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Seeking the First Victory in Ovarian Cancer

November 2024

Investor Pitch Presentation

Forward-Looking Statements

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Our Mission

We are advancing targeted anti-cancer hormonal therapies to address the root cause of cancers affecting women.

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Investment 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 not the problem

- Lead program demonstrated promising initial human clinical data
- Targeting prolactin; over expressed in ~80% of patients with ovarian cancer[1]
- Rapidly advancing into Phase 1 study in 2025
- Potential for Orphan Drug Designation with 7-years market exclusivity in US
- 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.

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

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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

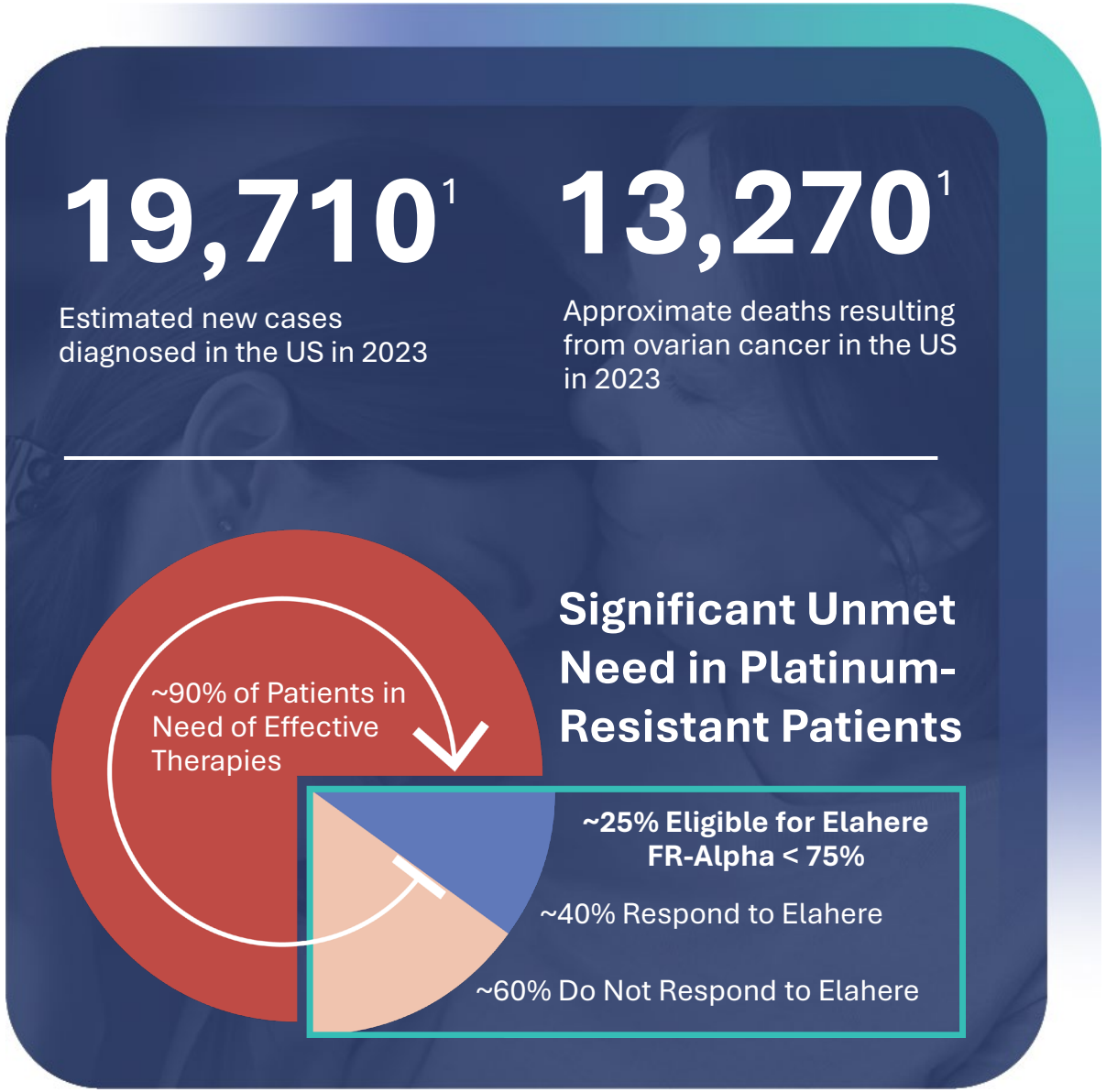
Typically identified when already late stage making treatment difficult and costly. Our focus is to target the cause that created the cancer.

