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BioPharma

Seeking Victory in the Fight Against Ovarian Cancer

May 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

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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 cancer[1]
- Kaida SAB lead, Anil K. Sood, MD – MDACC[2], 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 patients[3]

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***

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Kaida Scientific and Clinical Advisor: Anil K. Sood, M.D.

Internationally Recognized KOL in Gynecologic Oncology and Cancer Biology

Anil K. Sood, M.D.

Department of Gynecologic Oncology and Reproductive Medicine, Division of Surgery

University of Texas MD Anderson Cancer Center

Cell Reports

A Cell Press open access journal

Antagonism of Tumoral Prolactin Receptor Promotes Autophagy-Related Cell Death

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http://dx.doi.org/10.1016/j.celrep.2014.03.009

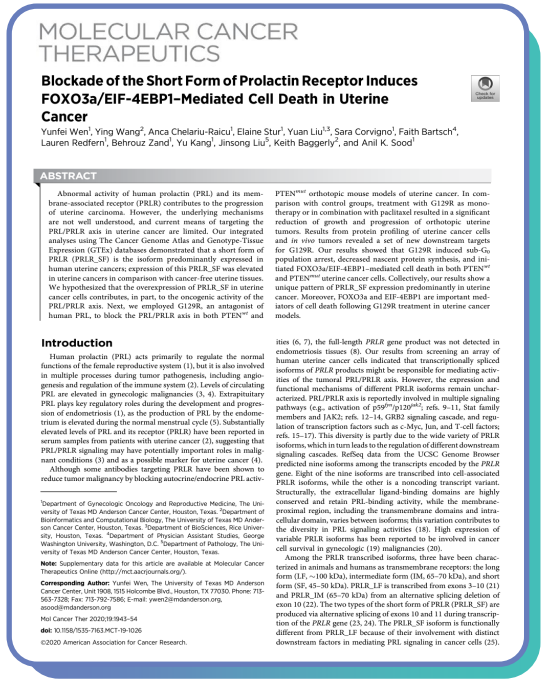
SUMMARY

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 roles as growth factors in tumor growth and progression. We utilized G129R, an antagonist peptide of PRL, to block activity of the tumoral PRL/PRLR axis, which resulted in inhibition of tumor growth in orthotopic models of human ovarian cancer. Prolonged treatment with G129R induced the accumulation of redundant autolysosomes in 3D cancer spheroids, leading to a type II programmed cell death. This inducible autophagy was a noncanonical beclin-1-independent pathway and was sustained by an astrocytic phosphoprotein (PEA-15) and protein kinase C zeta interactome. Lower levels of tumoral PRL/PRLR in clinical samples were associated with longer patient survival. Our findings provide an understanding of the mechanisms of tumor growth inhibition through targeting PRL/PRLR and 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 malignances, including ovarian and endometrial cancers (Levina et al., 2009; Mor et al., 2005; Tan et al., 2011). Human PRL has proproliferative effects on ovarian and endometrial cancer cells (Asai-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 (Fuj et al., 1994; Xie et al., 2002). 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 incomplete knowledge of its activity. G129R, 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 (Llovera et al., 2000).

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 (Rubinsztein et al., 2007). Key proteins regulate the formation and expansion of vesicular structures such as autophagosomes, which then fuse with lysosomes to form autolysosomes. Under normal conditions, basal levels of autophagy in proliferative cells function as a survival mechanism (Mathew et al., 2007). Prolonged exposure to therapeutic agents, however, can lead to progression of destructive autophagy and eventual programmed cell death (Dalby et al., 2010; White et al., 2010). Targeted molecular therapies that can induce sustained autophagy offer new therapeutic opportunities (Shimizu et al., 2004), 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 Wen[1], Ying Wang[2], Anca Chelariu-Raicu[1], Elaine Stur[1], Yuan Liu[1,3], Sara Corvigno[1], Faith Bartsch[4], Lauren Redfern[1], Behrouz Zand[1], Yu Kang[1], Jinsong Liu[5], Keith Baggerly[2], and Anil K. Sood[1]

