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Seeking the First Victory Against Ovarian Cancer

August 2024

Corporate Presentation

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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

- 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 Q4 2024
- Potential for Orphan Drug Designation with 7-years market exclusivity in US
- Pipeline expansion opportunity into breast cancer, 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 · OralBiolife · 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

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Novel Pipeline Targeting Ovarian Cancer

Program	Indication	Discovery	Preclinical	Phase 1	Phase 2	Highlights
KAD101	Ovarian Cancer					New formulation Rapidly advancing into Phase 1 study in Q4 2024
KAD102	Uterine Cancer					Enhanced pure antagonist of KAD101 New chemical entity

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Ovarian Cancer

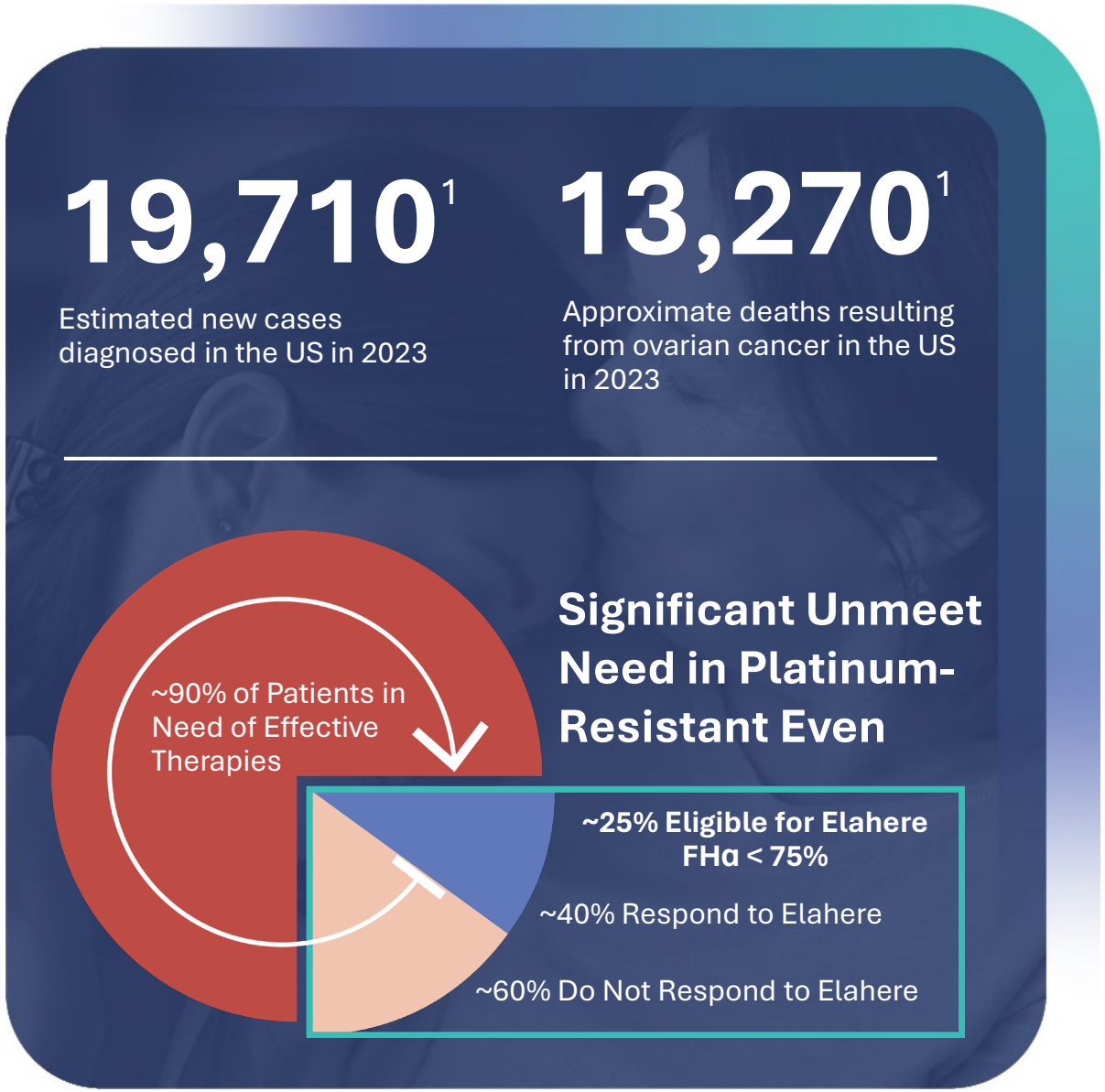
A malignant tumor that originates in the ovaries and is often detected at an advanced stage due to its subtle early symptoms.

