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.

SEPTEMBER 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 PAM[1] regimen, built for longer target coverage without class-defining toxicities

faeth

PIKTOR

serabelisib (PI3Kα) + 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

~$15–20B market opportunity in breast cancer[3]
~$2-3B market opportunity in endometrial cancer[4]

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 Capital[2]

[1] PAM: PI3K/AKT/mTOR, a pathway that regulates cell metabolism, growth, and survival and is altered in up to half of solid tumors; [2] as of February 2026 private placement
[3] Based on the combined sales of marketed CDK4/6 inhibitors (VERZENIO, KISQALI, and IBRANCE); [4] Based on JEMPERLI sales as the best available comp, and assuming market penetration into both dMMR and pMMR settings

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

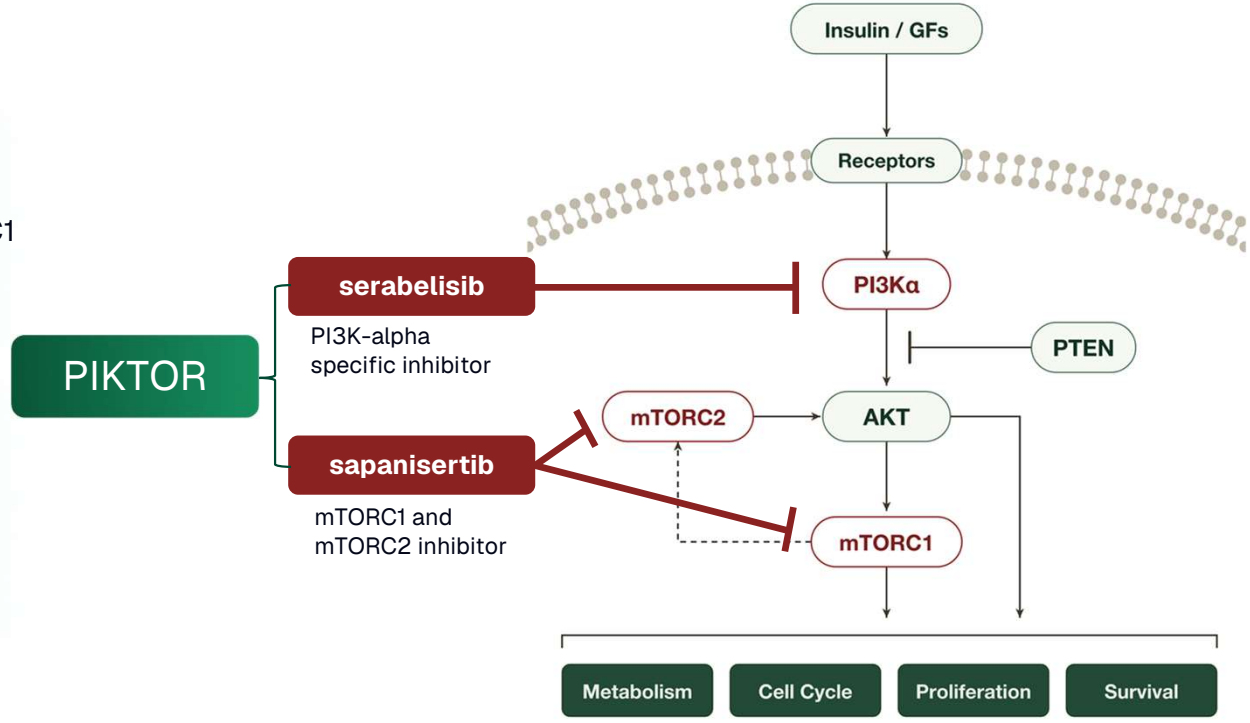
Scott Lowe, PhD
Chair of Cancer Biology & Genetics at Memorial Sloan Kettering, Founder: ORIC (NASDAQ)

Greg Hannon, PhD
Former Director of CRUK Cambridge Institute

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

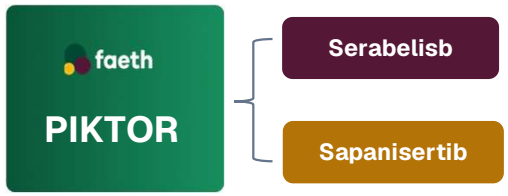
PIKTOR combines serabelisib (PI3K-alpha specific inhibitor) with sapanisertib (mTORC1 and mTORC2 inhibitor).

Targeting the PI3K/AKT/mTOR pathway to address one of the most-frequent genomic drivers in solid tumors[1].



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

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
Serabelisb	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 routes[4]
- 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 trials[4]

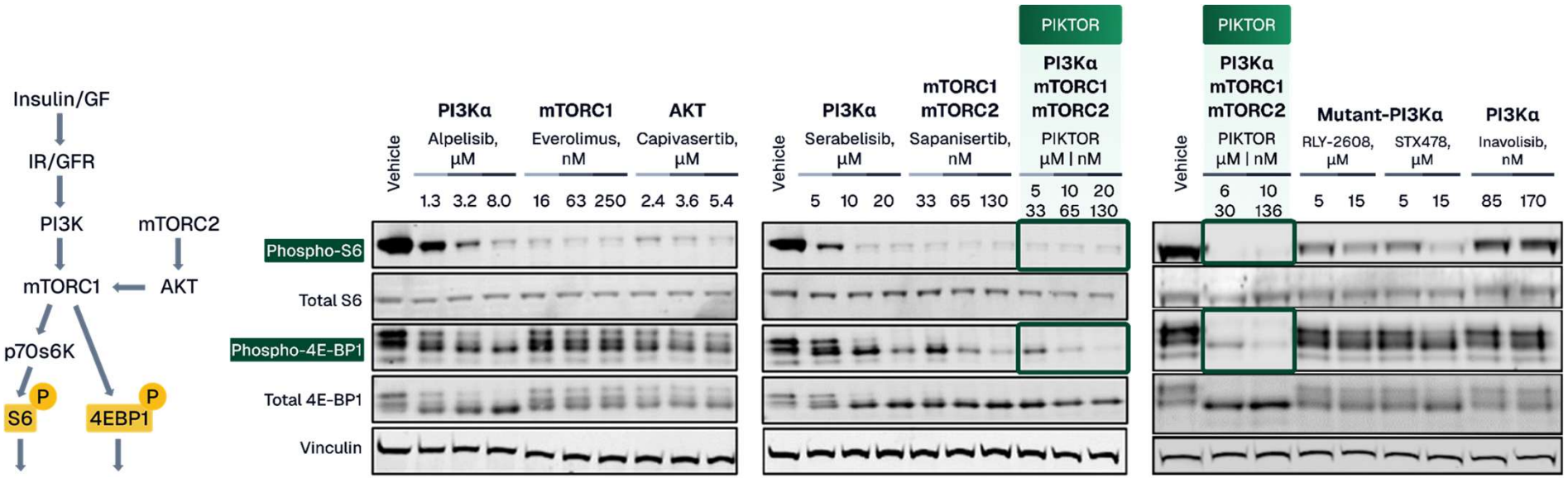
Deep clinical experience underpins the dose: serabelisib and sapanisertib have been studied in ~1,000 patients across Takeda-era and subsequent trials, including ~150 patients treated with the PIKTOR combination (sapanisertib 2–8 mg / serabelisib 100–400 mg).[5]