ABSTRACT

Abnormal activity of human prolactin (PRL) 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 PRL/PRLR axis in uterine cancer are limited. Our integrated analyses using The Cancer Genome Atlas and Genotype-Tissue Expression (GTEx) databases demonstrated that a short form of 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 PRL/PRLR axis. Next, we employed G129R, an antagonist of human PRL, to block the PRL/PRLR axis in both $PTEN^{wt}$ and $PTEN^{mut}$ orthotopic mouse models of uterine cancer. In comparison with control groups, treatment with G129R as monotherapy or in combination with paclitaxel resulted in a significant reduction of growth and progression of orthotopic uterine tumors. Results from protein profiling of uterine cancer cells and *in vivo* tumors revealed a set of new downstream targets for G129R. Our results showed that G129R induced sub-G_0 population arrest, decreased nascent protein synthesis, and initiated FOXO3a/EIF-4EBP1–mediated cell death in both $PTEN^{wt}$ and $PTEN^{mut}$ uterine cancer cells. Collectively, our results show a unique pattern of PRLR_SF expression predominantly in uterine cancer. Moreover, FOXO3a and EIF-4EBP1 are important mediators of cell death following G129R treatment in uterine cancer models.

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 gynecologic malignancies (3, 4). Extrapituitary 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/PRLR signaling may have potentially important roles in malignant conditions (3) and as a possible marker for uterine cancer (4).

Although some antibodies targeting PRLR have been shown to reduce tumor malignancy by blocking autocrine/endocrine PRL activities (6, 7), the full-length *PRLR* gene product was not detected in endometriosis 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. PRL/PRLR axis is reportedly involved in multiple signaling pathways (e.g., activation of $p59^{fyn}$/$p120^{ctn}$; refs. 9–11, Stat family 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 signaling cascades. RefSeq data from the UCSC Genome Browser predicted nine isoforms among the transcripts encoded by the *PRLR* gene. Eight of the nine isoforms are transcribed into cell-associated PRLR isoforms, while the other is a noncoding transcript variant. Structurally, 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 PRLR 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 exon 10 (22). The two types of the short form of PRLR (PRLR_SF) are 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).

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Note: Supplementary data for this article are available at Molecular Cancer Therapeutics Online (http://mct.aacrjournals.org/).

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Mol Cancer Ther 2020;19:1943–54

doi: 10.1158/1535-7163.MCT-19-1026

blood

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

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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

blood

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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)

