

FAETH THERAPEUTICS COMPANY OVERVIEW

Break through the PAM barrier.

Oral inhibition of the PI3K/AKT/mTOR pathway, one of the most frequently altered pathways in cancer.

OCTOBER 2026

Forward-Looking Statements

Certain matters discussed in this Presentation may contain forward-looking statements made by Faeth Therapeutics, Inc. (the “Company”) that are, by their nature, subject to significant risks and uncertainties. Forward-looking statements can be identified by words such as “may,” “will,” “should,” “would,” “could,” “believe,” “expect,” “anticipate,” “intend,” “plan,” “continue,” “seek,” “estimate,” “potential” or the negative of these terms or other similar terms. Forward-looking statements in this Presentation include, but are not limited to, statements about: the use of proceeds from the February 2026 financing and cash runway expectations therefrom, including such proceeds funding the Company through key clinical milestones; the Company’s product candidates and the potential benefits thereof; planned and ongoing pre-clinical and clinical studies, including the timing for data readouts and future development milestones; the potential peak sales and estimated total addressable markets, currently and in the future, for the Company’s product candidates; the Company’s CMC infrastructure plans and positioning for growth, including expectations regarding DS/DP supply for ongoing and proposed clinical trials; and expectations regarding patent protection for the Company’s product candidates, including plans to file patent applications and the timing thereof. Such forward-looking statements reflect the current views of the Company’s management regarding future events; they are not guarantees of future performance.

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Oral PAM¹ regimen, built for longer target coverage without class-defining toxicities



PIKTOR

serabelisib (PI3K α) +
sapanisertib
(mTORC1/2)

COMPLETE PAM BLOCKADE

Oral multi-node blockade that hits PAM at the **three nodes that we believe matter**.

PK EDGE (TOLERABILITY AND EFFICACY)

Intermittent dosing schedule that has been observed to flatten the peaks, providing **~2-3x more time above IC90** than other PAM inhibitors with a **low rate of stomatitis**

VALIDATED PATHWAY WITH LARGE MARKET OPPORTUNITIES

~\$1.5B market opportunity in endometrial cancer³
~\$5-10B market opportunity in breast cancer⁴

CAPITALIZED THROUGH CATALYSTS

Expected to be fully funded through 2 major catalysts

- Phase 2 endometrial topline data expected by year-end 2026
- Interim Phase 1b/2 breast data expected 2H 2027

Top-tier investors including Montanova, Avoro, Baker Brothers, Cormorant, Fairmount, RA Capital Management, and Vivo Capital²

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

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)

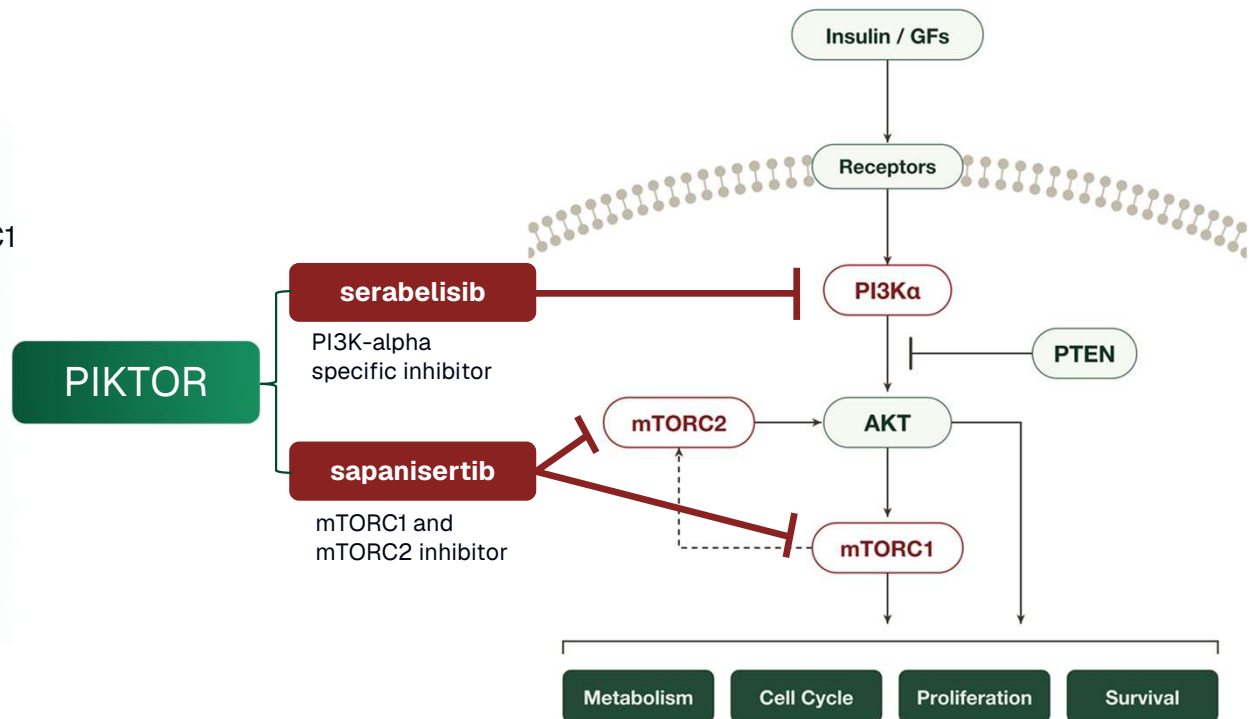
Greg Hannon, PhD

Former Director of CRUK Cambridge Institute

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.



Multi-node synergy designed to allow each agent to run using multiple-fold lower doses than monotherapy RP2D

		MONOTHERAPY RP2D	DOSE IN PIKTOR + PACLITAXEL ³	REDUCTION
	Serabelisib	900 mg QD×3dQW ¹	200 mg QD×3dQW	4.5× lower
	Sapanisertib	9 mg QD×3dQW ²	3 mg QD×3dQW	3× lower

