

FAETH THERAPEUTICS

Inaugural Investor/ Analyst Day

OCTOBER 6, 2026

Forward-Looking Statements

This Presentation contains forward-looking statements of Faeth Therapeutics, Inc. (the “Company”) within the meaning of the federal securities laws. Words such as “may,” “will,” “should,” “could,” “believe,” “expect,” “anticipate,” “intend,” “plan,” “estimate,” “potential” and similar terms identify forward-looking statements. These include statements about: the Company’s cash runway, including its expectation that existing cash, cash equivalents and marketable securities will fund operations through topline data readouts from its ongoing clinical trials; the potential benefits of its product candidates and of multi-node inhibition relative to other agents; the design, timing and results of ongoing and planned preclinical and clinical studies; its regulatory strategy, including the potential design and timing of any registrational trial and the outcome of interactions with the U.S. Food and Drug Administration; potential development in additional indications and in combination with other agents, including oral selective estrogen receptor degraders; the evolution of the treatment landscape and competitive environment, including third-party product candidates; estimated addressable markets and patient populations; and benchmarks the Company considers relevant to its clinical data. These statements reflect management’s current views and are not guarantees of future performance.

Actual results could differ materially from those expressed or implied, as a result of risks including: the Company’s ability to obtain additional capital; whether its clinical trials demonstrate acceptable safety and efficacy, and the risk that interim, preliminary or preclinical data will not be predictive of final or clinical results; delays in the commencement, enrollment or completion of clinical trials; the outcome of interactions with regulatory authorities; reliance on third parties to conduct and supply clinical trials; the size of market opportunities, which are estimates based on assumptions that may prove incorrect; competition; changes in law or regulation; and the other risks described under “Risk Factors” in the Company’s Annual Report on Form 10-K for the year ended December 31, 2025, as amended, its subsequent Quarterly Reports on Form 10-Q and its other filings with the U.S. Securities and Exchange Commission. The Company undertakes no obligation to update forward-looking statements except as required by law.

PIKTOR (serabelisib and sapanisertib) is investigational; its safety and efficacy have not been established and it has not been approved by any regulatory authority. This Presentation includes information from third-party studies, publications and other sources and the Company’s own estimates. The Company believes these sources are reliable but has not independently verified them. Data from different clinical trials are presented for context only: no head-to-head trials have been conducted, trials differ in design, populations and endpoints, cross-trial comparisons must be interpreted with caution, and no conclusions about comparative safety or efficacy should be drawn. Views expressed by the Company’s scientific co-founders and other non-employee speakers are their own.

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PIKTOR – one regimen, multiple high value opportunities

Designed for broad activity across PI3K-mutant and wild-type tumors, with near-term focus on endometrial and breast cancers

Co-founded by Dr. Lewis Cantley - discoverer of the PI3K pathway

Oral, multi-node PI3K/AKT/mTOR (PAM) inhibition - PIKTOR has preliminarily demonstrated activity and tolerability across PAM mutant and wild type tumors

Near term catalysts - endometrial Phase 2 topline expected by EOY 2026; breast Phase 1b/2 ongoing, interim expected 2H 2027

Premier investor support, with ~\$186M in cash to accelerate PIKTOR in EC and BC through proof-of-concept studies¹

¹ ~\$186M cash, cash equivalents and marketable securities as of June 30, 2026
Abbreviations: EC - endometrial cancer, BC - breast cancer

10:00 AM

Introduction

Anand Parikh, Co-founder and CEO

10:15 AM

PAM overview and single-node inhibitor escape

Dr. Marcus Goncalves, Scientific co-founder

10:30 AM

Preclinical and Translational data review

Dr. Oliver Maddocks, Co-founder and CSO

10:45 AM

Clinical

Dr. Debbie Chirnomas, CMO

11:00 AM

Market Opportunity

Brian Stephenson, CFO

11:15 AM

Closing Remarks/Q&A

Focused pipeline of potentially best-in-class product candidates, leveraging Faeth's unique metabolic insights

PIPELINE AND PROGRAM		TARGET POPULATION	PRECLINICAL	PHASE I	PHASE II	PHASE III
PIKTOR	PIK-201	Oncology: Endometrial				
PIKTOR	PIK-101	Oncology: Breast				
SAPANISERTIB	Other Indications	Oncology: Ovarian				
		Oncology: Lung				
OTHER	NEAAR: Formulated amino acids (+ Chemo / Radiation)	Oncology: Rectal (IIT)				
OTHER	IEM: Small molecule targeting amino acid metabolism	Pediatric: Rare Disease/Neuro				

Abbreviations: NEAAR - non-essential amino acid restriction, IEM - inborn error of metabolism

Built on work by Dr. Lewis Cantley, discoverer of the PI3K pathway. Molecules designed by Kevan Shokat, one of the world's leading medicinal chemists



Lew Cantley, PhD

Dana Farber Cancer Institute,
Harvard. Founder: Agios (NASDAQ),
Petra, Volastra
Co-founder: Faeth (NASDAQ)



Kevan Shokat, PhD

Professor of Cellular & Molecular
Pharmacology, UCSF; Professor
of Chemistry, UC Berkeley;
Howard Hughes Investigator

FAETH SCIENTIFIC CO-FOUNDERS

Sid Mukherjee, MD PhD

Asst. Prof., Columbia, Pulitzer Prize Winner, Time 100 Most Influential People, Founder: Vor
(NASDAQ)

Oliver Maddocks, MPharm, PhD

CSO & Co-Founder, Honorary Professor of Cancer Biology & Metabolism, University of Glasgow

Karen Vousden, PhD

Group Leader, Crick Institute, Former Chief Scientist of CRUK, Director, Bristol Myers Squibb

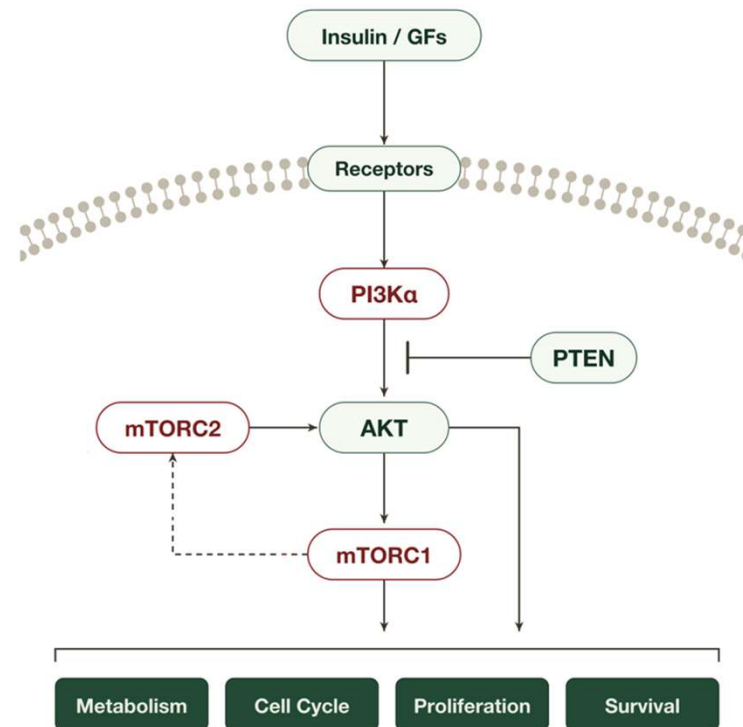
Scott Lowe, PhD

Chair of Cancer Biology & Genetics at Memorial Sloan Kettering, Founder: ORIC (NASDAQ)

Greg Hannon, PhD

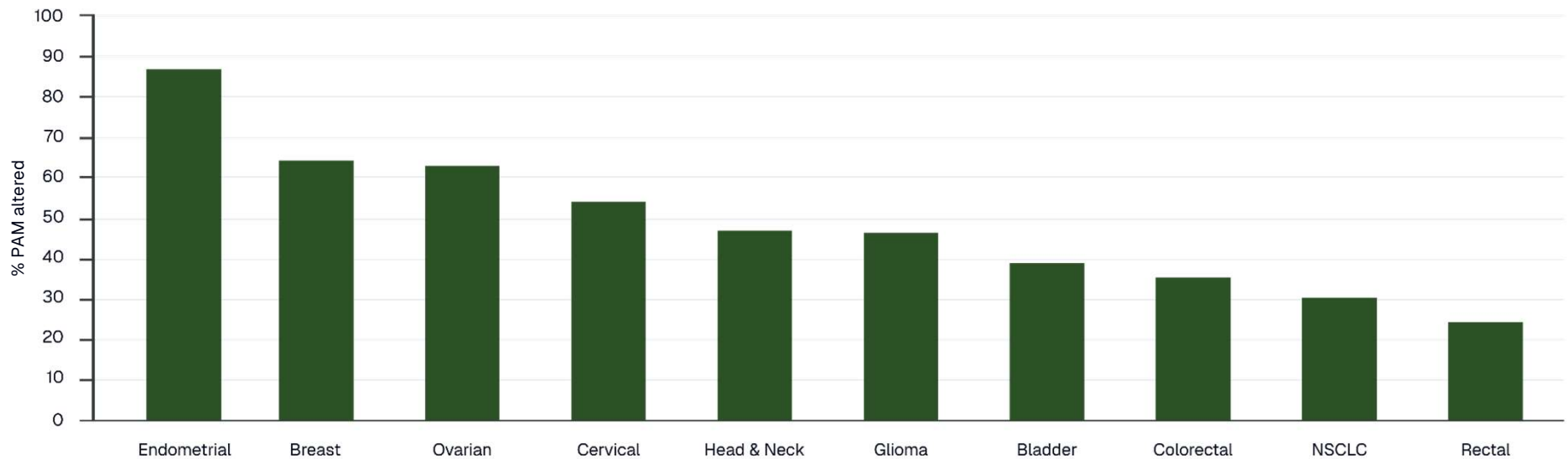
Former Director of CRUK Cambridge Institute

PAM is one of the most frequently altered pathways in cancer

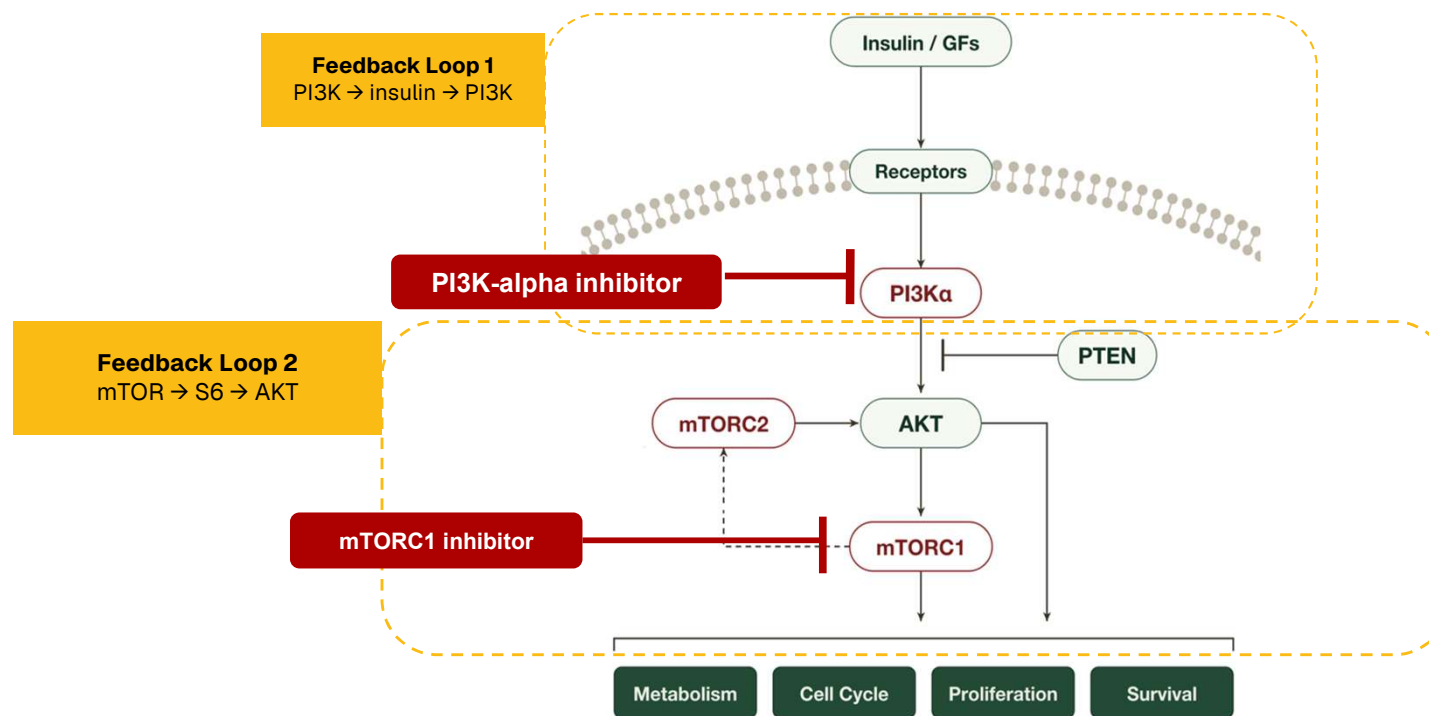


PAM is one of the most frequently-altered pathways in cancer

PAM pathway alterations by tumor type



Embedded feedback loops create a challenge for single-node inhibitors



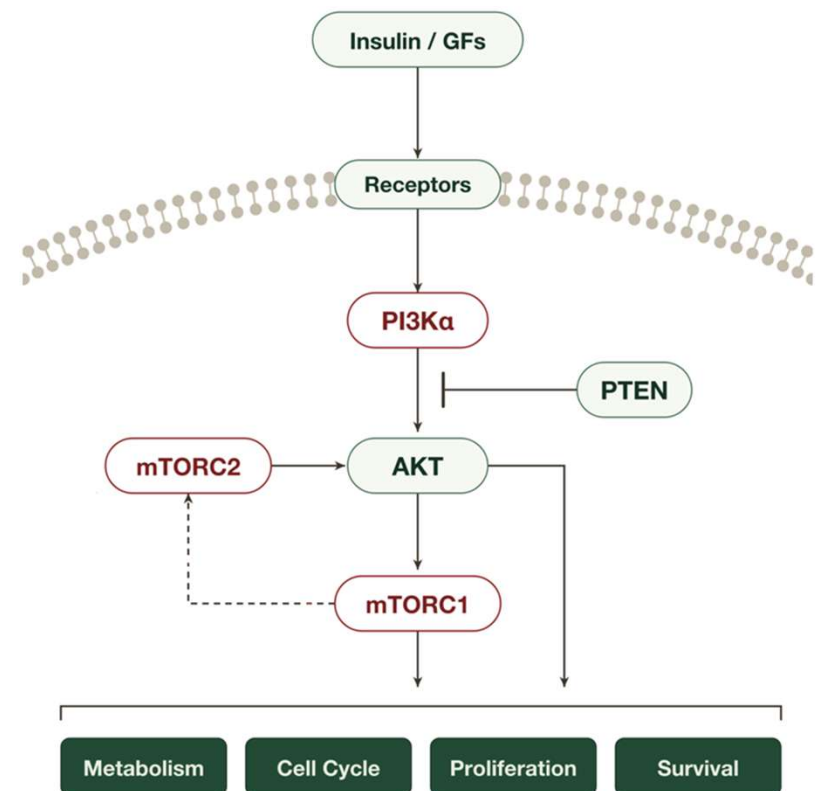
We believe that multi-node inhibition is the key to unlocking the promise of PAM inhibition

The Problem:

1. Feedback reactivation prevents single-node inhibitors from fully suppressing the pathway, even at high doses.
2. On-target toxicity limits the therapeutic window of single-node inhibitors (e.g. PI3Ki, AKTi, mTORi).

Our Solution:

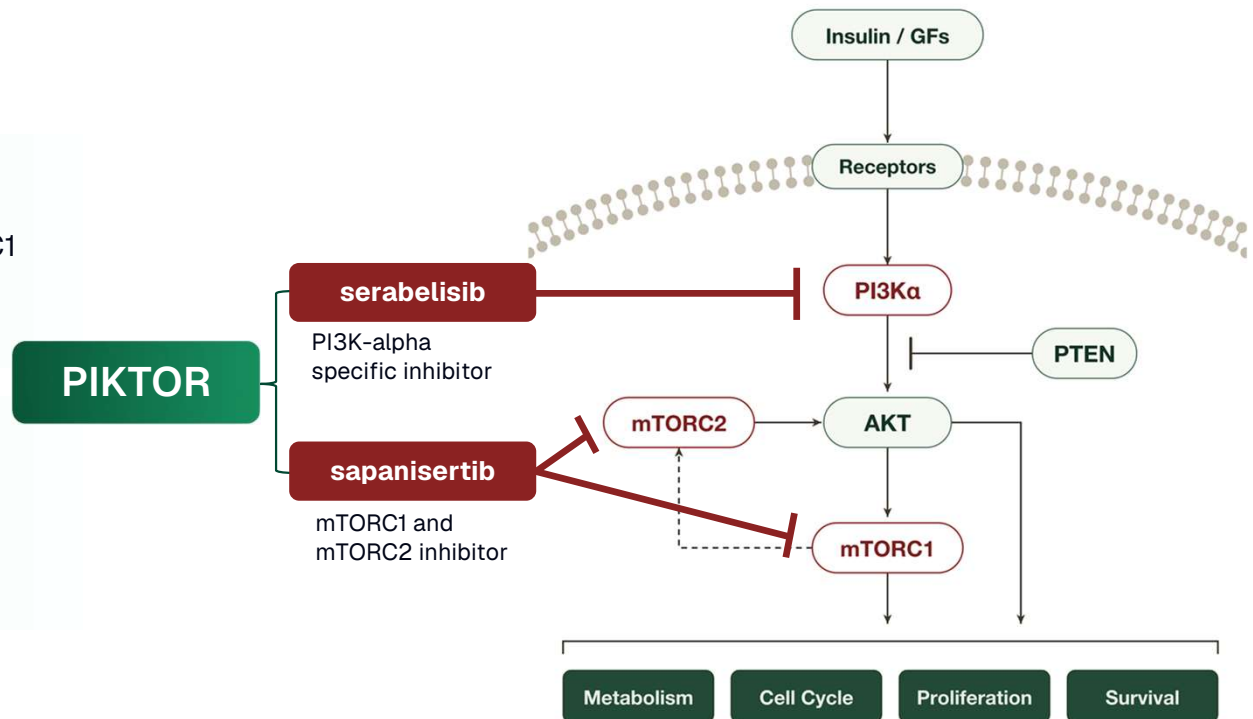
Combining single-node inhibitors at lower pharmacological doses is designed to increase pathway suppression while lowering on-target toxicity.



PIKTOR: Designed for potent and selective multi-node PI3K/AKT/mTOR pathway inhibition

PIKTOR combines serabelisib (PI3K-alpha specific inhibitor) with sapanisertib (mTORC1 and mTORC2 inhibitor).

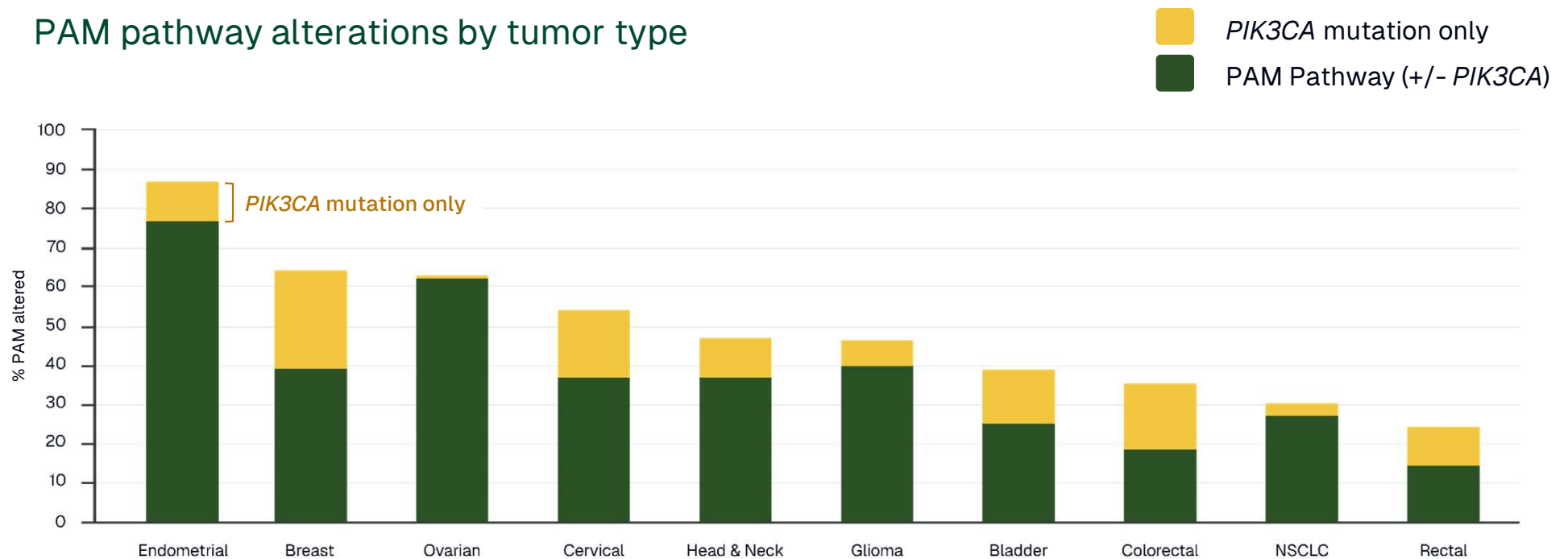
Targeting the PI3K/AKT/mTOR pathway to address one of the most-frequent genomic drivers in solid tumors¹.



