



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

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

January 2026
Investor Presentation



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Our Mission

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

Highlights

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

90% of patients are in need for an effective treatment

Targeting Primary Cause of Hormone-Driven Cancers: Prolactin

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

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

Kaida Management Team and Advisors

Proven leadership with drug development and business experience...

Dr. Stella Vnook

Chairperson, Co-Founder

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



Craig Pierson

Director, 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



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*

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

Internationally Recognized KOL in Gynecologic Oncology and Cancer Biology



Anil K. Sood, M.D.

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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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¹⁴http://dx.doi.org/10.1016/j.celrep.2014.03.029
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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 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 autophagosomes in 3D cancer spheroids, leading to a type II programmed cell death. This inducible autophagy was a mitochondrial β-actin-dependent pathway and was sustained by astrocytic phosphoprotein (PPA-15) and protein kinase C-β2a interference. 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 malignancies.

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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 preferentially 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^{+/+} and PTEN^{-/-} 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 proteomic 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-G1 population arrest, decreased nascent protein synthesis, and initiated FOXO3a/EIF-4EBP1-mediated cell death in both PTEN^{+/+} and PTEN^{-/-} 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 (3–5). Extrapancreatic PRL plays key regulatory roles during the development and progression of endometrial (1), as the production of PRL by the endometrium is elevated during the normal menstrual cycle (6). 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 multiplicity by blocking autocrine/paracrine PRL activity (6, 7), the full-length PRLR gene product was not detected in endometrial tissues (8). Our results from screening an array of human uterine cancer cells indicated that transcriptionally spliced isoforms of PRLR products might be responsible for mediating activities of the tumoral PRL/PRLR axis. However, the expression and functional mechanisms of different PRLR isoforms remain unclear. PRL/PRLR axis is reportedly involved in multiple signaling pathways (e.g., activation of p39^{+/+}p120^{+/+}, refs. 9–11, Stat family members and JAK2, refs. 12–14, GPCR 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 PRL gene. Eight of the nine isoforms are transcribed into cell-associated PRLR isoforms, while the other is a monocytic transcript variant. Structurally, the extracellular ligand-binding domains are highly conserved and retain PRL-binding activity, while the transmembrane-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 (IF; 60–70 kDa), and short form (SF; 45–50 kDa). PRLR_LF is transcribed from exons 3–10 (21) and PRLR_IF (60–70 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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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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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

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 Q1 2027 |
| KAD102 | Uterine Cancer |  | | | | New molecular entity – prolactin antagonist with improved half-life and bioavailability SRA with MDACC (2026) |

Ovarian Cancer

Patient Journey is Grim & Needs a Solution to Improve Outcomes

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

Market Opportunity¹

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

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

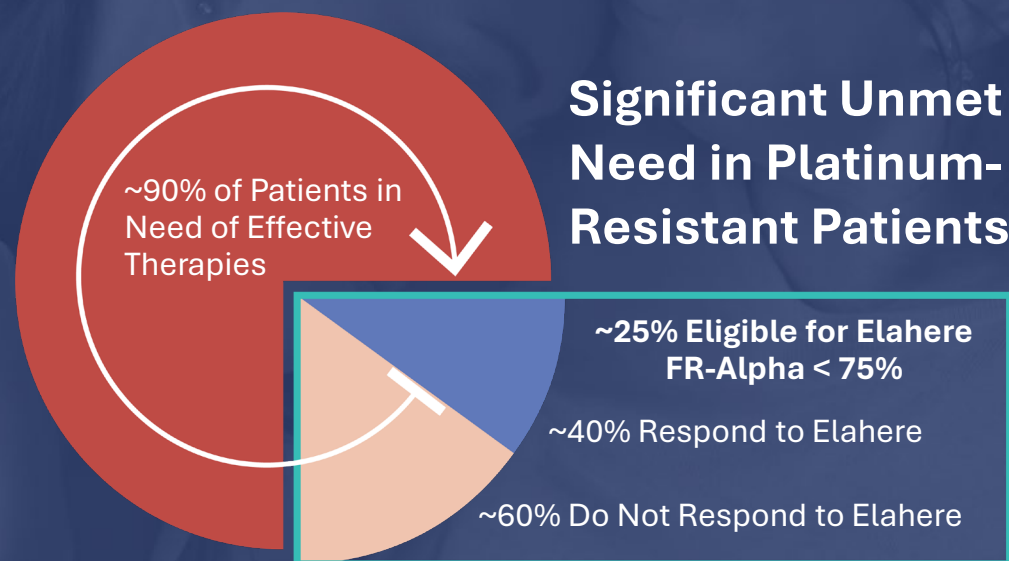
0 Therapeutics that offer a durable response

