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Seeking the First Victory in Ovarian Cancer

February 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 hormonal therapies to address the root cause of cancers affecting women.

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Highlights

Dedicated to advancing ovarian cancer treatment through innovative drug development

90% of patients are in need for an effective treatment

We Treat the Cause of the Cancer

- Lead program demonstrated promising initial human clinical data
- Targeting prolactin; over expressed in ~80% of patients with ovarian cancer[1]
- Potentially 1st in class prolactin receptor antagonist
- Rapidly advancing into Phase 1 study in H1 2026
- Pipeline expansion opportunity into Breast and Uterine cancer. Prolactin over expressed in ~90% of patients[2]

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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. 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.

Development Pipeline

Program	Indication	Discovery	Preclinical	Phase 1	Phase 2	Highlights
KAD101	Ovarian Cancer					New formulation Rapidly advancing into Phase 1 study targeted for H1 2026
KAD102	Uterine Cancer					Enhanced pure antagonist of KAD101 New molecular entity entering an SRA with MD Anderson in Q1 2025

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Leadership Team with Proven Track Record

Dr. Stella Vnook, MBA

Co-Founder

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

Likarda · Jazz Pharmaceuticals · MERCK · Catalent

Craig Pierson

Chairman, Founder

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

Lifetech Ventures · AiM Medical Robotics

John Langenheim, PhD

CSO, Co-founder

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

Thomas Jefferson University · Medical College of Wisconsin · MUSC Health Medical University of South Carolina

William Gannon Jr., MD, MBA

Director of Clinical & Medical Affairs

Clinical Trials Director, FDA Strategist

BioSTAT · Apthera · QUINTILES · Celsion Corporation

David Rosen

Foley & Lardner LLP

FDA Council, Former FDA Panel Member, Author of Orange Book

FDA · FOLEY & LARDNER LLP · DLA PIPER

Anil K. Sood, M.D.

Head of SAB

Department of Gynecologic Oncology and Reproductive Medicine, Division of Surgery

