P < 0.05$ versus Herceptin alone.

G129R Inhibited Tumor Growth in Ovarian Cancer Models⁽²⁾



G129R Inhibited Tumor Growth in Ovarian Cancer Models

(A) Left, PRLR-LF was detected in HeyA8 and SKOV3 cells treated with PRL (0.1 mg/ml), G129R (10 mg/ml), a combination (PRL+G129R), or control (CTL). Right, the fold gel density was normalized to that of b-actin.

(B) Effects of G129R and paclitaxel on tumor growth in HeyA8 and SKOV3 orthotopic mouse models. Weight of the tumor from each mouse is shown; left, HeyA8: * $p < 0.05$ (CTL versus G129R); ** $p < 0.001$ (CTL versus G129R+paclitaxel). Right, SKOV3: * $p < 0.05$ (CTL versus G129R); ** $p < 0.001$ (CTL versus G129R+paclitaxel).

(C) Left, representative images of cleaved caspase-3 in HeyA8 tumor tissues (inset shown at 2003 magnification); the scale bar represents 100 μ m. Results were confirmed with triplicate experiments. Right, percentage of tumor with cleaved caspase-3 staining is shown graphically (error bar = 95% confidence interval); differences between groups were compared by unpaired two tailed t test: $n = 5$, * $p < 0.05$ (paclitaxel versus G129R+paclitaxel).

Notes:

- (1) Wen Y Chen, Oncolix, Inc.
- (2) Wen, Yunfei et al. Cell Reports, Volume 7, Issue 2, 488 - 500



Accomplished & Upcoming Milestones

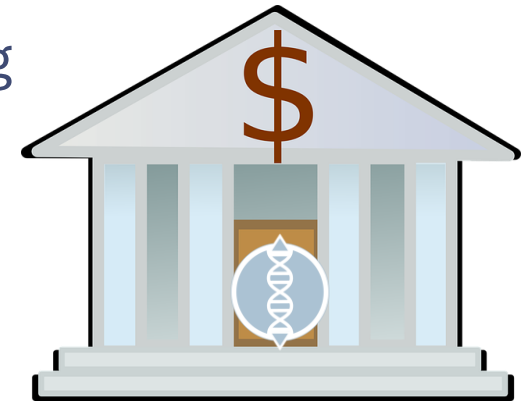
Completed

- Preclinical translational studies
- Re-formulation for KAD101 and IP through partner
- Regulatory path/strategy established
- Patents filed with further IP filings
- SAB in place with Dr Anil Sood from MD Anderson
- Commence manufacturing of KAD101 with US Based CDMO
















Upcoming Milestones and Events

- Re-open Existing IND. Securing Type C development meeting
- Complete animal "bridging" studies for the new formulation
- KAD101 limited drug substance toxicity studies anticipated
- Sponsored Research Agreement with MD Anderson
- Expanding IP portfolio with additional patent filings
- KAD101 Phase 1 study initiation expected Q4 2026/Q1 2027



Peer Valuations Suggest Potential for Significant Upside, Even at Earlier Stages

Company	Drug	Phase	ORR	mPFS (mo)	mOS (mo)	Deal Price / Market Cap
Acquisitions						
 immunogen	 irinotecan saravatinic-gym injection 400 mg	Approved	42%	5.6	16.5	\$10.1 Billion Acquired by  abbvie
 ProfoundBio	Rinatabart sesutecan: FR-alpha ADC	Phase 1/2	NA	NA	NA	\$1.8 Billion Acquired by  Genmab
Public Companies						
 SUTRO BIOPHARMA	Luvelta	Phase 2/3	~38%	NA	NA	\$350 Million
 Corcept THERAPEUTICS	Relacorilant	Phase 2	33%	5.6	13.9	\$2.8 Billion
 zentalis	Azenosertib	Phase 1/2		Study Ongoing		\$825 Million
 Nuvation Bio	NUV-1511	Phase 1/2		Study Ongoing		\$825 Million
 SHATTUCK LABS	SL-172154	Phase 1	9%	-	-	\$500 Million
 MACROGENICS	MGC026	Phase 1		Study Ongoing		\$280 Million
Recent Private Financings Underscores Interest and Value in Ovarian Cancer Space						
Company	Drug	Phase	Last Round	Total Raise	Total Raise to Date	
 TORL BIOTHERAPEUTICS	TORL-1-23: Claudin-6 ADC	Phase 1	Series B	\$158 Million	\$350	
 KAIDA BioPharma						

