FAETH THERAPEUTICS COMPANY OVERVIEW

Break through the PAM barrier.

JUNE 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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PIKTOR – one regimen, multiple high value opportunities

Designed for broad activity across PI3K-mutant and wild-type tumors, led by breast and endometrial cancer

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 by EOY 2026; breast Phase 1b/2 ongoing, interim expected 2H 2027

Premier investor support, with ~$203M in cash to accelerate PIKTOR in EC and BC to potential registrational study readiness[1]

[1] $202.8M cash and cash equivalents as of March 31, 2026

Abbreviations: EC – endometrial cancer, BC – breast cancer

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**				
OTHER	IEM: Small molecule targeting amino acid metabolism	Pediatric: **Rare Disease/Neuro**				

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

Faeth scientific founders: world leaders in cancer biology

Lew Cantley, PhD

Dana Farber Cancer Institute, Harvard. Founder: Agios (NASDAQ), Petra, Volastra

HARVARD MEDICAL SCHOOL

Sid Mukherjee, MD PhD

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

COLUMBIA UNIVERSITY

Oliver Maddocks, MPharm, PhD

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

CANCER RESEARCH UK

Karen Vousden, PhD

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

CANCER RESEARCH UK

Scott Lowe, PhD

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

Memorial Sloan Kettering Cancer Center

Greg Hannon, PhD

Former Director of CRUK Cambridge Institute

UNIVERSITY OF CAMBRIDGE

VIKTORIA-1 derisks PIKTOR MoA, with room for improvement

	faeth PIKTOR	Celcuity: GEDATOLISIB
Target Profile	**PI3K-alpha + mTORC1/2**	Pan-PI3K + mTORC1/2
Administration	**Oral**	Intravenous

Conclusion

We believe positive Celcuity data validates a multi-node PI3K + mTORC1/2 approach, while PIKTOR holds **potential advantages due to oral administration**, **more selective target profile and superior PK.**

PIK3CA wild type

HR+/HER2- ABC

vs. fulvestrant

Triplet:	Gedatolisib + Palbociclib + Fulvestrant	**9.3 mo** mPFS	**0.24** HR
Doublet:	Gedatolisib + Fulvestrant	**7.4 mo** mPFS	**0.33** HR

PIK3CA mutant

HR+/HER2- ABC

vs. alpelisib + fulvestrant

Triplet:	Gedatolisib + Palbociclib + Fulvestrant	**11.1 mo** mPFS	**0.50** HR
Doublet:	Gedatolisib + Fulvestrant	**11.3 mo** mPFS	**0.51** HR

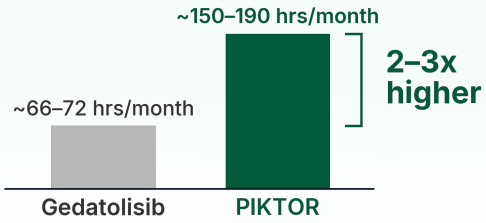
Gedatolisib is investigational. Different cohorts and comparators; not a direct comparison.
Hurvitz S, et. al., VIKTORIA-1, ASCO 2026
Abbreviations: ABC - Advanced Breast Cancer, mPFS - median progression free survival, HR - Hazard Ratio, PK - pharmacokinetics

PIKTOR has three potential ways to win: efficacy, tolerability and patient experience

Efficacy

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

Time above IC90



Modeled monthly exposure above IC90

Tolerability

Hyperglycemia AEs [3, 4]

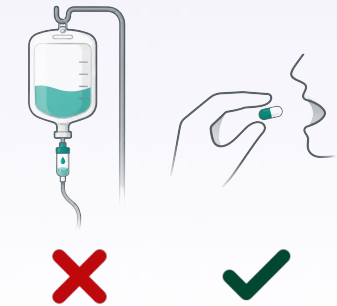
	ZOVEGALISIB	PIKTOR
Gr 1-2	35%	9%
Gr 3-4	**8%**	**5%**
Total	**43%**	**14%**

Stomatitis AEs [4, 5]