¹ cBioPortal References: Cerami et al., Cancer Discov. 2012, and Gao et al., Sci. Signal, 2013

Multi-nodal inhibition broadens the treatable landscape relative to single-node approaches

PAM pathway alterations by tumor type



Company history

2019



Founded

Faeth Therapeutics established around the hypothesis that simultaneously **suppressing multiple nodes of a pathway produces deeper, more durable tumor suppression** than targeting any single node alone

2021



Serabelisib in-licensed

Initial work exploring combinations of serabelisib with insulin-suppressing diet suggests as a means to overcome hyperglycemia-driven feedback bypass. **These findings could be recapitulated by adding an mTOR inhibitor.**

2023



Sapanisertib in-licensed

In-licensing followed topline readout from a Phase 1b legacy trial initiated by Takeda (Starks et al, 2022) in a basket of tumors. **The combination is being developed as PIKTOR.**

2025



PIK-201 Phase 2 trial initiates

Faeth initiates Phase 2 PIK-201 study with the PIKTOR regimen in advanced or recurrent endometrial cancer.

2026

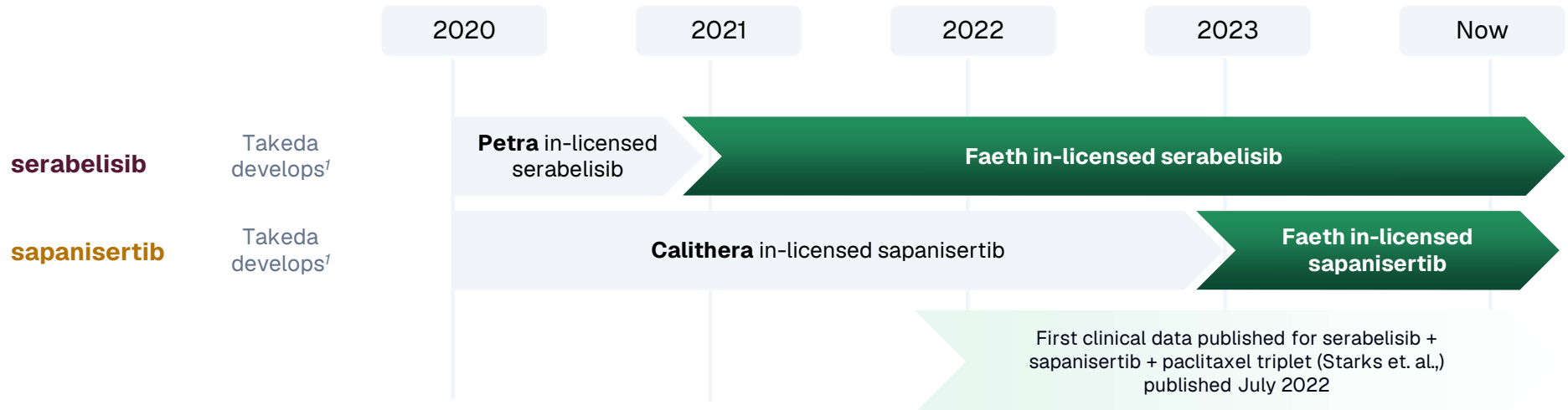


Faeth goes public

Faeth goes public via a reverse merger with Sensei Biotherapeutics. \$200M concurrent private placement. PIKTOR becomes the lead clinical program under the combined publicly-traded entity.

Takeda has already done much of the legwork for us...

PIKTOR asset history



Takeda developed **serabelisib** and **sapanisertib** focussed on **monotherapy** and **doublet** combinations. Takeda subsequently out-licensed both assets.

¹serabelisib and sapanisertib were developed by Kevan Shokat and spun into Intellikine, which was acquired by Takeda

PIKTOR: Designed to solve the challenges of drugging the PI3K pathway

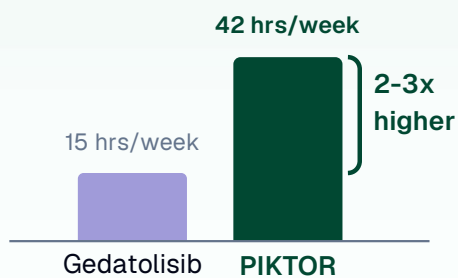
	MULTI-NODE		SINGLE-NODE	
	 faeth PIKTOR (Ph.2, Endometrial, Ph.1b Breast)	GEDATOLISIB Celcuity (Ph.3, Breast)	MUTANT-SPECIFIC Relay, Lilly, OnKure, Novartis (Ph.1/2, Breast)	FDA-APPROVED Alpelisib, Everolimus, Capiivasertib, Inavolisib
Mechanism of action Target profile	PI3K-alpha mTORC1 mTORC2	PI3K-alpha, PI3K-beta, PI3K-gamma, PI3K-delta, mTORC1 and mTORC2	Mutant PI3K-alpha	AKT or mTORC1 or PI3K-alpha
Multi-node inhibition	✓	✓	✗	✗
Potentially Immune Sparing (PI3K-alpha specific)	✓	✗	✓	✓
Broad pathway mutation coverage	✓	✓	✗	✗
Prevents wt-PI3K escape	✓	✓	✗	✓
Oral administration	✓	✗	✓	✓

PIKTOR has three potential advantages: efficacy, tolerability and patient experience

Efficacy

Oral multi-node inhibition designed for longer duration of target coverage, **achieving ~2–3x higher modeled exposure above IC90** than gedatolisib ^{1,2}

Time above IC90



Modeled weekly exposure above IC90

Tolerability

Hyperglycemia AEs ^{3,4}

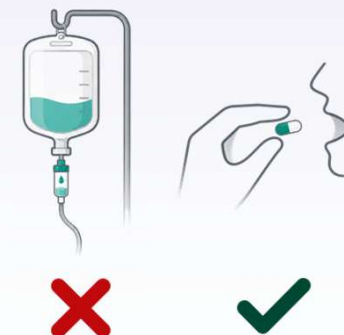
	ZOVEGALISIB	PIKTOR
Gr 1-2	35%	9%
Gr 3-4	8%	5%
Total	43%	14%

Stomatitis AEs ^{4,5}

	GEDATOLISIB	PIKTOR
Gr 1-2	48%	14%
Gr 3-4	15%	0%
Total	63%	14%

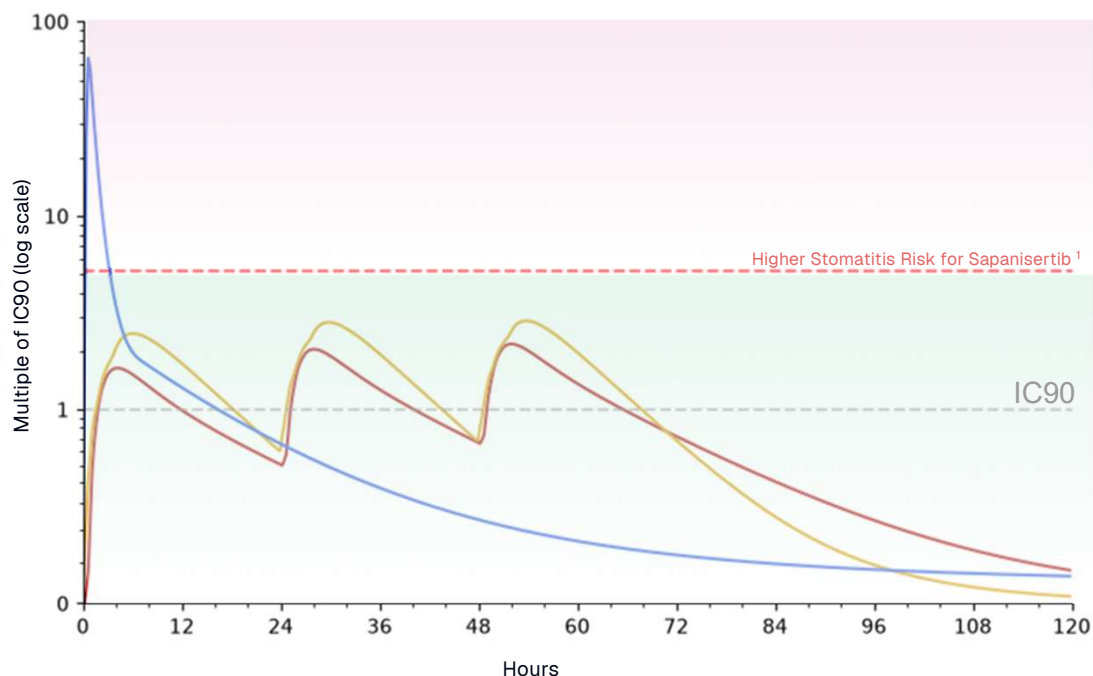
Patient Experience

Patient-centric **oral regimen** designed to replace IV infusion burden with convenient, at-home dosing



¹Faeth internal PK modeling based on Faeth internal human PK data. ²Based on Faeth internal analysis and estimates using publicly available data. ³RLY-2608 Ph1 (+fulvestrant): Varkaris et al., ESMO TAT 2026, 400 mg BID Fed, RP2D Cohort ⁴Data snapshot PIK-201 PIKTOR + Paclitaxel in patients with advanced endometrial cancer, 01-05-2026 (ongoing Faeth Sponsored Multicenter Phase 2 trial) ⁵Celcuity VIKTORIA-1 Phase 3 Results Presentation, Arms A + B, Oct 2025. The results are presented from different clinical trials at different points in time with differences in trial design. No head-to-head trials have been conducted among the results shown and cross-trial comparisons must be interpreted with caution. As a result, conclusive cross-trial comparisons cannot be made.

Oral PIKTOR dosing is designed for sustained exposure with reduced C_{max}-related toxicity risk



	Peak exposure (C _{max} : IC ₉₀ ratio)	Time above IC ₉₀ / week	Plasma protein binding / free fraction	
			Bound	Free
 Serabelisib	2.2	42 h	69%	31%
 Sapanisertib	2.9	44 h	70%	30%
 Gedatolisib	~65	15 h	98%	2%

The ratio of plasma drug concentration to cellular IC₉₀ from 0 - 120 hours was calculated for serabelisib, sapanisertib, and gedatolisib. Sapanisertib (3 mg) with serabelisib (200mg), with food, QDx3dQW plasma drug concentration was obtained from Maddocks *et al.*, 2026¹. Gedatolisib (180 mg QW) drug concentration data was derived from Shapiro *et al.*, 2015² by scaling 89 / 154 / 222 / 266 mg single dose data to 180 mg, assuming linearity. Mean gedatolisib plasma concentration at 180 mg was computed with a geometric mean per time-point across the scaled concentrations. The estimated time above IC₉₀ was computed as the time a stepwise linear interpolation crossed the threshold. IC₉₀ values for all drugs were calculated using IC₅₀ values presented in Tyrakis *et al.*, 2025³ and Slide 44 in this corporate deck. The cellular IC₅₀ values for all three drugs were derived from an average of six cell lines representing clinically relevant models i.e. HR+/HER2- breast cancer cell lines and endometrial cancer cell lines: MCF7, T47D, AN3CA, HEC1B, MFE-280, MFE-296. Dashed red line denotes the sapanisertib C_{max}/IC₉₀ ratio for the combined dose of sapanisertib and serabelisib (4 mg / 200 mg, QDx3dQW, fasted) resulting in Grade 3 stomatitis in a Phase 1b dose escalation study (NCT01899053, described in Maddocks *et al.*, 2026¹).

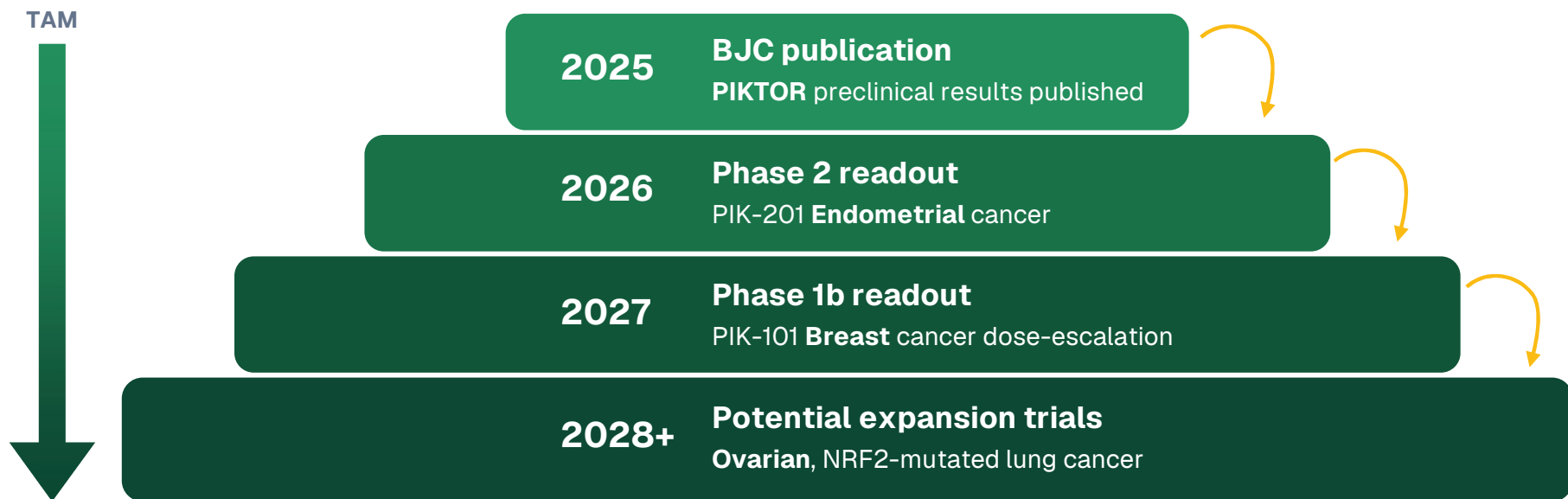
¹ Exposure/Response Analysis of an Oral Multi-Node PI3K/AKT/mTOR Pathway Inhibitor Combination of Serabelisib and Sapanisertib. Maddocks *et al.*, STOP Cancer Conference, 2026

² First-In-Human Study of PF-05212384 (PKI-587), a Small-Molecule, Intravenous, Dual Inhibitor of PI3K and mTOR In Patients With Advanced Cancer. Clin Cancer Res. 2015 April 15; 21(8): 1888-1895. doi:10.1158/1078-0432.CCR-14-1306.

³ Multi-node inhibition targeting mTORC1, mTORC2 and PI3Ka potentially inhibits the PI3K/AKT/mTOR pathway in endometrial and breast cancer models. British Journal of Cancer, 133: 144-154. <https://doi.org/10.1038/s41416-025-03035-z>

Where we are, Where we're going

Our strategy is to prove that PIKTOR's RP2D in endometrial is clinically active, potentially enabling us to rapidly dose-escalate in breast cancer, and sequentially **un-lock the potential of multi-node PAM inhibition in multiple cancers**



Marcus Goncalves, MD PhD



Dr. Marcus Goncalves,
MD PhD

Department of Medicine at
NYU Grossman School of
Medicine

Current roles

- Associate Professor of Endocrinology
- Associate Professor Department of Radiation Oncology
- Director, Holman Division of Endocrinology, Diabetes & Metabolism
- Director, Systemic Metabolism Research



Academic background



Weill Cornell Medicine
Postdoc / Ass. Professor



Memorial Sloan Kettering
Endocrinology Fellow



Perelman School of Medicine
MD PhD



Johns Hopkins University
BS Biomedical/Medical
Engineering

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PAM Overview and Single-Node Inhibitor Escape

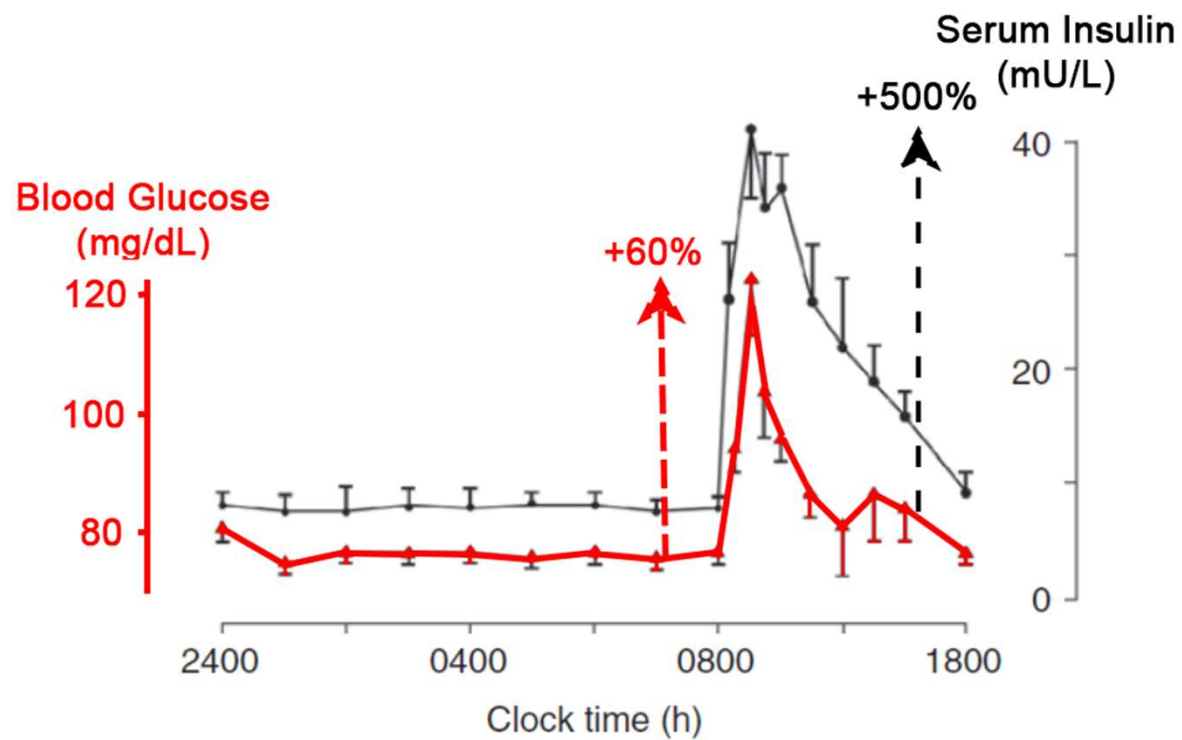
OCTOBER 6, 2026

Overview

Dr. Marcus Goncalves, Scientific co-founder

- 1 Uncovering the flaws in single-node PI3K inhibition approaches
- 2 Explaining the origins of PI3K-inhibitor induced hyperglycemia
- 3 Setting the scene for multi-node PAM inhibition