19,710¹

Estimated new cases diagnosed in the US in 2023

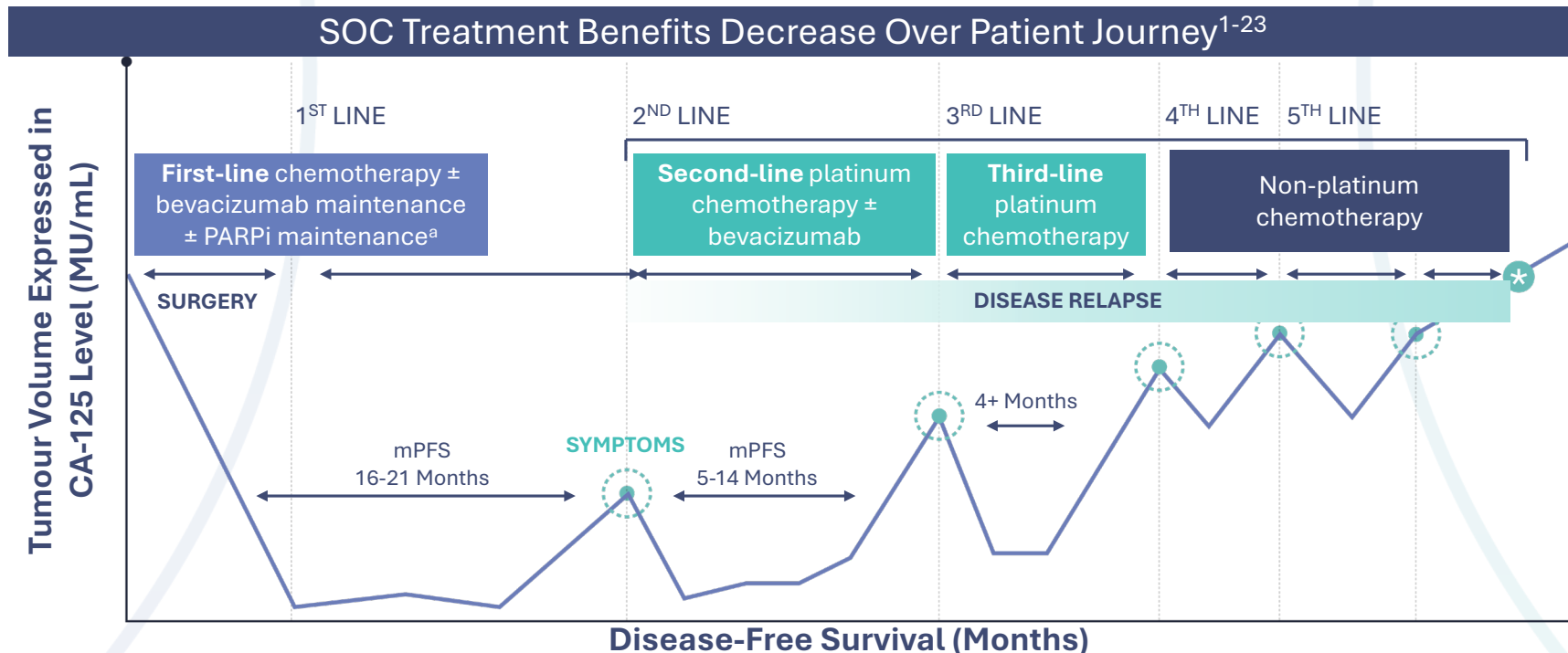
13,270¹

Approximate deaths resulting from ovarian cancer in the US in 2023



Competitive Landscape

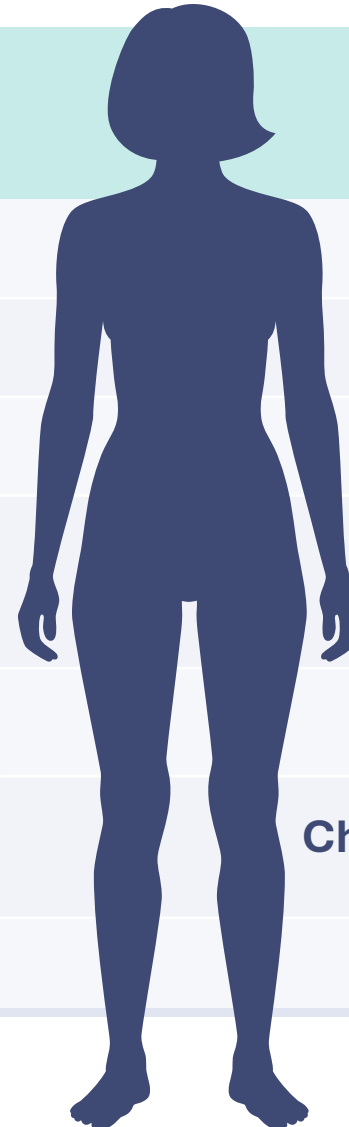
- **Standard of care**, predominantly chemotherapy-based across all 3 lines, **is ineffective** as up to **80% of patients with adv. disease will relapse within 12-18 months**
- Recent approvals **focused on patient subpopulations**:
 - Verstem's avutometinib + defactinib tablets (2025): KRAS-mutated recurrent low-grade serous ovarian cancer (LGSOC) who have received prior systemic therapy
 - Immunogen (AbbVie acq. for \$10.1bn in 2024) Elahere (2024): FRα +, platinum-res. epithelial ovarian, fallopian tube, or primary peritoneal cancer, post 1 to 3 systemic therapies