[1]Juric et. al., Clin. Cancer Res. 23 (17) 2017. [2]Voss et. al., Br. J. Cancer 123 (11) 2020. [3]Starks et. al., Gyn. Onc. 166, 2022. [4]Tyrakis et. al., Br. J. Cancer 133, 2025. [5]Maddocks et al., STOP Cancer 2026; RP2D – Recommended Phase 2 Dose; QD×3dQW – once daily, 3 days per week; FIH – first-in-human.

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 concentrations[1]

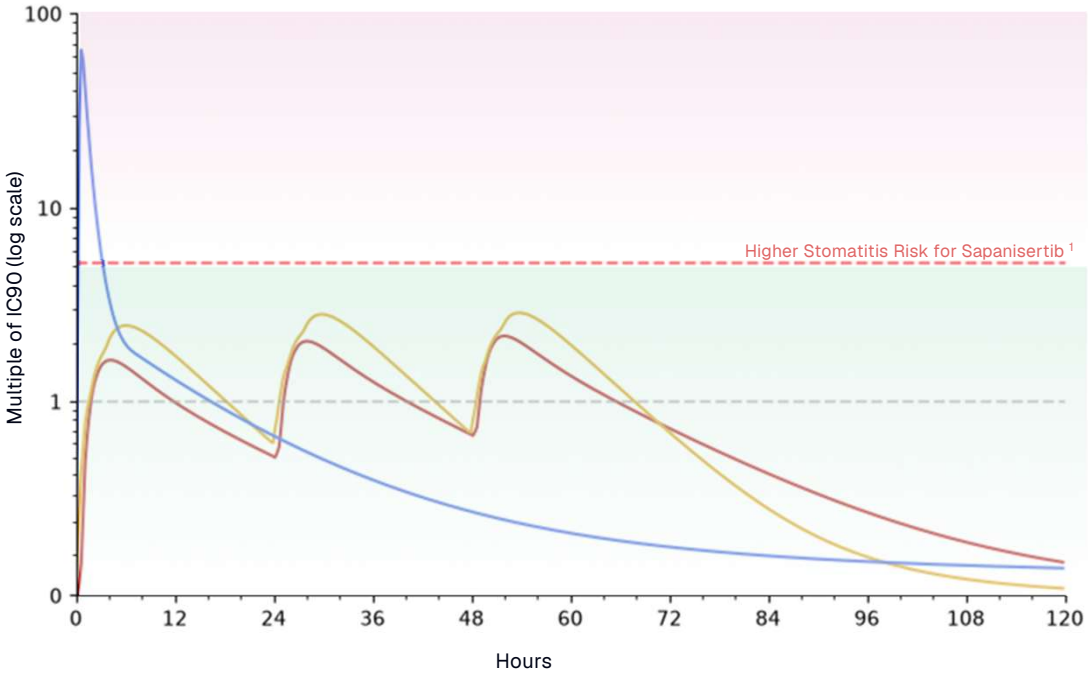
Preclinical data have been **recapitulated in human skin biopsy data** from clinical studies[2]



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 lines [1].

[1] Tyrakis et. al., (2025) British Journal of Cancer, 133: 144-154; [2] *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*

Oral PIKTOR dosing is designed for sustained exposure with reduced Cmax-related toxicity risk