Why the combination runs lower

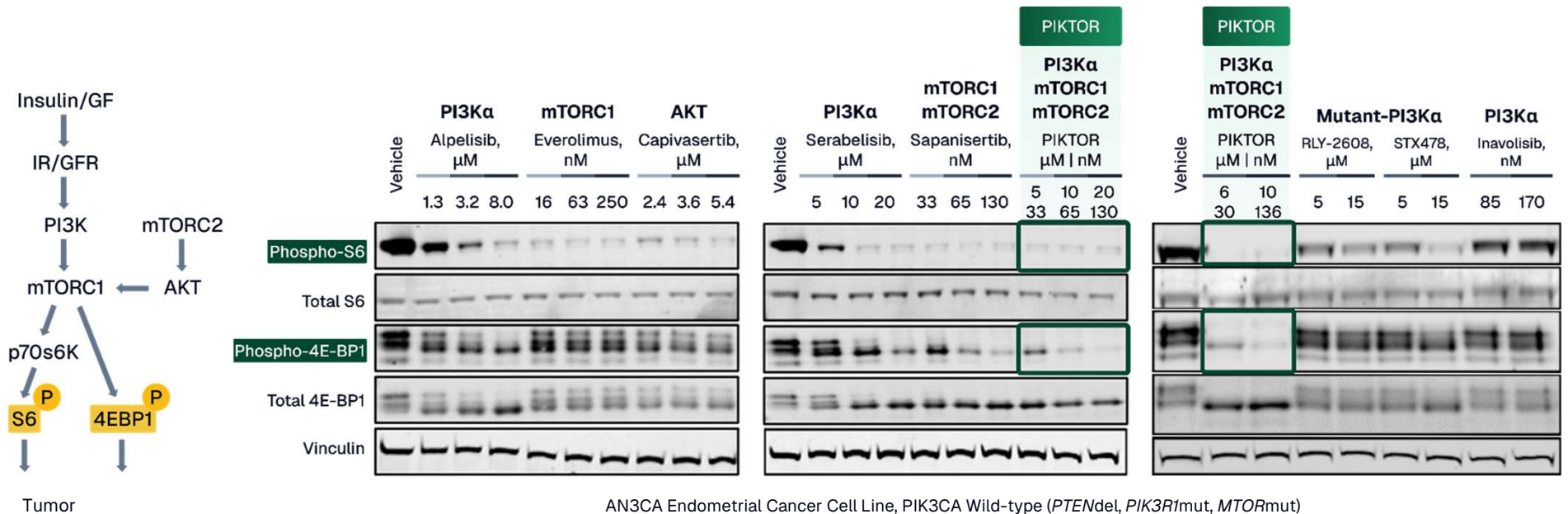
- Vertical blockade — PI3Kα upstream + mTORC1/2 downstream — designed to hit the pathway at three nodes and close feedback escape routes⁴
- Deep inhibition at lower exposure of each agent designed to widen the therapeutic window — e.g., hyperglycemia rates less than half those reported in single-node inhibitor trials⁴

Deep clinical experience underpins the dose: serabelisib and sapanisertib have been studied in ~1,000 patients across Takeda-era and subsequent trials, including ~150 patients treated with the PIKTOR combination (sapanisertib 2–8 mg / serabelisib 100–400 mg).⁵

Multi-node inhibition with PIKTOR achieved robust preclinical PI3K-pathway inhibition in cancer cell lines

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

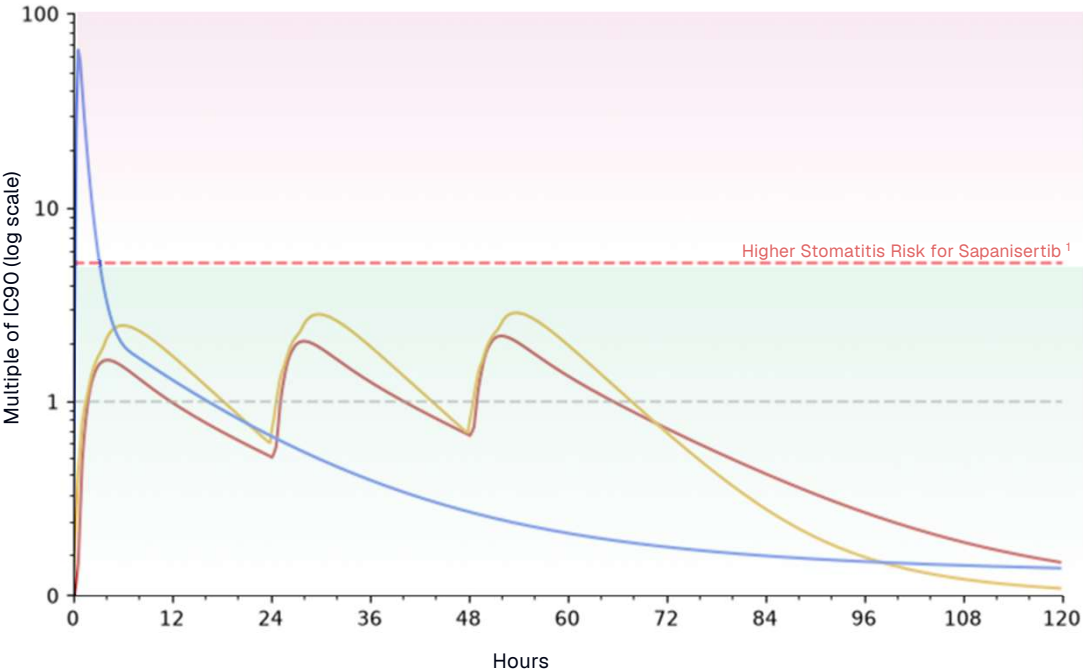
Preclinical data have been **recapitulated in human skin biopsy data** from clinical studies²



© 2026 Faeth Therapeutics. 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; ²Exposure/Response Analysis of an Oral Multi-Node PI3K/AKT/mTOR Pathway Inhibitor Combination of Serabelisib and Sapanisertib. Maddocks et. al., STOP Cancer Conference, 2026

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



	Peak exposure (Cmax : IC90 ratio)	Time above IC90 / 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 26 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 Cmax/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).

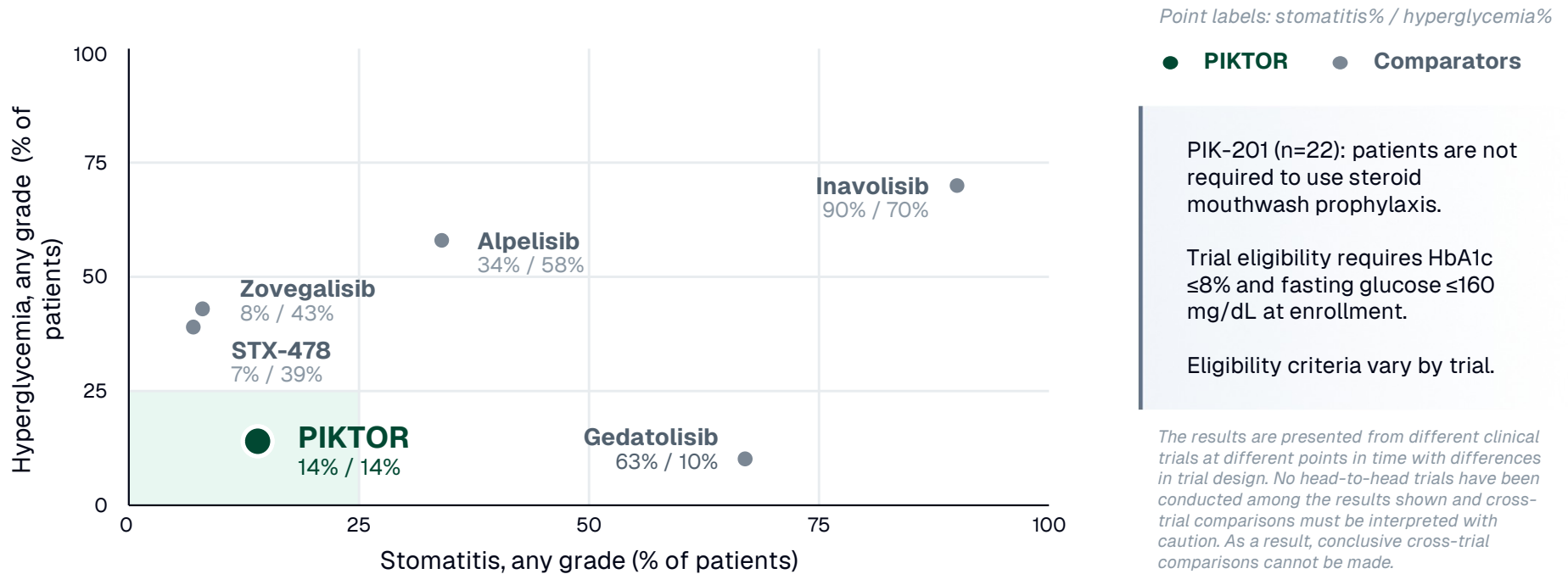
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¹ Exposure/Response Analysis of an Oral Multi-Node PI3K/AKT/mTOR Pathway Inhibitor Combination of Serabelisib and Sapanisertib. Maddocks *et. al.*, STOP Cancer Conference, 2026

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>

PIKTOR had low rates of both stomatitis and hyperglycemia in ongoing Phase 2 endo trial vs. other PAM agents



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

	MULTI-NODE		SINGLE-NODE	
	 PIKTOR (Ph.2, Endometrial, Ph.1b Breast)	GEDATOLISIB Celcuity (Approved)	MUTANT-SPECIFIC Relay, Lilly, OnKure, Novartis (Investigational)	FDA-APPROVED Alpelisib, Everolimus, Capivasertib, 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	✓	✗	✓	✓

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

Expanding market

Endometrial cancer is a growing market and recently overtook ovarian as the deadliest gynecological cancer¹.