	GEDATOLISIB	PIKTOR
Gr 1-2	61%	14%
Gr 3-4	**27%**	**0%**
Total	**88%**	**14%**

Patient Experience

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

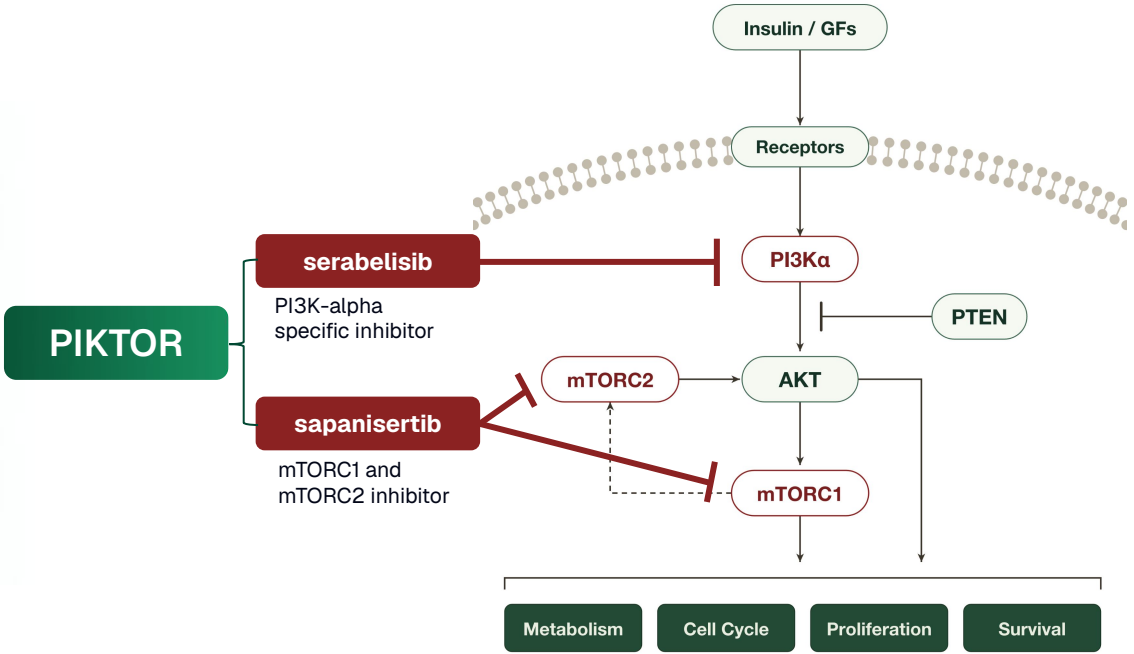


[1]Faeth internal PK modeling based on Faeth internal human PK data . [2]Based on Faeth internal analysis and estimates using publicly available data. [3]RLY-2608 Ph1 (+fulvestrant): Varkaris et al., ESMO TAT 2026, 400 mg BID Fed, RP2D Cohort [4]Data snapshot PIK-201 PIKTOR + Paclitaxel in patients with advanced endometrial cancer, 01-05-2026 (ongoing Faeth Sponsored Multicenter Phase 2 trial) [5]Layman et. al., 2022 (SABCS 2022 Poster). 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.

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 highest-frequency genomic driver across solid tumors[1].



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

PIKTOR: Four potential advantages of a multi-node approach

More complete pathway shut down

↓ Dose ↑ Tolerability

Simultaneous blockade of PI3Kα, mTORC1, and mTORC2 designed to prevent the feedback reactivation loops that limit single-node agents. PIKTOR components require lower doses than monotherapy while achieving deeper pathway suppression and better tolerability.[1, 5]

Evolved resistance is less likely

↑ Durability of response

Mutations that drive resistance to single-node inhibitors — including *PTEN* loss, *AKT1*, and *MTOR* alterations — have less capacity to reactivate the pathway under simultaneous multi-node blockade.[1, 2, 3, 4, 5]

Larger addressable market

MNI market vs. SNI

PIKTOR's multi-node mechanism targets PAM pathway activation beyond *PIK3CA* mutation alone — including *PTEN* loss and *AKT*/*MTOR* alterations prevalent in many solid tumors, as well as tumors not harboring PAM pathway mutations.[1, 5, 6, 7]

Oral administration

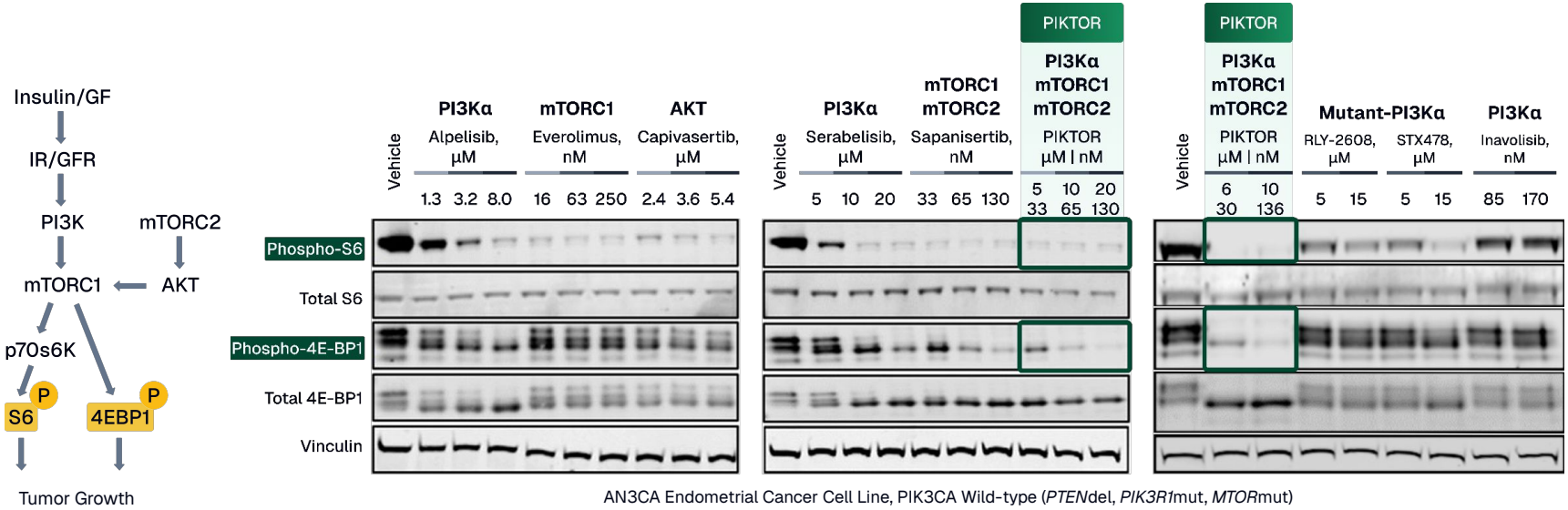
Convenient, ↑ time > IC90

Both components of PIKTOR are orally administered, eliminating the infusion burden associated with IV agents. PIKTOR's intermittent oral dosing is designed to limit the high Cmax peaks associated with toxicities such as stomatitis, while sustaining drug exposure above target-coverage thresholds.[8]

Data sources: [1]*Tyrakis et al., 2025;* [2]*Juric et al., 2015;* [3]*Coleman et al., 2021;* [4]*Varkaris et al., 2024;* [5]*Starks et al., 2022;* [6]*ACS/SEER 2025;* [7]*TCGA 2013*
[8]*Cross-trial stomatitis comparison: J Clin Oncol 2024;42:1–10 (inavolisib); André et al., N Engl J Med 2019;380:1929–40 (alpelisib); Layman et al., 2022 (SABCS poster, gedatolisib); PIK-201 PIKTOR + paclitaxel data snapshot, ongoing Faeth Phase 2 trial. (cross-ref slide 24)*
Abbreviations: MNI = multi-node inhibitors, SNI = single-node inhibitors

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

PIKTOR reduced PI3K-pathway signalling in cancer cell lines **more than single-node inhibitors** at clinically relevant concentrations[1]



AN3CA Endometrial Cancer Cell Line, PIK3CA Wild-type (*PTEN*del, *PIK3R1*mut, *MTOR*mut)

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.