Consistently Poor Results Across Therapies

Underpins Need for New Innovative Approach

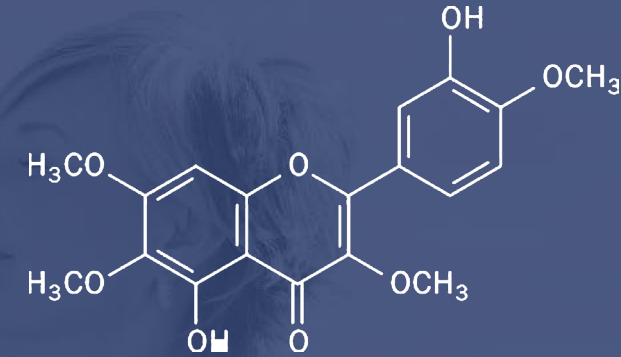
| Drug | Target | % of Patients Expression | ORR | mPFS (mo) | mOS (mo) |
|------------------------|------------------------------|--------------------------|--------------------------|-----------------|------------------|
| KAD101 KAD102 | Prolactin | ~80% | KAIDA Opportunity | | |
| Abbvie: Elahere | FR-Alpha $\geq 75\%$ | ~25-30% | 42% | 5.6 | 16.5 |
| Sutro: Luvelta | FR-Alpha $\geq 25\%$ | ~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 | ~3.5 Average | ~13.4 Average |
| Mersana: Upfitamab | NaPi2b | ~50% | 13% | Study Failed | |