High unmet need

Paclitaxel rechallenge: 15% ORR, 3–4 mo PFS².
ADCs are moving up in the treatment order, creating challenges for TOPO1 sequencing

Only PAM-Pathway option

PAM alterations occur in ~80% of all EC patients³
No PI3K inhibitor ever approved in EC⁴

ADC

6+ sponsors⁵ · Ph2/3

AstraZeneca 

 Daiichi-Sankyo

 MERCK

 Genmab

abbvie

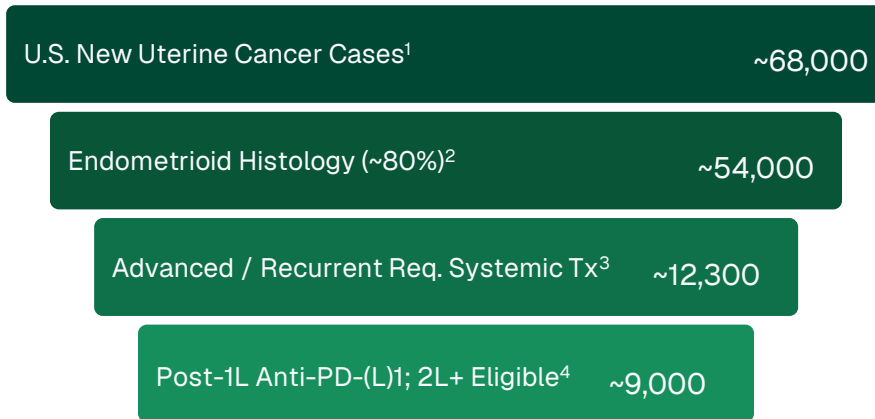
PAM

1 program · oral

 faeth

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

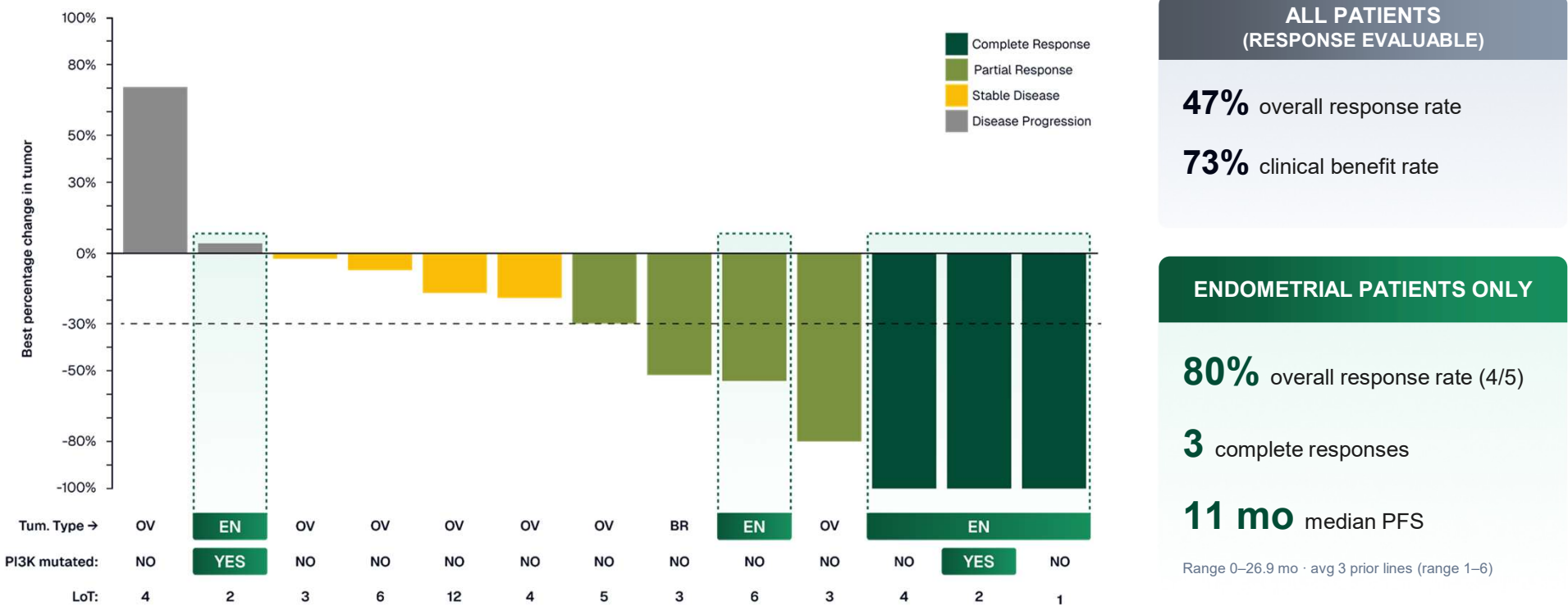
1. ACS 2026 / SEER 2025: ~68,000–69,000 new uterine corpus cases/yr. 2. Endometrioid histology ~80% ((SEER-based series, 80–85%; Clarke, JAMA Oncol 2019)).

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.

Ph1b: PIKTOR + paclitaxel drove deep responses in endometrial cancer



© 2026 Faeth Therapeutics. 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.

PIK-201: PIKTOR + paclitaxel in advanced endometrial cancer (currently enrolling)

Eligibility Criteria

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



Phase 2 Single arm (n≅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

NCT06463028

Study is being conducted in partnership with:



GOG-3111

We believe regulatory positioning supports accelerated path to registrational trial

Combination Dosing Precedent



Precedent supports a PI3K/mTOR multi-node strategy

- Recent gedatolisib Ph3 success reinforces the class and mechanism¹

Clin Pharm Gaps and Requirements Defined



Safety Database Supports Advancement



Strong safety foundation

- More than 240 subjects exposed; **no Hy's Law cases**, minimal bilirubin elevations, well-characterized toxicity on intermittent dosing to date

CMC Program on Track



Path to Registrational Trial



Contribution of Components

- Independent monotherapy activity observed for each component
- Contribution of components to be established prior to PIKTOR approval

Contribution of Components



Breast Cancer: Multi-node 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 mutation