The University of Texas MD Anderson Cancer Center

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Ovarian Cancer

Patient Journey is Grim & Needs a Solution to Improve Outcomes

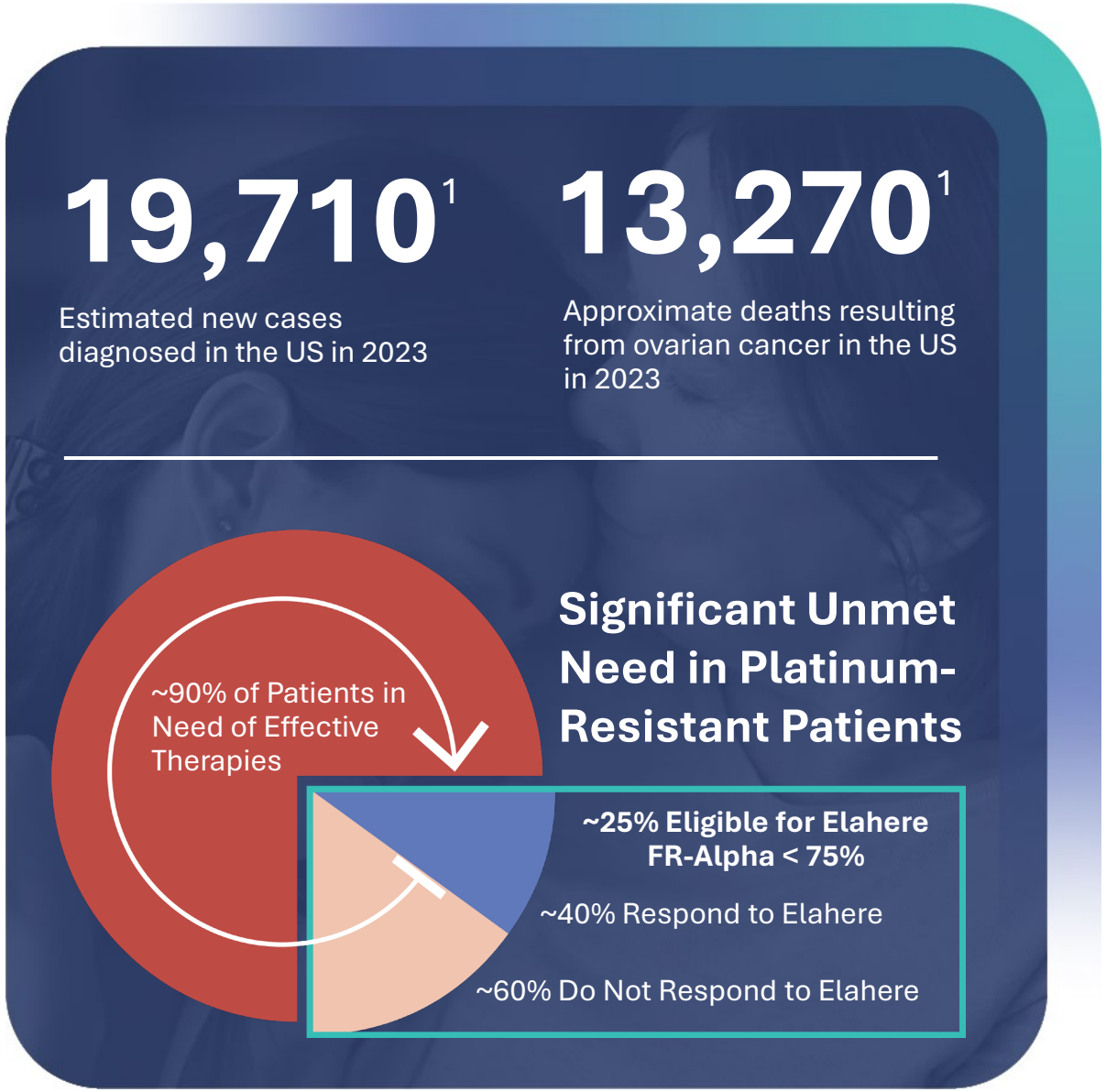
Typically identified when already late stage making treatment difficult and costly. Our focus is to target the cause that created the cancer.

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

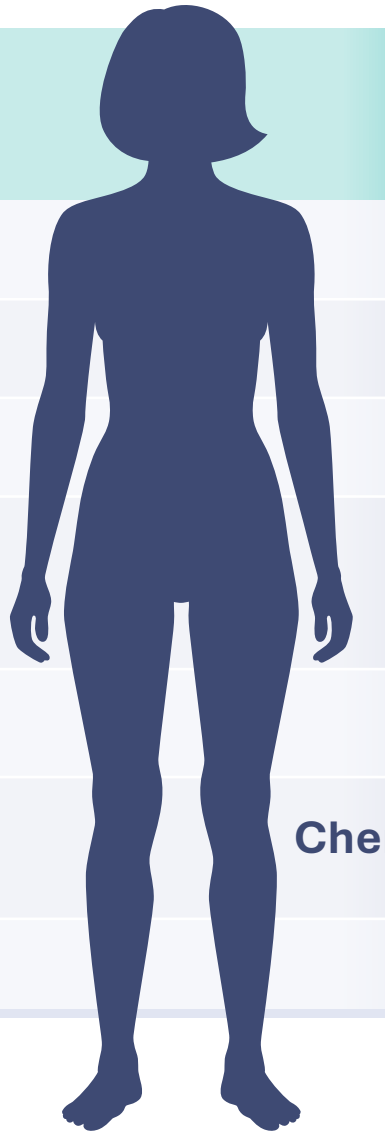


1. Research & Markets Ovarian Cancer drugs global Market Report 2023

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%***	***KAD102 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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Key Cancers Where Prolactin Plays a Role

1

Breast Cancer

Prolactin receptor overexpression is seen in up to 90% of breast cancer cases, especially in hormone receptor-positive subtypes. Prolactin promotes tumor growth and metastasis by activating the Jak2/STAT pathway and other downstream effectors that drive proliferation and survival.