KAD101 Initially Targeting Ovarian Cancer

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

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



Prolactin

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

Targeting Prolactin

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

Impact on Cell Signaling

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

Chemotherapy Resistance

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

KAD101: Best-in-Class Prolactin Antagonist

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

Activates Autophagy

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

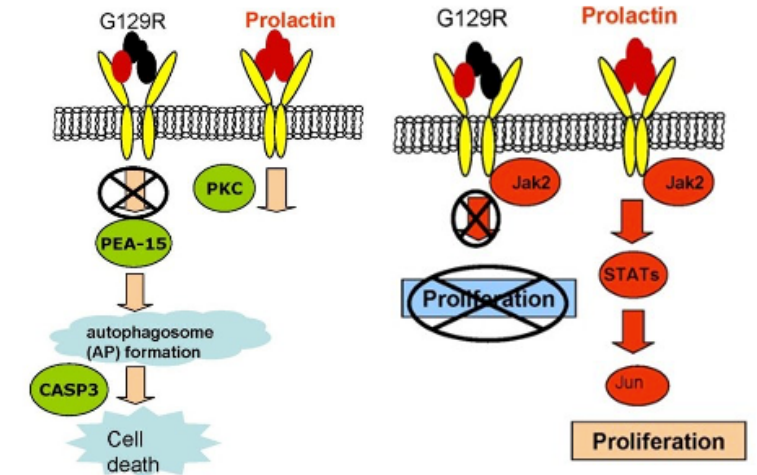
Cell Proliferation Halted

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

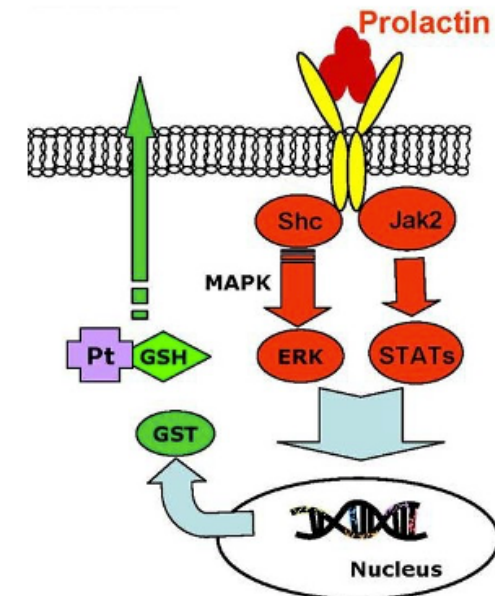
Chemoresistance Addressed

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

Autophagy: Programmed Cell Death



Downregulates GST: Chemoresistance



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

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

Patient 1

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

Patient 2

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

Patient 3

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

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

Notes:

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

Accomplished & Upcoming Milestones

Completed

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














Upcoming Milestones and Events


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



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

| Company | Drug | Phase | ORR | mPFS (mo) | mOS (mo) | Deal Price / Market Cap |
|---|--|-----------|------|---------------|----------|--|
| Acquisitions | | | | | | |
|  immunogen |  ELAHERE [®] nivolumab soravictasin-gyn injection 400 mg | Approved | 42% | 5.6 | 16.5 | \$10.1 Billion Acquired by  abbvie |
|  ProfoundBio | Rinatabart sesutecan: FR-alpha ADC | Phase 1/2 | NA | NA | NA | \$1.8 Billion Acquired by  Genmab |
| Public Companies | | | | | | |
|  SUTRO BIOPHARMA | Luvelta | Phase 2/3 | ~38% | NA | NA | \$350 Million |
|  Corcept THERAPEUTICS | Relacorilant | Phase 2 | 33% | 5.6 | 13.9 | \$2.8 Billion |
|  zentalis [®] | Azenosertib | Phase 1/2 | | Study Ongoing | | \$825 Million |
|  Nuvation Bio | NUV-1511 | Phase 1/2 | | Study Ongoing | | \$825 Million |
|  SHATTUCK LABS | SL-172154 | Phase 1 | 9% | - | - | \$500 Million |
|  MACROGENICS | MGC026 | Phase 1 | | Study Ongoing | | \$280 Million |

Recent Private Financings Underscores Interest and Value in Ovarian Cancer Space

| Company | Drug | Phase | Last Round | Total Raise | Total Raise to Date |
|---|--------------------------|---------|------------|---------------|---------------------|
|  TORL BIOTHERAPEUTICS | TORL-1-23: Claudin-6 ADC | Phase 1 | Series B | \$158 Million | \$350 |