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

Oral convenience

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

PI3K MUT

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



+ multiple others

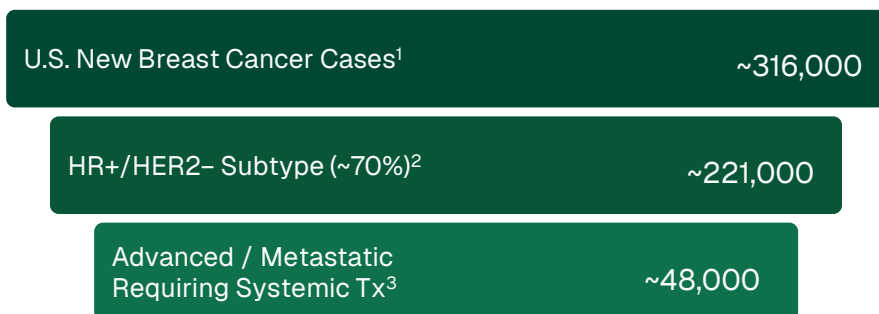
PI3K WT + PI3K MUT

100% of HR+/HER2- Breast Cancer



HR+/HER2- breast cancer: Large unmet need

U.S. Incident Therapeutic Build (Annual)



Three addressable patient populations from the ~48k advanced pool

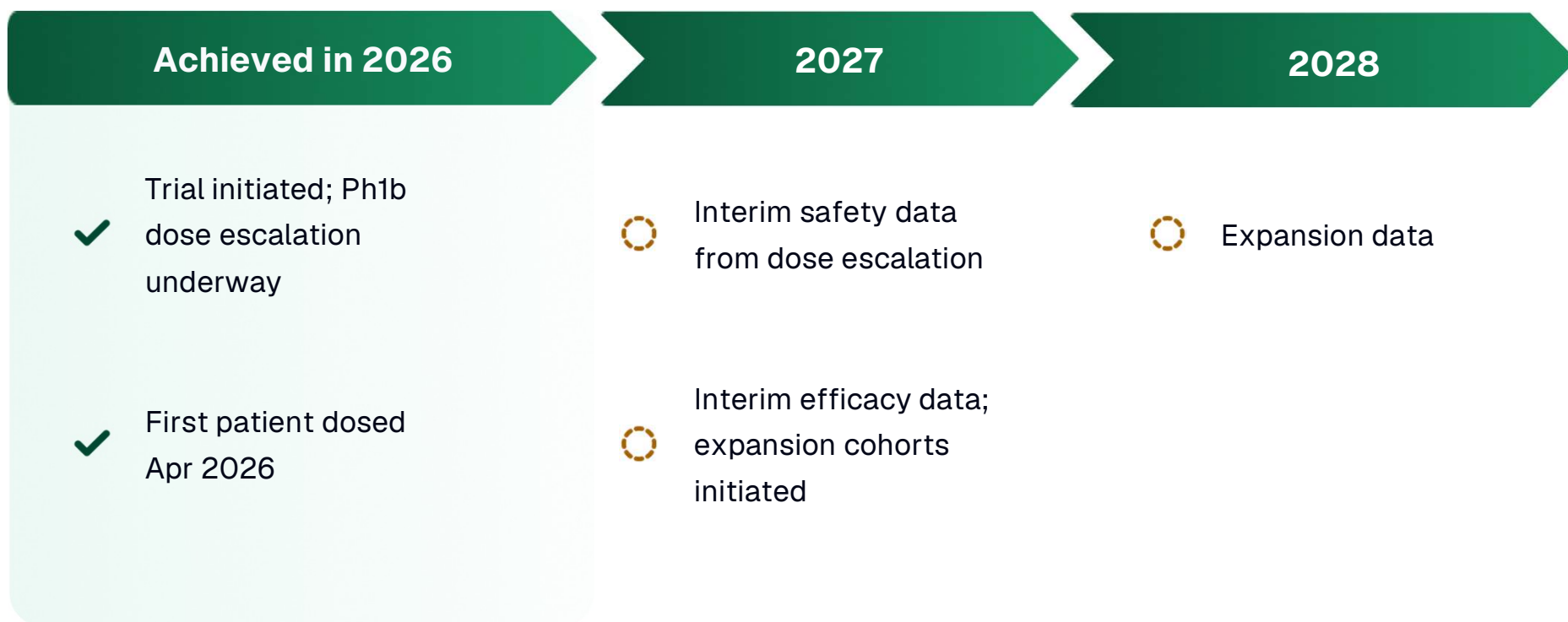
~48,000 1L patients receiving aromatase inhibitor + CDK4/6	~\$10B U.S. TAM⁴
~12,000 2L patients receiving SERD+/-CDK4/6 PI3K Mutant⁵	~\$2B U.S. TAM⁶
~18,000 2L patients receiving SERD+/-CDK4/6 PI3K WT⁵	~\$3B U.S. TAM⁷

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 (Howlander 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.

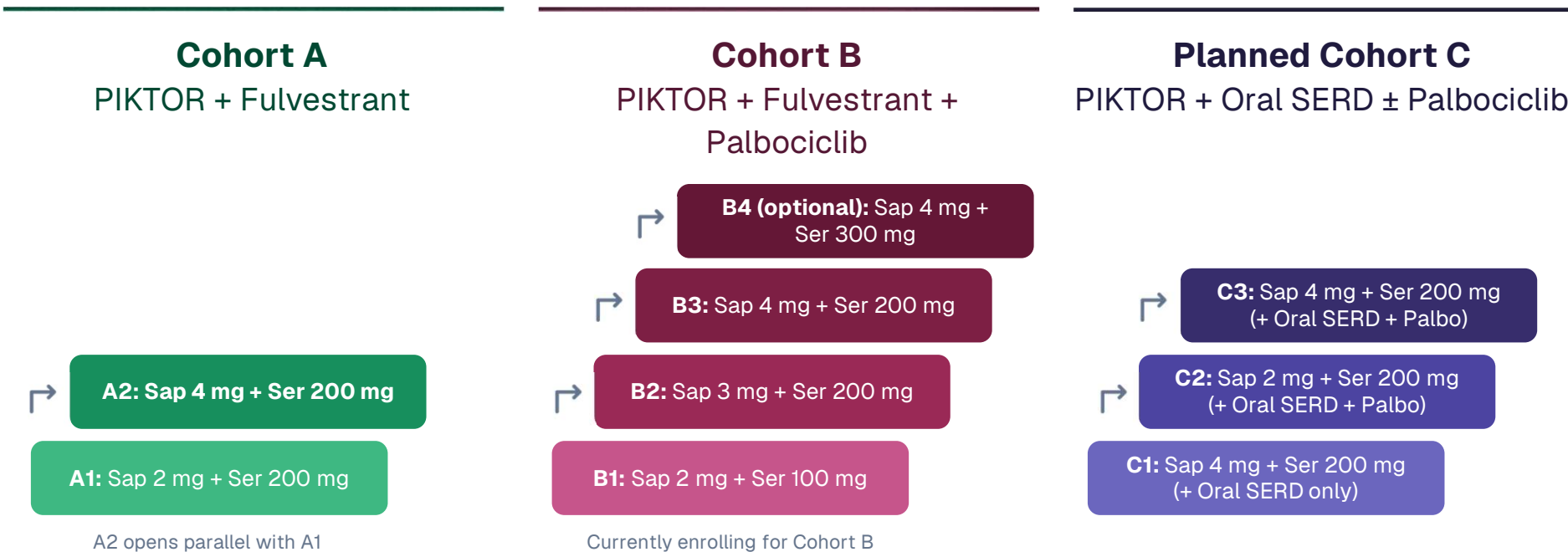
Serabelisib and Sapanisertib have each independently shown activity in HR+/HER2- breast cancer; Phase 1b/2 underway