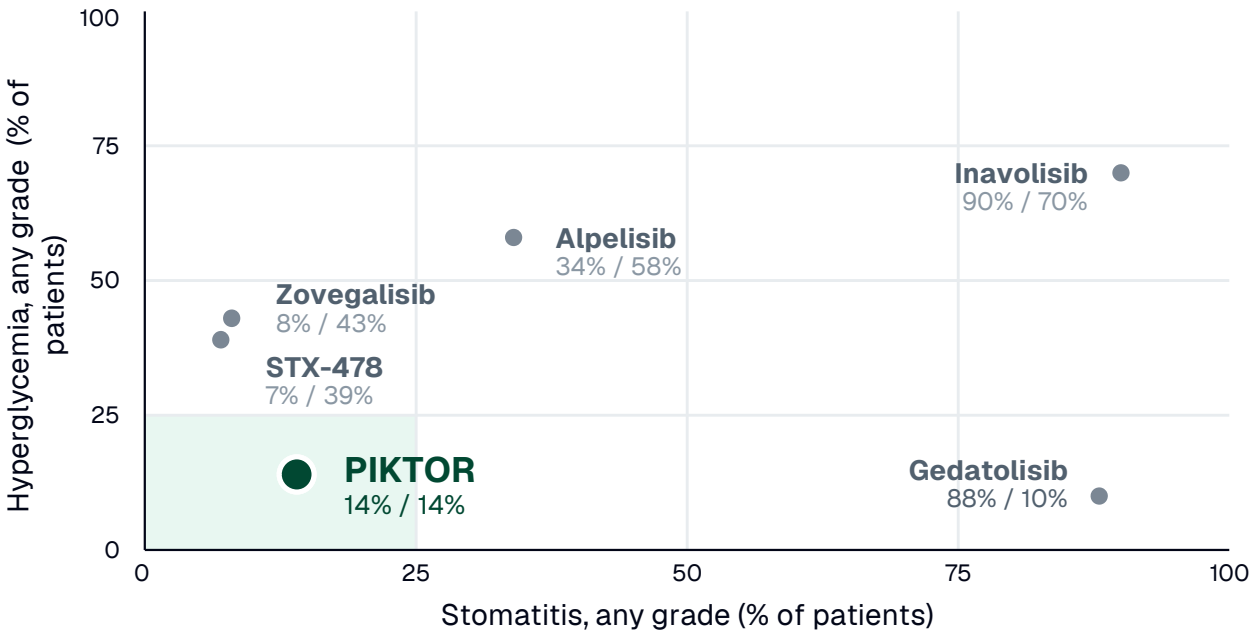


	Peak exposure (Cmax : IC90 ratio)	Time above IC90 / week	Plasma protein binding / free fraction	
			Bound	Free
Serabelisib	2.2	42 h	69%	31%
Sapanisertib	2.9	44 h	70%	30%
Gedatolisib	~65	15 h	98%	2%

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.,* 2026[1]. Gedatolisib (180 mg QW) drug concentration data was derived from Shapiro *et. al.,* 2015[2] 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.,* 2025[3] 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.,* 2026[1]).

[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 potently 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 hyperglycemia and stomatitis in ongoing Phase 2 endo trial vs. other PAM agents



Point labels: stomatitis% / hyperglycemia%

PIKTOR **Comparators**

PIK-201 (n=22): patients are not required to use steroid mouthwash prophylaxis.

Trial eligibility requires HbA1c ≤8% and fasting glucose ≤160 mg/dL at enrollment.

Eligibility criteria vary by trial.

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.

Hyperglycemia: Turner NEJM 2024 (inavolisib); Hurvitz ASCO 2026 (alpelisib, VIKTORIA-1); Varkaris ESMO TAT 2026 (zovegalisib/RLY-2608); Jhaveri ASCO 2026 (STX-478, PIKALO-1); Celcuity VIKTORIA-1 Oct 2025 (gedatolisib). Stomatitis: Layman SABCS 2022 (gedatolisib); cross-refs. PIKTOR: PIK-201 PIKTOR+paclitaxel snapshot 01-2026, n=22, ongoing Faeth Phase 2. Gedatolisib FDA-approved as Revtorpyk®, 14-Jul-2026.

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 (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 cancer[1].

High unmet need

Paclitaxel rechallenge: 15% ORR, 3–4 mo PFS[2].
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 patients[3]
No PI3K inhibitor ever approved in EC[4]

ADC

6+ sponsors[5] · Ph2/3

AstraZeneca
Daiichi-Sankyo
MERCK
Genmab
abbvie

PAM

1 program · oral

faeth

PIKTOR does not overlap with ADC dose-limiting toxicities (cytopenias, ILD)

1. ACS 2026 / SEER 2025. 2. Single-agent chemotherapy control arm, KEYNOTE-775 (Makker et al., NEJM 2022). 3. TCGA 201. 4. FDA approvals database. 5. Per publicly registered Phase 3 eligibility criteria (clinicaltrials.gov).

Endometrial: estimated PIKTOR addressable U.S. population

U.S. Incident Therapeutic Build (Annual)

U.S. New Uterine Cancer Cases[1]	~68,000
Endometrioid Histology (~80%)[2]	~54,000
Advanced / Recurrent Req. Systemic Tx[3]	~12,300
Post-1L Anti-PD-(L)1; 2L+ Eligible[4]	~9,000

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

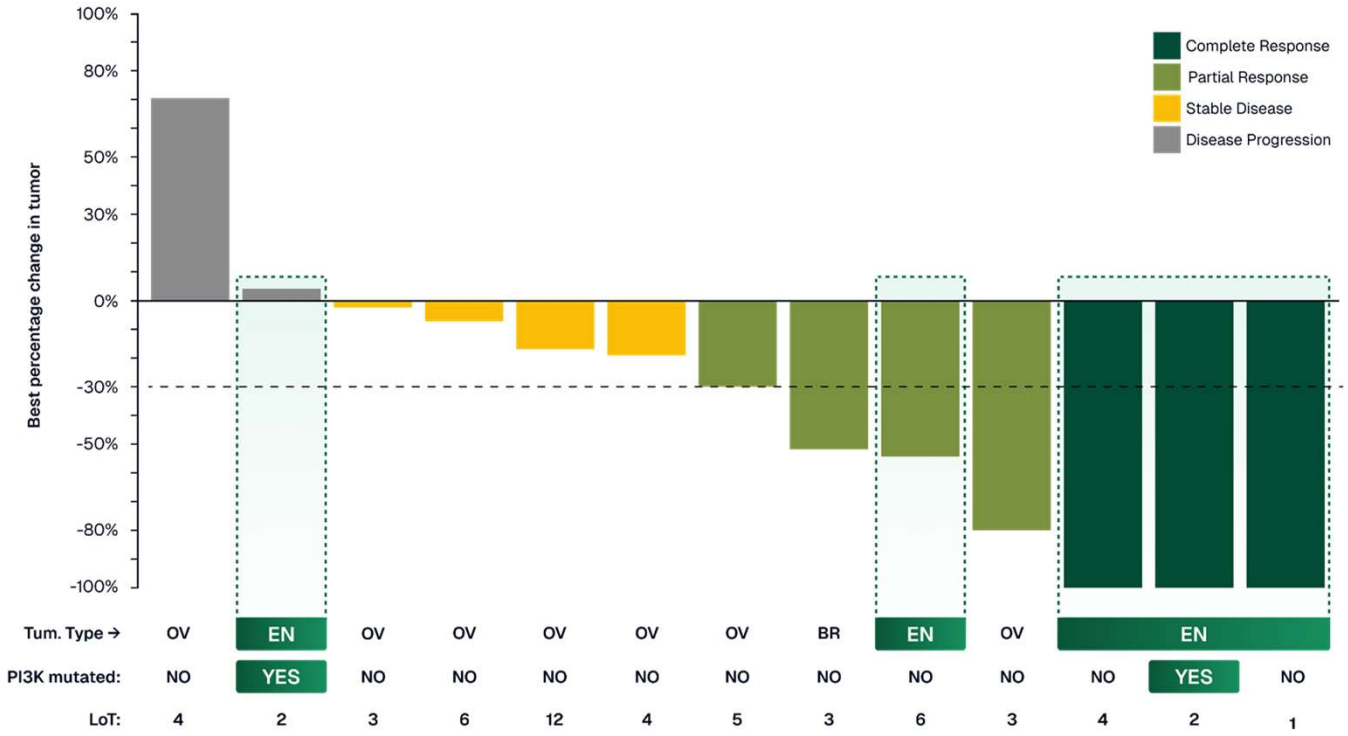
IO+chemo is now 1L SoC (NRG-GY018, RUBY, DUO-E; FDA 2024)