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Ovarian Cancer

Patient Journey is Grim & Needs a Solution to Improve Outcomes

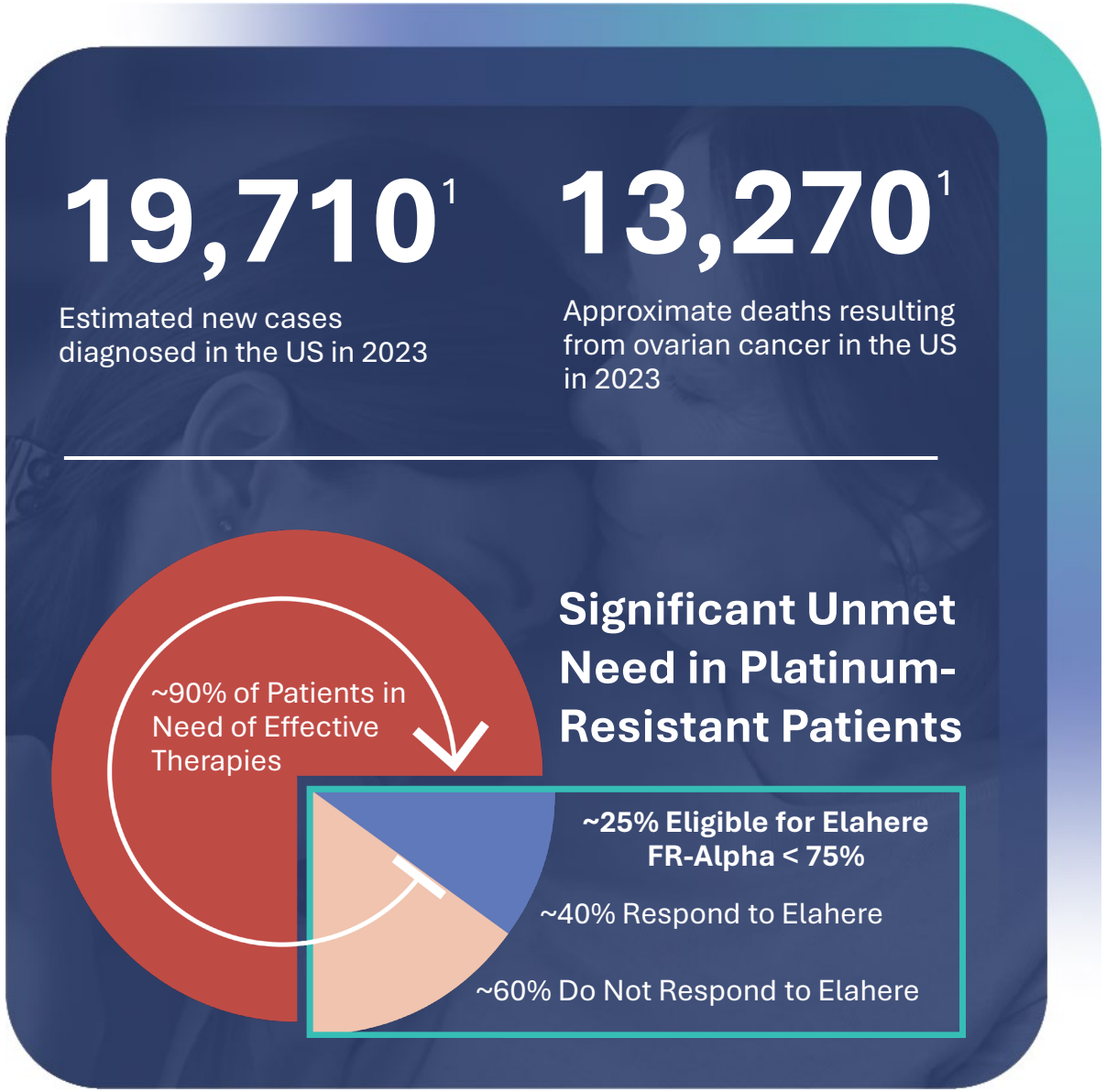
Typically identified when already late stage making treatment difficult and costly. Our focus is to target the cause that created the cancer.

Market Opportunity[1]

$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

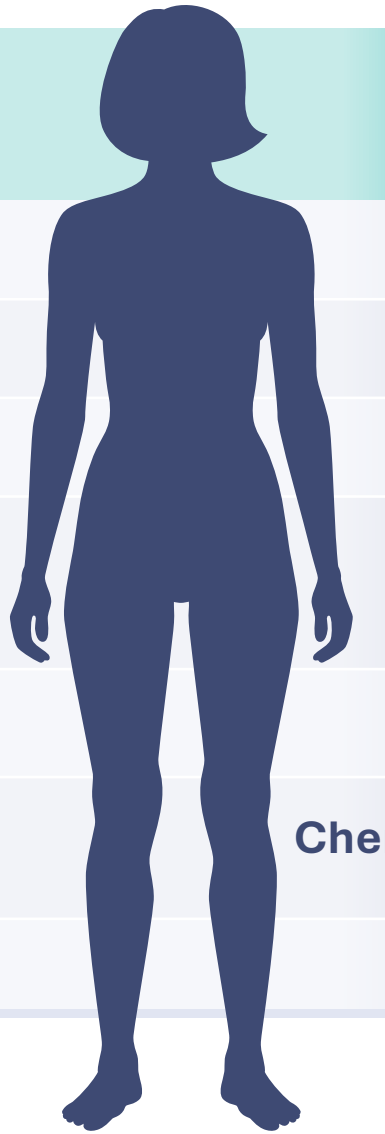


1. Research & Markets Ovarian Cancer drugs global Market Report 2023

Consistently Poor Results Across Therapies

Underpins Need for New Innovative Approach

Drug	Target	% of Patients Expression	ORR	mPFS (mo)	mOS (mo)
KAD101 | KAD102	***Prolactin***	***~80%***	***KAIDA Opportunity***		
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	