Market Opportunity[1]

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

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

0 Therapeutics that offer a durable response



1. Research & Markets Ovarian Cancer drugs global Market Report 2023
2. *Ovarian cancer survival rates: Ovarian cancer prognosis*. Ovarian Cancer Survival Rates | Ovarian Cancer Prognosis | American Cancer Society. (n.d.). https://www.cancer.org/cancer/types/ovarian-cancer/detection-diagnosis-staging/survival-rates.html

We Believe We Need to Link Ovarian Cancer to Prolactin to KAD101: If A=B and B=C then A=C

Research on Prolactin and Ovarian Cancer:

1. **Prolactin as a Biomarker**: Studies have shown that prolactin, a hormone primarily associated with lactation, can act as a biomarker in various cancers, including ovarian cancer. Elevated levels of prolactin have been found in ovarian cancer patients, suggesting a potential link between prolactin signaling and tumor progression .
2. **Prolactin Receptors**: Ovarian cancer cells often express prolactin receptors. The interaction between prolactin and its receptors can promote cell proliferation and survival, contributing to tumor growth and metastasis .
3. **Mechanism of Action**: Prolactin can activate several signaling pathways such as the JAK2/STAT5, PI3K/AKT, and MAPK pathways, which are involved in cell growth, differentiation, and survival. These pathways are often dysregulated in cancer, including ovarian cancer .

- **Therapeutic Implications of KAD101:**

1. **Regulating Prolactin Levels**: KAD101, a therapeutic agent, could potentially be designed to regulate prolactin levels or block prolactin receptors. By doing so, it could inhibit the prolactin-induced signaling pathways that contribute to ovarian cancer progression.
2. **Clinical Trials and Evidence**: Preclinical studies and early-phase clinical trials would be necessary to evaluate the efficacy of KAD101 in reducing prolactin levels or blocking its receptors, thereby inhibiting ovarian cancer cell growth. Evidence from similar therapeutic strategies in other cancers can provide a rationale for this approach.
3. **Potential Benefits**: Regulating prolactin with KAD101 could slow down tumor growth, reduce metastasis, and improve survival rates in ovarian cancer patients. It might also enhance the effectiveness of existing treatments when used in combination.

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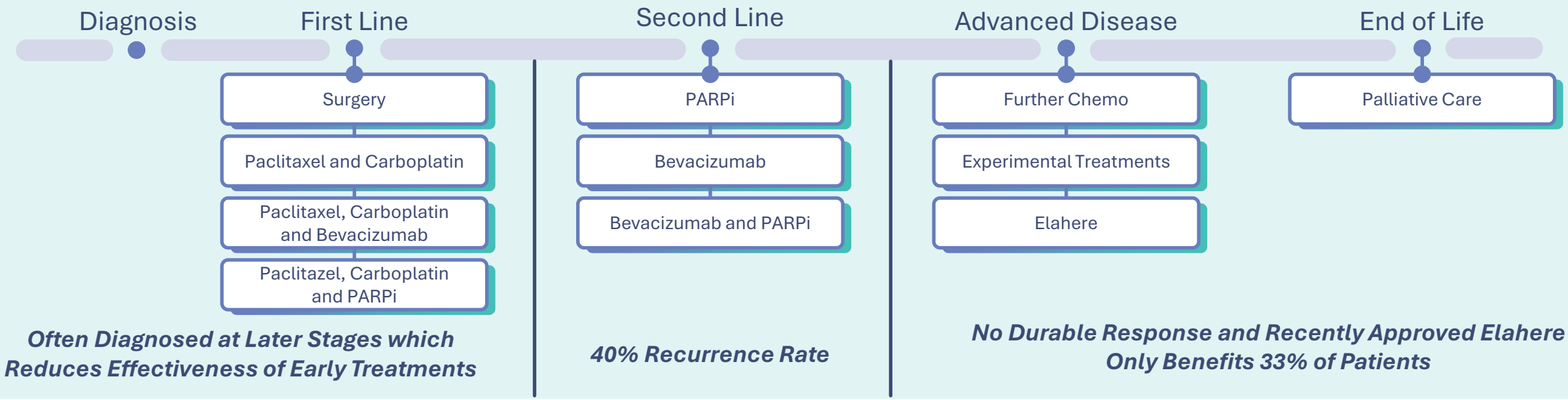
Patient Journey is Grim