~9,000
U.S. incident patients per year post-anti-PD-(L)1, endometrioid EC

~12,500
U.S. prevalent eligible TAM[5]

1. ACS 2026 / SEER 2025: ~68,000–69,000 new uterine corpus cases/yr. 2. Endometrioid histology ~80% (TCGA 2013; SEER).
3. Advanced/recurrent requiring systemic Tx (~12,300/yr): de novo advanced ~9,700 (54K × 18% FIGO III/IV; SEER ~9% distant + ~50% of 18% regional as FIGO III, consistent w/ 20–30% high-risk fraction in literature) + steady-state early-stage recurrence ~2,550/yr (44K early-stage entrants/yr × 8% lifetime cumulative recurrence; 27% vaginal-vault-only excluded per Gynecol Oncol 2022; 73% require systemic Tx).
4. ~9,000 addressable 2L+ patients/yr at steady state: ~11,685 receive 1L IO+chemo (12,300 × 95%; IO+chemo is NCCN preferred/universal SOC post NRG-GY018/RUBY) × ~77% lifetime progression ≈ 9,000/yr addressable. 5. Prevalent (~12,500): ~9,000 incident/yr × ~1.3–1.5yr mean post-2L survival (KEYNOTE-775 mOS 18mo len/pembro, 12mo chemo; real-world ~10mo; blended ~15mo). Cross-checked via mortality: ~13,500 EC deaths/yr × 60% endometrioid = ~8,100 endometrioid deaths/yr × 1.4yr mean survival ≈ ~11,300 prevalent, consistent with Method 1 estimate.
Abbreviations: TAM - Total Addressable Market

Ph1b: PIKTOR + paclitaxel drove deep responses in endometrial cancer



ALL PATIENTS (RESPONSE EVALUABLE)

47% overall response rate

73% clinical benefit rate

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-Espaillat 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 PARTNERS A GOG Foundation Program GOG-3111

We believe regulatory positioning supports accelerated path to registrational trial

Combination Dosing Precedent	✓
Clin Pharm Gaps and Requirements Defined	✓
Safety Database Supports Advancement	✓
CMC Program on Track	✓
Path to Registrational Trial	✓
Contribution of Components	◌

Precedent supports a PI3K/mTOR multi-node strategy

- Recent gedatolisib Ph3 success reinforces the class and mechanism[1]

Strong safety foundation

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

Contribution of Components

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

[1]Hurvitz, S. et. al., (2025) ESMO Presentation
Abbreviations: PK/BA - Pharmacokinetics / Bioavailability, EC - endometrial cancer, CoC - contribution of components
Subject to feedback from FDA

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 patients[1]

Most patients lack PI3K mutation

PIKTOR is designed to **work in all patients**; not just the ~40% that have an activating *PIK3CA* mutation, or the ~20% with *PIK3CA* as the sole PAM pathway alteration

Oral convenience

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

PI3K MUT

~20% of HR+/HER2- breast cancer with *PIK3CA* as sole PAM alteration [2-5]

RELAY THERAPEUTICS

LOXO ONCOLOGY at Lilly

OnKure® THERAPEUTICS

+ multiple others

PI3K WT + PI3K MUT

100% of HR+/HER2- Breast Cancer

faeth

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- breast cancer opportunity

U.S. Incident Therapeutic Build (Annual)

U.S. New Breast Cancer Cases[1]	~316,000
HR+/HER2– Subtype (~70%)[2]	~221,000
Advanced / Metastatic Requiring Systemic Tx[3]	~48,000

Three addressable patient populations from the ~48k advanced pool

~32,000 U.S. incident patients per year 2L post-CDKi + AI ABC[4]	**~37,000** U.S. prevalent 2L+ patients TAM[5]
~30,000 U.S. incident patients per year 1L ETR ABC[6]	**~33,000** U.S. prevalent 1L ETR patients TAM[7]
~18,000 U.S. incident patients per year 1L ETS ABC[8]	**~60,000** U.S. prevalent 1L ETS patients TAM[9]