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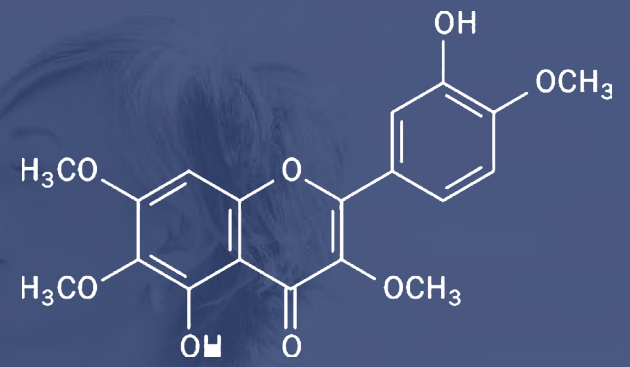
KAD101 Initially Targeting Ovarian Cancer

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

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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

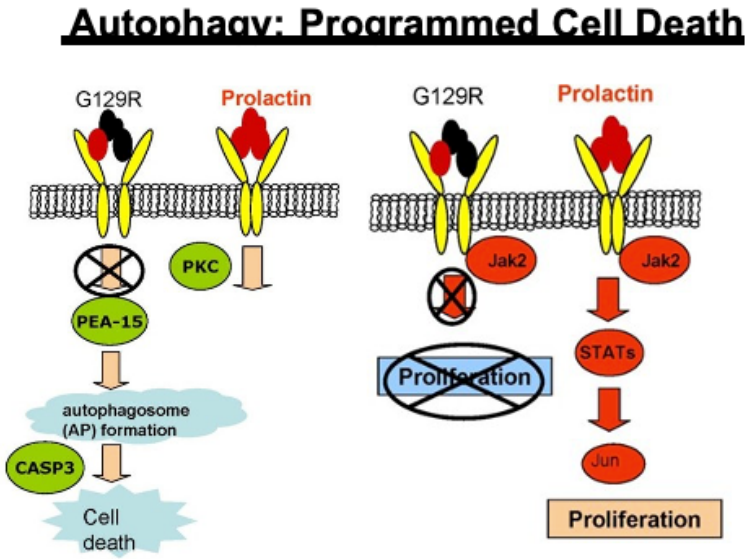
- Triggers cell 'self-eating' process, leading to the death of cancer cells

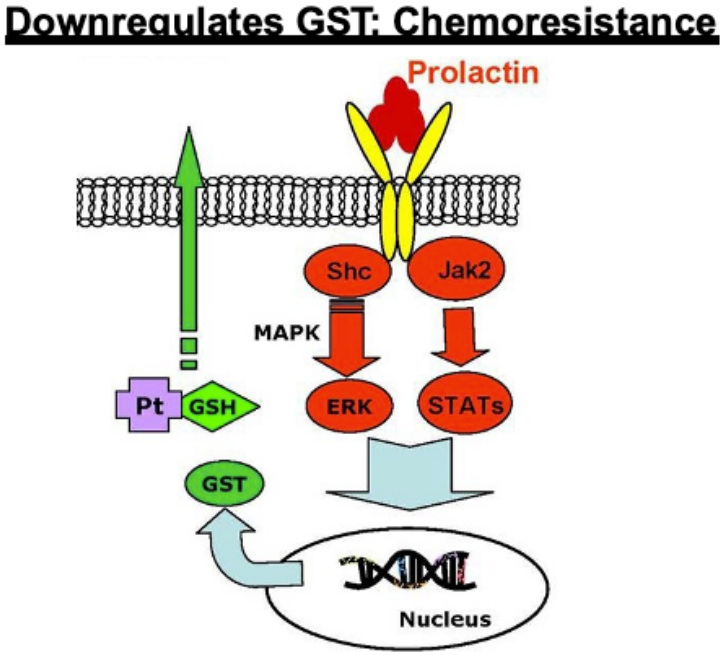
Cell Proliferation Halted

- 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





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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