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: 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, 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	✓	✗	✓	✓

Oral PIKTOR dosing is designed to enable sustained exposure while limiting risk of potential Cmax related toxicity

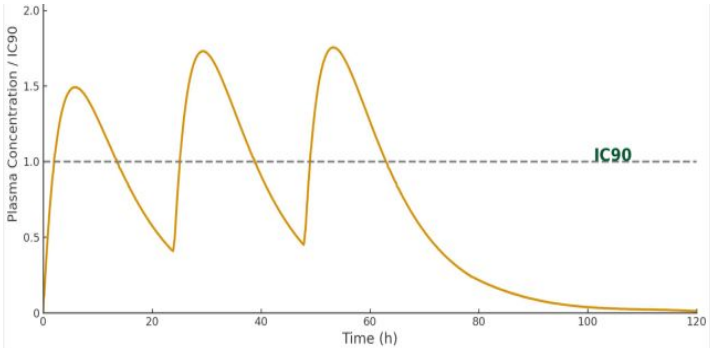
	ROUTE	SCHEDULE	EXPOSURE ≥ IC 90	CMAX: IC90
faeth PIKTOR	**Oral**	**3 days per week, every week**	**~150-190 hours per month [1]**	**~2:1 [1]**
GEDATOLISIB	IV	Once weekly, 3 weeks a month	~66-72 hours per month [2]	~50:1 [2]

PIKTOR's 3-day oral regimen is designed to deliver repeated intra-week exposure above critical efficacy thresholds

PIKTOR is given every week (4 out of 4 weeks per month), allowing sustained drug exposure

Multiple weekly doses **designed to avoid extreme Cmax** which may be associated with AEs

Human skin biopsy data showed dose-dependent reduction of 4EBP1 phosphorylation[3], a critical biomarker correlated with in vitro/in vivo efficacy[4]



Plot shows ratio of human plasma drug concentration to in vitro (cellular) IC90 for sapanisertib (cellular IC90 determined in HR+ breast cancer cell lines for sapanisertib + serabelisib). Pharmacokinetic (PK) data is modeled from Faeth internal human PK studies and represents sapanisertib oral capsule 3 mg given once a day with food for 3 days with serabelisib.

[1]Faeth internal PK modeling based on Faeth internal human PK data . [2]Based on Faeth internal analysis and estimates using publicly available data.
[3]Faeth internal data from PK/PD study in human subjects . [4]Tyrakis et. al., (2025) British Journal of Cancer, 133: 144-154.

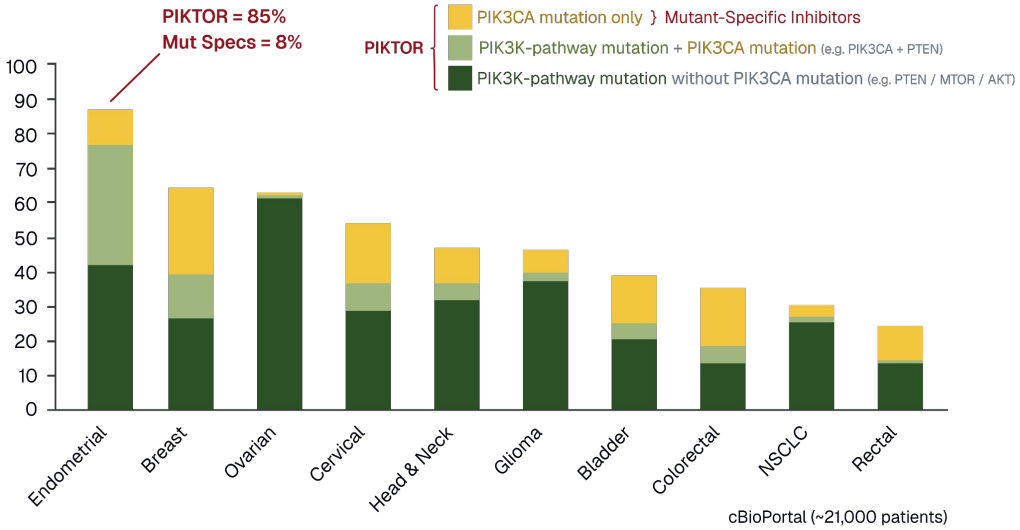
PIKTOR clinical development: Endometrial

	ENDOMETRIAL	BREAST	OVARIAN	LUNG
Phase	**Phase 2 enrolling**	Phase 1b enrolling	Phase 2 complete (Sapa + Pac); FDA interaction planned in 2026	Phase 2 protocol drafted LungMAP consortium IIT
Global TAM	**~$1.5-2B[1]**	~$20-25B[1]	~$1.5-2B[1]	~$0.75-1.25B[2]
First indication	**2L advanced EC**	HR+/HER2- advanced BC	Advanced platinum-resistant OC	*PIK3CA*, *NFE2L2*, or *KEAP1* mut adv NSCLC
Description	• **Likely first approval if development is successful** • **>80% PI3K/AKT/mTOR pathway mutated** • **Large unmet clinical need in 2L**	• Well understood mechanism with potential for broad label • 60% PI3K/AKT/mTOR pathway mutated • Oral dosage form advantage vs. gedatolisib	• Demonstrated activity in an all-comers population • 60% PI3K/AKT/mTOR pathway mutated • Successful Phase 2 complete	• Promising single agent activity of sapanisertib in *NFE2L2*/*KEAP1* mutated NSCLC

[1]Deallus' February 12, 2024 report to the Company [2]Faeth internal analysis
Abbreviations: TAM - total addressable market

Endometrial cancer is our most advanced PIKTOR indication

PAM pathway alterations by tumor type:



No PI3K inhibitor has ever been approved in endometrial cancer. Concurrent PAM mutations defeat the single-node agents that work in breast — the gap multi-node PIKTOR is built to close.