		STUDY	DRUG / COMBINATION	ORR	DCR	mPFS	CR
HR+/HER2- (Phase 2)	Sapanisertib	Garcia-Saenz <i>et. al.</i> , 2022 ¹	Sapanisertib + Fulvestrant (n=47 arm B)	21.3%	75% PR+CR+SD	7.2 m	2/47
	Control Arm	Garcia-Saenz <i>et. al.</i> , 2022 ¹	Fulvestrant (n=46 arm A)	10.9%	61% PR+CR+SD	3.5 m	0/46
VIKTORIA-1 (Phase 3)	Gedatolisib	Hurvitz, S. <i>et al.</i> , 2025 ²	Gedatolisib + Fulvestrant (n=130)	28.3%	77% PR+CR+SD	7.4 m	NA
		STUDY	DRUG / COMBINATION	ORR	DCR	mPFS	CR
Breast Cancer Subjects from Phase 1	Serabelisib	Juric <i>et. al.</i> , 2017 ³	Serabelisib (n=21)	14%	76% PR+CR+SD	NR	0/21
	Comparator Drug Data	Juric <i>et. al.</i> , 2018 ⁴	Alpelisib (n=23)	4%	61% PR+CR+SD	NR	0/23

Breast: First patient dosed in Phase 1b trial in April 2026



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



Key eligibility: HR+/HER2- breast cancer · ≥1 prior therapy

Treatment: Until disease progression, unacceptable toxicity, withdrawal of consent, or death

Two clinically de-risked indications outside of endometrial cancer and breast cancer add optionality to pipeline

Ovarian

Platinum-resistant ovarian cancer (PROC)

HR 0.66

PFS benefit in randomized Phase 2 DICE trial (90% CI: 0.45–0.96)

- Phase 2 complete (n=134): sapanisertib + paclitaxel vs. paclitaxel alone; late-breaking oral presentation at ESMO 2025
- Mean PFS 5.8 vs. 4.0 months with no meaningful added toxicity (Grade 3/4 AEs: 7.0% vs. 6.6%)
- Supported by ovarian responses in Phase 1b PIKTOR triplet (2 PRs, 4 SDs ≥6 months)

FDA interaction planned 2026 • TAM ~\$1.5–2B

Lung

NFE2L2/KEAP1-mutated advanced NSCLC

25% ORR

Single-agent sapanisertib; mPFS 8.9 months (squamous NSCLC)

- Genotype-directed monotherapy activity — potentially the first genotype-directed therapy for squamous NSCLC
- FDA Fast Track designation granted based on these results
- PIKTOR adds PI3Kα blockade to the validated mTORC1/2 backbone — potential to deepen and extend responses

Phase 2 protocol in process • TAM ~\$0.75-1.25B

\$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	 Received Fast Track Designation	Topline data readout	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 (sapanisertib)			Preclinical data on rare disease	

Robust IP portfolio and registration-ready CMC

Intellectual property

May 2037 — Composition of matter

Issued COM patents for both APIs; serabelisib expiry includes PTA and expected 5 years of PTE

ISSUED

May 2039 — Combination MoT

Issued patent covering PIKTOR + ISD method of treatment across a range of tumor types

ISSUED

~2048 — Pending Filings

Pending MoT filings (endometrial/breast; ovarian) plus novel formulation COM filing targeted 2027

PENDING

CMC & supply

Mature DS/DP materials

Both drug substances highly stable; batches with 36–60 months of stability data and proven manufacturing history

Scale-up on track

DS/DP process optimization being completed; demo batches planned for 2026; registrational readiness on track

Supply secured

Ample DS/DP supply expected for ongoing and proposed Phase 1b / Phase 2 studies

Global exclusive licenses from Takeda for serabelisib and sapanisertib across all oncology indications

Faeth team has deep drug development experience

Management team



Anand Parikh, JD
Chief Executive Officer



Oliver Maddocks, MPharm PhD
Chief Scientific Officer



Brian Stephenson, PhD, CFA
Chief Financial Officer



Debbie Chirnomas, MD MPH
Chief Medical Officer



Christopher Gerry, JD
General Counsel

Board



Anand Parikh, JD
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Karen Vousden, PhD
Board Member

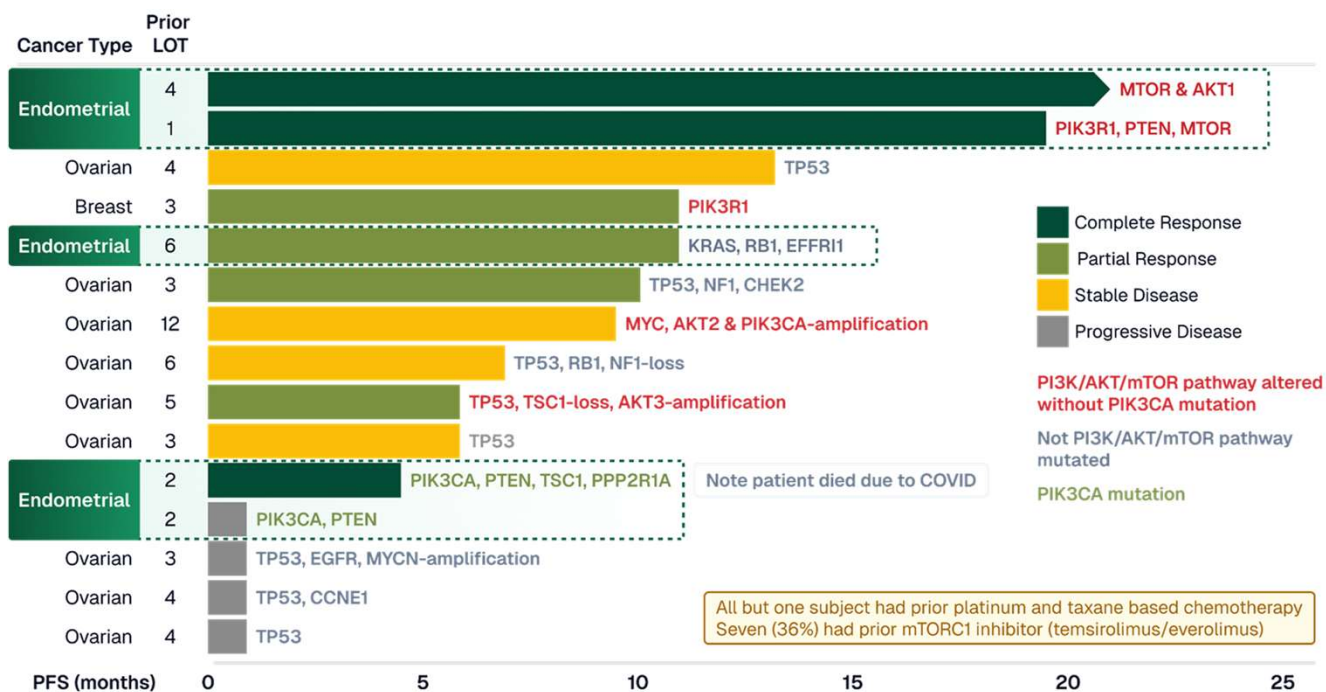
Appendix

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

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



PIKTOR AND PACLITAXEL (all RECIST evaluable)