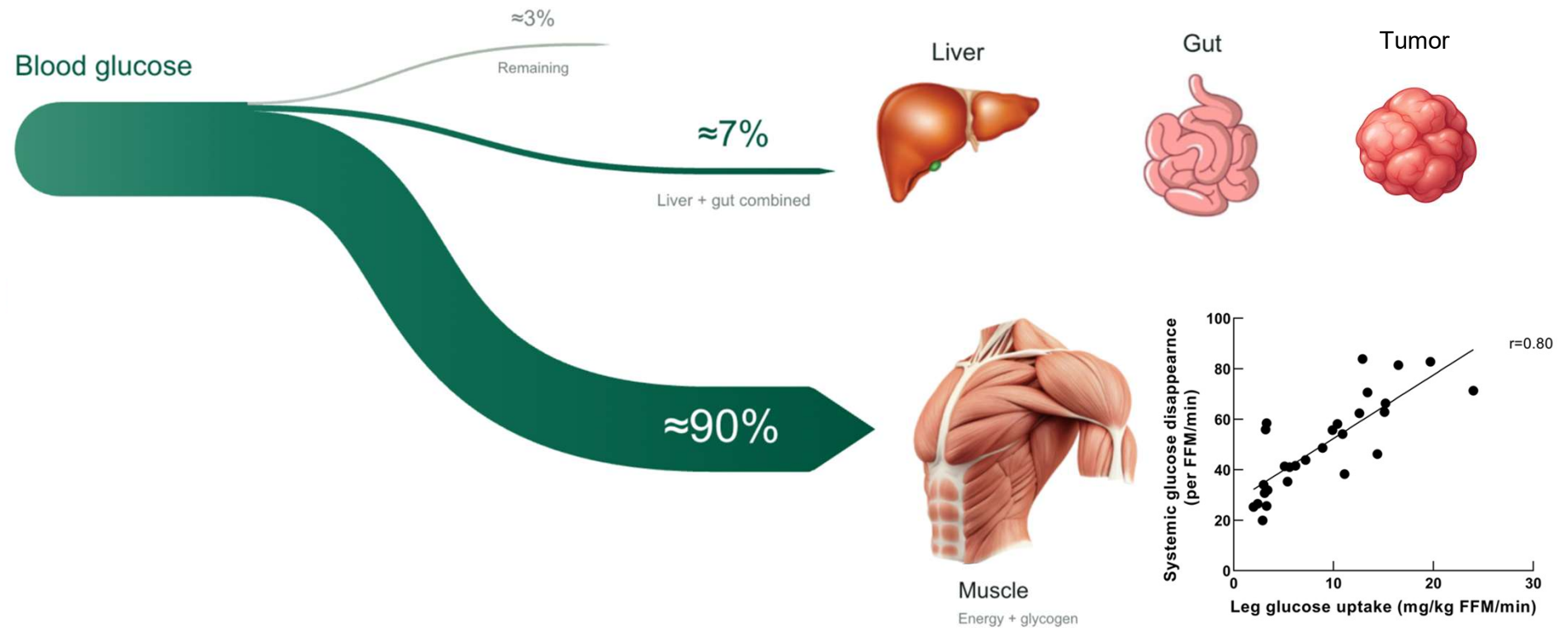
Post-meal Glucose and Insulin



Adapted from P.D. Home Diabetes, Obesity and Metabolism 17: 1011–1020, 2015

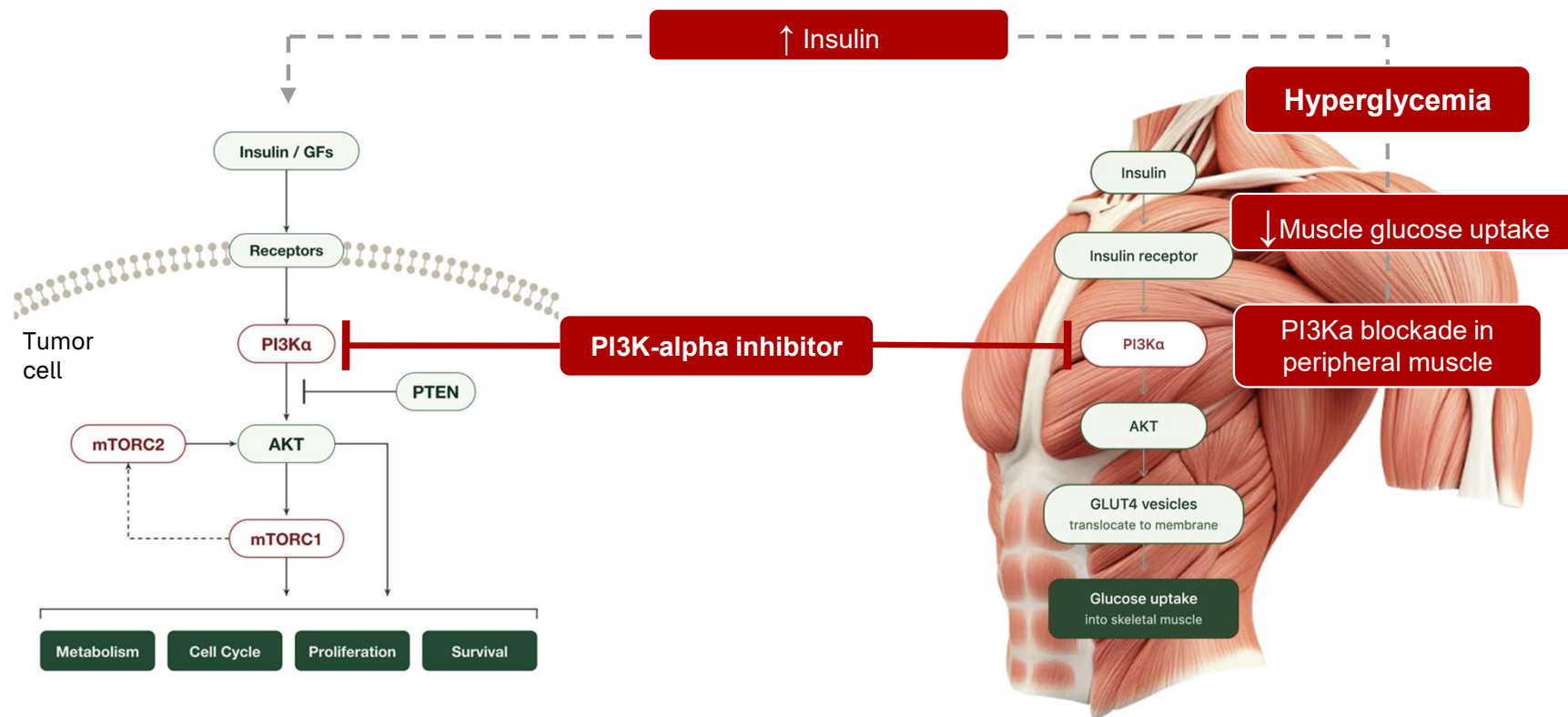
Muscle dominates insulin-stimulated glucose disposal

INSULIN-STIMULATED STATE / HUMAN CLAMP DATA



Width of glucose flows proportional to uptake. Rounded Study estimates. Glucose uptake by brain not included as not insulin dependent. DeFronzo et al JCI 1985

Identifying the flaws in Single-Node PAM inhibition



01 Normal insulin response

02 PI3K α inhibition

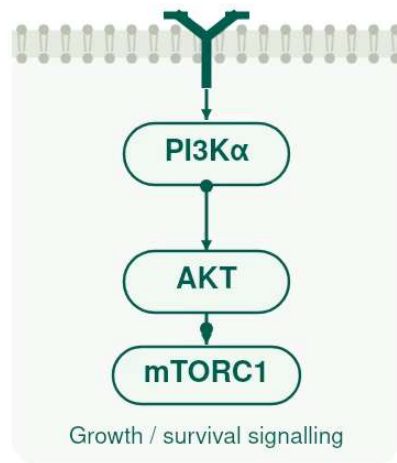
03 Reduced glucose uptake

04 Hyperglycaemia

05 Insulin feedback

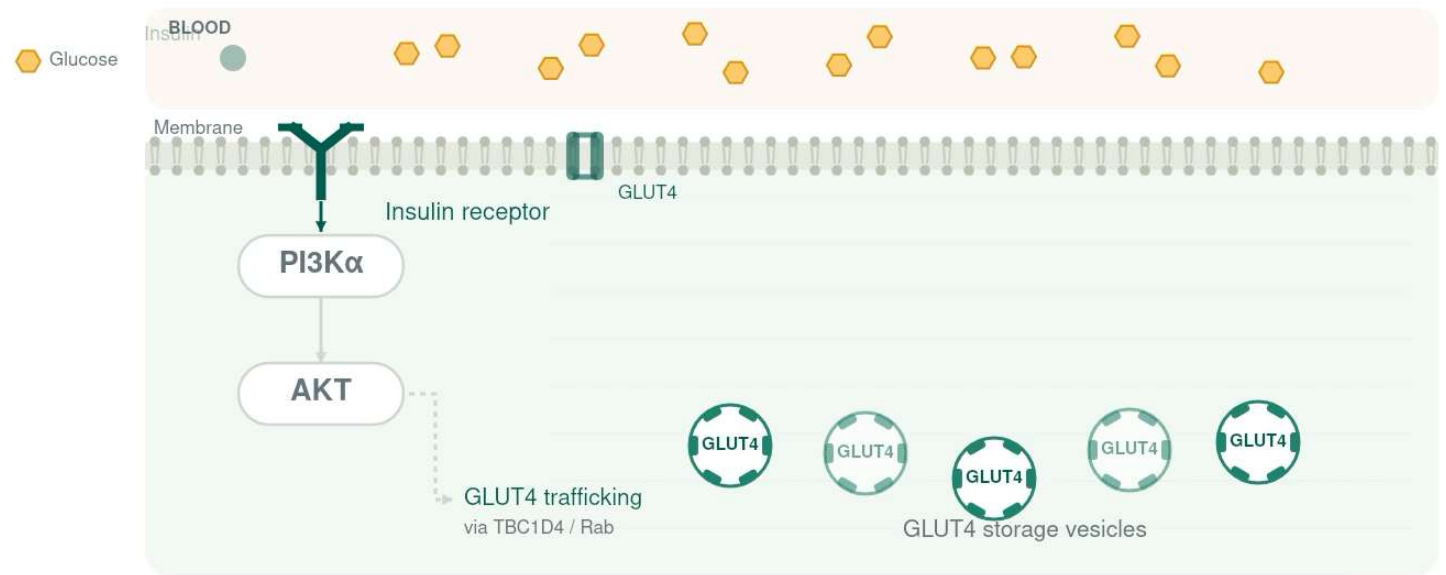
TUMOUR CELL

Therapeutic target

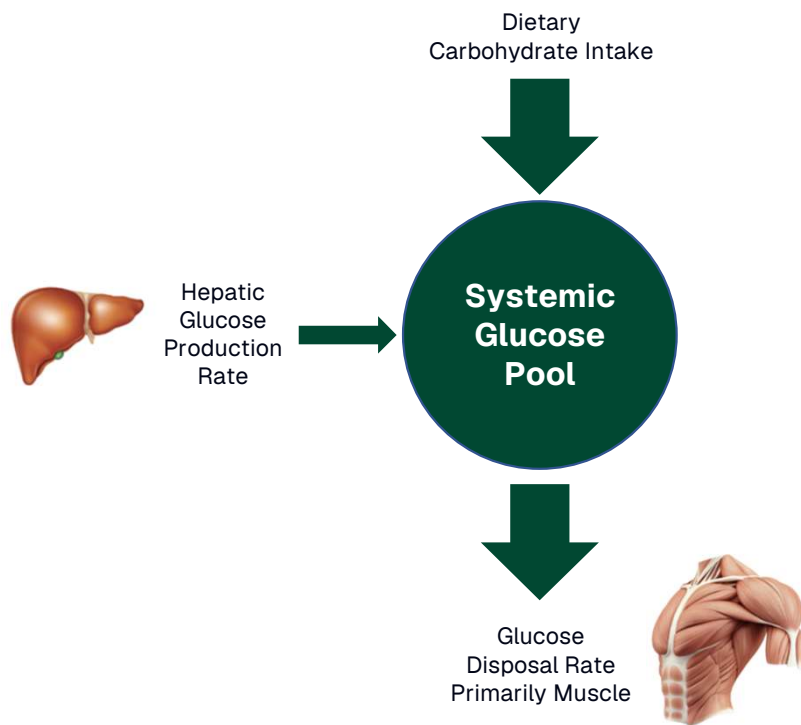


SKELETAL MUSCLE

Insulin-responsive glucose disposal



Influence of hepatic glucose production is outweighed by glucose disposal

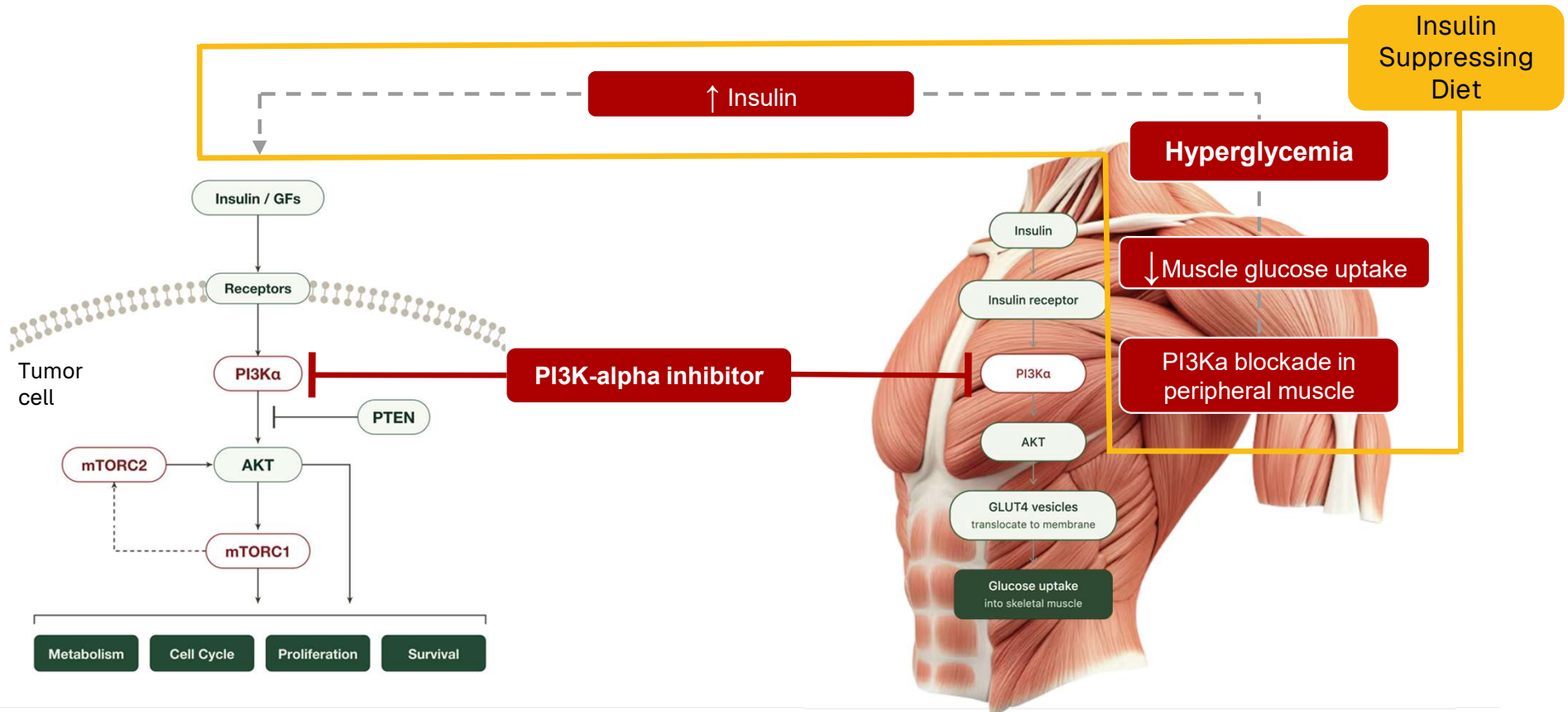


Insulin dependent (PI3K inhibitor responsive) glucose metabolism

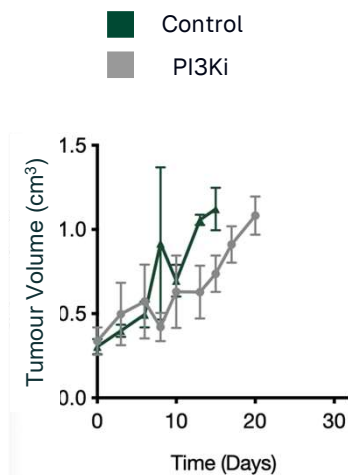


Nine healthy adults underwent a hyperinsulinaemic–euglycaemic clamp after overnight fasting. Glucose production and disposal were compared at baseline and 60–120 minutes; bars show mean ± SEM.

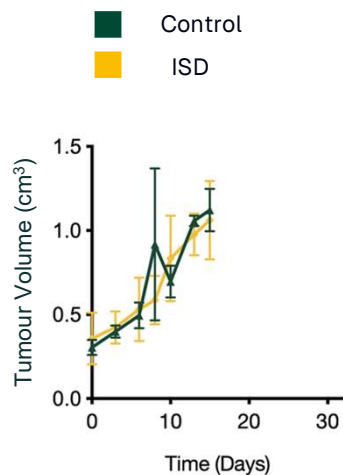
Breaking the glucose/insulin feedback loop improves efficacy



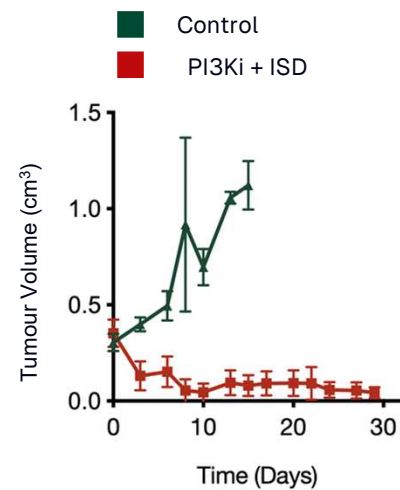
Breaking the glucose/insulin feedback loop improves efficacy



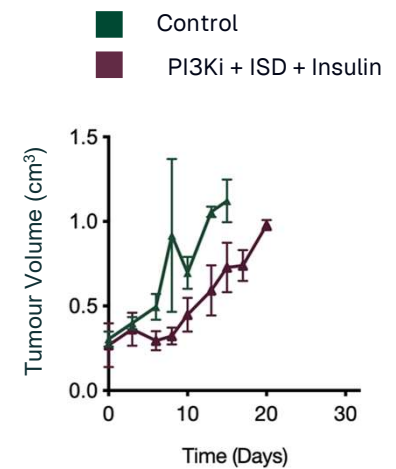
Drug alone
Efficacy: minimal



Insulin suppression alone
Efficacy: none

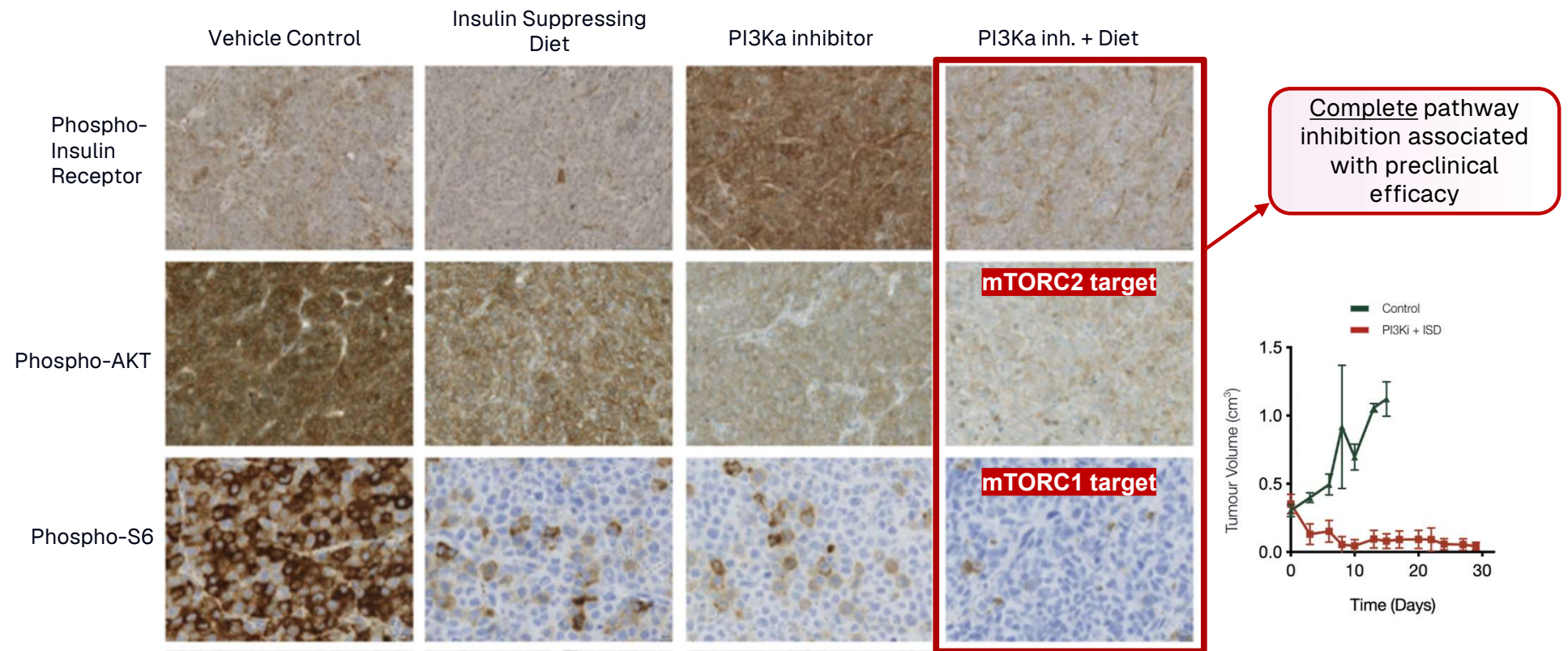


Drug + insulin suppression
Efficacy: outstanding



Drug + insulin suppression +
insulin (proof of principle)
Efficacy: reversed

Towards Multi-Node: PI3K inh. + ISD converges on **mTOR**

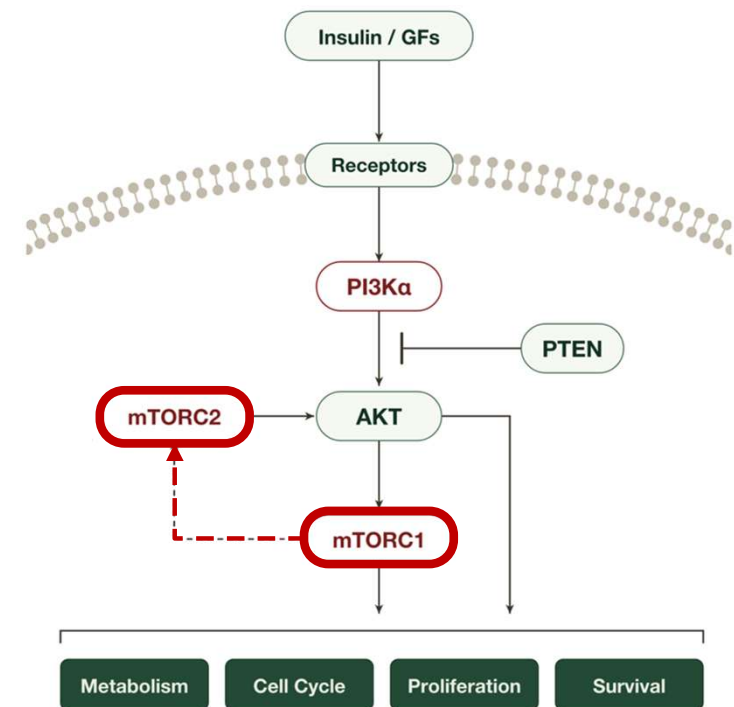


mTORC1 and mTORC2 signaling are vital for PAM activity

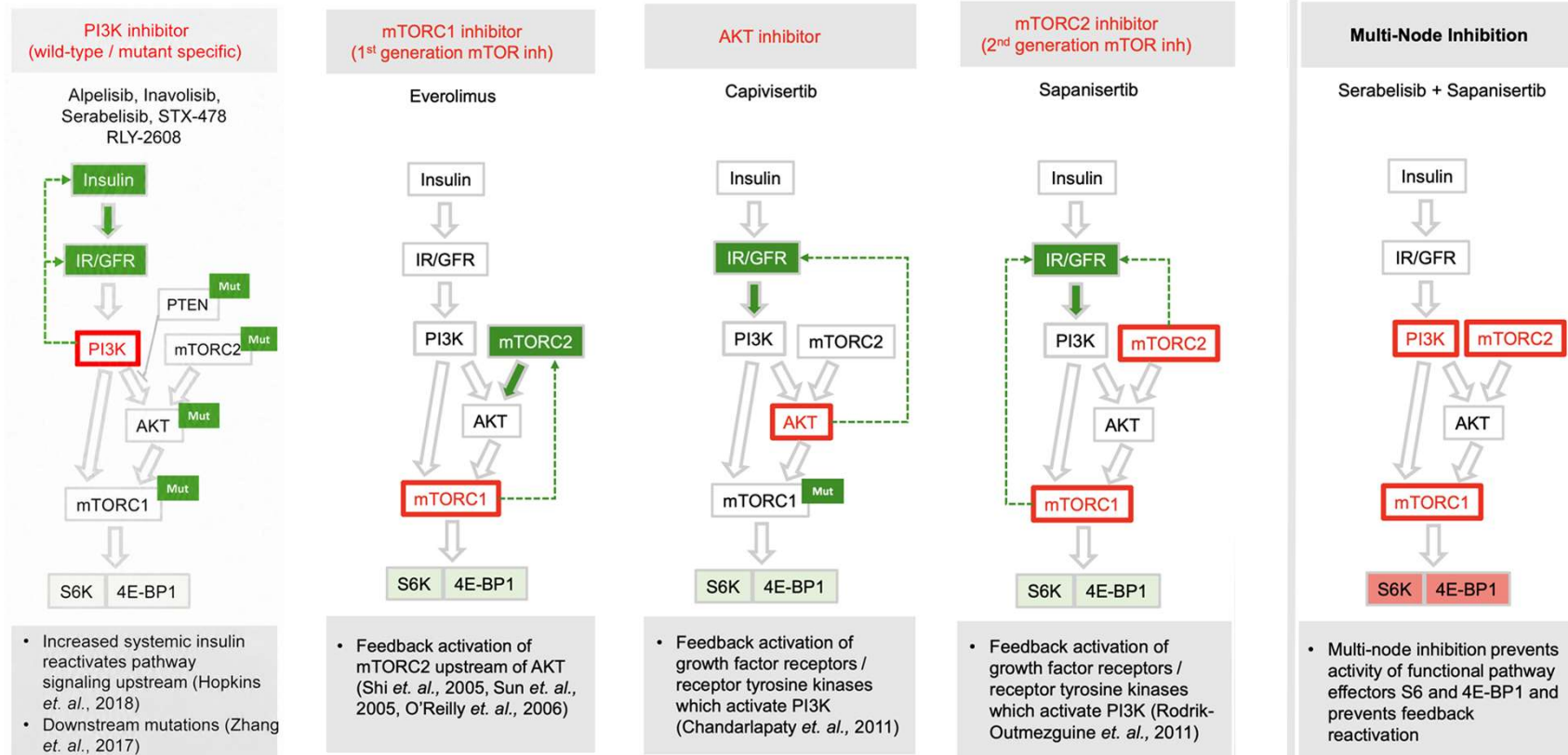
mTOR is a vital coordinator of pathway activity and reactivation in the setting of PI3K inhibitors