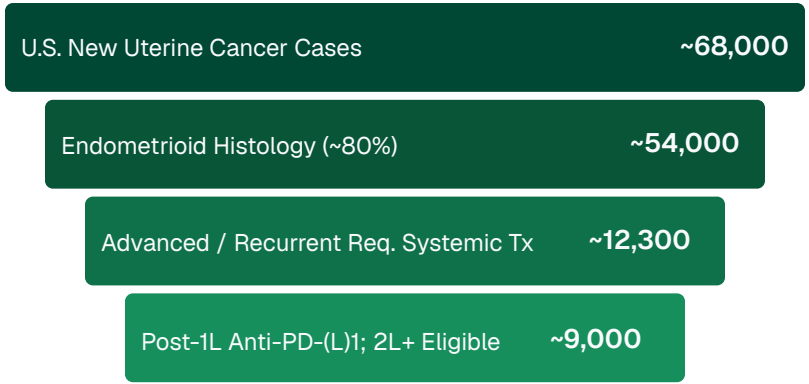
Cancer with **highest incidence of PAM pathway** alterations

EC patients are metabolically sicker. Obesity, T2DM, and hypertension frequently co-occur ("triple syndrome") and most are insulin-resistant at diagnosis, priming them for hyperglycemia, the class's signature toxicity that PIKTOR is designed to limit.[3,4,5]

[1] Luevano-Gonzalez et al. "Prospective evaluation of insulin resistance among endometrial cancer patients." *Am J Obstet Gynecol.* 2011; PMID 21324431, [2]Akhan et al. "Evaluation of hemoglobin A1c levels in endometrial cancer patients: a retrospective study." *Arch Gynecol Obstet.* 2015; PMID 25773830, [3]Morice et al. "Endometrial cancer." *Frontiers in Oncology.* 2019; doi:10.3389/fonc.2019.00744, [4]Klopp et al. "Addressing the Role of Obesity in Endometrial Cancer Risk, Prevention, and Treatment." *J Clin Oncol.* 2019; PMC5455320, [5]Nout et al. "Survival outcomes in endometrial cancer patients according to diabetes: a systematic review and meta-analysis." *BMC Cancer.* 2022; PMC9019948. 'Mut-Specs' = Mutant specific PI3K inhibitors.

Endometrial: estimated PIKTOR addressable U.S. population

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

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

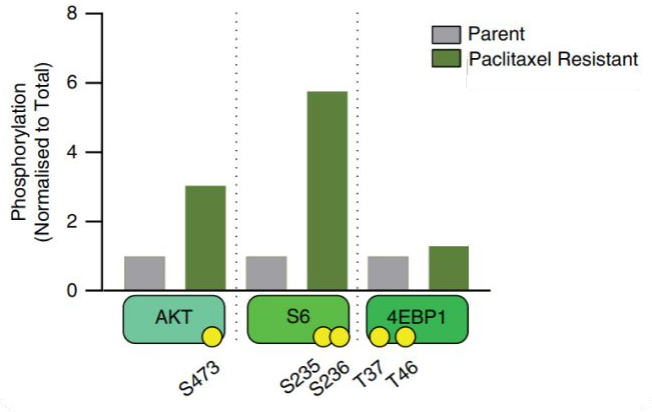
~9,000
U.S. incident patients per year post-anti-PD-(L)1, endometrioid EC

~12,500
U.S. prevalent eligible TAM

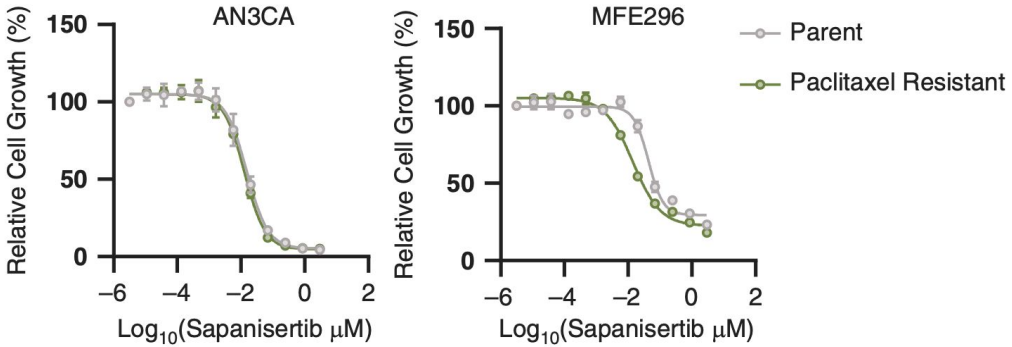
1. ACS 2026 / SEER 2025: ~68,000–69,000 new uterine corpus cases/yr. 2. Endometrioid histology ~80% (TCGA 2013; SEER).
3. Advanced/recurrent requiring systemic Tx (~12,300/yr): de novo advanced ~9,700 (54K × 18% FIGO III/IV; SEER ~9% distant + ~50% of 18% regional as FIGO III, consistent w/ 20–30% high-risk 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

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.