1. 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 × 6%; Malmgren 2018) + steady-state distant recurrence ~35K (208K early-stage entrants/yr × 17% lifetime cumulative distant recurrence; Pan 2017 NEJM, adjusted for distant-only events and competing mortality). 4. [2L] ~32K incident 2L+: 38K × ~85% lifetime progression on 1L CDK4/6i+ET (PALOMA-2/MONALEESA-2 5yr data; SONIA trial). 5. [2L] Prevalent ~37K: 32K incident/yr × ~1.15yr mean treatment-eligible survival from 2L+ entry; post-CDK4/6i median OS ~22mo (CAPItello-291) but prevalent pool discounted for hospice/declining PS/comorbidities; consistent with Celcuity VIKTORIA-1 investor estimate of ~37K (March 2026 investor presentation; SEER 2024, Pan 2017 NEJM, Salvo 2021). 6. [1L ETR] ~30K incident 1L ETR: 48K × ~62% endocrine-resistant fraction; ETR defined as recurrence during or within 12mo of adjuvant ET, de novo metastatic with aggressive features, or primary endocrine resistance (Dowsett 2009; Salvo 2021; Pan 2017 NEJM). 7. [1L ETR] Prevalent ~33K: 30K incident/yr × ~1.1yr mean treatment-eligible survival in 1L ETR; SoC mPFS ~7–13mo for CDK4/6i+fulvestrant in ETR populations (MONALEESA-3 ETR subgroup; RIGHT Choice trial). 8. [1L ETS] ~18K incident 1L ETS: 48K × ~38%; ETS = de novo metastatic or recurrence >12mo after completing adjuvant ET; ETS and ETR are mutually exclusive and exhaustive partitions of the 1L pool (38% + 62% = 100%; Pan 2017 NEJM; Dowsett 2009). 9. [1L ETS] Prevalent ~60K: 18K incident/yr × ~3.3yr mean time on 1L therapy; SoC mPFS ~25mo on CDK4/6i+AI (PALOMA-2, MONALEESA-2); real-world mean DoT ~36–40mo; largest of the three BC subpopulations by prevalence.
Abbreviations: TAM - Total Addressable Market ETS - endocrine therapy sensitive ETR - endocrine therapy resistant

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 *et. al.*, 2022 [1]	Sapanisertib + Fulvestrant (n=47 arm B)	21.3%	75% PR+CR+SD	**7.2 m**	2/47
	Control Arm	Garcia-Saenz *et. al.*, 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. *et. al.*, 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 *et. al.*, 2017 [3]	Serabelisib (n=21)	14%	76% PR+CR+SD	NR	0/21
	Comparator Drug Data	Juric *et. al.*, 2018 [4]	Alpelisib (n=23)	4%	61% PR+CR+SD	NR	0/23

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.

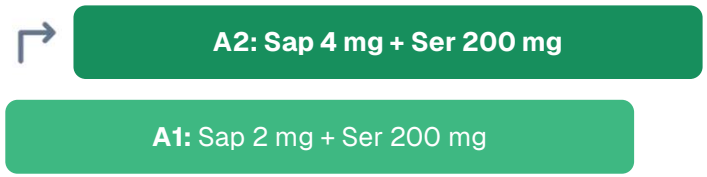
[1]Garcia-Saenz et. al., (2022) J. Clinical Cancer Research (2022) 28 (6): 1107–1116 [2]Hurvitz, S. et. al., (2025) ESMO Presentation [3]Juric et. al., (2017) Clin. Cancer Res. 23 (17) [4]Juric et. al., (2018) Journal of Clinical Oncology 36 (13),1291-1299

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

Achieved in 2026	2027	2028
✓ Trial initiated; Ph1b dose escalation underway	○ Interim safety data from dose escalation	○ Expansion data
✓ First patient dosed Apr 2026	○ Interim efficacy data; expansion cohorts initiated	

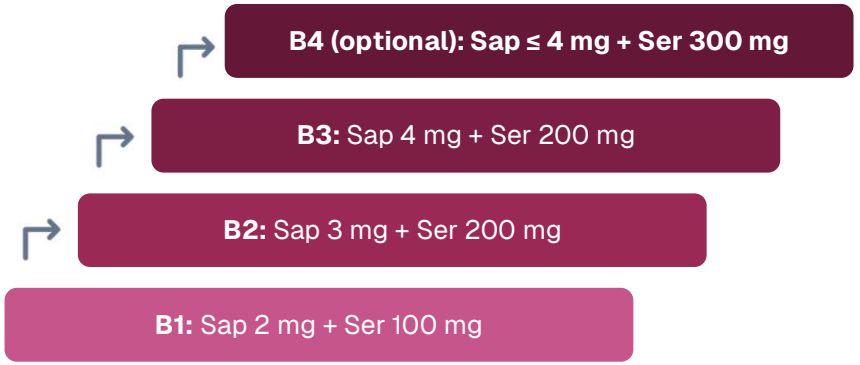
Breast: Ongoing Phase 1b/2 trial designed to rapidly identify recommended dose and initiate expansion cohorts

Cohort A
PIKTOR + Fulvestrant



A2 opens parallel with A1

Cohort B
PIKTOR + Fulvestrant + Palbociclib



Concurrently enrolling in Cohort B

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

Krell J et al., ESMO 2025; Paik PK et al., J Thorac Oncol 2023;18(4):516–526; Starks DC et al., Gynecol Oncol 2022. TAM per Deallus report (Feb 2024) and company analysis. Abbreviations: PFS – progression-free survival; HR – hazard ratio; CI – confidence interval; ORR – overall response rate; AE – adverse event; IIT – investigator-initiated trial; TAM – total addressable market.

$186.4M[1] in cash expected to fund PIKTOR through anticipated readouts in endometrial cancer and breast cancer

PROGRAM	Achieved in 2026	2H 2026	2027	2028+
ENDOMETRIAL	✓ Phase 2 enrollment ongoing	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	

[1] $186.4M cash and cash equivalents as of June 30, 2026,
Abbreviations: EC - endometrial cancer, BC - breast cancer