Multi-node inhibition at PI3K and mTOR are associated with strong anti-tumor response

Pharmacologic mTOR inhibition may improve the efficacy of PI3K inhibition and mimic the drug-diet combination therapy.



Single-Node PAM inhibitors have numerous resistance mechanisms



Multi-Node Inhibition & Hyperglycemia: Part 1

Why the Multi-Node mechanism reduces hyperglycemia

More complete inhibition of the pathway enables lower relative doses versus single-node
e.g. serabelisib 4.5x lower dose as multi-node combo

Multi-Node 'spreads' the hit to mTOR
PI3K/AKT are the core nodes for signaling glucose uptake by muscle

PIKTOR and Gedatolisib leverage intermittent dosing, lowering the chronic systemic impact on PI3K/AKT

Multi-Node Inhibition & Hyperglycemia: Part 2

Why route of administration is unrelated

Muscle, not liver, is the primary site of glucose disposal and PI3K inhibition in muscle is what drives hyperglycemia; IV drugs impact muscle as much or more-so than oral drugs

Intravenous single-node pan-PI3K inhibitor administration does not avoid hyperglycemia	Study	Regimen	Any grade	Grade 3	Grade 4
	CHRONOS-1	IV: Copanlisib monotherapy	50.0%	33.1%	7.0%
	CHRONOS-3	IV: Copanlisib + rituximab	69.4%	48.2%	8.1%
	SOLAR-1	Oral: Alpelisib + fulvestrant	63.7%	32.7%	3.9%

PIKTOR, our oral Multi-Node PAM inhibitor has shown low hyperglycemia with very permissive HbA1c and fasting glucose inclusion criterion in a high risk population

Multi-node oral PAM inhibition has the potential to improve efficacy and tolerability

Building on the clinical success of Gedatolisib

Blocking multiple nodes at once keeps the feedback loops that reactivate the pathway from operating.

With no escape route, no single node has to be inhibited as deeply and can be given intermittently

Multi-Node → resistance pathways stay closed → less depth required → intermittent exposure becomes possible

...With a deep understanding of the biology

PI3K α mediates insulin signaling in skeletal muscle.

Inhibiting PI3K blocks glucose uptake in muscle → blood glucose rises. This is a known, on-target class effect — seen with oral and IV agents alike.

So the question isn't route — it's how much PI3K inhibition reaches muscle outside the tumor.

Oliver Maddocks



Dr. Oliver D. K. Maddocks,
MPharm PhD

Current roles

- Chief Scientific Officer and Co-Founder of Faeth
- Honorary Professor of Cancer Biology and Metabolism, University of Glasgow

Academic background



University of Glasgow
Professor of Cancer Biology and Metabolism



Beatson Institute
Postdoctoral Fellow



University of Maryland School of Medicine
Fulbright Scholar, Postdoc



University of Edinburgh
PhD



Cardiff University
MPharm

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Preclinical → Clinical

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Overview

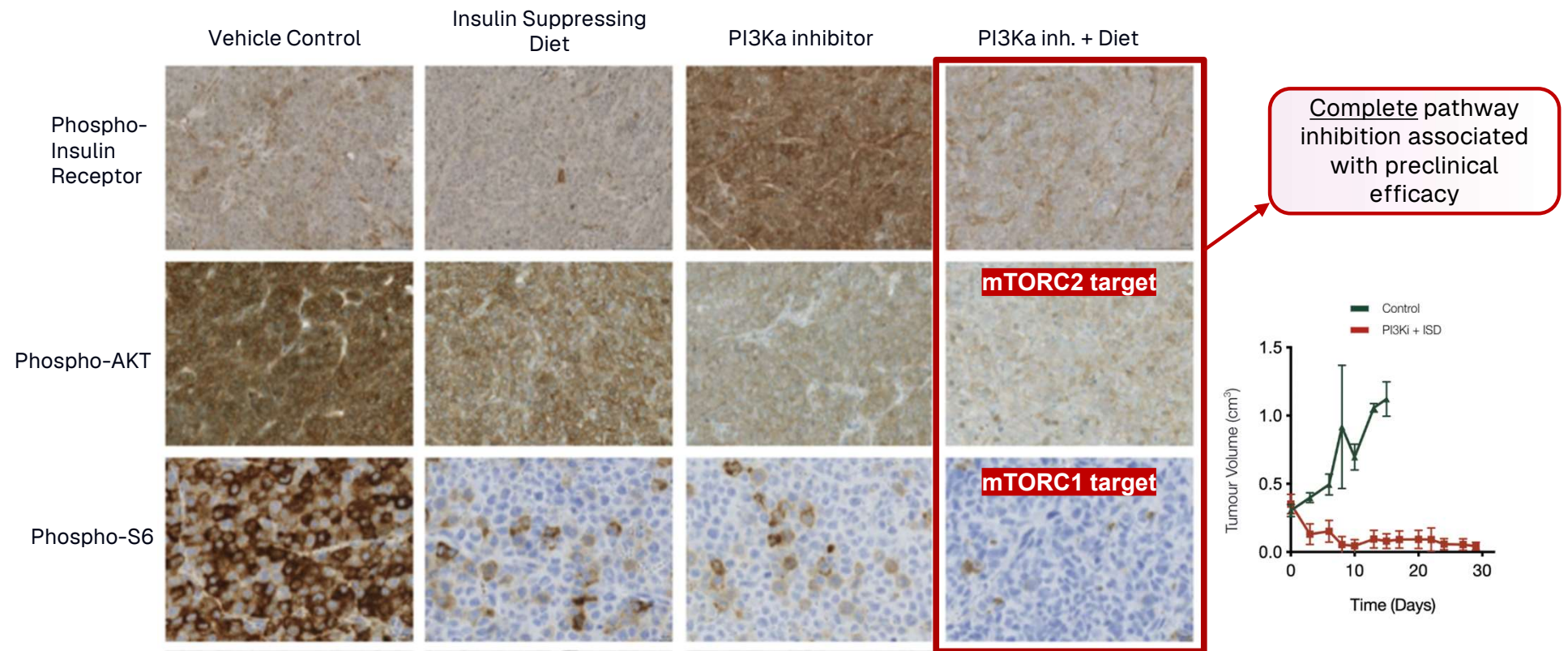
Dr. Oliver Maddocks, Co-founder and CSO

1 PIKTOR Mechanism of Action

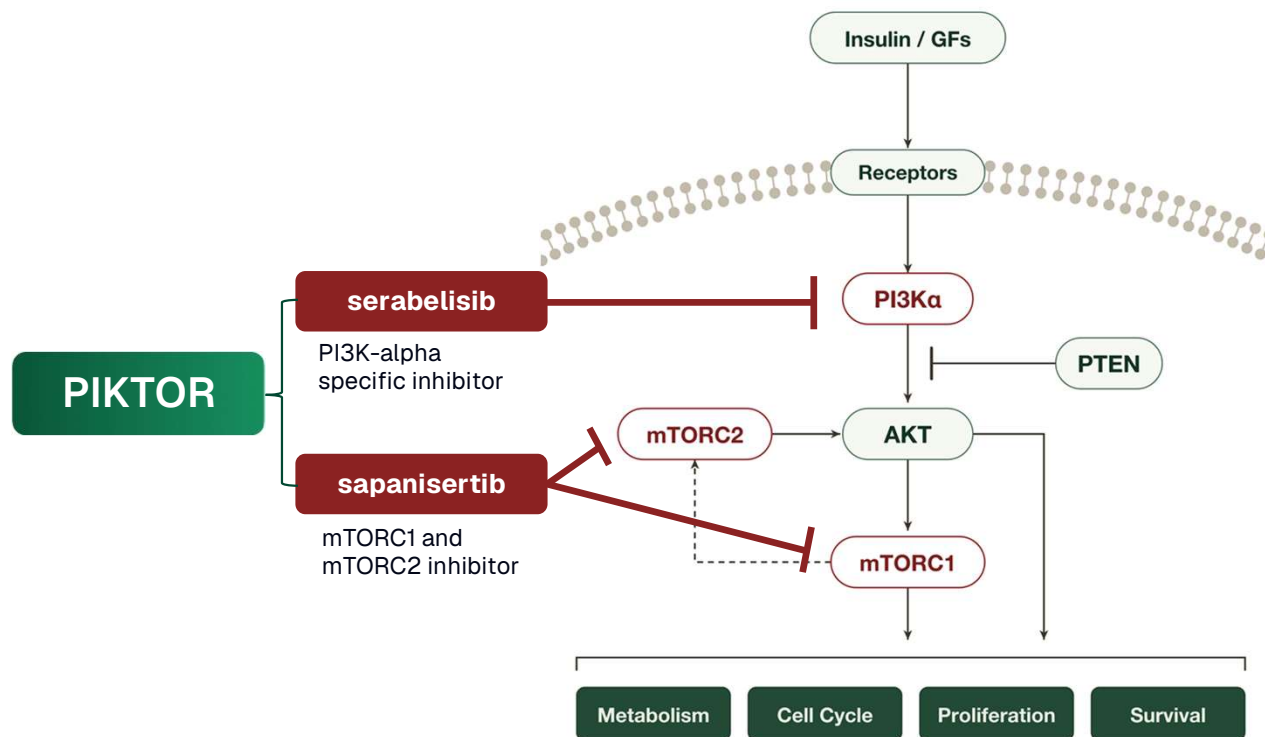
2 Preclinical Data

3 PK-PD → Dose Selection

Towards Multi-Node: PI3K inh. + ISD converges on **mTOR**

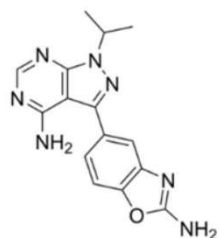


Adding sapanisertib - highly potent 2nd gen. mTOR inh



Sapanisertib

Sapanisertib
mTORC & mTORC2 inhibitor

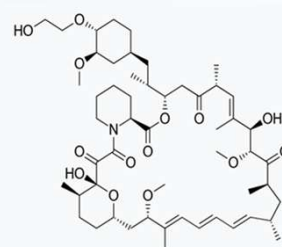


Sapanisertib Chemical Structure
CAS No. : 1224844-38-5

Other names:
TAK-228
MLN0128
INK-128

Recombinant Enzyme	IC50 (nM)
mTOR	1.0
PI3K alpha	219.0
PI3K beta	5293.0
PI3K delta	230.0
PI3K gamma	221.0

First generation (rapalog) inhibitors of mTOR only target the mTORC1 complex via drug-FKBP12 complexation, they are not ATP competitive or rationally designed



Everolimus structure. Rapamycin was derived from *Streptomyces hygroscopicus*, Easter Island.

Rapalogs target FKBP12 which inhibits mTORC1 complex, not mTORC2

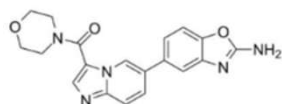
Sapanisertib is a rationally designed ATP-competitive kinase inhibitor that is designed to directly bind mTOR, and is highly differentiated from rapalogs; it **targets two distinct multi-protein complexes; mTORC1 and mTORC2** which have distinct functions in controlling cell growth, survival and proliferation.

Sapanisertib is administered as immediate release capsules (1mg and 3mg)

Serabelisib

Serabelisib

PI3K alpha inhibitor



Serabelisib Chemical Structure

CAS No. : 1268454-23-4

Other names:

TAK-117

MLN1117

INK1117

Recombinant Enzyme	IC50 (nM)
PI3K alpha	21.4
PI3K beta	5647.0
PI3K delta	12147.0
PI3K gamma	988.0
mTOR	2680.0

Serabelisib is an investigational, orally available, selective small molecule inhibitor of the class I phosphoinositide 3-kinase (PI3K) alpha isoform (PIK3 α , encoded by PIK3CA gene).

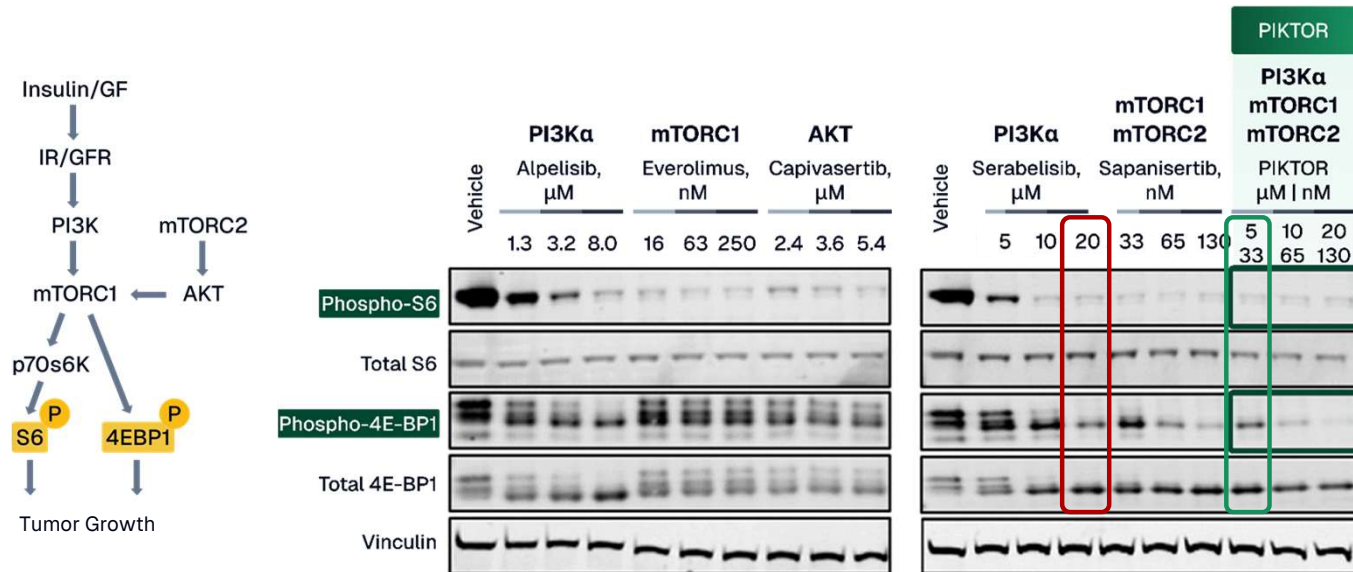
PI3K enzymes rely on ATP to phosphorylate phosphatidylinositol-4,5-bisphosphate (PIP2) to produce phosphatidylinositol-3,4,5-trisphosphate (PIP3), a critical lipid signaling molecule that activates downstream AKT and mTOR pathways. **Serabelisib is designed to competitively bind to the ATP-binding pocket of PI3K alpha, blocking the enzyme's ability to convert PIP2 to PIP3, thereby halting the downstream signaling.**

Serabelisib is designed to selectively inhibit the PI3K alpha isoform, one of the four PI3K class IA isoforms (alpha, beta, delta, and gamma). This isoform selectivity minimizes off-target effects associated with the inhibition of other PI3K isoforms, which can lead to unwanted side effects.

Serabelisib is administered as immediate release tablets (100mg and 300mg)

Multi-node pharmacologic inhibition has the potential to be a more durable approach.

PIKTOR reduced PI3K-pathway signalling in cancer cell lines more than single-node inhibitors at clinically relevant concentrations¹



AN3CA Endometrial Cancer Cell Line

- *PIK3CA* Wild-type
- *PTEN* deletion
- *PIK3R1* mutant
- *MTOR* mutant

In DRC assays, reduction of **phos-4E-BP1**, but not phos-S6, is correlated with drug potency (cellular IC50)

Tyrakis et. al., 2025

Western blot data shown is from AN3CA endometrial cancer cells (mutations in *PTEN*, *PIK3R1*, *mTOR*) and is replicated in multiple PI3K-mutated endometrial and breast-cancer cell lines¹.

¹Tyrakis et. al., (2025) British Journal of Cancer, 133: 144-154.

PIKTOR: Consistent pathway inhibition at S6/4E-BP1/AKT in breast and endometrial models irrespective of genotype

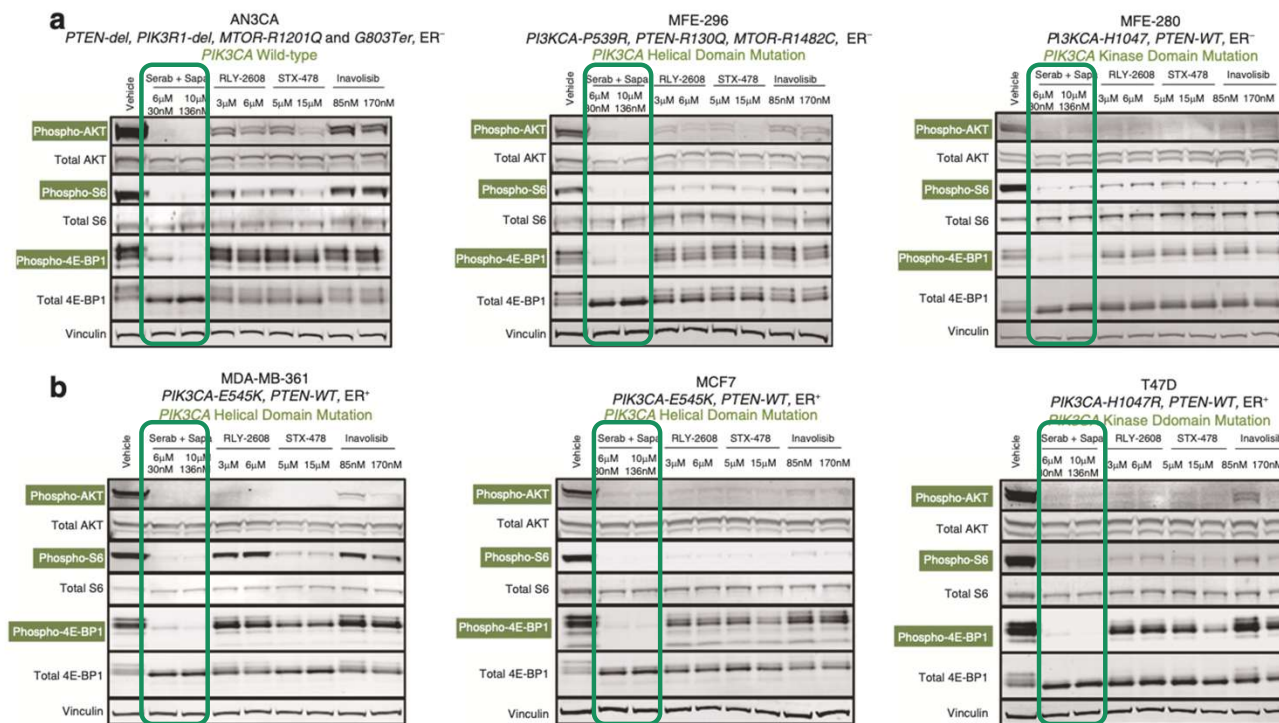


Figure 2a, b: ¹Tyrakis et. al., (2025) British Journal of Cancer, 133: 144-154.