Ph1b: PIKTOR + paclitaxel dose escalation using multiple-fold lower doses than monotherapy RP2D

Advanced Solid Tumors

Advanced solid tumors

All-comers (mutation agnostic) Second line or later [3]

Sapanisertib / Serabelisib

Cohort 1: 2mg / 100mg (Pac 60mg/m^2)

Cohort 2: 2mg / 200mg (Pac 60mg/m^2)

Cohort 3: 2mg / 200mg (Pac 80mg/m^2)

Cohort 4: 3mg / 200mg (Pac 80mg/m^2) — RP2D Expansion

Cohort 5: 4mg / 200mg (Pac 80mg/m^2)

Monotherapy RP2D

Serabelisib 900 mg QD x 3dQW PO[1]

Combo RP2D **4.5x** less than mono

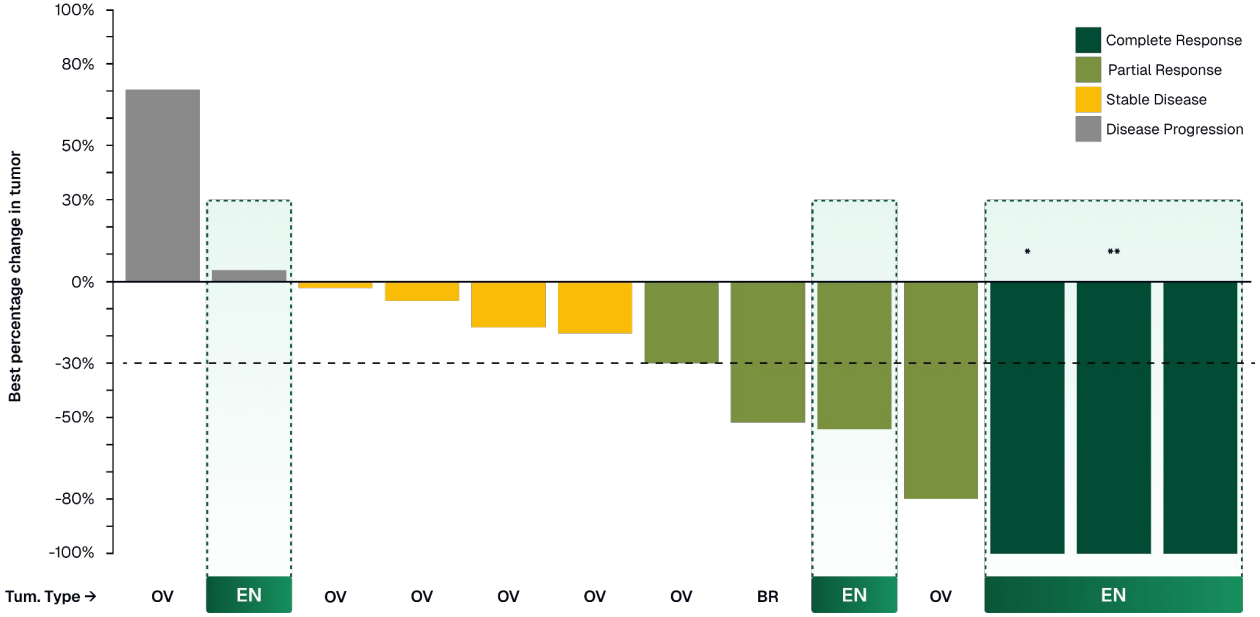
Sapanisertib 9 mg QD x 3dQW PO[2]

Combo RP2D **3x** less than mono

Data sources: [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.

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

Ph1b: PIKTOR + paclitaxel achieved 47% ORR in R/R patients, including 3 CRs



PIKTOR AND PACLITAXEL (all RECIST evaluable)