Highlights

Lead product candidate (KAD101, best-in-class next generation prolactin antagonist) indication: platinum resistant ovarian cancer (PROC)

90% of patients are in need for an effective treatment

Targeting Primary Cause of Hormone-Driven Cancers: Prolactin

- KAD101 demonstrated promising initial human clinical data
- Leadership has extensive preclinical, clinical, and drug development experience/expertise to execute clinical development plan
- Targeting prolactin; over expressed in ~80% of patients with ovarian cancer¹
- Kaida SAB lead, Anil K. Sood, MD – MDACC², is internationally recognized PROC KOL and leading expert on prolactin (PRL) biology
- KAD101 Phase 1 study initiation expected Q4 2026/Q1 2027
- Pipeline expansion opportunity into Breast and Uterine cancer. Prolactin over expressed in ~90% of patients³

1. V. Levina et al. Biological Significance of Prolactin in Gynecologic Cancers. Cancer Res 15 June 2009; 69 (12): 5226-5233. <https://doi.org/10.1158/0008-5472.CAN-08-4652>; 2. MDACC: The University of Texas MD Anderson Cancer Center 3. Faupel-Badger et al. Prolactin receptor expression and breast cancer: relationships with tumor characteristics among pre- and post-menopausal women in a population-based case-control study from Poland. Horm Cancer. 2014 Feb;5(1):42-50. doi: 10.1007/s12672-013-0165-7. Epub 2013 Nov 19. PMID: 24249584; PMCID PMC3906637.



K A I D A
B i o P h a r m a

Thank You!

Dr. Vnook
KAIDA BioPharma
Email: skvnook@gmail.com
www.KAIDA-BioPharma.com



Glossary of Terms

Autophagy	A process where cells recycle their components to stay healthy and respond to stress.
CAGR	Compounded Annual Growth Rate: The annualized average growth rate of an investment or metric over a specified period.
Cell Proliferation	The process by which cells divide and multiply, leading to growth or tissue repair.
Chemoresistance	The ability of cancer cells to resist the effects of chemotherapy.
Endometrial Cancer	Cancer that begins in the lining of the uterus (endometrium).
GST Enzyme	Glutathione S-transferase: An enzyme involved in detoxifying harmful compounds within cells.
Half-Life	The time it takes for a substance, like a drug, to reduce to half its original amount in the body.
Jak2 Pathway	A signaling pathway involved in cell growth and development, often linked to certain cancers.
Malignancy	Cancerous growth that can spread to other parts of the body.
mOS	Medium overall survival.
Orphan Drug Designation	A special status for drugs targeting rare diseases affecting fewer than 200,000 patients in the U.S., providing 7 years of market exclusivity.
ORR	Overall Response Rate: The percentage of patients whose cancer shrinks or disappears due to treatment.
Ovarian Cancer	Cancer that begins in the ovaries, part of the female reproductive system.
PFS	Progression Free Survival: The length of time during and after treatment that a patient's cancer does not worsen.
Platinum-Resistant Ovarian Cancer (PROC)	A type of cancer that does not respond well to platinum-based chemotherapy.
Prolactin	A hormone primarily responsible for stimulating milk production in mammals.
RECIST-measured	A standard way to measure tumor response to treatment, using defined criteria.
SRA	Sponsored Research Agreement.
Tumor	An abnormal mass of tissue resulting from uncontrolled cell growth.
Uterine Cancer	Cancer that starts in the uterus, commonly involving the endometrium.