PIKTOR demonstrated preclinical pathway suppression at lower concentrations than other PI3K/AKT/mTOR targeted agents

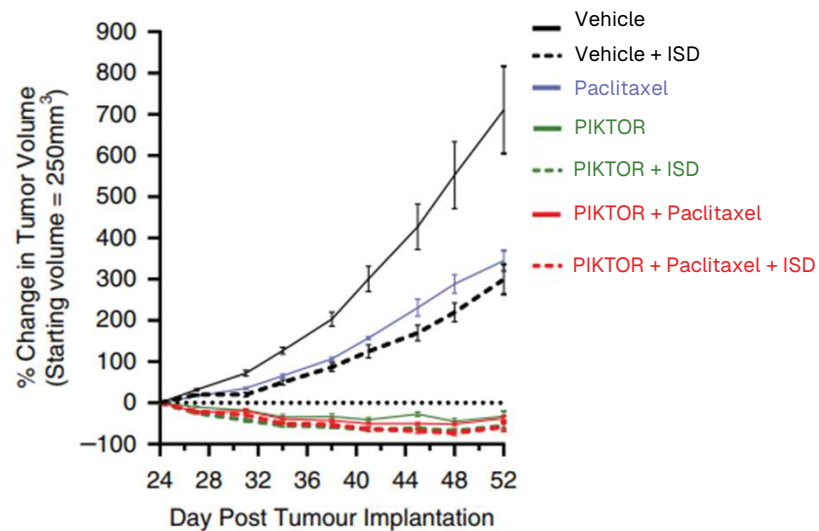
Cell Line	PI3K-Pathway Status	Serabelisib ¹	Sapanisertib ¹	PIKTOR ¹	Gedatolisib ²	Alpelisib ¹	Everolimus ¹	Capivasertib ¹	RLY-2608 ¹	STX-478 ¹	Inavolisib ¹	Paclitaxel ¹
MFE-296	PIK3CA-P539R, PTEN-R130Q, MTOR-R1482C	5.17	0.0070	0.0067	0.0158	0.286	15.4	0.11	3.86	5.92	0.648	0.0088
AN3CA	PTEN-del, PIK3R1-del, MTOR-R1201Q	3.377	0.0108	0.0050	0.0040	1.653	15.0	0.34	4.85	6.97	1.44	0.0007
HEC1B	PIK3CA-G1049R, RICTOR-D939G	2.346	0.0153	0.0054	0.0500	0.438	15.2	0.57	8.41	10.5	1.35	0.0042
MFE-280	PIK3CA-H1047	2.19	0.0187	0.0047	0.0520	1.779	14.8	>100	2.15	1.06	0.113	0.014
MFE-296 Paclitaxel Resistant ²	PIK3CA-P539R, PTEN-R130Q, MTOR-R1482C	3.521	0.0058	0.0045	ND	0.438	46.1	0.06	ND	ND	ND	0.035
AN3CA Paclitaxel Resistant ²	PTEN-del, PIK3R1-del, MTOR-R1201Q	2.671	0.0072	0.0038	ND	0.494	15.2	0.14	ND	ND	ND	0.015
Endometrial Cancer Average IC50:		3.213	0.0108	0.0050	0.0305	0.8480	20.28	16.87	4.818	6.113	0.8878	0.0130

Cell Line	PI3K-Pathway Status	HR/HER2 Status	Serabelisib ¹	Sapanisertib ¹	PIKTOR ¹	Gedatolisib ²	Alpelisib ¹	Everolimus ¹	Capivasertib ¹	RLY-2608 ¹	STX-478 ¹	Inavolisib ¹	Paclitaxel ¹
MDA-MB-361	PIK3CA-E545K, MTOR-E1427Q	HR+/HER2+	3.82	0.010	0.0075	0.023	1.34	>100	0.879	5.94	2.93	0.162	0.237
T47D	PIK3CA-H1047R	HR+/HER2-	1.34	0.012	0.0033	0.047	0.20	28.8	0.255	0.61	0.13	0.056	0.009
MCF-7	PIK3CA-E545K	HR+/HER2-	1.33	0.005	0.0021	0.014	0.407	>100	0.455	1.57	0.63	0.063	0.026
MDA-MB-231	Wild-type PI3K pathway	TNBC (HR-/HER2-)	6.01	0.024	0.0111	0.024	19.89	14.8	87.5	31.6	>100	>100	0.011
Breast Cancer Average IC50:			3.13	0.013	0.006	0.027	5.46	60.9	22.3	9.93	25.92	25.1	0.071

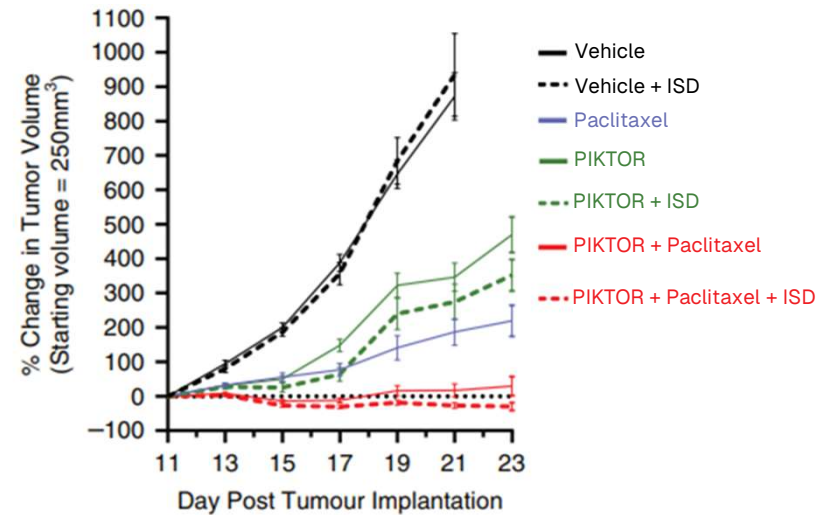
¹ Tyrakis et. al, 2025, British Journal of Cancer ² Faeth Internal Data
Abbreviations: ND - not done

PIKTOR achieved effective *in vivo* tumor growth inhibition

MDA-MB-361 Breast Cancer Model



AN3CA Endometrial Cancer Model



Overview

Dr. Oliver Maddocks, Co-founder and CSO

1 PIKTOR Mechanism of Action

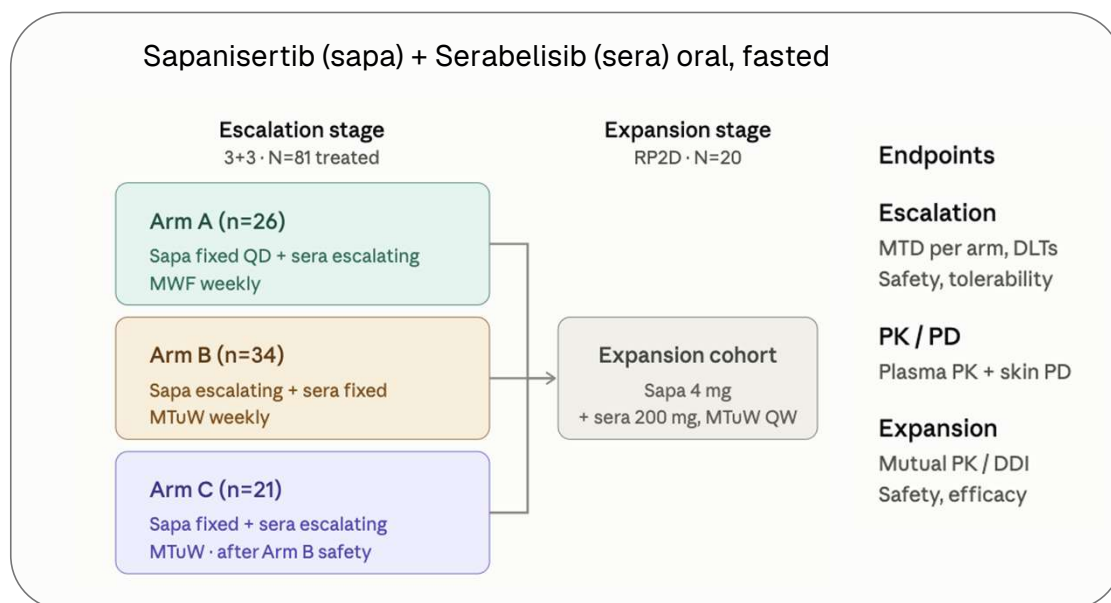
2 Preclinical Data

3 PK-PD → Dose Selection

Takeda C32001 trial informed PIKTOR dose and schedule

Typical later-line, treatment-refractory Phase 1 cohort with predominantly metastatic cancer (NCT01899053)

- Heavily pretreated, advanced solid-tumour population that had exhausted standard therapy
- ~62% had Stage IV disease at entry

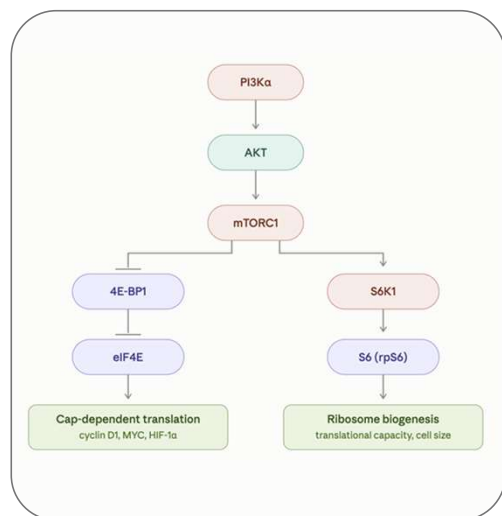


Efficacy summary:

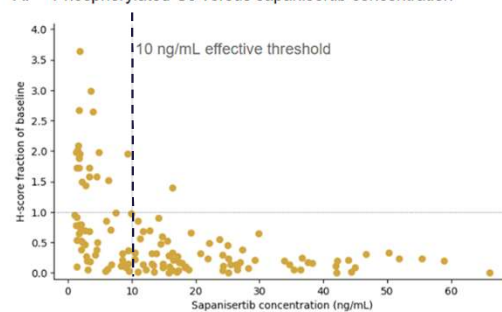
Stage	Response-evaluable N	ORR (CR+PR)	Clinical benefit rate
Escalation (all arms)	63	0%	51%
Expansion (RP2D)	16	~13% (2 PR)	44%

Two confirmed partial responses were observed. Both occurred in patients with heavily-pretreated endometrial cancer.

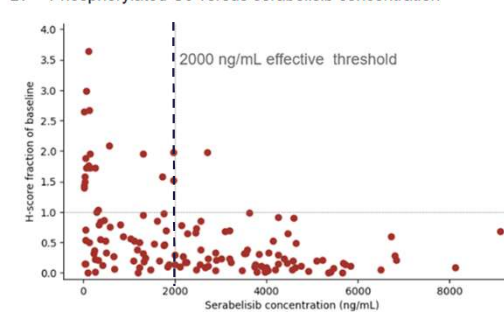
PK-PD Analysis in human subjects (C32001)



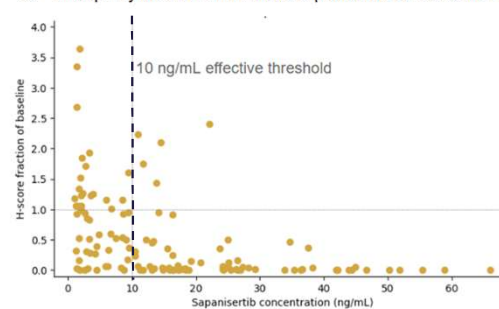
A. Phosphorylated-S6 versus sapanisertib concentration



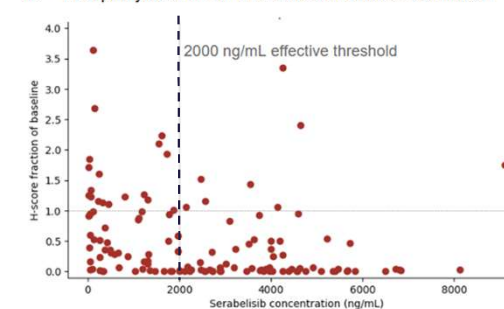
B. Phosphorylated-S6 versus serabelisib concentration



C. Phosphorylated-4E-BP1 versus sapanisertib concentration

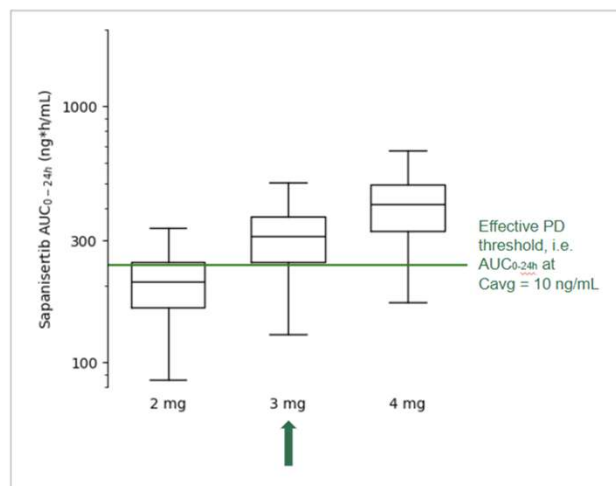


D. Phosphorylated-4E-BP1 versus serabelisib concentration



Dose selection based on human PK-PD (C32001)

Sapanisertib AUC 2-4 mg doses versus effective PD threshold



Serabelisib AUC 100-300 mg doses versus effective PD threshold

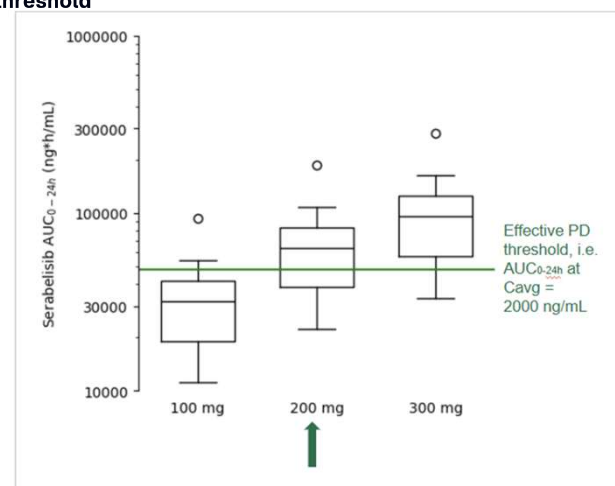
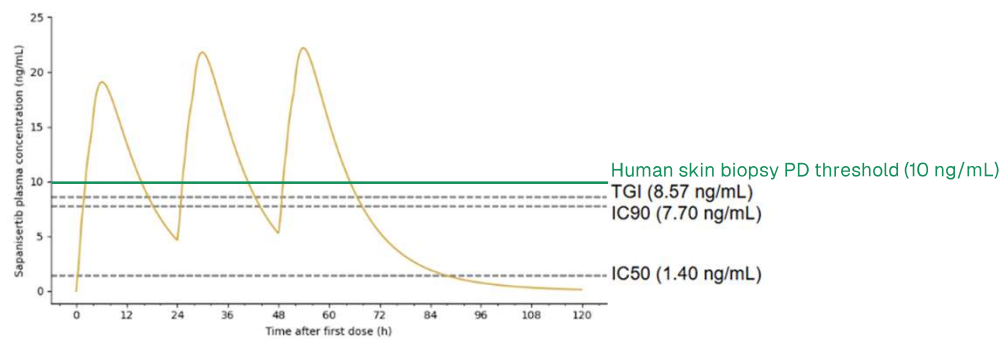


Figure 3. Dose selection based on effective drug thresholds from human PK-PD data. PK data from the study described in Figure 2 was used to compare the exposure (AUC) for (A) sapanisertib at 2 mg, 3 mg and 4 mg doses, and (B) serabelisib at 100 mg, 200 mg and 300 mg doses with the AUCs determined in Figure 2 to be pharmacodynamically effective drug concentrations. The plots show that doses of 3 mg sapanisertib + 200 mg serabelisib represent to lowest doses where the mean AUC is above the effective drug threshold. This data suggests that these doses will provide drug exposures which combine effective on-target PI3K/AKT/mTOR pathway inhibition while subjecting patients to pharmacodynamically effective drug exposures with the lowest risk of drug-related toxicities.

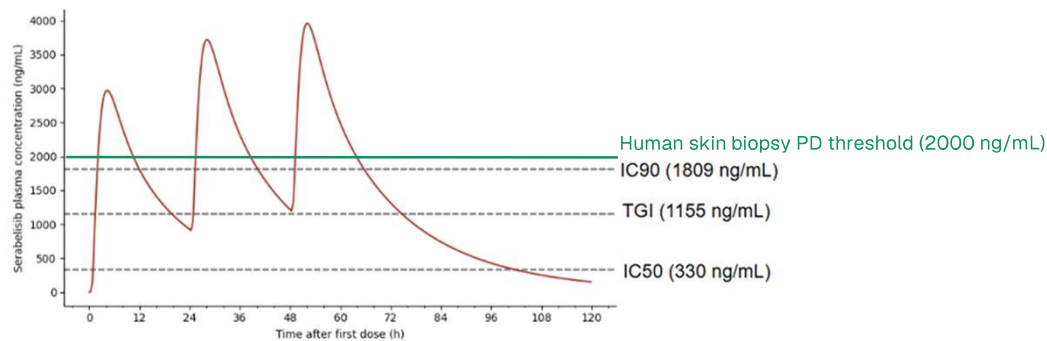
Endometrial RP2D PK versus PD / efficacy thresholds

Sapanisertib



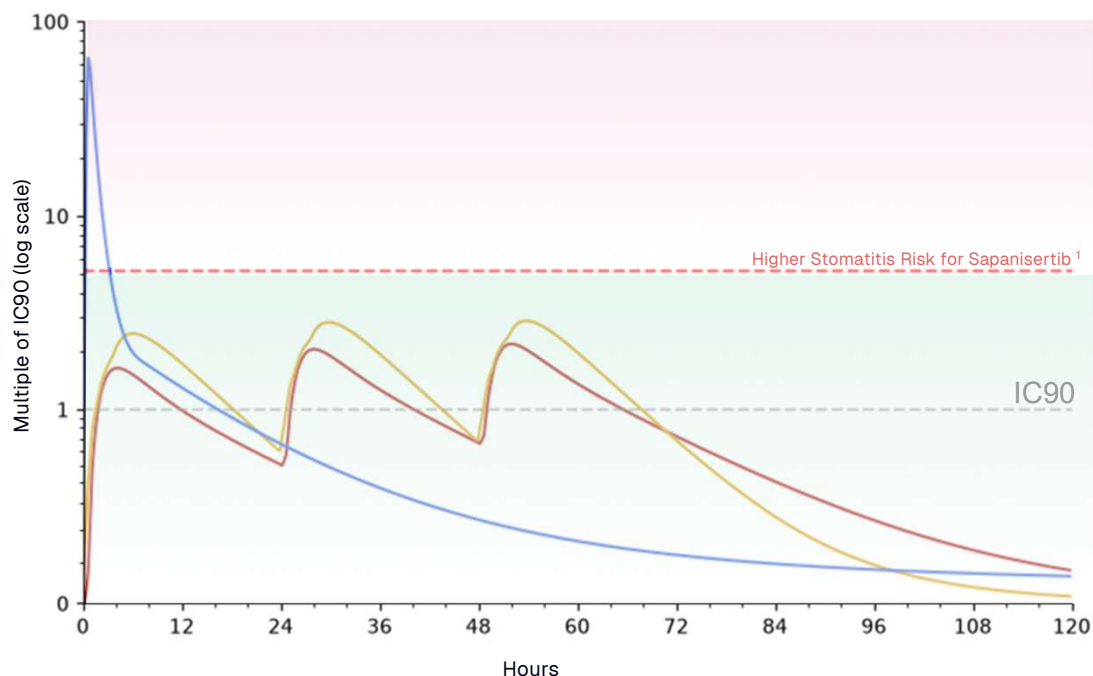
Sapanisertib	Per week	Per 28-day cycle
Time above IC90 threshold (hours)	44.4	177.5
Time above IC50 threshold (hours)	77.6	310.4
Time above TGI threshold (hours)	43.4	173.6
Time above PD threshold (hours)	41.2	164.7

Serabelisib



Serabelisib	Per week	Per 28-day cycle
Time above IC90 threshold (hours)	42.3	169.3
Time above IC50 threshold (hours)	94.8	379.0
Time above TGI threshold (hours)	62.8	251.0
Time above PD threshold (hours)	37.6	150.2

Oral PIKTOR dosing is designed for sustained exposure with reduced C_{max}-related toxicity risk



	Peak exposure (C _{max} : IC ₉₀ ratio)	Time above IC ₉₀ / week	Plasma protein binding / free fraction	
			Bound	Free
 Serabelisib	2.2	42 h	69%	31%
 Sapanisertib	2.9	44 h	70%	30%
 Gedatolisib	~65	15 h	98%	2%

The ratio of plasma drug concentration to cellular IC₉₀ from 0 - 120 hours was calculated for serabelisib, sapanisertib, and gedatolisib. Sapanisertib (3 mg) with serabelisib (200mg), with food, QDx3dQW plasma drug concentration was obtained from Maddocks *et al.*, 2026¹. Gedatolisib (180 mg QW) drug concentration data was derived from Shapiro *et al.*, 2015² by scaling 89 / 154 / 222 / 266 mg single dose data to 180 mg, assuming linearity. Mean gedatolisib plasma concentration at 180 mg was computed with a geometric mean per time-point across the scaled concentrations. The estimated time above IC₉₀ was computed as the time a stepwise linear interpolation crossed the threshold. IC₉₀ values for all drugs were calculated using IC₅₀ values presented in Tyrakis *et al.*, 2025³ and Slide 44 in this corporate deck. The cellular IC₅₀ values for all three drugs were derived from an average of six cell lines representing clinically relevant models i.e. HR+/HER2- breast cancer cell lines and endometrial cancer cell lines: MCF7, T47D, AN3CA, HEC1B, MFE-280, MFE-296. Dashed red line denotes the sapanisertib C_{max}/IC₉₀ ratio for the combined dose of sapanisertib and serabelisib (4 mg / 200 mg, QDx3dQW, fasted) resulting in Grade 3 stomatitis in a Phase 1b dose escalation study (NCT01899053, described in Maddocks *et al.*, 2026¹).