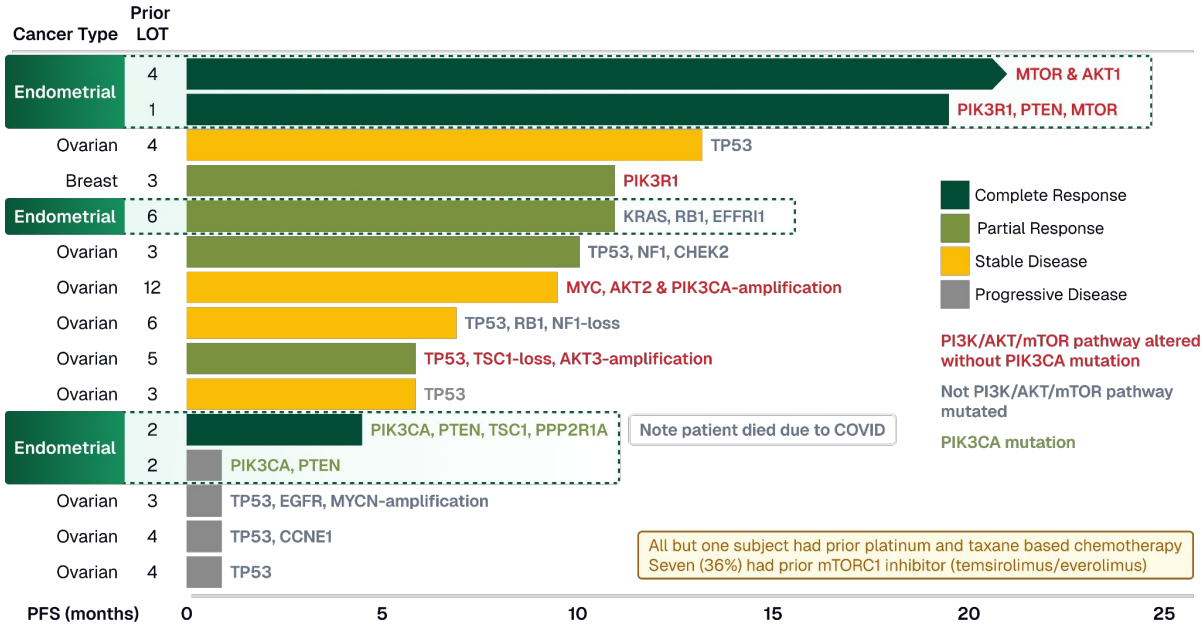
47%
Overall Response Rate

71%
ORR in patients with PI3k pathway mutation

73%
Clinical Benefit Rate in evaluated patients

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: ORR - Overall Response Rate, R/R - Relapsed Refractory, CR - Complete Response, uCR - Unconfirmed Complete Response, CBR - Clinical Benefit Rate, LOT - Lines of Therapy, Ov - Ovarian Cancer, En - Endometrial Cancer, Br - Breast Cancer

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

Ph 1b Safety Highlights: PIKTOR was generally well tolerated

Favorable toxicity profile compared to published results for approved agents

	faeth PIKTOR AND PACLITAXEL[1]	LENVATINIB AND PEMBROLIZUMAB[2]	SINGLE AGENT CHEMO IN 2L ENDOMETRIAL[3]
Grade 3 AEs	**58%**	89%	73%
Discontinuation	**~5%**	33%	8%

Primarily low grade AEs: Common AEs included Gr 1, 2 events for nausea, decreased appetite, diarrhea, fatigue, neutropenia and anemia (potentially due to paclitaxel)[1]

One dose-limiting toxicity (renal failure secondary to hyperglycemia) occurred at dose level 5 (4mg of sapanisertib, 200mg of serabelisib and 80 mg/m2 of paclitaxel).

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.
Data sources: (1) Starks et al. 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. (2) Makker et al., NEJM 2022 (KEYNOTE-775) (3) Chemotherapy: Control arm from KEYNOTE trial; Treating investigator's choice (doxorubicin 60 mg/m2 IV weekly or paclitaxel 80 mg/m2 IV weekly with 3 weeks on and 1 week off. Data cutoff per publication 10/1/21.

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

Eligibility Criteria

- Endometrial-endometrioid cancer
- 2nd line or later
- Post Pembrolizumab
- 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

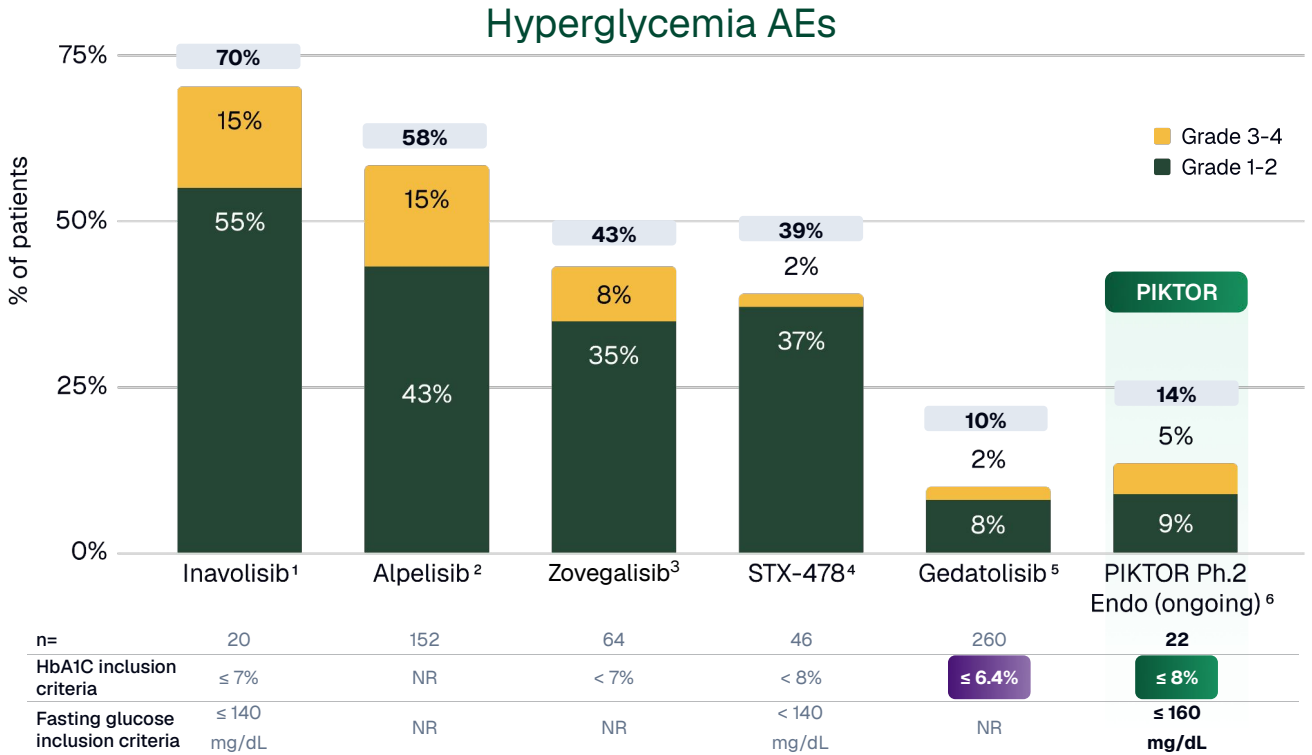
- PFS
- CBR
- Safety/ tolerability
- DOR
- OS

Exploratory endpoints

- PK
- Efficacy endpoints vs. specific mutations

Study is being conducted in partnership with: GOG PARTNERS A GOG Foundation Program

Hyperglycemia: **PIKTOR** compared to other PI3K compounds



	Inavolisib[1]	Alpelisib[2]	Zovegalisib[3]	STX-478[4]	Gedatolisib[5]	PIKTOR Ph.2 Endo (ongoing)[6]
n=	20	152	64	46	260	**22**
HbA1C inclusion criteria	≤ 7%	NR	< 7%	< 8%	**≤ 6.4%**	**≤ 8%**
Fasting glucose inclusion criteria	≤ 140 mg/dL	NR	NR	< 140 mg/dL	NR	**≤ 160 mg/dL**

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.