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%

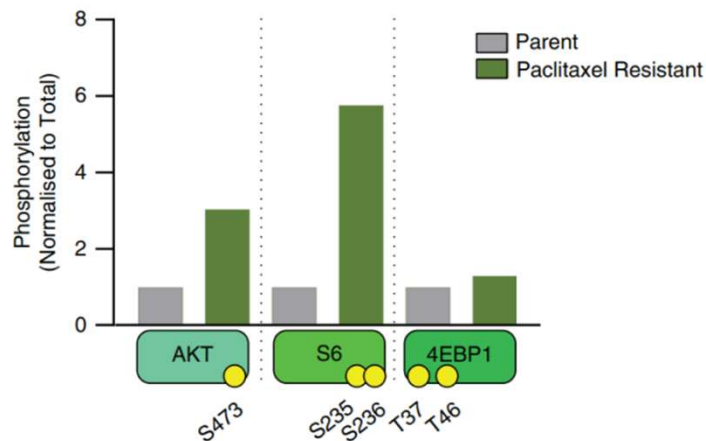
Clinical Benefit Rate

11 mo

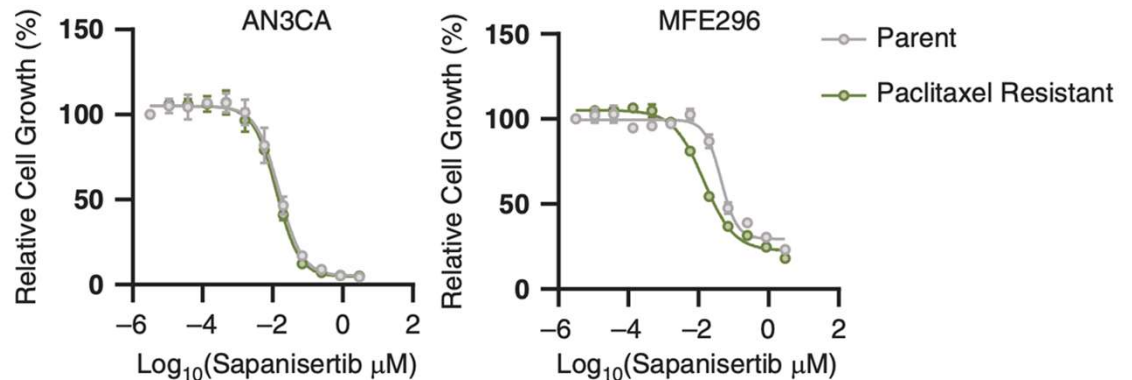
 Median PFS (Range 0 - 26.9 mo)
 Avg 3 prior lines of therapy (range 1 - 6)

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



Phase 1 Clinical Data Comparisons (all monotherapy)

		REFERENCE	COHORT	ORR	DCR (PR+CR+SD)
	Serabelisib	Juric et. al., 2017	All Comers (n= 71)	5.2%	40.3%
			Breast Cancer (n=21)	14%	76%
	Sapanisertib	Voss et. al., 2020	All Comers (n= 198)	5.0%	42.4%
			Breast Cancer (n=16)	NA	NA
	Zovegalisib	Varkaris et. al., 2023	All Comers (n= 19)	5.0%	NA
			Breast Cancer (n=2)	50% (n=1)	100% (n=2)
	Gedatolisib	Shapiro et. al. 2015	All Comers (n= 77)	3.9%	39%
			Breast Cancer (n=4)	0%	NA

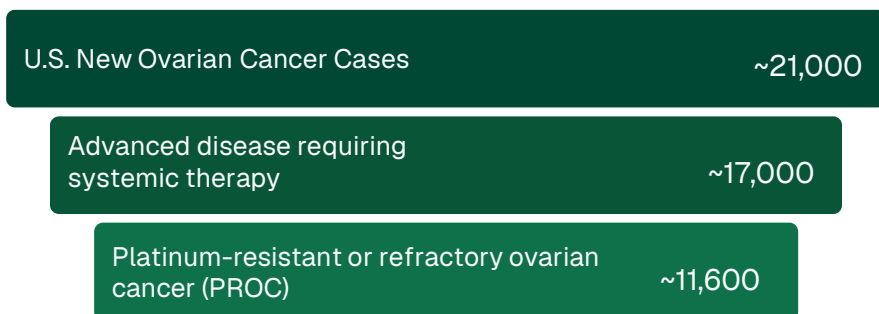
Comparison of First in human Phase 1 Clinical Trials including BC sub-groups

Note that patient populations and drug doses are naturally highly variable across trials and not directly comparable

Data shown to illustrate that early clinical data for PIKTOR agents (used separately) is similar to the related drugs zovegalisib and gedatolisib

Ovarian cancer: estimated PIKTOR addressable U.S. population

U.S. Incident Therapeutic Build (Annual)



Anticipated FDA Label Population

Advanced platinum resistant ovarian cancer (PROC)

- Epithelial ovarian, fallopian tube, and peritoneal cancers
- Platinum-resistant or refractory (progression during or within 6mo of last platinum)
- All-comers (no biomarker selection required)
- SoC mPFS ~3–4mo; mOS ~12mo; significant unmet need

~11,600

U.S. incident patients per year
advanced, platinum-resistant or
refractory, PI3K/AKT/mTOR
pathway altered

~12,000

U.S. prevalent eligible patients
TAM

1. ACS/SEER 2025: ~20,890 new OC cases/yr; ~12,730 deaths/yr. 2. ~17,000 patients/yr requiring 1L systemic Tx: de novo advanced ~15,750 (21K x 75% FIGO III/IV; Torre et al. CA Cancer J Clin 2018) + early-stage recurrence ~1,050.
3. PROC ~11,600/yr at steady state: (a) Primary platinum-resistant/refractory ~3,060 (18% of 17K; Lheureux Lancet 2019: ~20% are primary resistant, conservatively 18% after excluding refractory-who-die-early). (b) Acquired platinum resistance ~8,570/yr: ~12,240 platinum-sensitive patients enter follow-up/yr; at steady state, 70% lifetime cumulative conversion to PROC yields 12,240 x 70% = ~8,570 new PROC patients/yr across all overlapping cohorts. Total: 3,060 + 8,570 = ~11,630, rounded to ~11,600.
4. Prevalent ~12,000: 11,600 incident/yr x ~1.0yr = mOS. PROC median OS ~12mo across contemporary trials: AURELIA control mOS 13.3mo, bev+chemo mOS 16.6mo (Pujade-Lauraine JCO 2014); single-agent chemo (PLD, topotecan, weekly paclitaxel) mOS ~9–12mo; blended real-world mOS ~12mo. When mOS = 1yr, prevalent pool = annual incidence. Mortality cross-check: ~12,730 OC deaths/yr (SEER 2025) x ~91% pass through PROC = ~11,600 PROC deaths/yr = incident flow. Converges.
Abbreviations: TAM - Total Addressable Market

Phase 2 DICE Trial: Sapanisertib + Paclitaxel in PROC

Platinum Resistant Ovarian Cancer



Phase 2 DICE Trial: Sapanisertib + Paclitaxel demonstrated benefits in PROC

Late breaking oral pres at ESMO



Mean PFS

Arm A: 4.0 months

Arm B: 5.8 months

HR=0.66; 90% CI: 0.45–
0.96 (P=0.0776)