Highlights

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

90% of patients are in need for an effective treatment

Targeting Primary Cause of Hormone-Driven Cancers: Prolactin

- KAD101 demonstrated promising initial human clinical data
- Leadership has extensive preclinical, clinical, and drug development experience/expertise to execute clinical development plan
- Targeting prolactin; over expressed in ~80% of patients with ovarian cancer¹
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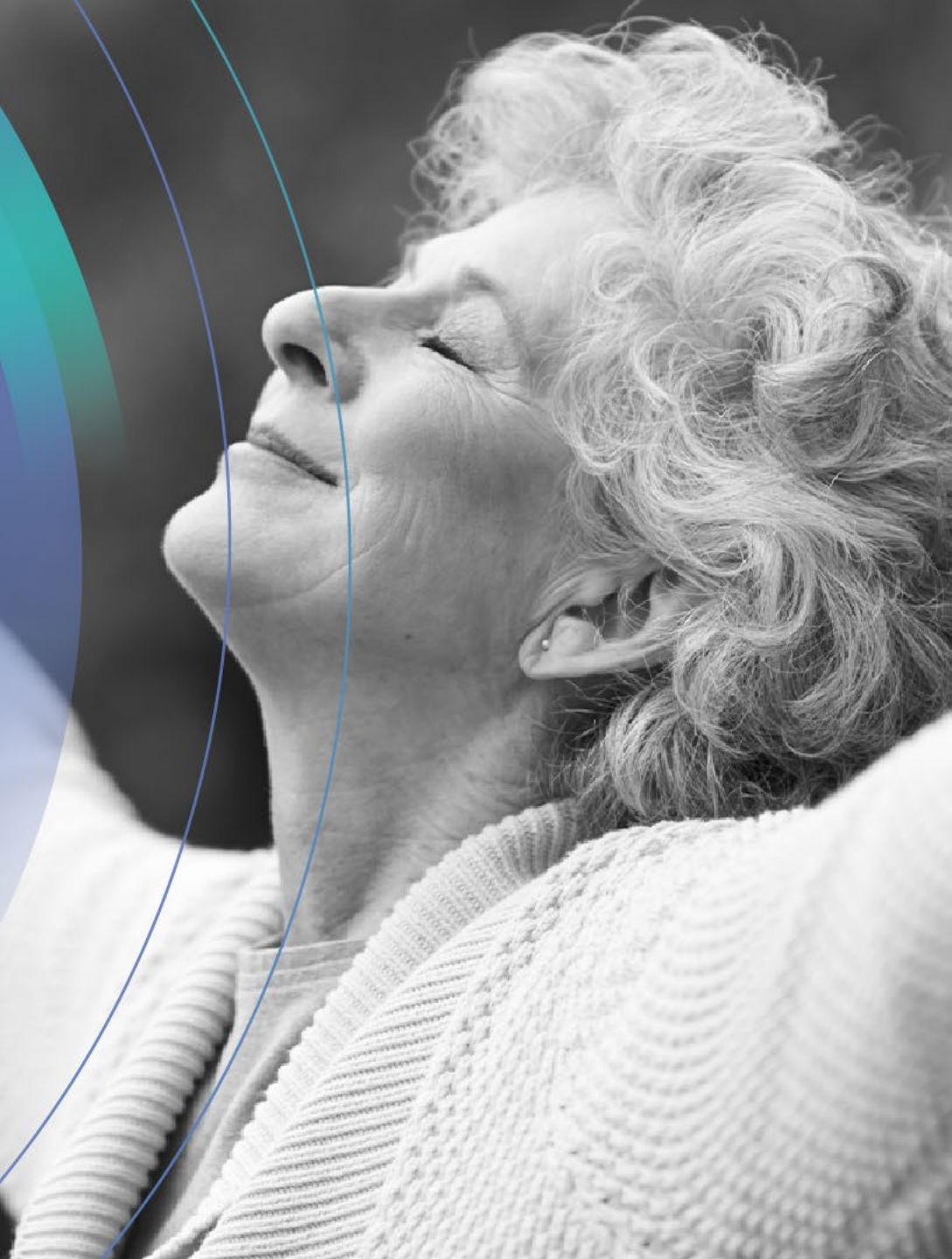
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KAIDA
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Thank You!

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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. |