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

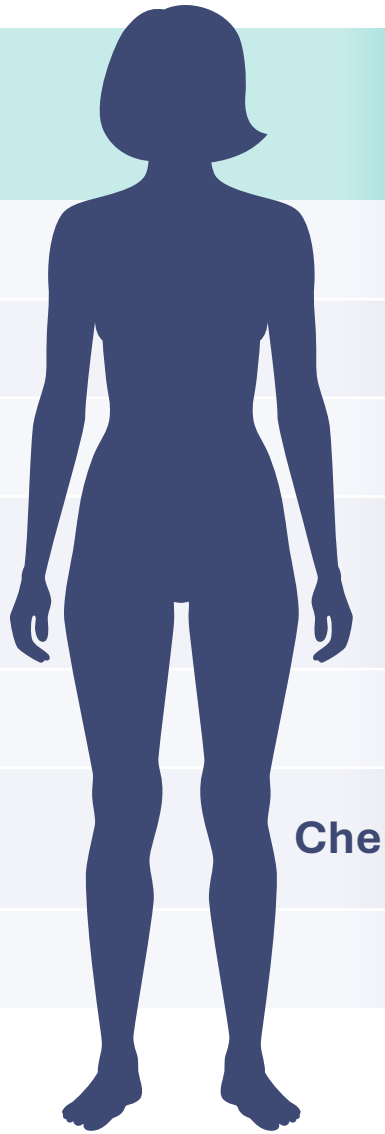


1. Research & Markets Ovarian Cancer drugs global Market Report 2023

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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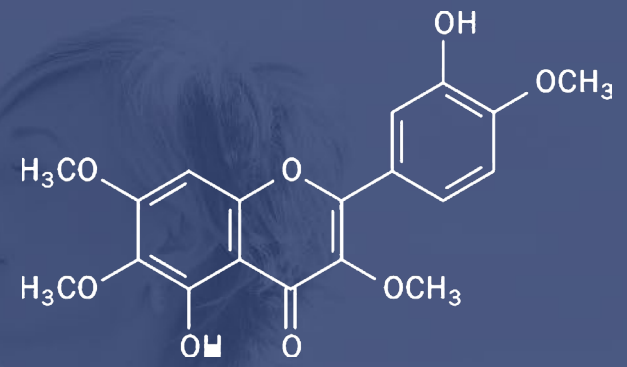
Lead Program Initially Targeting Ovarian Cancer

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
- 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

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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Unique 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

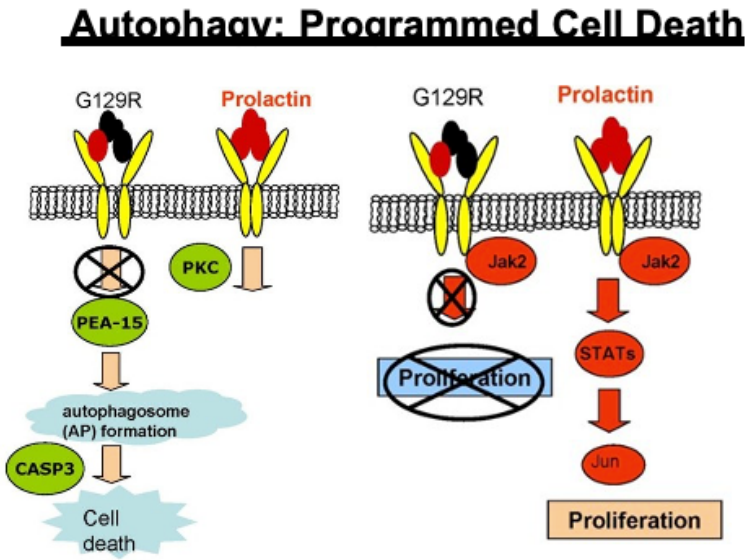
- Triggers cell 'self-eating' process, leading to the death of cancer cells

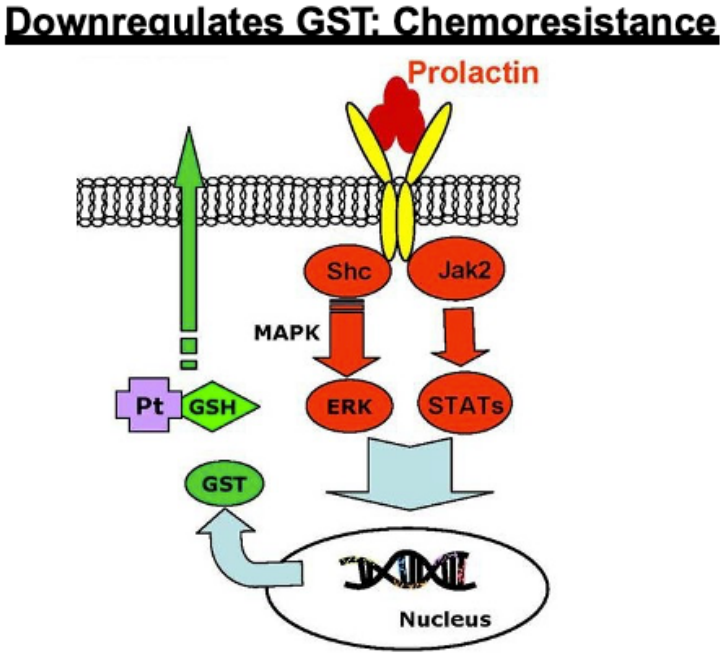
Cell Proliferation Halted

- 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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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 First in Low Dosing Toxicity 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 Decreases Injections and Should Provide Improved Activity and Efficacy in the Patient