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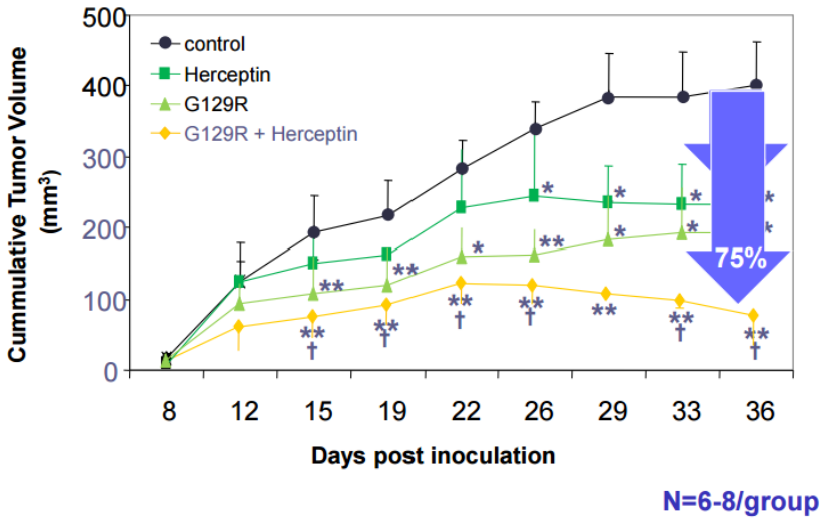
G129R Preclinical Experiments

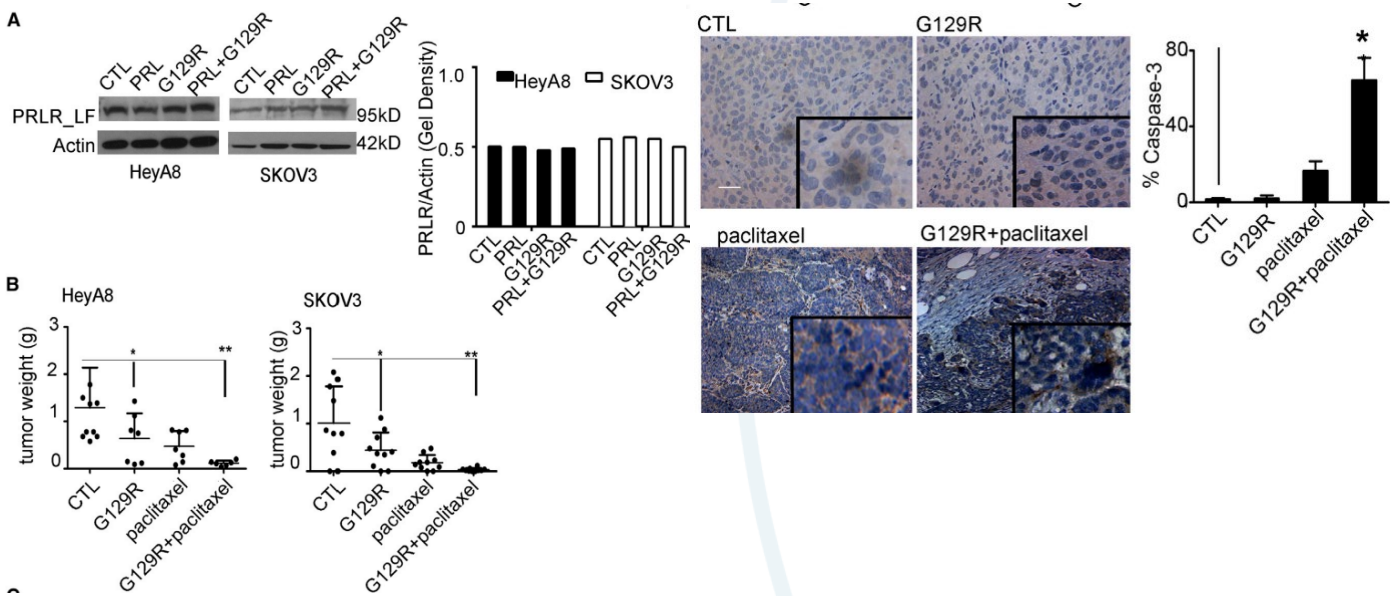
Preclinical data of KAD101 + standard of care therapies highlight therapeutic synergy

G129R Potentiates Herceptin Effects in T-47D Xenografts in Nude Mice[1]



T-47D cells (8.5 x10^6) 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 ± 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 mm. 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).

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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

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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	ELAHERE mirvetuximab soravtansine-gynx injection 100 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**

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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 cancer[1]
- Kaida SAB lead, Anil K. Sood, MD – MDACC[2], 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 patients[3]

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.

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Thank You!

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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.