¹ Exposure/Response Analysis of an Oral Multi-Node PI3K/AKT/mTOR Pathway Inhibitor Combination of Serabelisib and Sapanisertib. Maddocks *et al.*, STOP Cancer Conference, 2026

² First-In-Human Study of PF-05212384 (PKI-587), a Small-Molecule, Intravenous, Dual Inhibitor of PI3K and mTOR In Patients With Advanced Cancer. Clin Cancer Res. 2015 April 15; 21(8): 1888-1895. doi:10.1158/1078-0432.CCR-14-1306.

³ Multi-node inhibition targeting mTORC1, mTORC2 and PI3Ka potentially inhibits the PI3K/AKT/mTOR pathway in endometrial and breast cancer models. British Journal of Cancer, 133: 144-154. <https://doi.org/10.1038/s41416-025-03035-z>

Dr. Debbie Chirnomas



Dr. Debbie Chirnomas,
MD MPH

Current role

- Chief Medical Officer, Faeth

Background



Arvinas

VP, Oncology Clinical Development



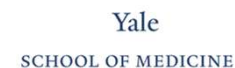
Gamida Cell

VP, Clinical Development



Pfizer

Medical Director / Medical Strategy Head



Yale School of Medicine

Assistant Professor, Director of Pediatric Stem Cell Transplant

FAETH THERAPEUTICS

Clinical

OCTOBER 6, 2026

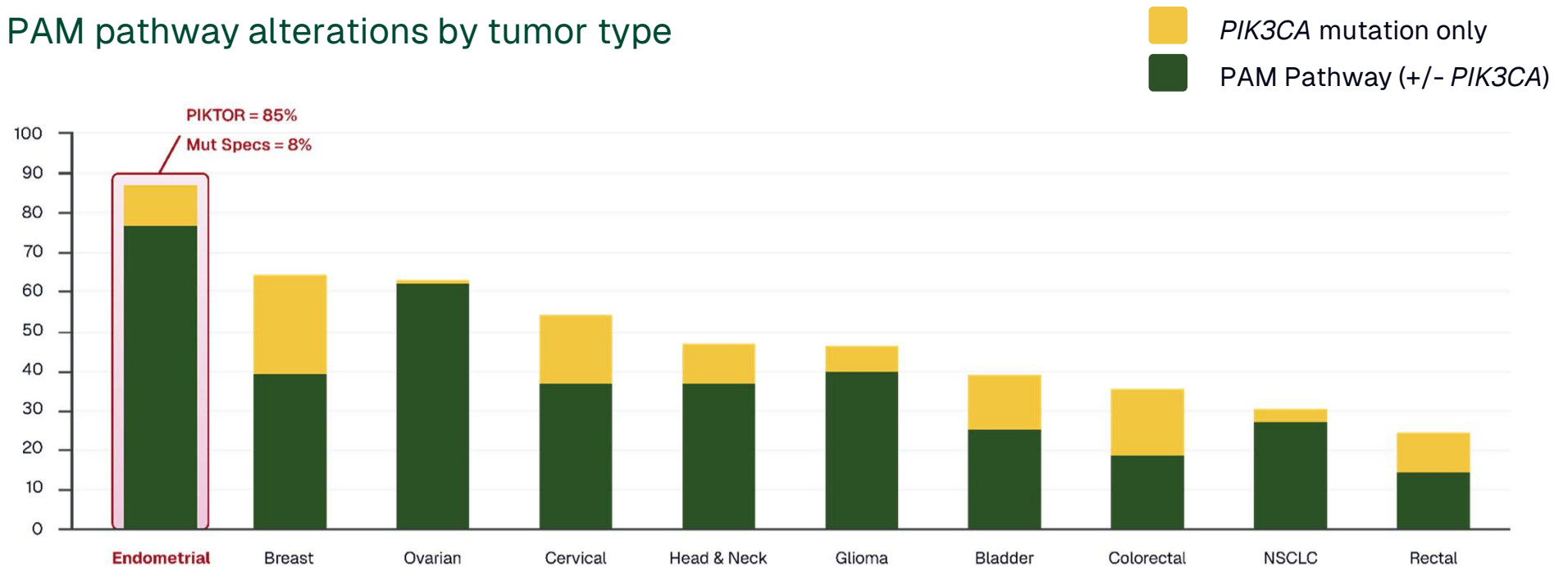
Overview

Dr. Debbie Chirnomas, CMO

- 1 The 'Why' Behind Endometrial Endometrioid Cancer
- 2 How We Got Here
- 3 PIK-201 Today & Read-Through to Breast

Multi-nodal inhibition potentially broadens the treatable landscape relative to single-node approaches

PAM pathway alterations by tumor type



Endometrial Cancer (EC) is a rising and underserved indication

The most common gynecological cancer (+68k new U.S. cases annually)

Unlike many cancers, both new cases and mortality are increasing

High unmet need

Chemotherapy remains the backbone, with very limited 2L treatment options

Dominated by PI3K-pathway mutations

Found in 80-90% of tumors with no approved PI3K-pathway targeting agent

Metabolic in nature

Associated with metabolic syndrome and obesity

From 2015 - 2025

↑26%

Incidence

↑36%

Mortality

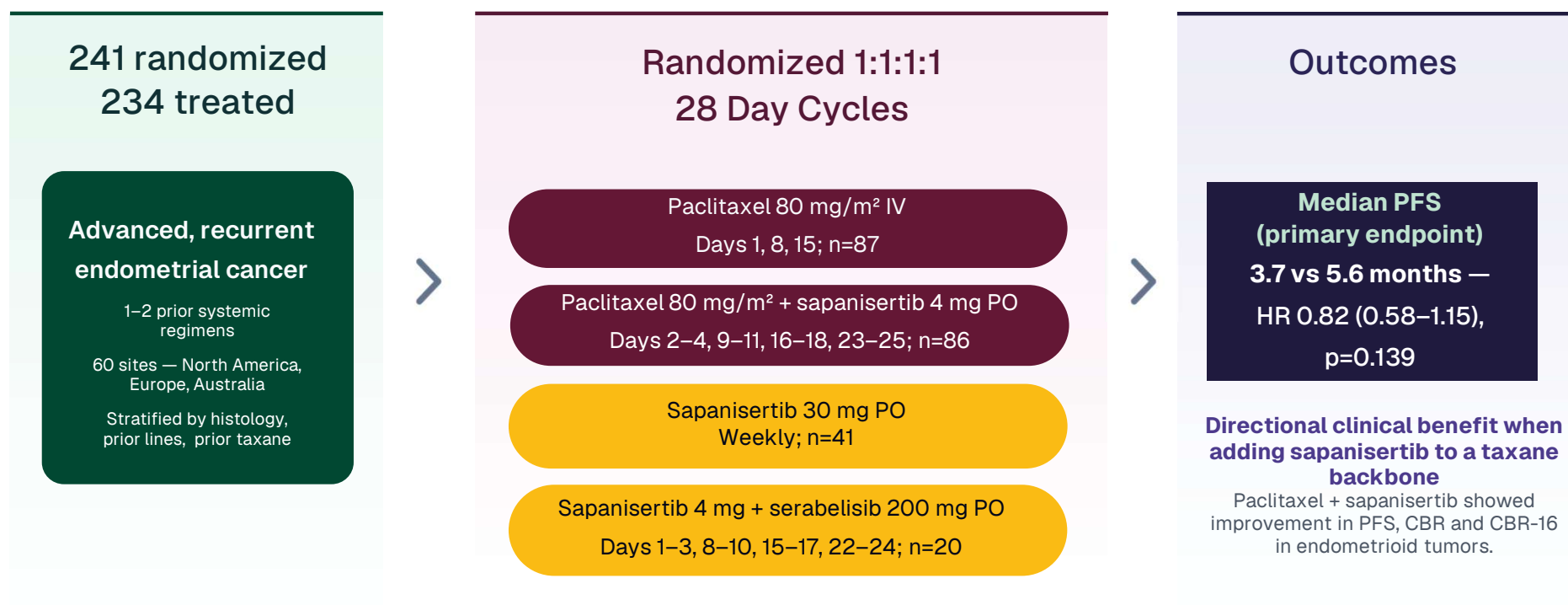
+68k

New U.S. cases/yr

ACS Cancer Facts and Figures, 2015 and 2025

How we got here: Han et al, 2023 Study Design

Randomized phase 2, open-label, four arms (NCT02725268)



Source: Han SN, Oza A, Colombo N, et al. A randomized phase 2 study of sapanisertib in combination with paclitaxel versus paclitaxel alone in women with advanced, recurrent, or persistent endometrial cancer. *Gynecologic Oncology* 2023;178:110–118. NCT02725268.
CBR, clinical benefit rate; CBR-16, CBR at 16 weeks; HR, hazard ratio; PFS, progression-free survival; TEAE, treatment-emergent adverse event.

How we got here: Han et al, 2023

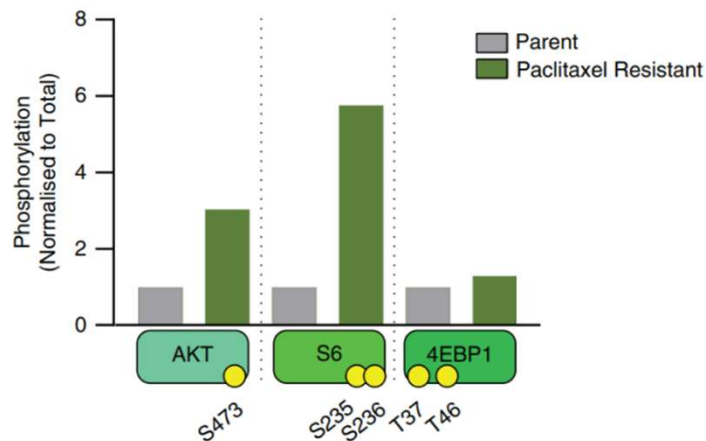
Safety population, n (%)	Paclitaxel n=87	Paclitaxel + Sapanisertib n=86	Sapanisertib n=41	Sapanisertib + Serabelisib n=20
CR	2 (2.3)	2 (2.3)	0	0
PR	14 (16.1)	19 (22.1)	2 (4.9)	0
SD	34 (39.1)	48 (55.8)	12 (29.3)	7 (35.0)
SD ≥16 weeks	16 (18.4)	23 (26.7)	5 (12.2)	1 (5.0)
ORR	16 (18.4)	21 (24.4)	2 (4.9)	0
<i>Odds ratio (95% CI) vs paclitaxel</i>	–	1.34 (0.64–2.81)	0.22 (0.04–1.19)	0.00
CBR	50 (57.5)	69 (80.2)	14 (34.1)	7 (35.0)
<i>Odds ratio (95% CI) vs paclitaxel</i>	–	2.84 (1.43–5.63)	0.42 (0.19–0.92)	0.47 (0.16–1.37)
PFS in months	3.7	5.6	–	–
<i>Hazard ratio (95% CI) vs paclitaxel</i>	–	0.82 (0.58–1.15)	–	–

• Phase 2, open-label, 4-arm randomized study (1:1:1:1) in advanced, recurrent or persistent endometrial cancer after 1–2 prior regimens; 28-day cycles. Primary endpoint was investigator-assessed PFS (RECIST v1.1); ORR, CBR and CBR-16 were secondary endpoints.
 • Arms: paclitaxel 80 mg/m² IV days 1, 8, 15; paclitaxel 80 mg/m² + sapanisertib 4 mg PO days 2–4, 9–11, 16–18, 23–25; sapanisertib 30 mg PO weekly; sapanisertib 4 mg + TAK-117 200 mg PO days 1–3, 8–10, 15–17, 22–24. The sapanisertib and sapanisertib + TAK-117 arms closed early for futility.

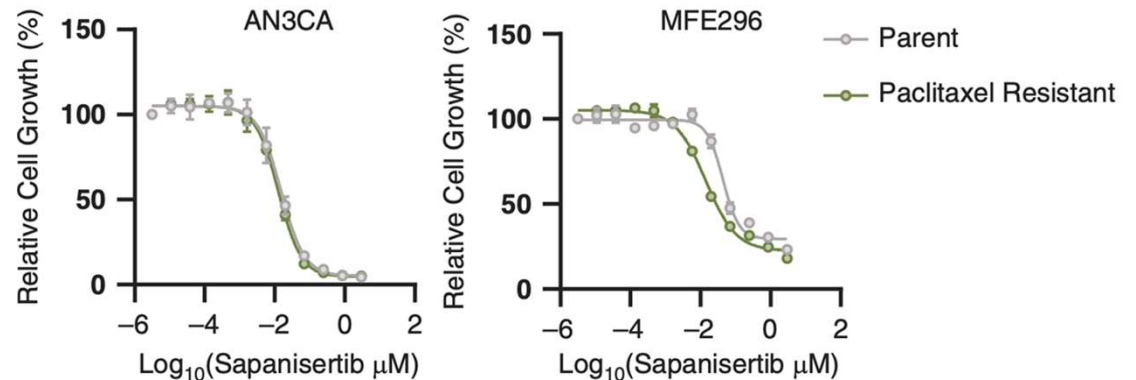
PFS, progression-free survival; CBR, clinical benefit rate (ORR + stable disease); CBR-16, CBR at 16 weeks; CI, confidence interval; CR, complete response; ORR, overall response rate; PR, partial response; SD, stable disease. Median PFS (primary endpoint) was 3.7 months with paclitaxel versus 5.6 months with paclitaxel + sapanisertib (HR 0.82; 95% CI 0.58–1.15; p = 0.139); the primary endpoint was not met. Source: Han SN, Oza A, Colombo N, et al. A randomized phase 2 study of sapanisertib in combination with paclitaxel versus paclitaxel alone in women with advanced, recurrent, or persistent endometrial cancer. *Gynecologic Oncology* 2023;178:110–118 (Table 2). NCT02725268.

Taxane resistance activates PI3K/AKT/mTOR pathway signaling in cancer models, sensitive to inhibition by PIKTOR

Taxane resistance activates PAM Pathway



Taxane resistant cells retain sensitivity to PIKTOR



Building on the Starks et al Phase 1b

Advanced Solid Tumors

Advanced solid tumors

All-comers (mutation agnostic)

Second line or later³



Sapanisertib / Serabelisib

Cohort 1: 2mg / 100mg (Pac 60mg/m²)

Cohort 2: 2mg / 200mg (Pac 60mg/m²)

Cohort 3: 2mg / 200mg (Pac 80mg/m²)

Cohort 4: 3mg / 200mg (Pac 80mg/m²)

Cohort 5: 4mg / 200mg (Pac 80mg/m²)

RP2D Expansion

**RP2D = <1/3
Monotherapy Dose**

Serabelisib

900 mg

QD x 3dQW PO¹

Combo RP2D **4.5x** less than mono

Sapanisertib

9 mg

QD x 3dQW PO²

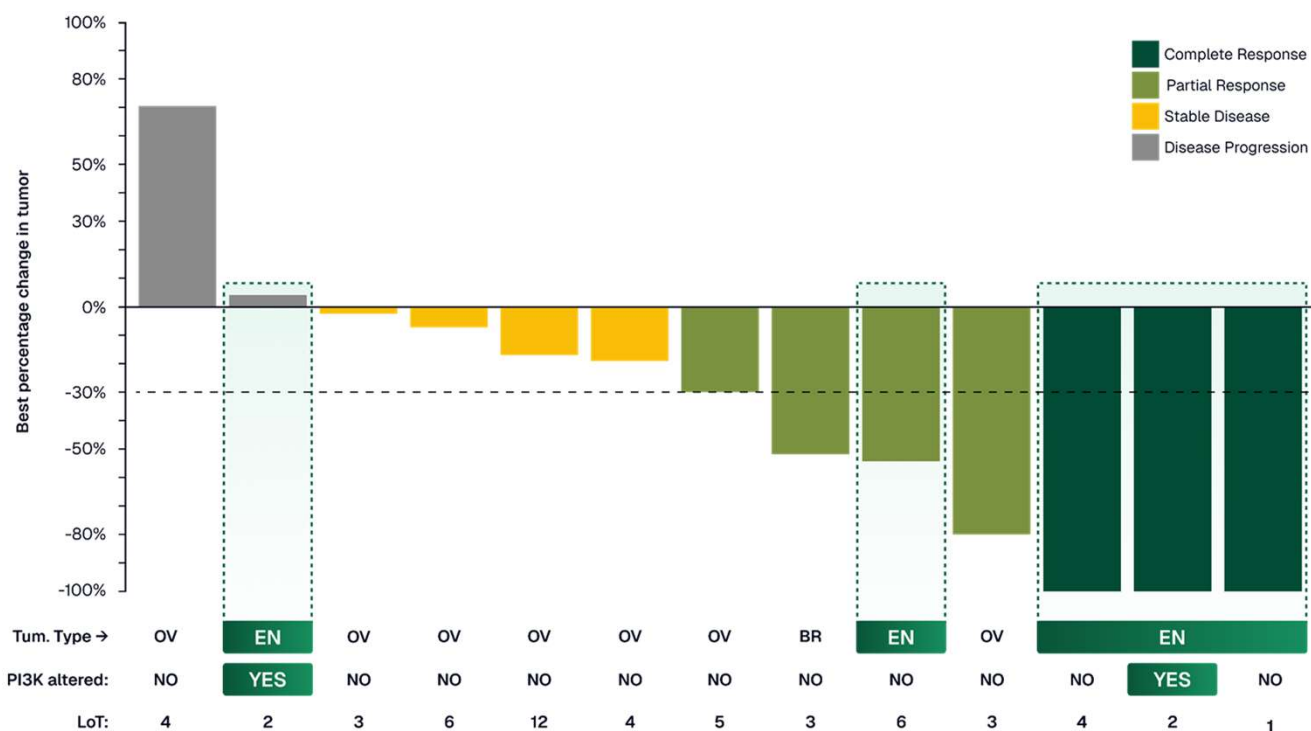
Combo RP2D **3x** less than mono

Data sources: ¹Juric et. al., Clin. Cancer Res. 23 (17) 2017. ²Voss et. al., Br. J. Cancer 123 (11) 2020. ³Starks et. al., Gyn. Onc. 166, 2022.

Abbreviations: RP2D - Recommended Phase 2 Dose, 'Pac' - Paclitaxel

Ph1b: PIKTOR + paclitaxel drove deep, durable responses

80% ORR in endometrial cancer



ALL PATIENTS (RECIST EVALUABLE)

47% overall response rate

73% clinical benefit rate

71% ORR in patients with PI3K pathway mutation

- 7 response evaluable patients with PI3K pathway mutation
- 5 responses (3 CR, 2PR)

ENDOMETRIAL PATIENTS ONLY

80% overall response rate (4/5)

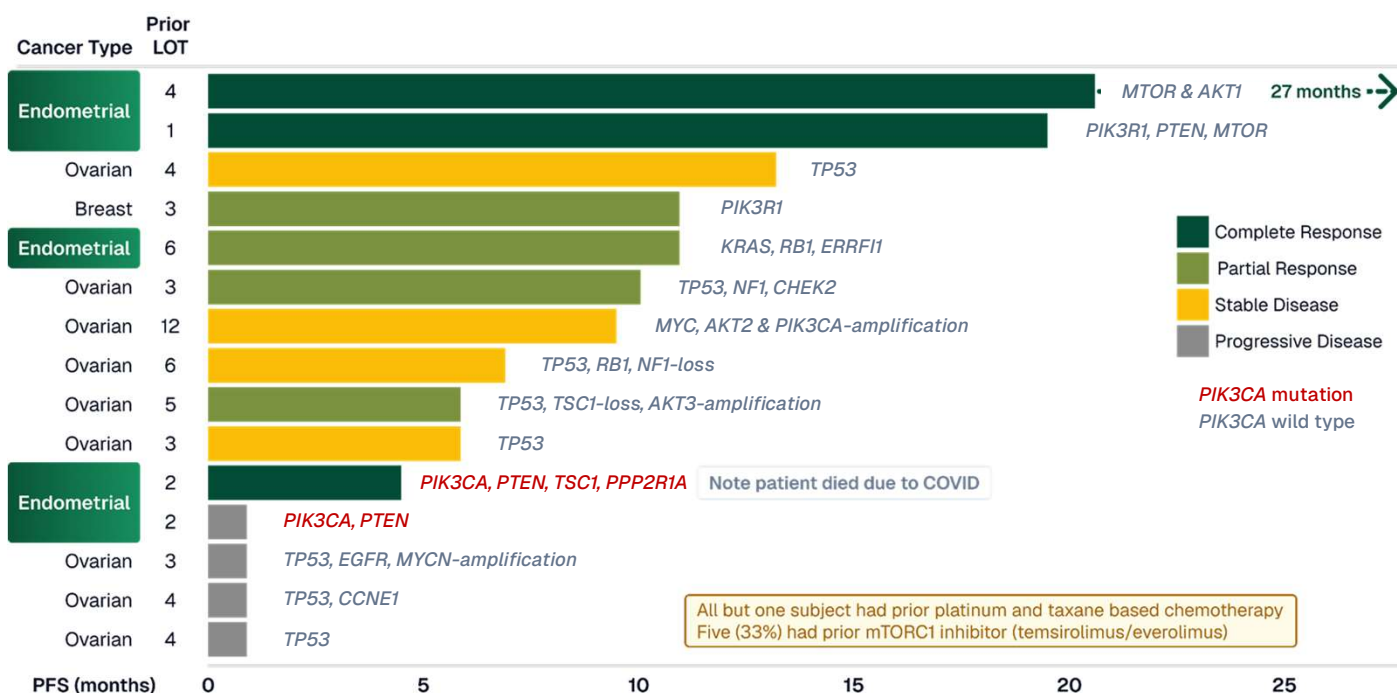
3 complete responses

11 mo median PFS

Range 0–26.9 mo · avg 3 prior lines (range 1–6)

n=19 enrolled (15 response evaluable, 13 RECIST evaluable); endometrial subgroup n=5. Data cutoff per publication 10/1/21. Starks DC, Rojas-Espallat L, Meissner T, Williams CB. Phase I dose escalation study of dual PI3K/mTOR inhibition by sapanisertib and serabelisib in combination with paclitaxel in patients with advanced solid tumors. Gynecologic Oncology. 2022 Jul 15. Abbreviations: ORR – Overall Response Rate; CBR – Clinical Benefit Rate; CR – Complete Response; PFS – Progression-Free Survival; Ov – Ovarian; En – Endometrial; Br – Breast.