Disease Progression vs. Diagnosis: 5-Year Relative Survival Rates[1]

- Localized: 93% 5-Year Survival
  - **~15% Rate of Diagnosis**
- Regional: 75% 5-Year Survival
  - **~20% Rate of Diagnosis**
- Distant: 31% 5-Year Survival
  - **~60% Rate of Diagnosis**

Significant Unmet Need for Innovative Treatments[1]



Often Diagnosed at Later Stages which Reduces Effectiveness of Early Treatments

40% Recurrence Rate

No Durable Response and Recently Approved Elahere Only Benefits 33% of Patients

Benchmark for Overall Survival with a Chemotherapy is ~12-13 Months

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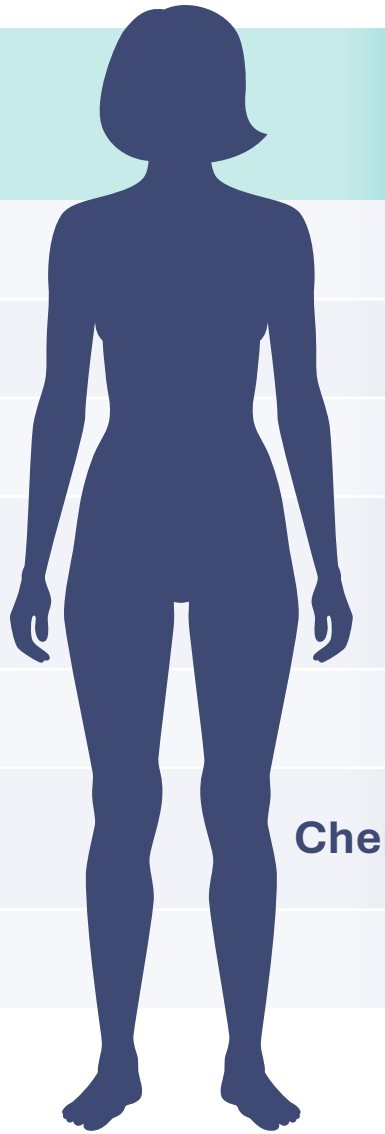
1. *Ovarian cancer survival rates: Ovarian cancer prognosis*. Ovarian Cancer Survival Rates | Ovarian Cancer Prognosis | American Cancer Society. (n.d.). https://www.cancer.org/cancer/types/ovarian-cancer/detection-diagnosis-staging/survival-rates.html

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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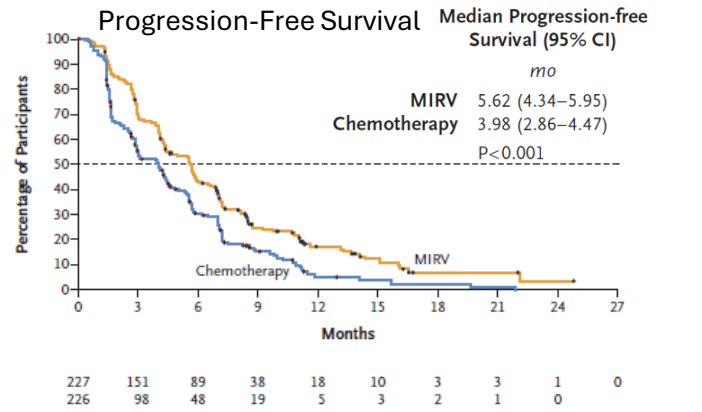
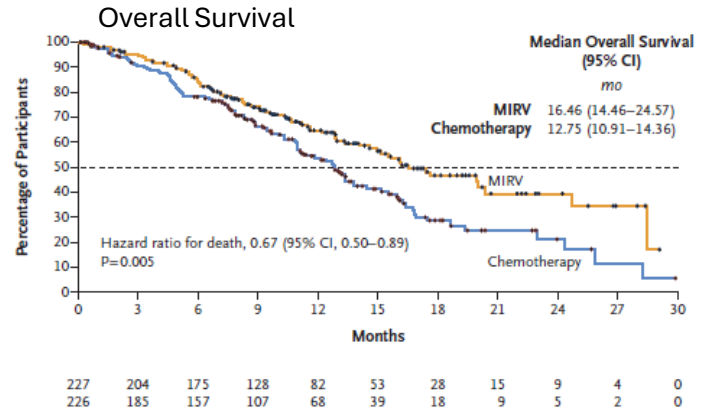
Recently Approved Elahere Sets a Low Bar for Approval While Realizing High-Value Exit