2

Uterine Cancer:

Prolactin overexpression and its signaling through PRLR have been implicated in endometrial cancer. Prolactin stimulates cell proliferation and protects cancer cells from apoptosis (programmed cell death).

3

Prostate Cancer:

In prostate cancer, prolactin is thought to enhance tumorigenesis through both autocrine and paracrine mechanisms, activating the Jak2/STAT and PI3K/AKT pathways. This increases cancer cell survival, proliferation, and resistance to apoptosis.

4

Pancreatic Cancer

Pancreatic cancer is notoriously difficult to treat, but prolactin signaling has emerged as a novel target. Studies suggest that prolactin promotes the survival of pancreatic cancer cells through the PRLR-Jak2-STAT axis, aiding in tumor growth and chemoresistance.

5

Colorectal Cancer:

Research has shown that PRLR is upregulated in colorectal cancer, contributing to cancer cell growth and survival. Prolactin may interact with other growth factors to enhance the malignant potential of colorectal tumors.

Backed by Over 25 Years of Published Research from Leading Institutions in the US, Israel and France

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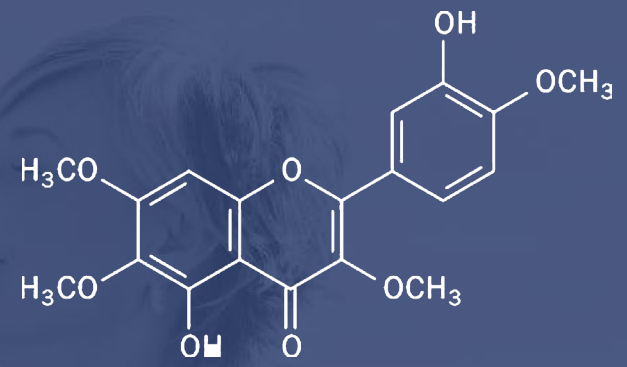
Lead Program Initially Targeting Ovarian Cancer

Novel Biologic that Blocks the Prolactin Receptor to Prevent Cancer Cell Growth Signals and Incite Autophagy

- Novel formulation of de-risked asset, KAD101, which has seen promising initial human clinical data
- New Patents filed to secure our future
- 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
- G129R prolactin 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

Novel mechanism significantly weakens the tumor, and in some cases leads to complete remission

Activates Autophagy

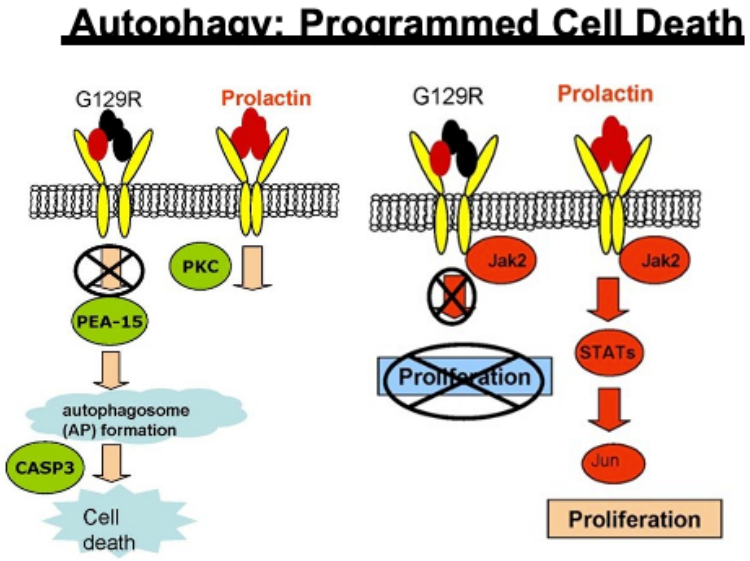
- Triggers cell 'self-eating' process, leading to the death of cancer cells