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
Board Chair

Phil Donenberg
Board Member

Stephen Hahn, MD
Board Member

Saira Ramasastry
Board Member

Bob Holmen, JD
Board Member

Appendix

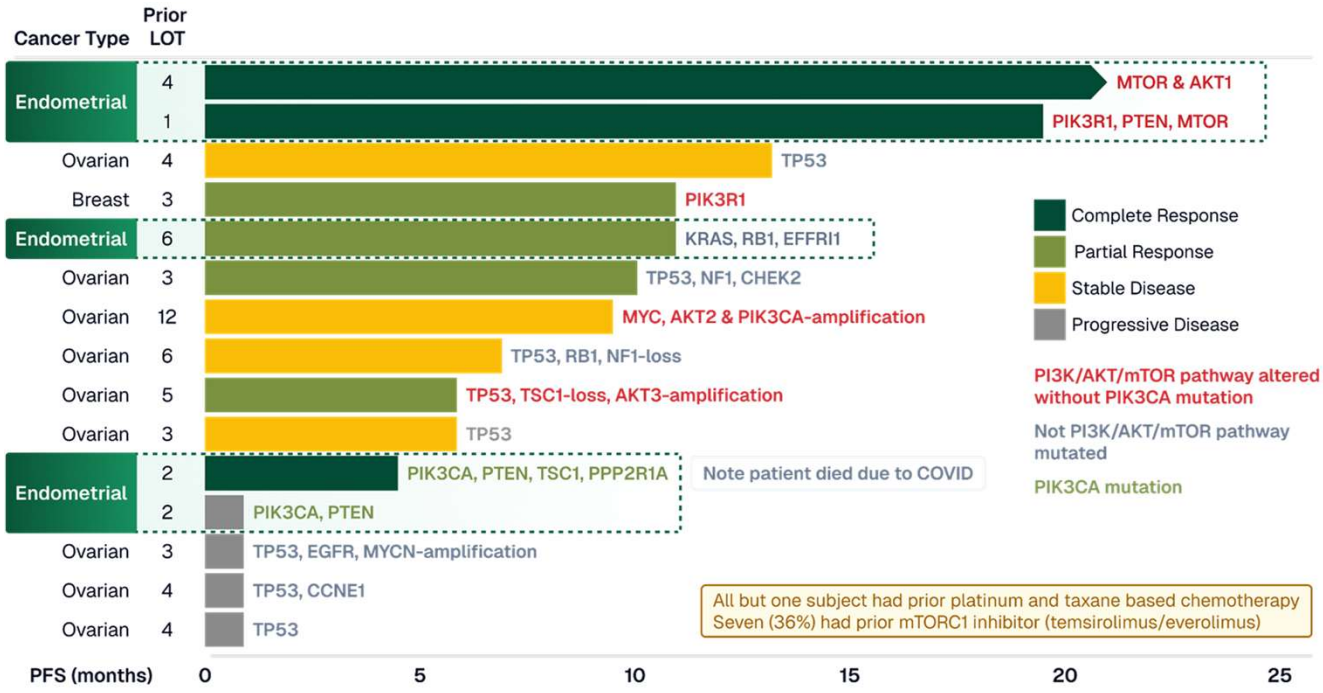
PIKTOR demonstrated preclinical pathway suppression at lower concentrations than other PI3K/AKT/mTOR targeted agents

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**

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

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

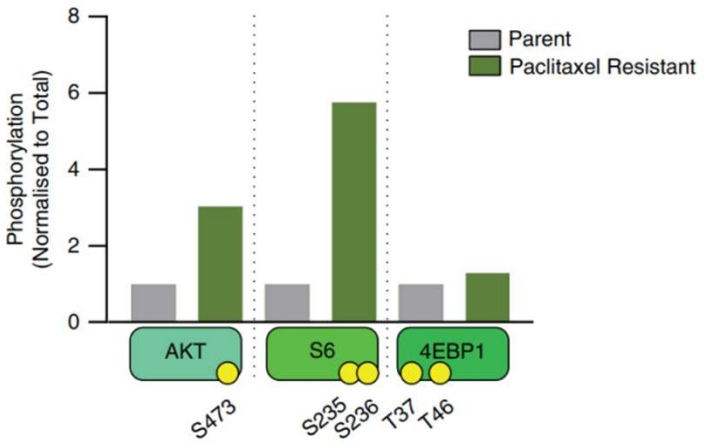
Endometrial patients only

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)

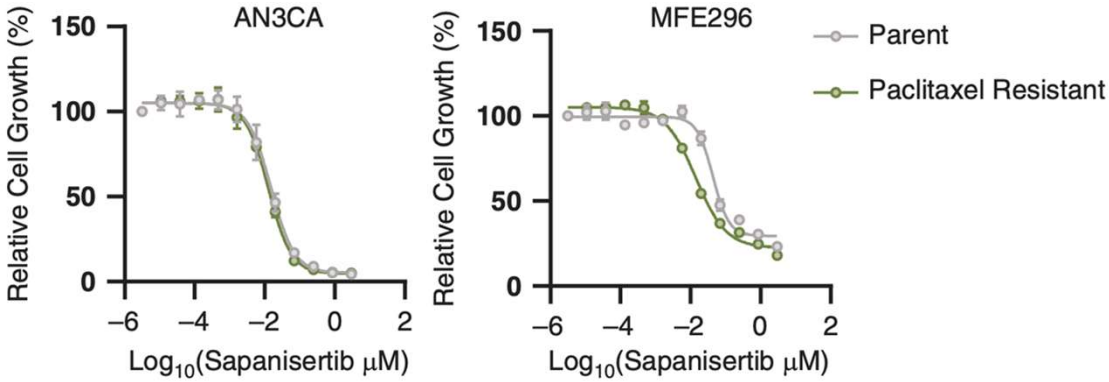
n=19 enrolled (15 response evaluable, 13 RECIST evaluable). Data cutoff per publication 10/1/21.
Starks DC, Rojas-Espaillat L, Meissner T, Williams CB. Phase I dose escalation study of dual PI3K/mTOR inhibition by Sapanisertib and Serabelisib in combination with paclitaxel in patients with advanced solid 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

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

Taxane resistance activates PAM Pathway

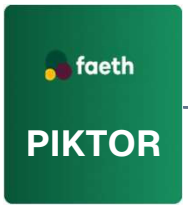


Taxane resistant cells retain sensitivity to PIKTOR



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

Phase 1 Clinical Data Comparisons (all monotherapy)



	REFERENCE	COHORT	ORR	DCR (PR+CR+SD)
Serabelisb	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

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.