Ph1b: PIKTOR + paclitaxel showed durable responses in both *PIK3CA*mut and wt patients



All RECIST Available Patients

47% Overall Response Rate

71% ORR in patients with PI3k pathway mutation

73% Clinical Benefit Rate in evaluated patients

Endometrial patients only

80% Overall Response Rate

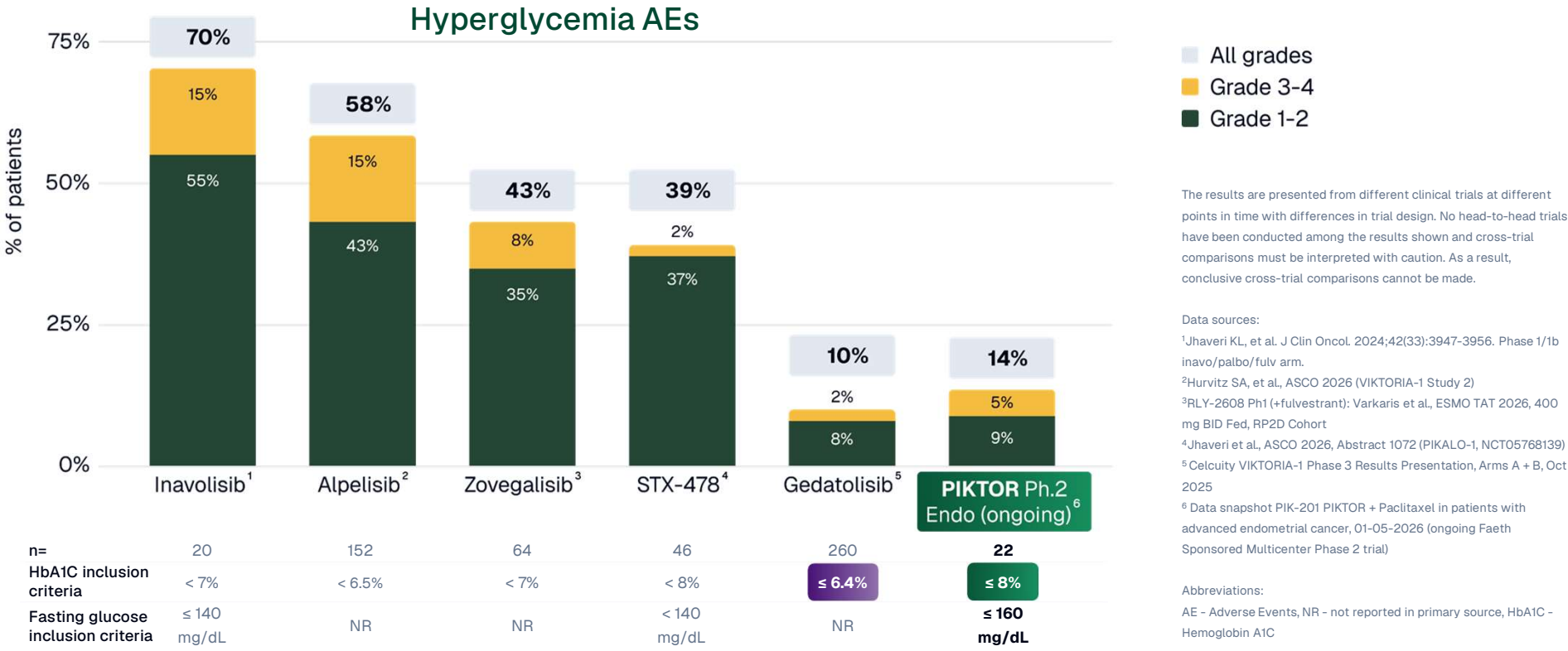
80% Clinical Benefit Rate

11 mo Median PFS (Range 0 – 26.9 mo)
Avg 3 prior lines of therapy (range 1 – 6)

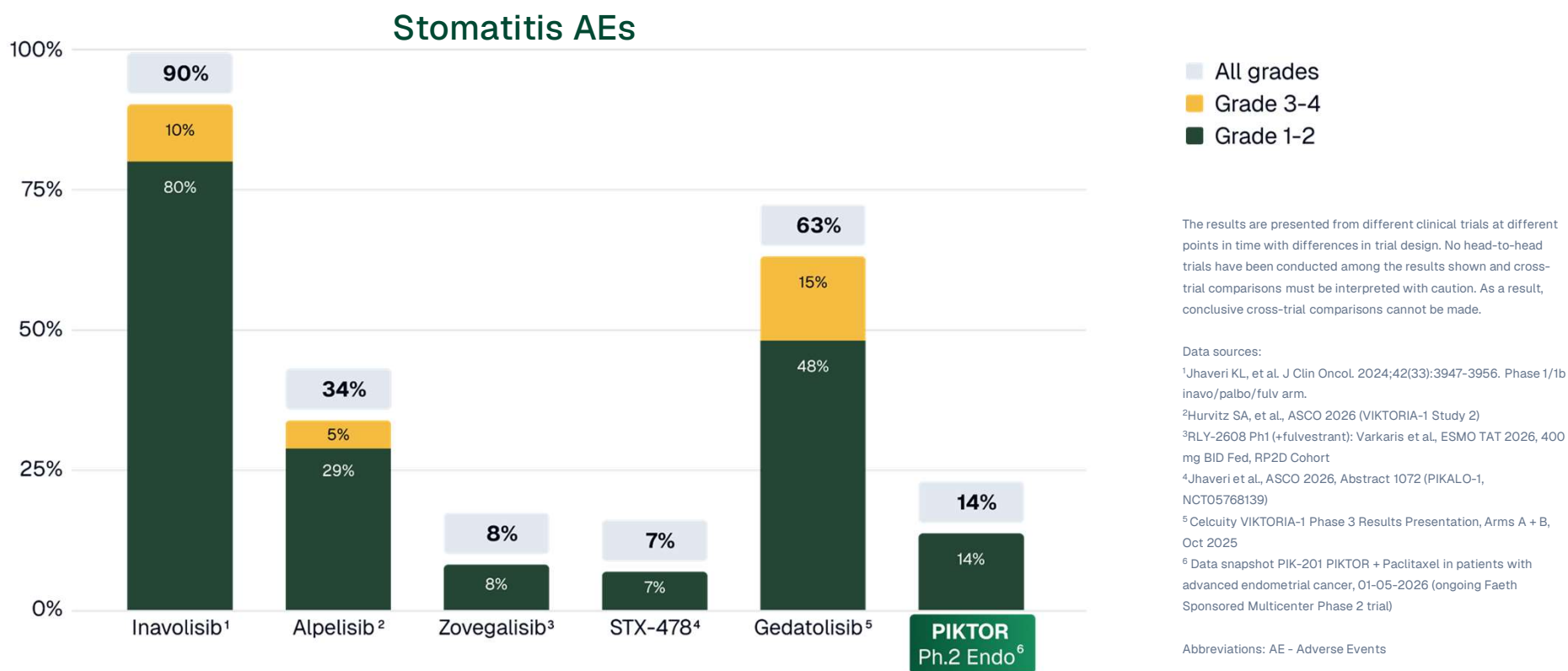
n=19 enrolled (15 response evaluable, 13 RECIST evaluable). Data cutoff per publication 10/1/21.

Starks DC, Rojas-Espallat L, Meissner T, Williams CB. Phase I dose escalation study of dual PI3K/mTOR inhibition by Sapanisertib and Serabelisib in combination with paclitaxel in patients with advanced solid tumors. Gynecologic Oncology. 2022 Jul 15. Abbreviations: PI3KCAmt - with mutation in PI3KA, wt - wild type, LOT - Lines of Therapy, ORR - Overall Response Rate, CBR - Clinical Benefit Rate, PFS - Progression Free Survival

Hyperglycemia: PIKTOR compared to other PI3K compounds



Stomatitis: PIKTOR compared to other PI3K compounds



PIK-201: A Ph2 Study with PIKTOR + paclitaxel in advanced endometrial cancer (currently enrolling)

Eligibility Criteria

- Endometrioid endometrial cancer
- 2nd line or later
- Post ICI and chemotherapy
- Must have PI3K/AKT/mTOR pathway mutation



Single arm (n $\cong$ 40)

- Sapanisertib 3 mg
- Serabelisib 200 mg
- Paclitaxel 80 mg/m²

Optional sub-study; diet to suppress glucose / insulin

Primary endpoint

- ORR

Secondary endpoints

- PFS
- CBR
- Safety/ tolerability
- DOR
- OS

Exploratory endpoints

- PK
- Efficacy endpoints vs. specific mutations

Study is being conducted in partnership with:



Endometrioid and HR+/HER2- BC share ER/PR dependence

Endometrioid

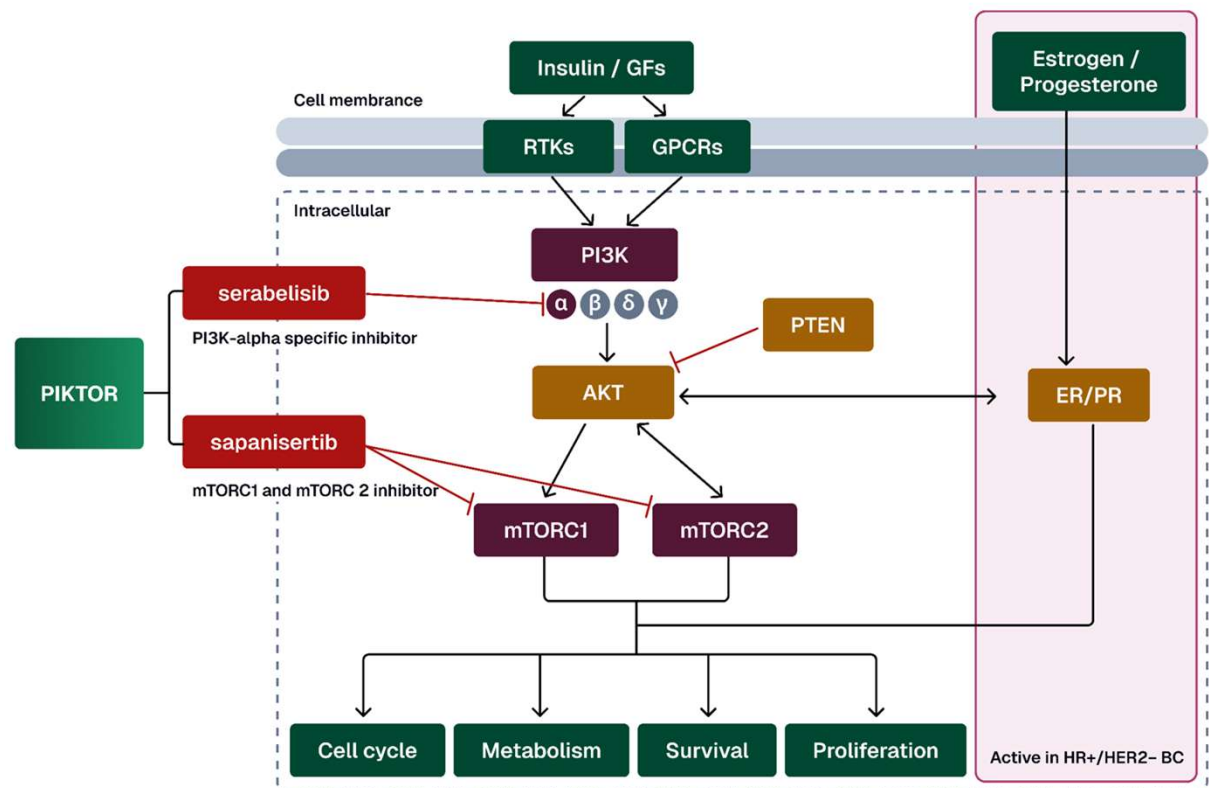
~80% of cases | hormone-dependent

- Estrogen driven — unopposed estrogen from obesity, anovulation, PCOS, and other causes
- **ER/PR high**
- **PAM alterations in >80% of cases**

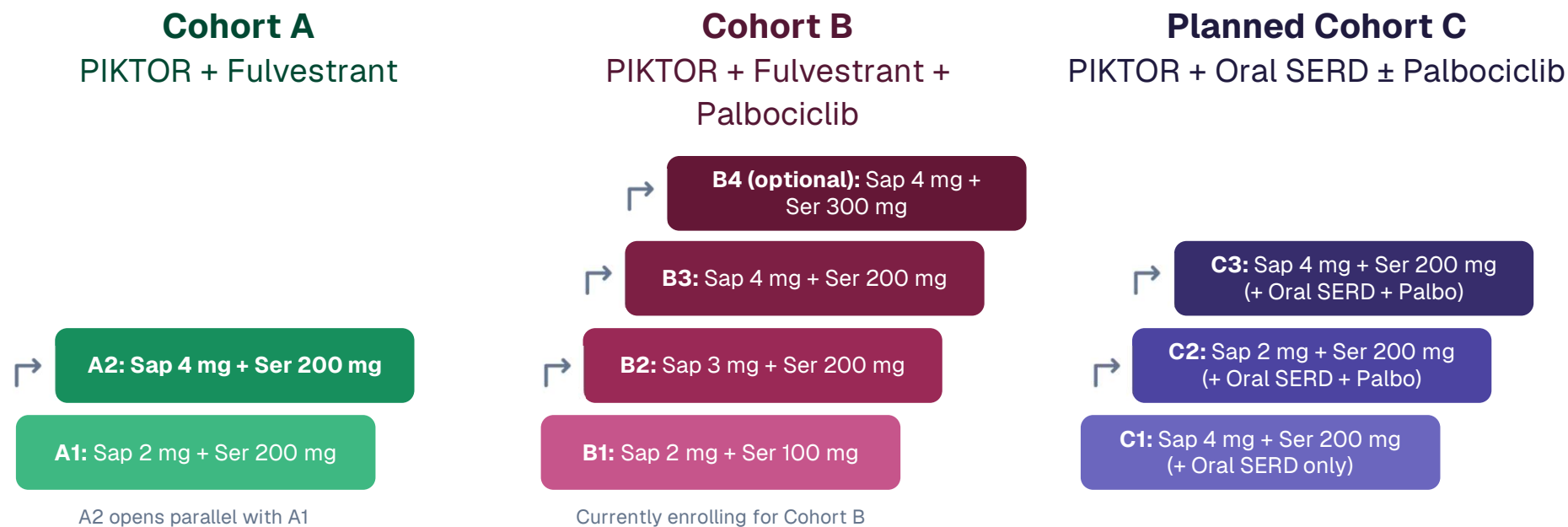
Non-endometrioid (serous, clear cell, undifferentiated)

~20% of cases | hormone-independent

- Not estrogen-driven: arises in atrophic endometrium, older patients, often lower BMI
- **ER/PR low or absent**
- **PAM alterations uncommon (<15%).** TP53 mutated ~90%. HER2 amplified ~25–30%



Breast: Ongoing Phase 1b/2 trial designed to rapidly identify recommended dose and initiate expansion cohorts



Brian Stephenson



Brian Stephenson,

PhD, CFA

Current roles

- Chief Financial Officer, Faeth

Background



BridgeBio Pharma
CFO and Co-founder



Capital IP Investment Partners
Partner and Head of Life Sciences



Leerink Partners
Director and Vice President



McKinsey & Company
Manager



Massachusetts Institute of Technology
PhD and MS, Chemical Engineering

FAETH THERAPEUTICS

Market Opportunity

OCTOBER 6, 2026

Overview

Brian Stephenson, CFO

- 1 Endometrial Cancer: Market opportunity
- 2 PIK-201 Benchmarks for success
- 3 HR+ Breast Cancer: Market opportunity

2L+ endometrial market, rising unmet need, one PAM-pathway option

Expanding market

Endometrial cancer is a growing market and recently overtook ovarian as the most deadly gynecological cancer (per SEER statistics)

High unmet need

Paclitaxel rechallenge: 15% ORR, 3–4 mo PFS¹.
ADCs are moving up in the treatment order, creating challenges for TOPO1 sequencing

Only multi-nodal PAM option

PAM alterations occur in ~80% of all EC patients.
No PI3K inhibitor ever approved in EC²

ADC

6+ sponsors · Ph2/3³

AstraZeneca 

 Daiichi-Sankyo

 MERCK

 Genmab

abbvie

PAM

1 oral multi-node program

 faeth

PIKTOR

Oral, intermittent dosing

1. Single-agent chemotherapy control arm, KEYNOTE-775 (Makker et al., NEJM 2022); 2. TCGA 2013; FDA approvals database. 3. Per publicly registered Phase 3 eligibility criteria (clinicaltrials.gov).

Relevant benchmarks for 2L+ Endometrial Cancer

- **Patients lack a consensus 2L+ standard-of-care once surgery with curative intent is no longer an option**
- Re-challenge with taxane-based chemotherapies (paclitaxel or docetaxel) is the most common approach
- Patients with endometrioid histology are sometimes offered everolimus + letrozole (which is on NCCN guidelines, not formally approved by FDA)

Approved and investigational benchmarks for 2L+

Treatment	Class	Prior Chemo + I/O	ORR	mPFS	Tolerability (d/c rate)
Rechallenge ¹	Chemo	No	15%	3.8 months	10%
Len + Pem ¹	I/O + VEGF	No	32%	7.2 months	35%
TOPO1 ²	ADC	Yes/No	30-60%	6-7 months	5%
Everolimus + Letrozole ^{3,4}	Hormonal	No	20-30%	3-6 months	10%

**Numbers have been rounded to the nearest whole for consistency*

1. Makker V et al, *N Engl J Med* 2022;386:437-48 (primary analysis; histology subgroup hazard ratios per supplementary appendix); 2. *Nat Rev Clin Oncol*. 2026 Sep;23(9):672-693; 3. *Gynecol Oncol*. 2022 Mar;164(3):481-491; 4. *J Clin Oncol*. 2015 Mar 10;33(8):930-6

KEYNOTE-775 in more detail

- **KEYNOTE-775** readout in 2022 and at the time was the largest ever randomized trial in 2L endometrial cancer
- It tested **pembrolizumab + lenvatinib** vs. **chemotherapy rechallenge**.
- Chemotherapy rechallenge had **15% ORR (95% CI between 11–18%)** and **median PFS of 3.8 months**

Efficacy — Primary analysis

Endpoint	Len + Pembro (n = 411)	Chemo (n = 416)	Histology	PFS — HR (95% CI)	OS — HR (95% CI)
ORR	32% (27-37%)	15% (11-18%)	Endometrioid	0.52 (0.41–0.65)	0.65 (0.49–0.84)
Median PFS (BICR)	7.2 mo	3.8 mo	Non-endometrioid	0.56 (0.43–0.73)	0.55 (0.42–0.72)
Median OS	18.3 mo	11.4 mo			

Endometrioid histology was ~60% of enrolled patients. **Outcomes were similar in endometrioid and non-endometrioid**

TroFuse-005: The first large readout in a post-chemo + I/O setting

- **Chemo I/O has since replaced platinum chemotherapy as the frontline treatment of choice in endometrial cancer**
- KEYNOTE-775 is no longer the most contemporaneous bar. All patients in PIK-201 require prior treatment with chemo I/O
- **TroFuse-005**, which tested Merck's Sac-TmT against chemotherapy rechallenge, **will readout at**  (LBA11)

TroFuse-005 is testing Sac-TmT at the 4 mg/kg dose. In Phase 2, the 4 mg/kg dose demonstrated a 31% ORR (vs. 34% at the higher 5 mg/kg dose), with a 6 month median PFS.