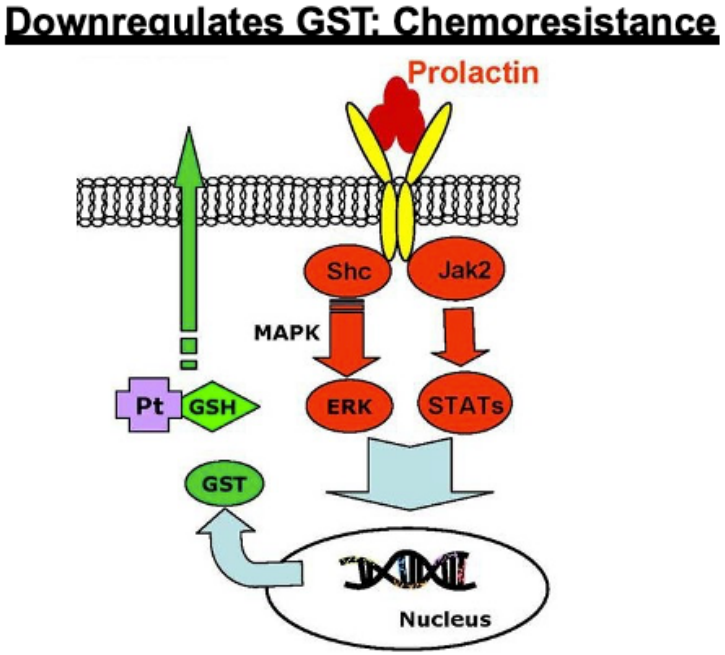
Cell Proliferation Halted

- 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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KAD101 (Originally G129R) Demonstrated Promising Initial Human Clinical Data (daily injectable)

All Patients Showed Tumor Reduction with a Clean Safety Profile as a Daily Injectable
A low toxicity with the tested doses in prior Clinical Trials for Ovarian Cancer!

Patient 1

- Received Taxol with Neulasta
- Had 2 significant RECIST-measured tumors
- Stable disease achieved; no new cancer growths observed
- Treatment with low-dose KAD101 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
- Demonstrated significant response to low-dose KAD101

Patient 3

- Prior treatments: Gemzar, Carboplatin, Doxil, Avastin
- Started the three RECIST-measured tumors
- Post-treatment, tumors reduced by 15-20% in size
- KAD101 showed a marked reduction in tumor volume

Kaida Improved the Formulation to Achieve a Longer Half-Life that will Decrease the Number of Injections and Should Provide Improved Activity and Efficacy in the Patient

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Data Published in Prestigious Journals

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Cell Reports

A Cell Press open access journal

Antagonism of Tumoral Prolactin Receptor Promotes Autophagy-Related Cell Death

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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,

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MOLECULAR CANCER THERAPEUTICS

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

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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^{RAS}; 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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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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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

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Accomplished & Upcoming Milestones

COMPLETED

- PK and Preclinical Translational Studies
- RE-Formulation for KAD101 (new patent)
- Regulatory Path forward implemented FDA
- Patents filed with further IP filings
- SAB in place with Dr Anil Sood from MD Anderson

UPCOMING*

- Commence Manufacturing of KAD-101 with US Based CDMO
- Re-open Existing IND. Securing Type C development meeting
- Complete animal "bridging" studies for the new formulation-
- G129R (KAD101) drug substance toxicity studies completed
  Sponsored Research Agreement with MD Anderson
- Expanding IP portfolio with additional patent filings
- Target Commencement of Phase 1 H1 2026

* And Use of Proceeds

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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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The Kaida Opportunity

Dedicated to advancing ovarian cancer treatment through innovative drug development

Lead program demonstrated promising initial human clinical data

Rapidly advancing into Phase 1 study in H1 2026

Targeting $6.4 billion[1] market opportunity where current therapies have limited efficacy

KAD 101 leverages autophagy induction to promote cancer cell death, represents a novel and exciting mechanism of action

Represents a novel and exciting MOA that significantly weakens the tumor and in some cases you may see complete remission. When combined with targeted therapeutics we believe we will see Victory!

KAIDA BioPharma

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

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Thank You!

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