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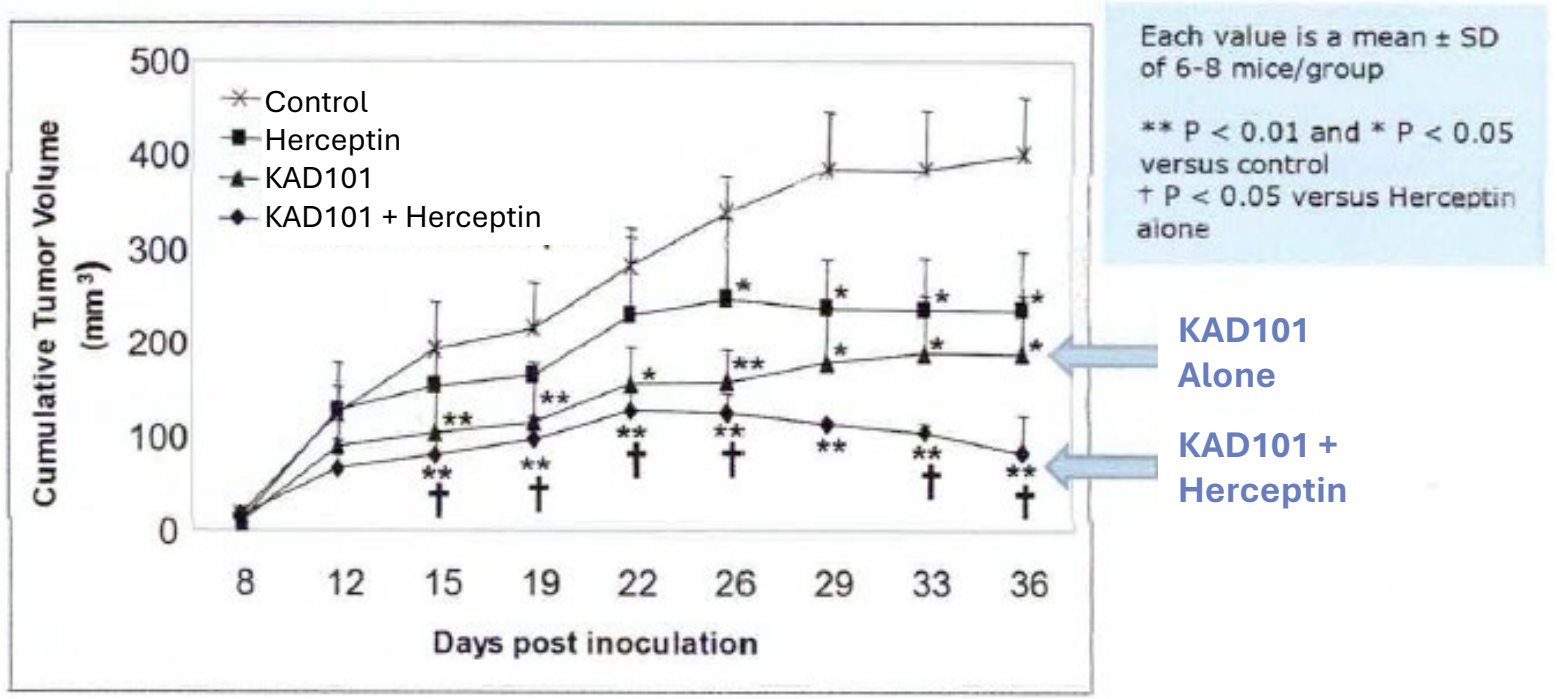
Demonstrated Significant Tumor Reduction

Further Validates Homological Approach to Tumor Reduction

KAD101 Demonstrated Synergistic Effect with Herceptin in Breast Cancer Models



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Development Pipeline

Program	Indication	Discovery	Preclinical	Phase 1	Phase 2	Highlights
KAD101	Ovarian Cancer					New formulation Rapidly advancing into Phase 1 study targeted for 2025
KAD102	Uterine Cancer					Enhanced pure antagonist of KAD101 New molecular entity entering an SRA with MD Anderson in 2024

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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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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,

MD Anderson Cancer Network

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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Mol Cancer Ther 2020;19:1943–54

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

AACR American Association for Cancer Research

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

blood

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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 2025

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

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

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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.
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	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.