Antibody-Drug Conjugate (ADC) Approved for Platinum-Resistant Ovarian Cancer





$10.1 Billion Acquisition

immunogen → abbvie

- Only approved for ~33% of patients
- ~1.6-month improvement in progression-free survival
- ~3.7-month improvement in overall survival
- Significant ocular toxicity

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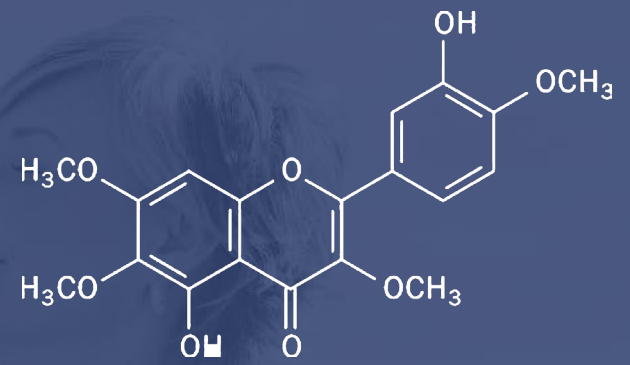
Lead Programs Initially Targeting Ovarian Cancer

Modified Prolactin that Blocks the Prolactin Receptor to Prevent Cancer Cell Growth Signals

- Novel formulation of de-risked asset, KAD101, which has promising initial human clinical data
- Multiple expansion opportunities into endometrial and breast cancers



Prolactin

Higher Expression Correlates with Reduced Survival

Elevated Prolactin's Role

- Contributes to tumor growth and gynecologic malignancies' development

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

New formulation KAD101 is a modified prolactin that blocks the receptor to prevent cancer cell growth signals

Activates Autophagy

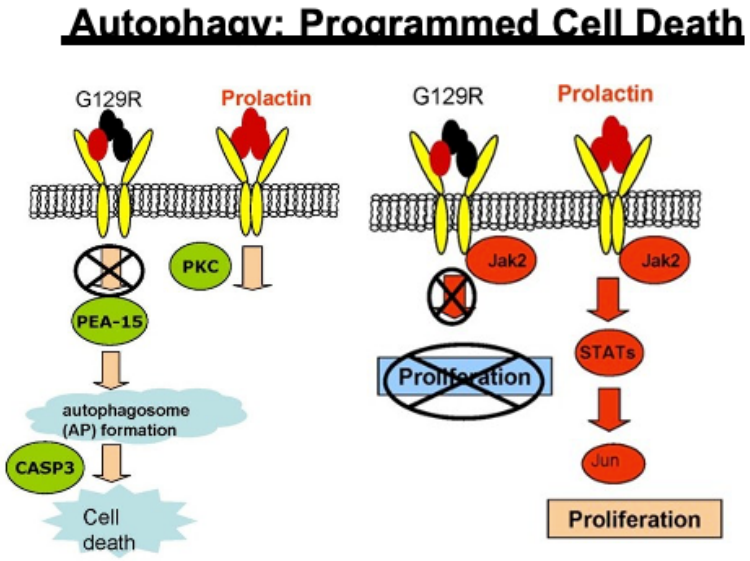
- Triggers cell 'self-eating' process, leading to the death of cancer cells