Data sources:

[1]Turner NC, et al. N Engl J Med. 2024;391(17):1584-1596

[2]Hurvitz SA, et al., ASCO 2026 (VIKTORIA-1 Study 2)

[3]RLY-2608 Ph1 (+fulvestrant): Varkaris et al., ESMO TAT 2026, 400 mg BID Fed, RP2D Cohort

[4]Jhaveri et al., ASCO 2026, Abstract 1072 (PIKALO-1, NCT05768139)

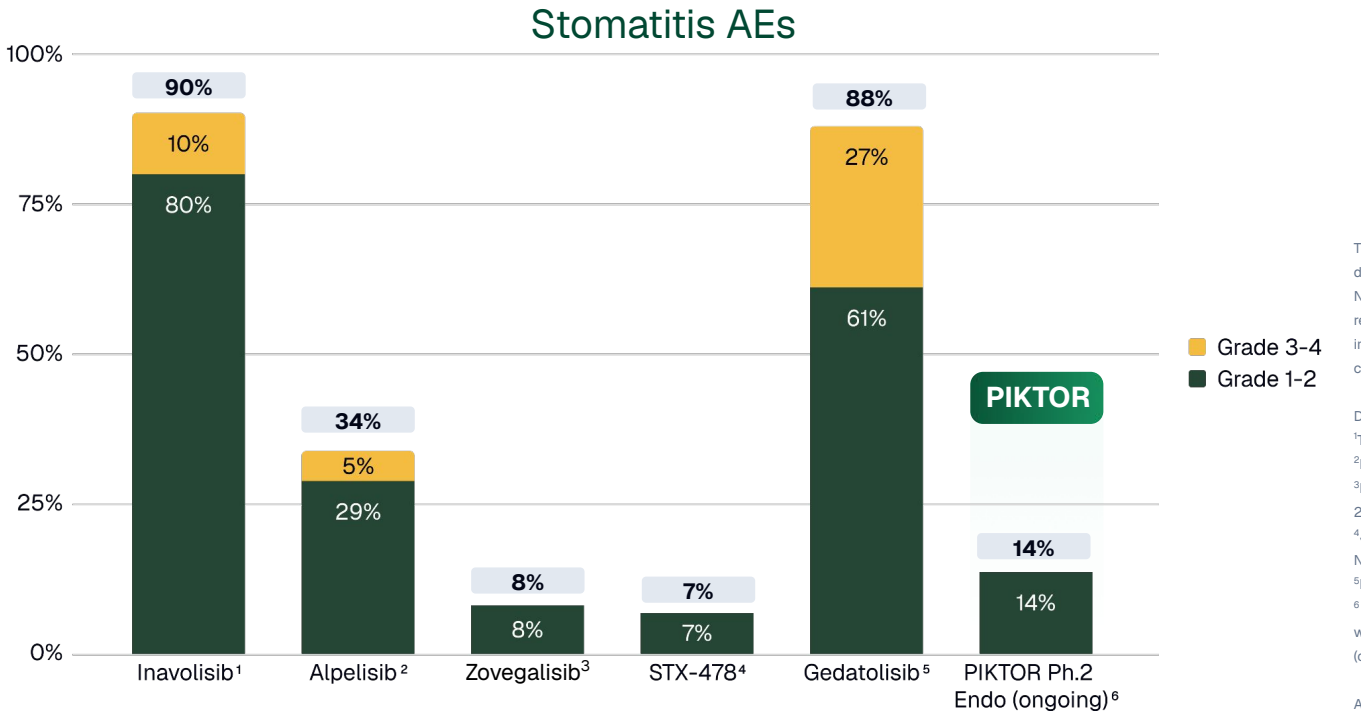
[5]Celcuity VIKTORIA-1 Phase 3 Results Presentation, Arms A + B, Oct 2025

[6] Data snapshot PIK-201 PIKTOR + Paclitaxel in patients with advanced endometrial cancer, 01-05-2026 (ongoing Faeth Sponsored Multicenter Phase 2 trial)

Abbreviations:

AE - Adverse Events, NR - not reported in primary source, HbA1C - Hemoglobin A1C

Stomatitis: **PIKTOR** compared to other PI3K compounds



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.

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[1]Turner NC, et al. N Engl J Med. 2024;391(17):1584-1596

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[3]RLY-2608 Ph1 (+fulvestrant): Varkaris et al., ESMO TAT 2026, 400 mg BID Fed, RP2D Cohort

[4]Jhaveri et al., ASCO 2026, Abstract 1072 (PIKALO-1, NCT05768139)

[5]Layman et. al., 2022 (SABCS 2022 Poster)

[6] Data snapshot PIK-201 PIKTOR + Paclitaxel in patients with advanced endometrial cancer, 01-05-2026 (ongoing Faeth Sponsored Multicenter Phase 2 trial)