Ovarian cancer: estimated PIKTOR addressable U.S. population

U.S. Incident Therapeutic Build (Annual)

- U.S. New Ovarian Cancer Cases — ~21,000
  - Advanced disease requiring systemic therapy — ~17,000
    - Platinum-resistant or refractory ovarian cancer (PROC) — ~11,600

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 × 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 × 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 × ~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) × ~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

Eligibility Criteria

- Epithelial ovarian / fallopian tube / peritoneal
- Platinum-resistant or refractory
- ≥1 prior line; measurable disease (RECIST 1.1)

R 1:1
(N=134)

Arm A:
Control (n=66)

Paclitaxel 80 mg/m² IV
Days 1, 8, 15 (q28d)

Arm B:
Experimental (n=68)

Paclitaxel 80 mg/m²
Sapanisertib 4 mg PO 3d/w

Endpoints

- Primary endpoint: PFS
- Secondary endpoints: ORR, OS, DoR, CBR, QoL, Safety
- Experimental endpoint: Genetic biomarkers in tissue / blood

Krell, J. et. al., ESMO 2025.
Abbreviations: ORR - Overall Response Rate, OS - Overall Survival, DoR - Duration of Response, CBR - Clinical Benefit Rate, QoL - Quality of Life, PFS - progression free survival, HR - Hazard Ratio, CI - Confidence Interval

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

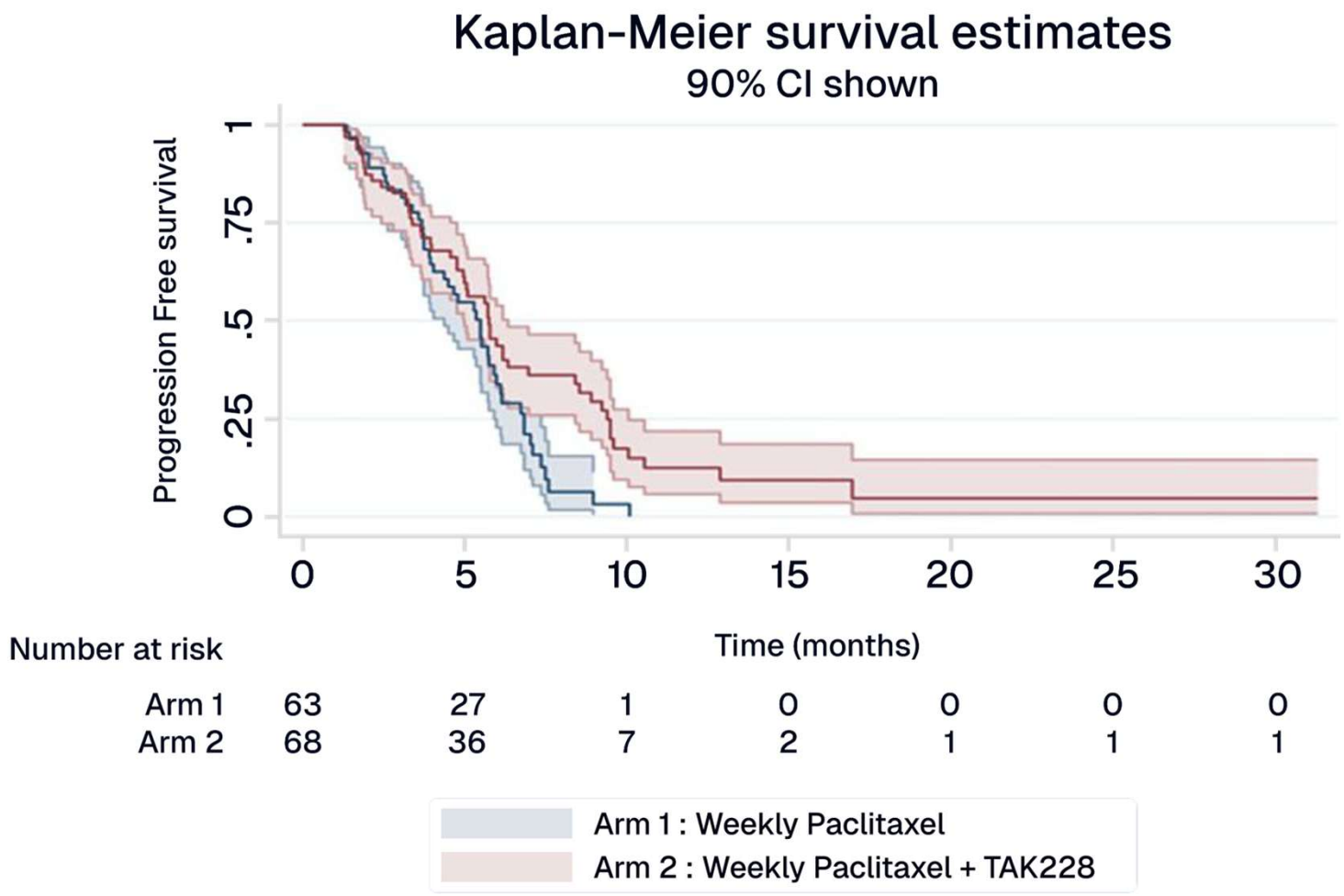
- OS, ORR and detailed safety data to come
- Potential FDA interaction in 2026 regarding future development, including potential registrational trial

Krell, J. et. al., ESMO 2025.

