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# Seeking the First Victory Against Ovarian Cancer

September 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.
- 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 cancer, and Uterine. Prolactin over expressed in ~90% of patients

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# Leadership Team with Proven Track Record

## Dr. Stella Vnook, MBA

**Co-Founder, CEO-Elect**

*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 FOLEY & LARDNER LLP DLA PIPER

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# Novel Pipeline Targeting Ovarian Cancer

| Program | Indication | Discovery | Preclinical | Phase 1 | Phase 2 | Highlights |
|---|---|---|---|---|---|---|
| **KAD101** | Ovarian Cancer | | | | | New formulation<br>Rapidly advancing into Phase 1 study targeted for 2025 |
| **KAD102** | Uterine Cancer | | | | | Enhanced pure antagonist of KAD101<br>New Molecular Entity Entering into an SRA with MD Anderson in 2024 |

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# Ovarian Cancer

## *Patient Journey is Grim & Needs a Solution to Improve Outcome*

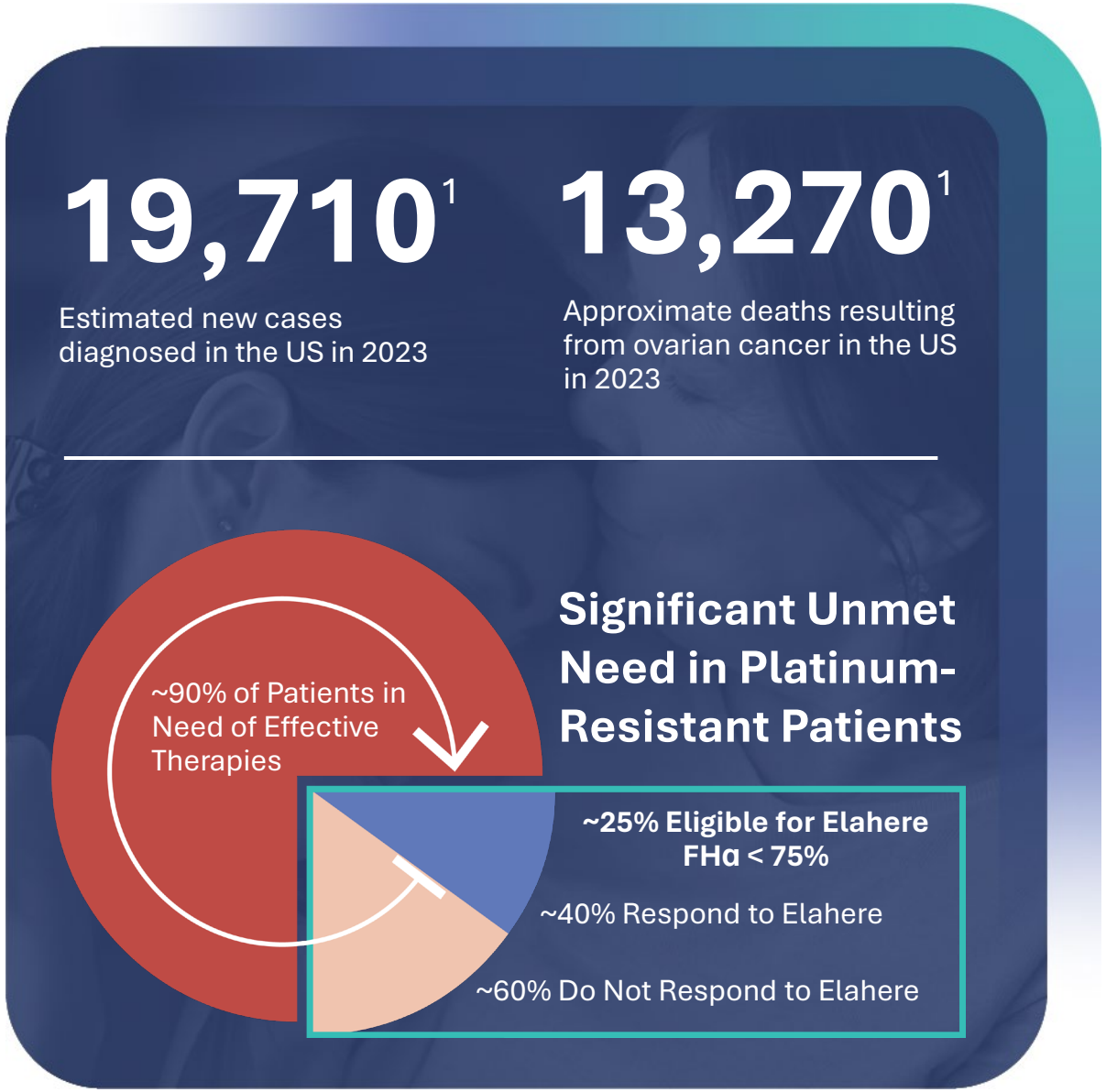
*Typically identified when already late stage making treatment difficult and costly. Our focus is to target the cause that created the cancer.*