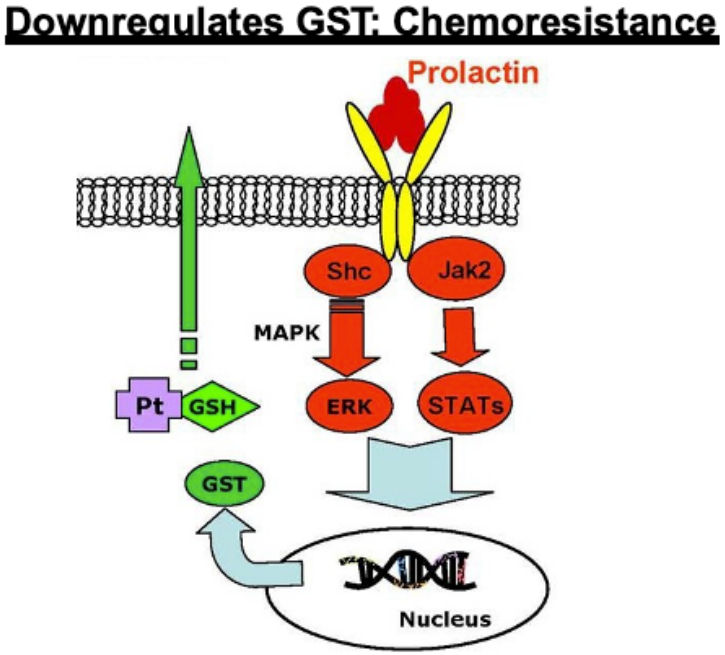
Cell Proliferation Halted

- 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 Demonstrated Promising Initial Human Clinical Data

All Patients Showed Tumor Reduction with Clean Safety Profile
A First in Low Dosing Toxicity Clinical Trials for Ovarian

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

Reintroducing the Improved Formulation Should Offer Enhanced Activity and Efficacy

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Rapidly Advancing Towards Phase 1 Study

Expect to File IND and Enter Phase 1 in Q1 2025

- Engaging with clinical experts
- Finalizing trial design
- Planning Pre-IND meeting with FDA

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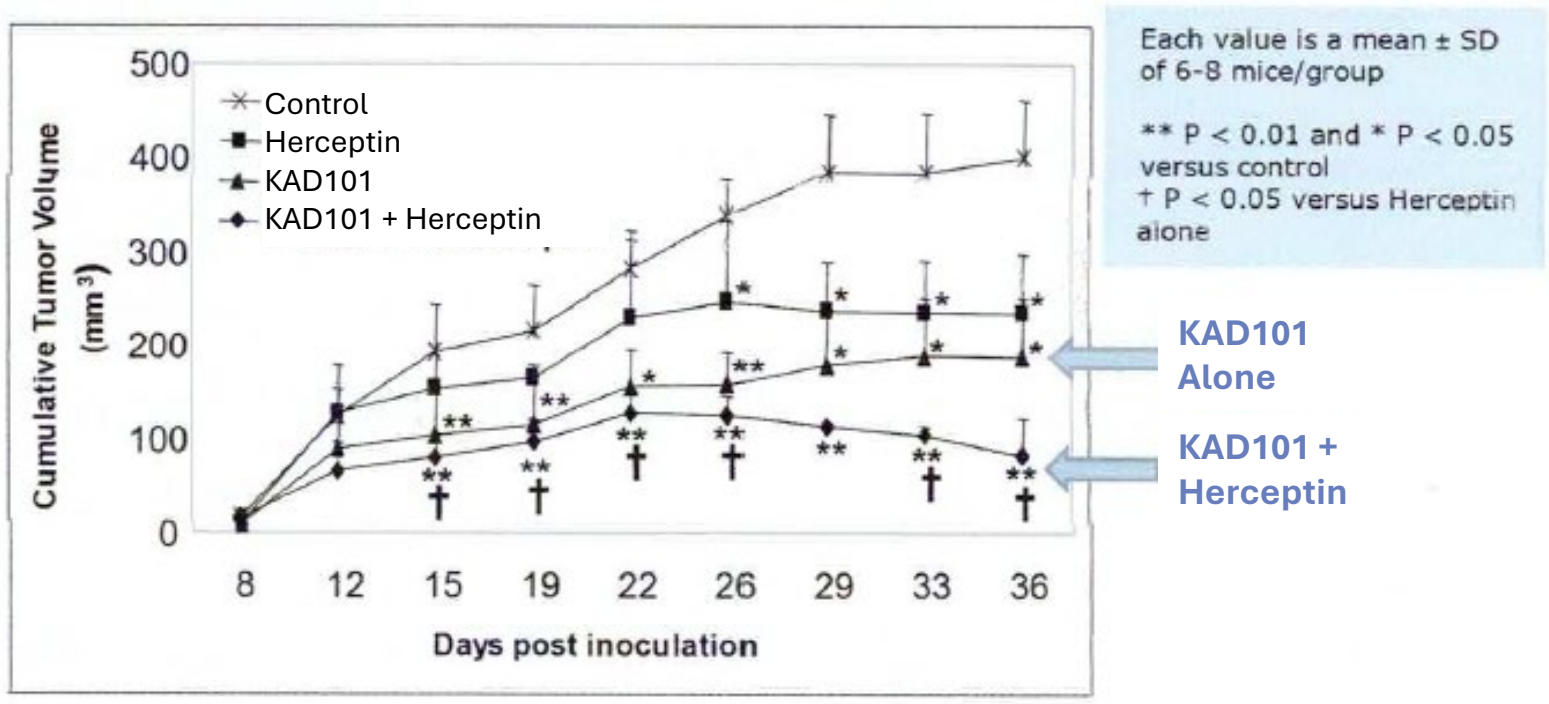
Expansion Opportunity Into Breast Cancer