Abbreviations: ORR - Overall Response Rate, OS - Overall Survival, DoR - Duration of Response, CBR - Clinical Benefit Rate, QoL - Quality of Life, PFS - progression free survival, HR - Hazard Ratio, CI - Confidence Interval

Phase 2 DICE Trial PFS

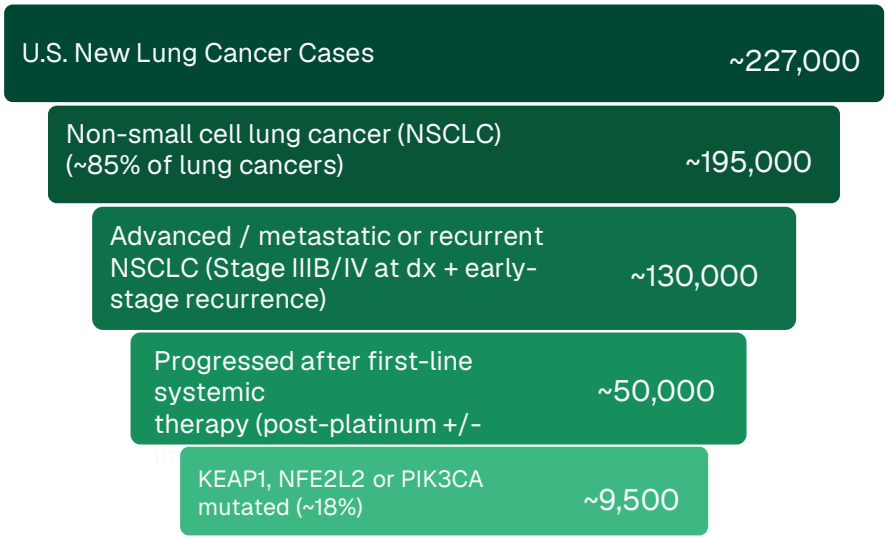
Sapanisertib (aka TAK 228) and Paclitaxel **showed PFS benefit in PROC**



Krell, J. et. al., ESMO 2025
Abbreviations: PFS - progression free survival, CI - Confidence Interval

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 × 65% Stage IIIB/IV; SEER stage distribution) + steady-state early-stage recurrence ~12K (68K early-stage entrants/yr × 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 × 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%×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 × ~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 × ~8.4% biomarker-positive 2L-eligible = ~8,900 deaths/yr; at mOS ~8mo, prevalent ≈ 8,900 × (8/12) ≈ 5,900. Both methods bracket ~6,000–7,500; point estimate ~7,000.
Abbreviations: TAM - Total Addressable Market

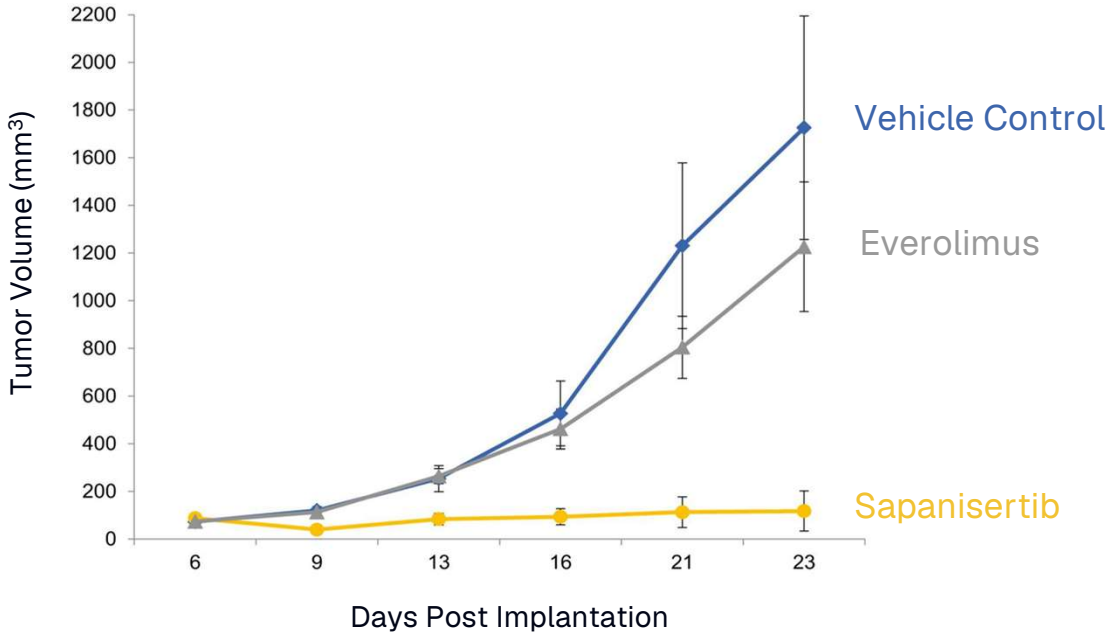
Sapanisertib shows preclinical efficacy in lung cancer models

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

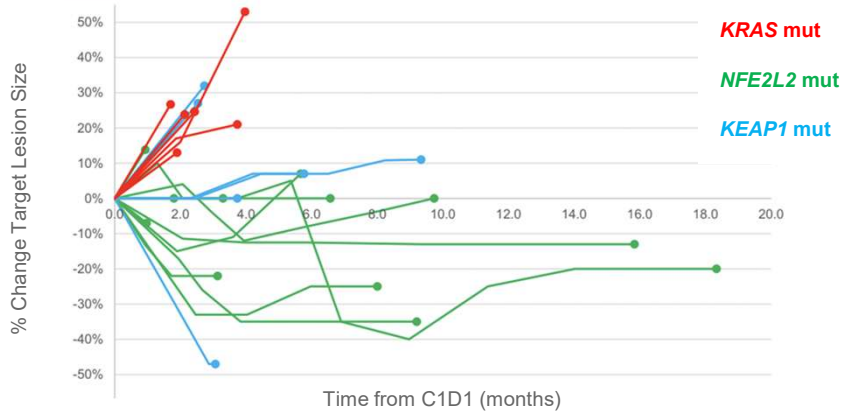


 Paik PK et al. *J Thorac Oncol.* 2023;18(4):516–526

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

Paik PK et al. *J Thorac Oncol.* 2023;18(4):516–526.
Abbreviations: ORR - Overall Response Rate, OS - Overall Survival, DoR - Duration of Response, CBR - Clinical Benefit Rate, QoL - Quality of Life, PFS - progression free survival, HR - Hazard Ratio, CI - Confidence Interval

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

 Global exclusive licenses for Serabelisib and Sapanisertib, from Takeda for all oncology indications

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

Thank you