### 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

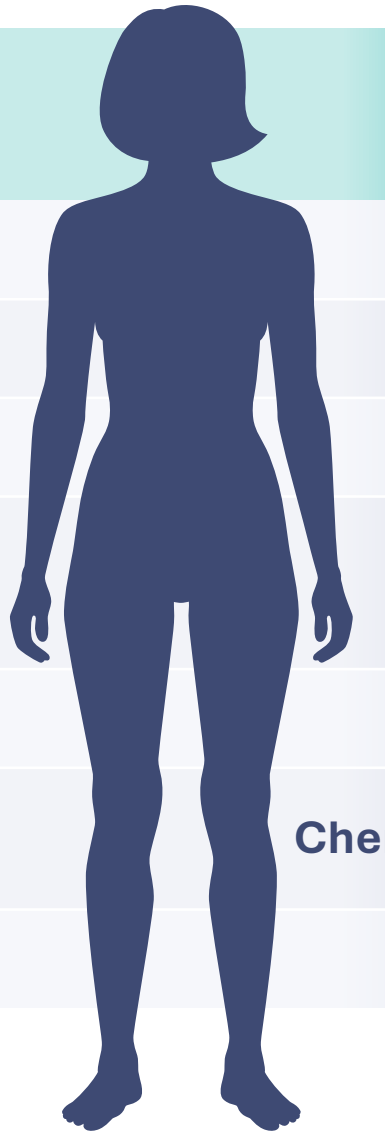



1. Research & Markets Ovarian Cancer drugs global Market Report 2023

# 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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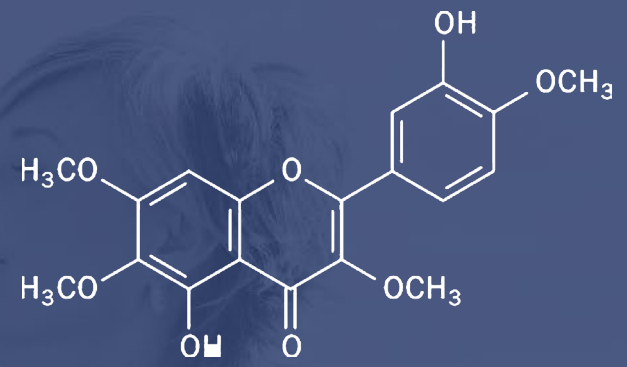
# Lead Program Initially Targeting Ovarian Cancer

*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

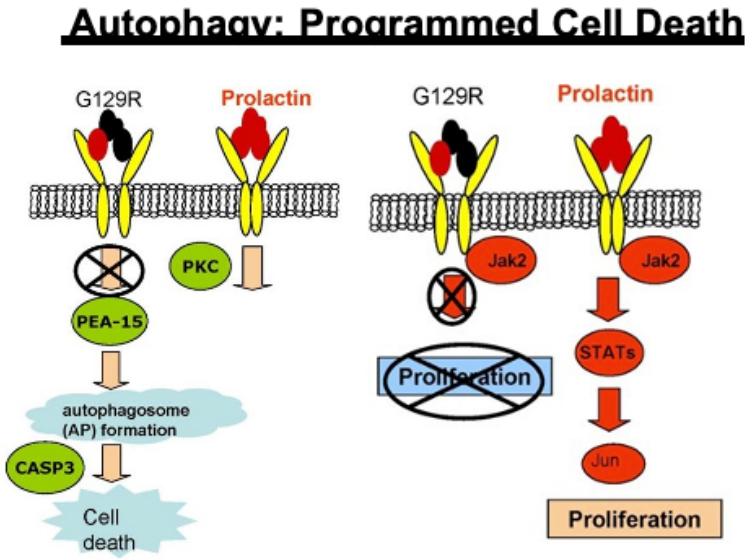
- Triggers cell 'self-eating' process, leading to the death of cancer cells