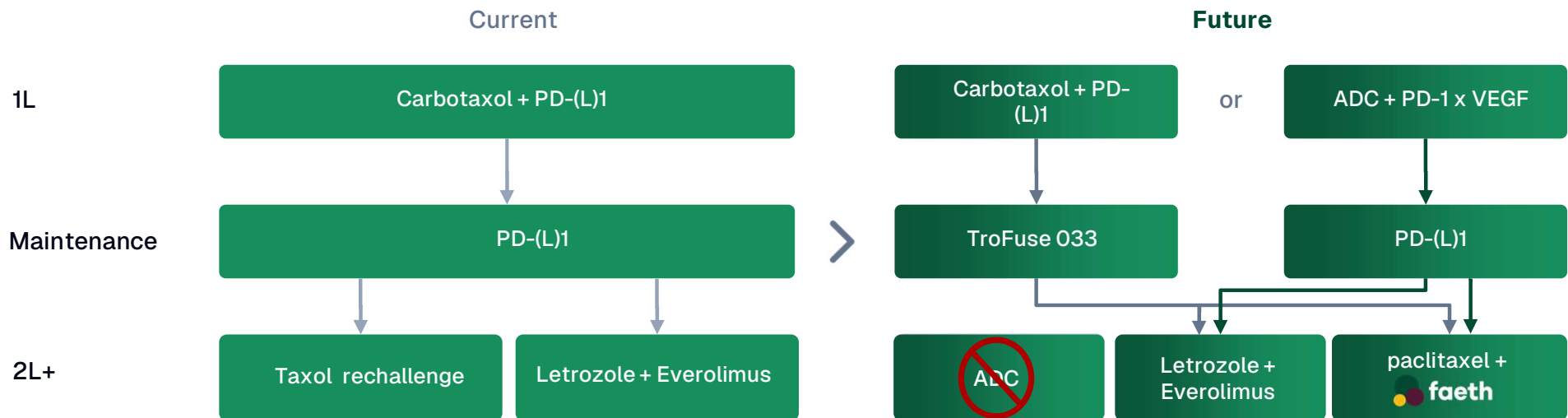
Efficacy benchmarks from Sac-TmT's China-based Phase 1/2 trial¹

Endpoint	4 mg/kg (RP2D)	5 mg/kg
ORR	31%	34%
95% CI	(22%-40%)	(20%-50%)
Median PFS	6 months	7.3 months
Median OS	14 months	18 months

1. Phase 1/2 trial (2870-001/KL264-01) presented at IGCS 2025; abstract LB014

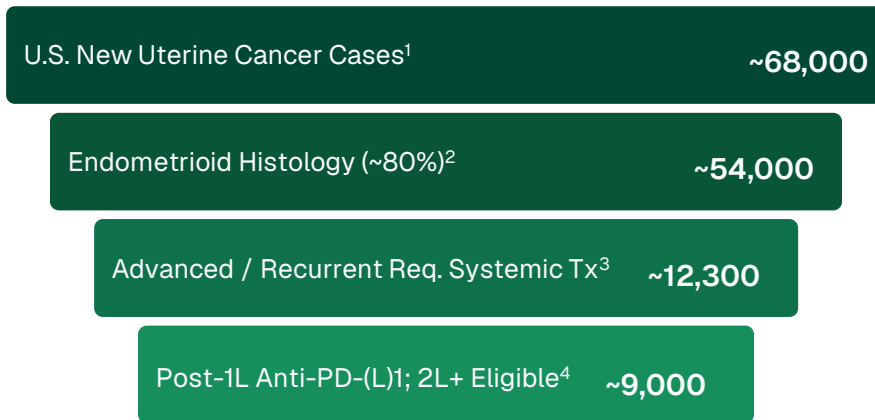
Potential changes to frontline could favorably impact PIKTOR

- Merck is currently exploring Sac-TmT in the frontline maintenance setting (**TroFuse-033**)
- Given the inability to sequence TOPO1-based ADCs, we would expect other developers to follow-suit
- We anticipate that increased usage of TOPO1-based ADCs in frontline settings would lessen their use later line



Estimated PIKTOR addressable U.S. population (2L+)

U.S. Incident Therapeutic Build (Annual)



Anticipated FDA Label Population

Post-1L advanced/recurrent endometrioid EC

- Endometrioid histology
- Advanced or recurrent, not amenable to curative surgery/radiation
- Progressed on/after prior anti-PD-1/PD-L1-containing regimen and TOPO1-based ADC (if available)

~9,000

U.S. incident patients per year
post-anti-PD-(L)1, endometrioid EC

~\$1.5B

US TAM⁵

1. ACS 2026 / SEER 2025: ~68,000–69,000 new uterine corpus cases/yr. 2. Endometrioid histology ~80% ((SEER-based series, 80–85%; Clarke, JAMA Oncol 2019)).

3. Advanced/recurrent requiring systemic Tx (~12,300/yr): de novo advanced ~9,700 (54K × 18% FIGO III/IV; SEER ~11% distant + ~half of 18% regional; conservatively 18%) + steady-state early-stage recurrence ~2,550/yr (44K early-stage entrants/yr × ~8% cumulative recurrence, 27% vaginal-only excluded (Åkesson, Gynecol Oncol 2023; all-stage endometrioid cohort); 73% require systemic Tx).

4. ~9,000 addressable 2L+ patients/yr at steady state: 12,300 × ~95% (company assumption; IO+chemo NCCN-preferred post NRG-GY018/RUBY) × ~77% progression (company estimate) ≈ 9,000/yr addressable .5. Total Addressable Market (TAM): ~\$20K/28-day supply (TRUQAP WAC) × 231 days (median duration of treatment, lenvatinib + pembrolizumab arm, KEYNOTE-775; Makker et al, NEJM 2022) × ~9,000 patients ≈ \$1.5B.

Market size estimates reflect the stated assumptions on patient numbers, price and duration of therapy. They estimate the size of the treatment setting and are not forecasts of PIKTOR revenue or market share.

Breast Cancer: PAM blockade more than doubles addressable market relative to mutant-selective inhibitors

High unmet need

HR+/HER2- patients comprise ~70% of all metastatic breast cancer patients¹

Most patients lack PI3K mutations

PIKTOR is designed to **work in all patients**; not just the ~40% that have an activating *PIK3CA* mutation²⁻⁵

Oral convenience

PIKTOR is taken with food on a 3 days on/4 days off schedule

PI3K MUT

~40% of HR+/HER2- breast cancer have *PIK3CA* mutation²⁻⁵



+ multiple others

PI3K WT + PI3K MUT

100% of HR+/HER2- Breast Cancer



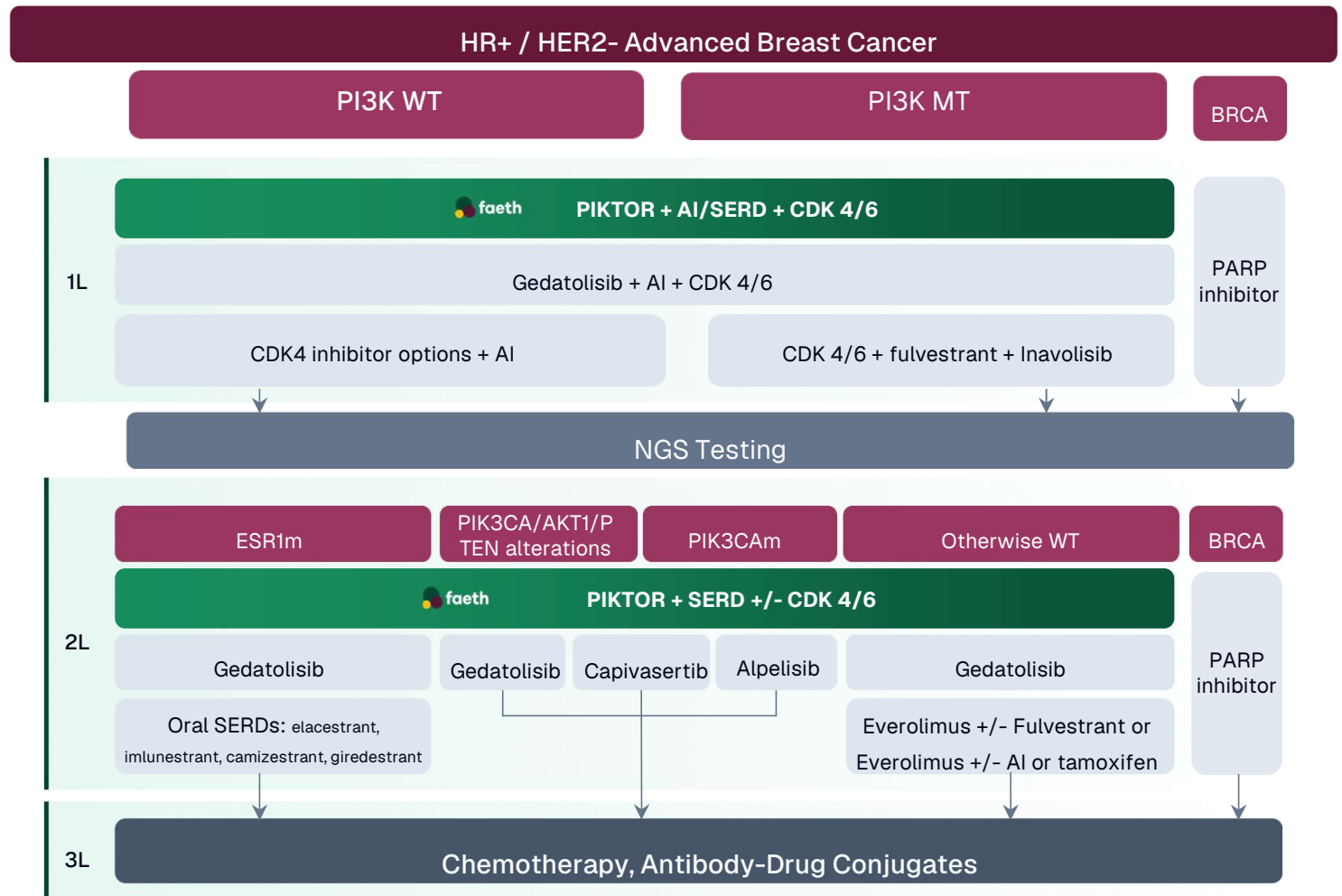
celcuity

1. Howlader 2014; SEER subtype rates 91.3/130.8 per 100K) = ~221K/yr

2. Tao JJ, et al. *JCO Precis Oncol* 2025 · 3. Zhang Y, et al. *Cancer Cell* 2017;31:820–832.e3 · 4. *Breast Cancer Res Treat* 2025, PMID 40931235 · 5. Krishnamurthy N, et al. *Front Oncol* 2020;10:1475

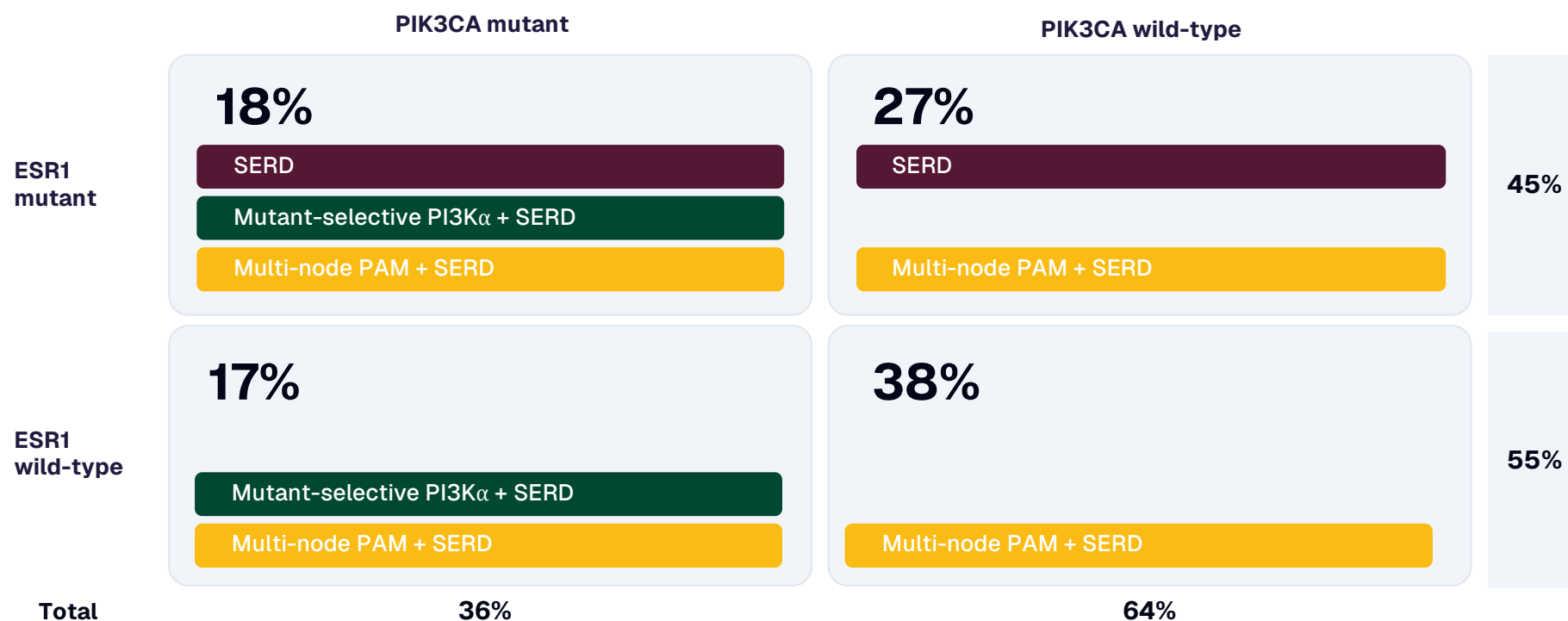
HR+/HER2- Advanced Breast Cancer potential future state

We believe **PIKTOR** has the potential to become **a differentiated all-oral option across first- and second-line settings**, with relevance across future standards of care.



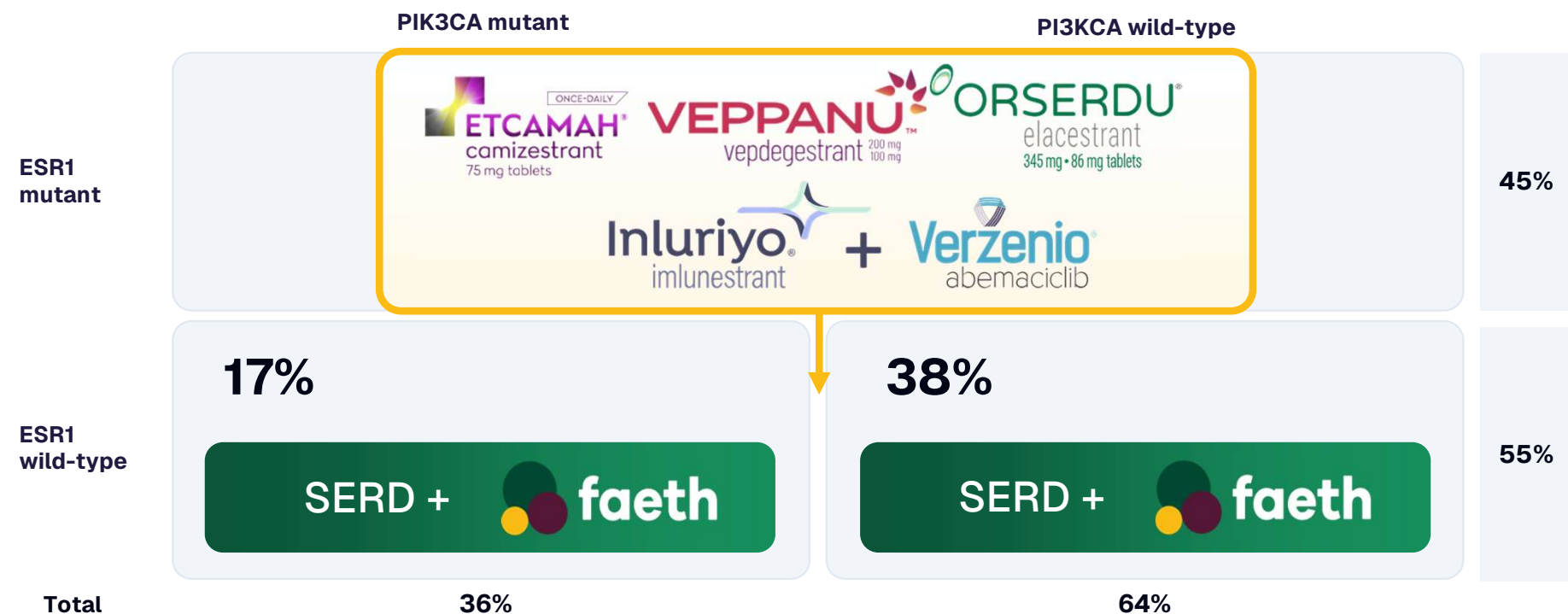
PIKTOR's reach potentially extends to virtually all 2L patients

Multi-nodal inhibitors (PIKTOR and gedatolisib) are the only PAM treatments that are designed to work **agnostic to mutational status**



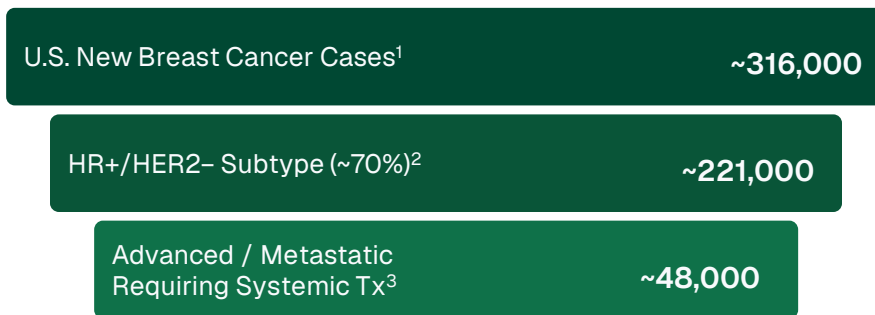
We believe PIKTOR could expand the reach of oral SERDs

To date, oral SERDs have only been approved in ESR1-mutant patients. Combining with PIKTOR **could enhance activity in wild-type patients** because **endocrine resistance often runs through PAM**.

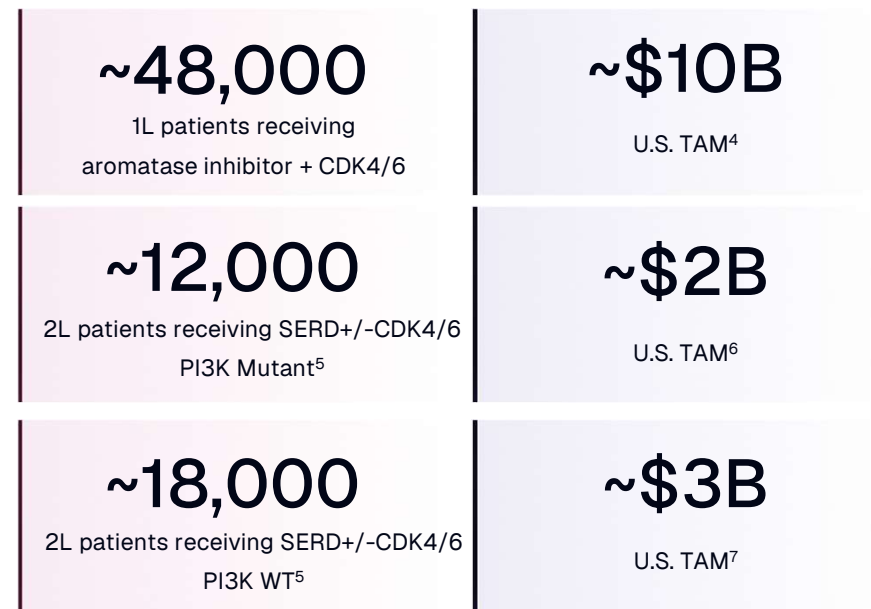


HR+/HER2- breast cancer: Large unmet need

U.S. Incident Therapeutic Build (Annual)



Three addressable patient populations from the ~48k advanced pool



SEER 2025: ~317K new U.S. female BC cases; ~42K BC deaths/yr (baseline for all three markets shown). 2. HR+/HER2- subtype ~70% of all BC (Howlader 2014; SEER subtype rates 91.3/130.8 per 100K) = ~221K/yr. 3. Advanced/metastatic requiring systemic Tx ~48K/yr: de novo stage IV ~13K (221K x ~6% stage IV at diagnosis (SEER)) + steady-state distant recurrence ~35K (208K early-stage entrants/yr x ~17% cumulative distant recurrence (company blend; Pan, NEJM 2017: 13-41% by stage)). 4. 1L TAM: ~48,000 incident 1L patients x ~\$17K/month (CDK4/6 inhibitor list price) x 12 months = \$10B. PIKTOR is not currently being studied in the first-line setting; 5. [2L post-AI+CDK4/6] Assume 42K annual deaths in US, of which ~70% are HR+/HER2- (per SEER statistics; ACS Facts and Figures 2026) as conservative estimate for the number of patients who progress post AI+CDK4/6. Of these, 40% are PI3K mutant, and 60% are PI3K wild-type. 6. 2L TAM for PI3K mutant patients: ~\$20K/28 days (Truqap WAC, conservative) x 11 mo (median PFS, VIKTORIA-1 PIK3CA-mutant triplet arm) x 12,000 = \$2.6B, rounded to ~\$2B; 7. 2L TAM for PI3K wild-type patients: ~\$20K/28 days (as fn 6) x 9 mo (median PFS, VIKTORIA-1 PIK3CA wild-type triplet arm) x 18,000 = \$3.2B, rounded to ~\$3B. Market size estimates reflect the stated assumptions on patient numbers, price and duration of therapy. They estimate the size of the treatment setting and are not forecasts of PIKTOR revenue or market share.

FAETH THERAPEUTICS

Closing Remarks

OCTOBER 6, 2026

\$186.4M¹ in cash expected to fund PIKTOR through anticipated readouts in endometrial cancer and breast cancer

PROGRAM	Achieved in 2026	2H 2026	2027	2028+
ENDOMETRIAL	✓ Fast Track Designation	Topline data readout; Ph2 enrollment ongoing	Last patient dosed + 6 months data	
BREAST	✓ Trial initiated; Ph1b dose escalation underway (first patient dosed Apr 2026)		- Interim safety data from dose escalation - Interim efficacy data; expansion cohorts initiated	Expansion data
INDICATION EXPANSION			Preclinical data on rare disease	

¹ \$186.4M cash, cash equivalents and marketable securities as of June 30, 2026.
Abbreviations: EC - endometrial cancer, BC - breast cancer

Thank you