Grade 3/4 AEs

Arm A: 6.6%

Arm B: 7.0%

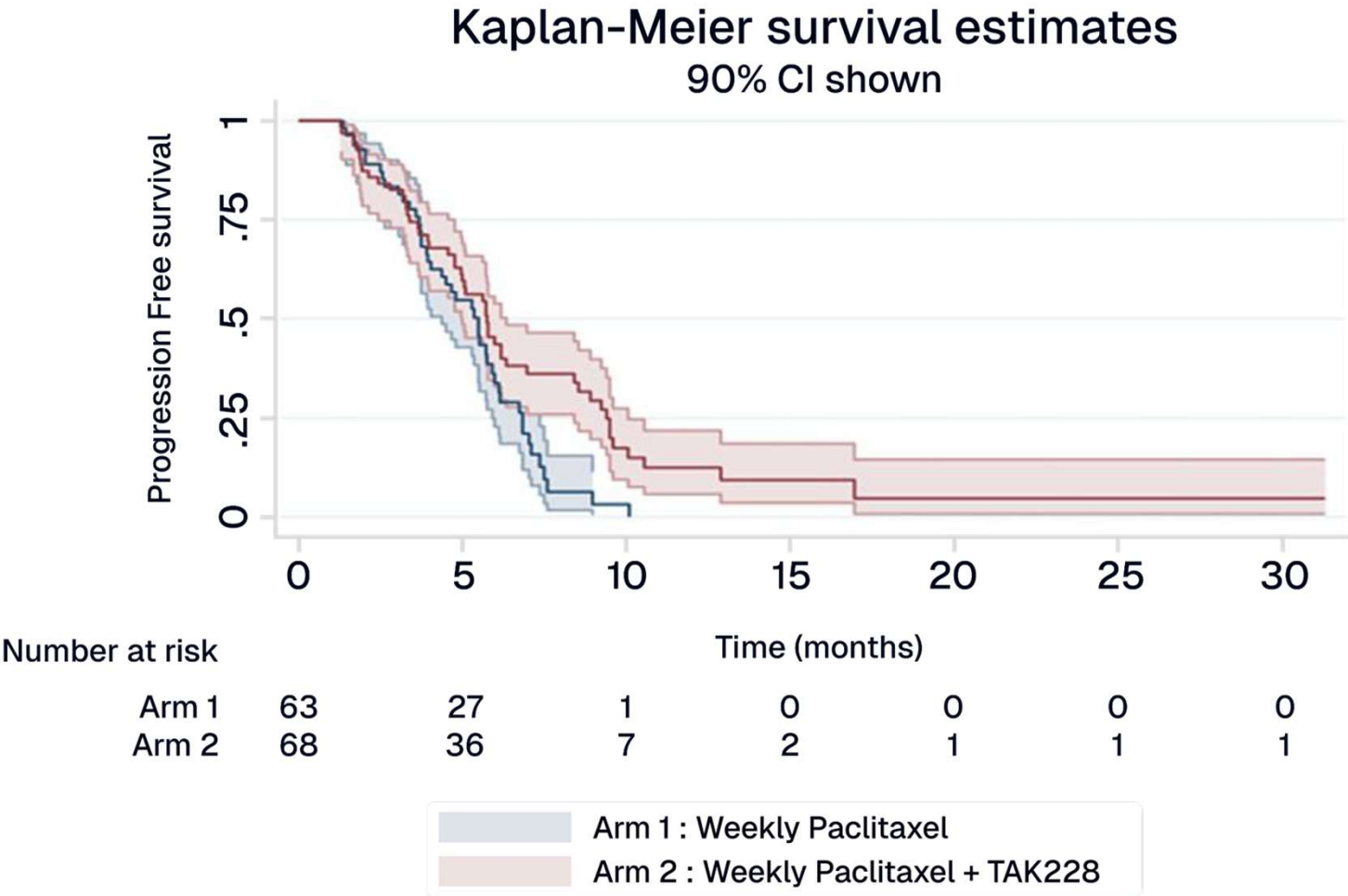
Gastrointestinal AEs (0% vs. 11.4%)
and Rash (0% vs. 2.9%) more
common in Arm B but were
manageable

Next steps

- OS, ORR and detailed safety data to come
- Potential FDA interaction in 2026 regarding future development, including potential registrational trial

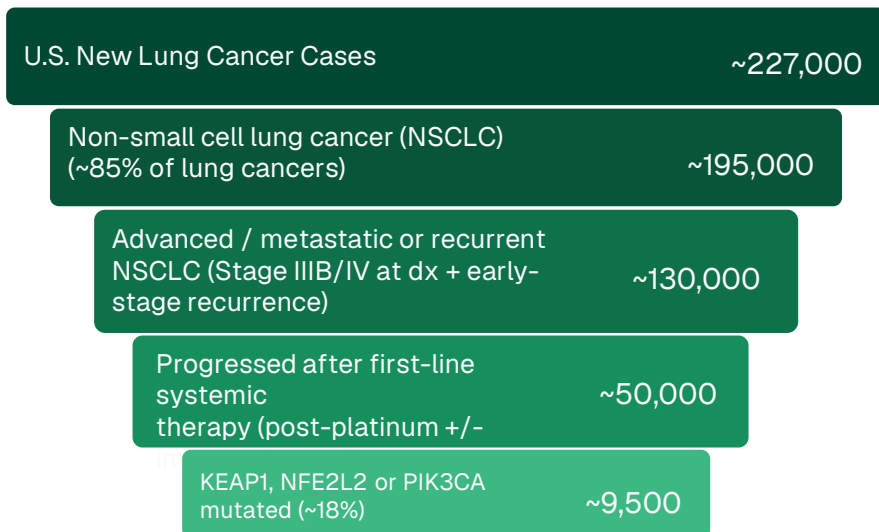
Phase 2 DICE
Trial PFS

Sapanisertib (aka
TAK 228) and
Paclitaxel **showed**
PFS benefit in
PROC



Lung cancer: estimated PIKTOR addressable U.S. population

U.S. Incident Therapeutic Build (Annual)



Anticipated FDA Label Population

Advanced or metastatic non-small cell lung cancer (NSCLC)

- Progressed on prior systemic therapy (platinum-based chemo +/- anti-PD-1/PD-L1)
- KEAP1, NFE2L2 or PIK3CA pathway gene alteration
- All histologies (adenocarcinoma and squamous)

Label population may be broader than biomarker-selected subsets

~9,500

U.S. incident patients per year
advanced or metastatic NSCLC,
KEAP1, NFE2L2 or PIK3CA pathway
gene alteration