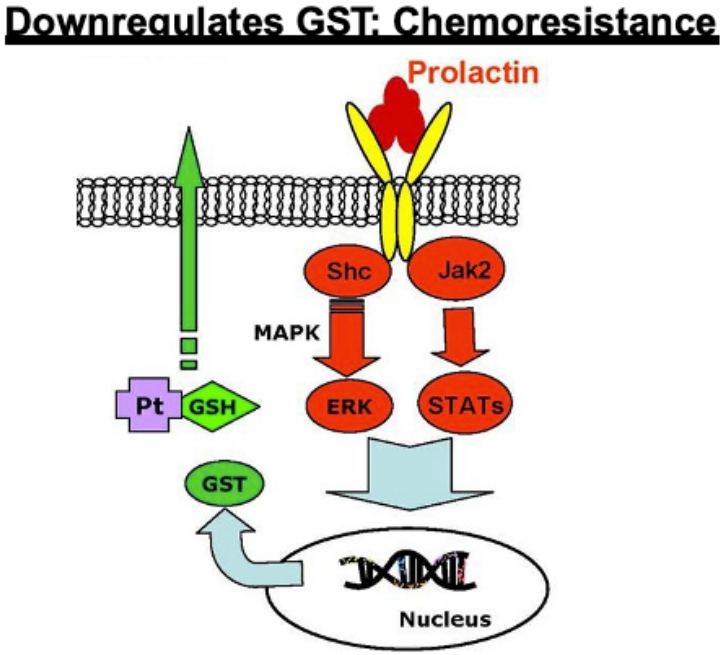
## Cell Proliferation Halted

- 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

Improved Formulation with Longer Half-Life Decreases Injections and Should Offer Improved Activity and Efficacy

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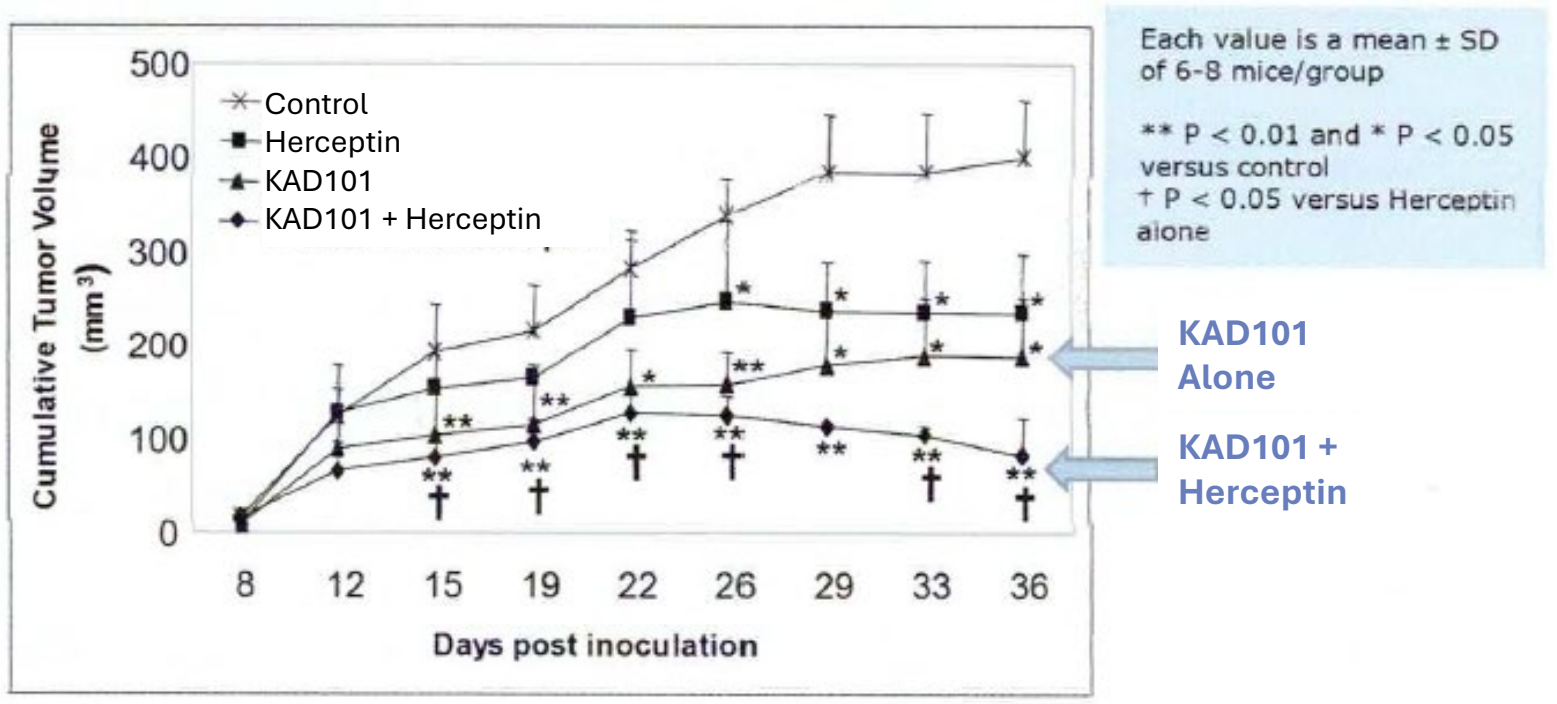
# Demonstrated Significant Tumor Reduction