KAD101 Demonstrated Synergistic Effect with Herceptin in Breast Cancer Models



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Data Published in Prestigious Journals

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., 2005). 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 (Rui et al., 1994; Xie et al., 2002). Despite the importance of the PRL/PRLR-signaling complex in tumor growth, the underlying mechanisms are not well understood, and the ability to target this pathway is limited by incomplete knowledge of its activity. G129R, a variant of normal human PRL that differs by a single amino acid substitution mutation, inhibited PRL-induced oncogenic signaling responsible for cancer cell proliferation (Llovera et al., 2000).

Autophagy is a lysosome-dependent cellular degradation pathway that can be triggered by many stimuli, including metabolic stress, hypoxia, or treatment with chemotherapy agents or radiation (Rubinsztein et al., 2007). Key proteins regulate the formation and expansion of vesicular structures such as autophagosomes, which then fuse with lysosomes to form autolysosomes. Under normal conditions, basal levels of autophagy in proliferative cells function as a survival mechanism (Mathew et al., 2007). Prolonged exposure to therapeutic agents, however, can lead to progression of destructive autophagy and eventual programmed cell death (Dalby et al., 2010; White et al., 2010). Targeted molecular therapies that can induce sustained autophagy offer new therapeutic opportunities (Shimizu et al., 2004), particularly in breast,

MOLECULAR CANCER THERAPEUTICS

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

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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 $p59^{fyn}$/$p120^{jak2}$; 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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MD Anderson Cancer Network®

AACR American Association for Cancer Research

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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
90% of Patients Need an Effective Therapy

- Demonstrated encouraging results in human clinical study
- Targeting prolactin; over expressed in ~80% of patients with ovarian cancer
- Opportunity to expand pipeline into breast cancer

Recent Acquisitions and Peer Valuations Highlight Potential for Significant Upside

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Appendix

Consistently Poor Results Across Platinum-Resistant Therapies

Company	Trial	Year	Therapy	N	Measurable Disease %)	ORR	mPFS (mo)	mOS (mo)
ImmunoGen	MIRASOL (FRa ≥75%)	2022	IC Chemo	226	100%	16%	4.0	12.8
			MIRV	227	100%	42%	5.6	16.5
Genentech	AURELIA	2011	IC Chemo	182	79%	12%	3.4	13.3
			IC Chemo + Bev	179	80%	27%	6.7	16.6
Chugai Pharmaceutical	JGOG-3018	2020	PLD 50 mg/m2	137	60%	17%	4.0	14.0
			PLD 40 mg/m2	135	58%	13%	4.0	14.0
ImmunoGen	FOWARD 1 (FRa)	2018	IC Chemo	118	100%	10%	4.4	12.0
			MIRV	248	100%	24%	4.1	17.3
Sanofi	CORAIL	2020	lurbinectedin	221	100%	15%	3.5	12.6
			IC Chemo	221	100%	13%	3.6	13.0
Millennium	PRECEDENT	2010	vintafolide + PLD	100	100%	18%	5.0	
			PLD	49	100%	12%	2.7	
Corcept	CORT-P2b	2022	relacorilant + NabPac	118	93%	33%	5.6	13.9
			NabPac	60	88%	32%	3.8	12.2
Sutro Biopharma	STRO-P2	Ongoing	Luvelta	92	100%	38%	-	-
Shattuck Labs	STTK-P2	Ongoing	SL-172154 + PLD	11	100%	27%	-	-
Mersana	MRSN-P2b	FAILED	Upfitamab Rils	268	100%	13%	-	-
PMV Pharmaceuticals	PMVP P1	Completed	rezatapopt	22	91%	32%	-	-
AstraZeneca	JAVELIN-200	2017	avelumab	188	100%	4%	1.9	11.8
			avelumab + PLD	188	100%	13%	3.7	15.7
			PLD	190	100%	4%	3.5	13.1
Merck	KEYNOTE-100	2018	pembro	285	100%	5%	2.1	18.7
			IC Chemo	91	100%	8%	2.1	17.6

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