~7,000

U.S. prevalent patients
TAM

1. ACS/SEER 2025: 226,650 new LC cases; ~124,730 deaths. 2. NSCLC ~85% (ACS 2024; Molina et al. Mayo Clin Proc 2008). 3. ~130,000 advanced/recurrent requiring systemic Tx: de novo advanced ~125K (195K x 65% Stage IIIB/IV; SEER stage distribution) + steady-state early-stage recurrence ~12K (68K early-stage entrants/yr x 20% 5yr CIR ~13,600 at steady state; NLST, Ann Thorac Surg 2023). CIR already incorporates competing mortality. Total ~137K; presented as ~130K (conservative round). De novo advanced dominates (~91%).
4. Post-1L treatment-eligible (~50,000): ~130K x 80-85% receive 1L systemic → ~40% initiate 2L (clinical attrition: rapid progression, declining PS, death on 1L; ESMO 2023 review 30-46%; SEER-Medicare 47%). Unlike BC/EC where most patients survive to 2L, NSCLC 1L mOS ~12-15mo and many patients deteriorate before 2L is feasible.
5. KEAP1, NFE2L2 or PIK3CA mutated (~9,500, ~18%); KEAP1/NFE2L2 ~15% (Frank et al. CCR 2018: KEAP1 11.3%, NFE2L2 3.5%, largely mutually exclusive; Jeong et al. CCR 2020: 17%). PIK3CA adds ~4% of NSCLC (Frank et al. Oncotarget 2014, n=1,144: 3.7%; range 2-4%; squamous ~9% vs. adeno ~3%; TCGA LUSC). PIK3CA co-occurrence with KEAP1/NFE2L2 not significantly enriched (Frank et al. CCR 2018, n.s.), so overlap ~0.6% (15% x 4%); union ~15% + 4% - 0.6% ~18%. Reflects proposed LUNG-MAP expansion adding PIK3CA to the NFE2L2/KEAP1 (NRF2) subset; PI3K/AKT signaling is mechanistically linked to NRF2. Applied to 2L pool as a stable tumor genotype.
6. Prevalent (~7,000): 9,500 incident/yr x ~0.75yr mean post-2L survival. Post-2L NSCLC mOS ~7-9mo (SEER-Medicare 7.3mo); KEAP1/NFE2L2 mutants have HR ~2.0 for death vs. WT (POPLAR/OAK, Front Oncol 2021), mOS ~5-7mo from 2L start, while PIK3CA mutants do not carry an independent adverse prognosis (Frank et al. Oncotarget 2014), so the blended pool survival is slightly more favorable than KEAP1/NFE2L2 alone. Mortality cross-check: ~106K NSCLC deaths x ~8.4% biomarker-positive 2L-eligible = ~8,900 deaths/yr; at mOS ~8mo, prevalent ~8,900 x (8/12) ~5,900. Both methods bracket ~6,000-7,500; point estimate ~7,000.
Abbreviations: TAM - Total Addressable Market

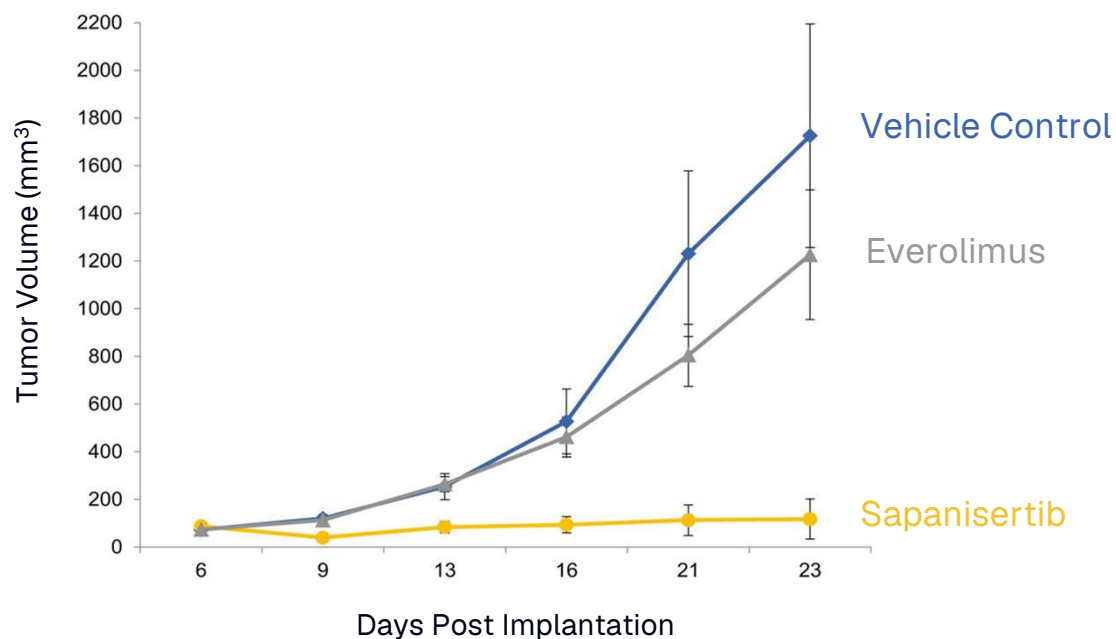
Sapanisertib shows preclinical efficacy in lung cancer models

LK-2 Squamous cell lung cancer xenograft tumor model (*NFE2L2 E79K* mutant)

Sapanisertib showed benefit versus everolimus

Sapanisertib 3 mg/kg PO QD

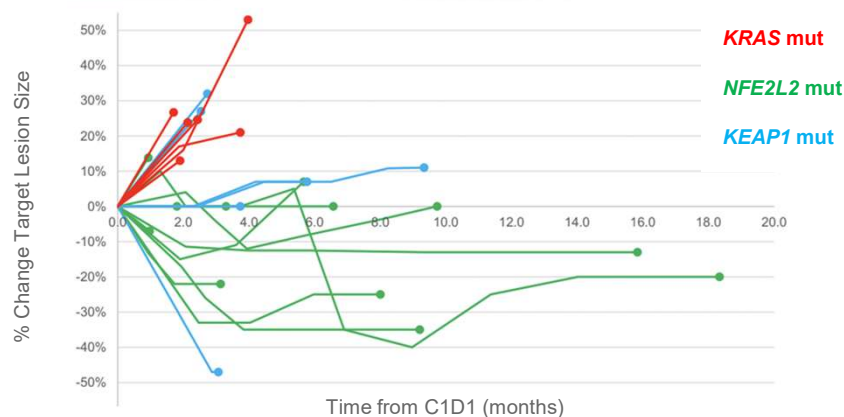
Everolimus 3 mg/kg PO QD



Phase 2 NSCLC Trial: Sapanisertib demonstrated benefits

Key results

Sapanisertib monotherapy: **25% ORR and mPFS of 8.9 months** in patients with NFE2L2-altered squamous NSCLC
First successful metabolic targeting of NSCLC and potentially the first genotype-directed therapy for LUSC



PIKTOR implications

Established **single-agent proof of concept** for mTORC1/2 inhibition in this biomarker-defined population.

PIKTOR adds PI3K-alpha inhibition (serabelisib) on top of sapanisertib — **multi-node blockade may further enhance depth and durability of response**

FDA granted Fast Track designation to sapanisertib for NRF2-mutated squamous lung cancer based on these results

Key patents

SUBJECT MATTER	PATENT EXPIRATION DATE	NOTES
Composition of Matter - Serabelisib API - Sapanisertib API	May 2037	- Issued COM patents for each API - COM expiry date includes patent term adjustment (PTA) and expected 5 years of patent term extension (PTE) added to serabelisib COM patent
PIKTOR + ISD Method of Treatment for Cancer	May 2039	- Issued Patent - Covers use of PIKTOR + ISD in a range of tumor types
PIKTOR Method of Treatment for Cancer	Pending (March 2046 = 20-year)	- Patent Filed March 2025 - Endometrial and Breast cancer
Sapanisertib Method of Treatment for Cancer	Pending (Oct. 2046 = 20-year)	- Patent Filed Oct. 2025 - Ovarian Cancer
Opportunity for novel composition of matter IP Formulation development ongoing	Target patent filing = 2027 (20-year expiry ~2048)	Development work ongoing

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				

Thank you