*KAD101 Demonstrated Synergistic Effect with Herceptin in Breast Cancer Models*




*Further Validates Homological Approach to Tumor Reduction*

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# Data Published in Prestigious Journals


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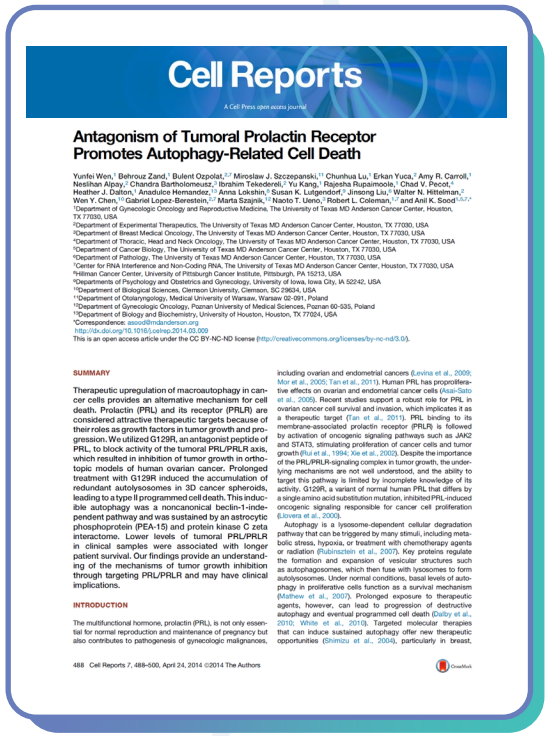

Cell Reports

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., 2006). 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 (Liu 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 (Rabinowitz 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 (Daltoy 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 $p38^{MAPK}$/$p120^{ctn1}$, 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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AACR American Association for Cancer Research

**HHS Public Access**

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**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) contribute 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 TCGA and 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 to 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. Besides, our results showed that G129R induced sub-$G_0$ population, decreased nascent protein synthesis, and initiated FOXO3a/



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Conception and design: Wen Y, Sood AK

Analysis and interpretation of data (e.g., statistical analysis, biostatistics, computational analysis): Wen Y, Wang Y, Baggerly KA

Development of methodology: Wen Y, Sood AK

Acquisition of data (provided patients samples and animals, acquired and managed cell lines, etc.): Wen Y, Wang Y, Chelariu-Raicu A, Stur E, Liu Y, Corvigno S, Bartsch F, Redfern L, Zand B, Kang Y

Writing, review, and/or revision of the manuscript: Wen Y, Sood AK

Technical, resources, or material support: Yunfei Wen, Ph.D. and Anil K. Sood, M.D.

**Disclosure of Potential Conflicts of Interest:** AKS: Consulting (Merck, Kiyatec); shareholder (BioPath); research funding (M-Trap). The remaining authors declare no potential conflicts of interest.


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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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# Investment Summary

*Rapidly Advancing Toward Phase 1 Clinical Study in Ovarian Cancer*

- Pre-IND meeting with FDA to solidify regulatory pathway
- Demonstrated encouraging results in human clinical study with daily injectable
- Targeting prolactin; over expressed in ~80% of patients with ovarian cancer. We see this as the cause for women's cancer
- Opportunity to expand pipeline into other gynecologic cancers, including breast

**Recent Acquisitions and Peer Valuations Highlight Potential for Significant Upside**

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