Abbreviations: AE - Adverse Events

PIKTOR clinical development: Breast

	ENDOMETRIAL	BREAST	OVARIAN	LUNG
Phase	Phase 2 enrolling	**Phase 1b enrolling**	Phase 2 complete (Sapa + Pac); FDA interaction planned in 2026	Phase 2 protocol drafted LungMAP consortium IIT
Global TAM	~$1.5-2B[1]	**~$20-25B[1]**	~$1.5-2B[1]	~$0.75-1.25B[2]
First indication	2L advanced EC	**HR+/HER2- advanced BC**	Advanced platinum-resistant OC	*PIK3CA*, *NFE2L2*, or *KEAP1* mut adv NSCLC
Description	• Likely first approval if successful • >80% PI3K/AKT/mTOR pathway mutated • Large unmet clinical need in 2L	• **Well understood mechanism with potential for broad label** • **60% PI3K/AKT/mTOR pathway mutated** • **Oral dosage form advantage vs. gedatolisib**	• Demonstrated activity in an all-comers population • 60% PI3K/AKT/mTOR pathway mutated • Successful Phase 2 complete	• Promising single agent activity of sapanisertib in NFE2L2/KEAP1 mutated NSCLC

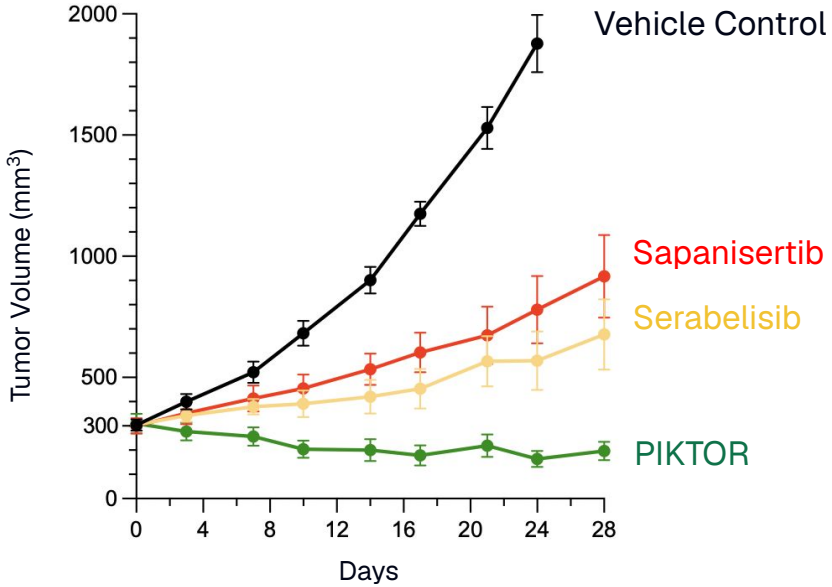
[1]Deallus' February 12, 2024 report to the Company [2]Faeth internal analysis
Abbreviations: TAM - total addressable market

PIKTOR showed preclinical efficacy in breast cancer models

MDA-MB-361, Breast cancer xenograft tumor model
(ER+, HER2+, *PIK3CA mutant)*

Dosing Schedule Mirrors Clinical Schedule:
Once per day, 3 days per week on, 4 days per week off

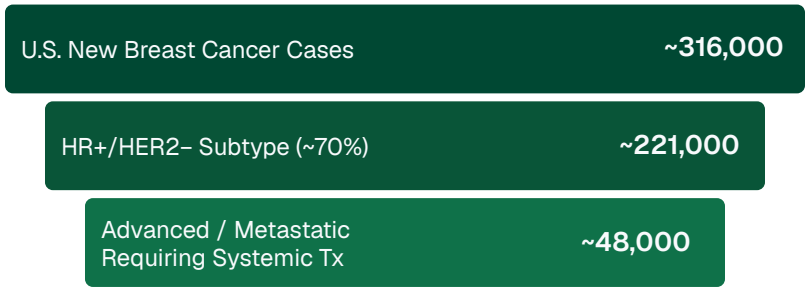
Sapanisertib 0.5 mg/kg PO QD3dQW
Serabelisib 75 mg/kg PO QD3dQW



 Faeth internal data / Tyrakis et al., Br J Cancer 2025;133:144–154.

HR+/HER2- breast cancer: estimated PIKTOR addressable U.S. populations

U.S. Incident Therapeutic Build (Annual)



Three addressable patient populations from the ~48k advanced pool

~30,000 U.S. incident patients per year 1L ETR ABC	**~33,000** U.S. prevalent 1L ETR patients TAM
~18,000 U.S. incident patients per year 1L ETS ABC	**~60,000** U.S. prevalent 1L ETS patients TAM
~32,000 U.S. incident patients per year 2L post-CDKi + AI ABC	**~37,000** U.S. prevalent 2L+ patients TAM

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 post-CDK4/6i] ~38K start 1L CDK4/6i+ET (48K × 80%; Gao et al. Frontiers Pharmacology 2025 citing Parikh 2024; corroborated by Flatiron P-VERIFY n=9,146 and ESMO 2025 Brufsky n=11,557); 20% residual = ET-alone (indolent/low-volume), chemo-first (visceral crisis), frail/BSC, or non-CDK4/6i trial. 5. [2L] ~32K incident 2L+: 38K × ~85% lifetime progression on 1L CDK4/6i+ET (PALOMA-2/MONALEESA-2 5yr data; SONIA trial). 6. [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). 7. [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). 8. [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). 9. [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). 10. [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

Serabelisib and Sapanisertib have each independently shown activity in HR+/HER2- breast cancer

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

Phase 1b/2 trial (PIK-101) - announced first patient dosed in May 2026

1

Phase 1b dose escalation

Dose escalation in WT and MT HR+/HER2- breast cancer with adaptive Bayesian design; starting dose near anticipated therapeutic exposure, with backfill cohorts during escalation.

Establish RP2D

First patient — 1H 2026

2

Phase 2 expansion

Expansion cohorts to be defined based on Phase 1b data; may include novel–novel combinations.

Response & durability

Interim data — 2027

3

Phase 3 registrational

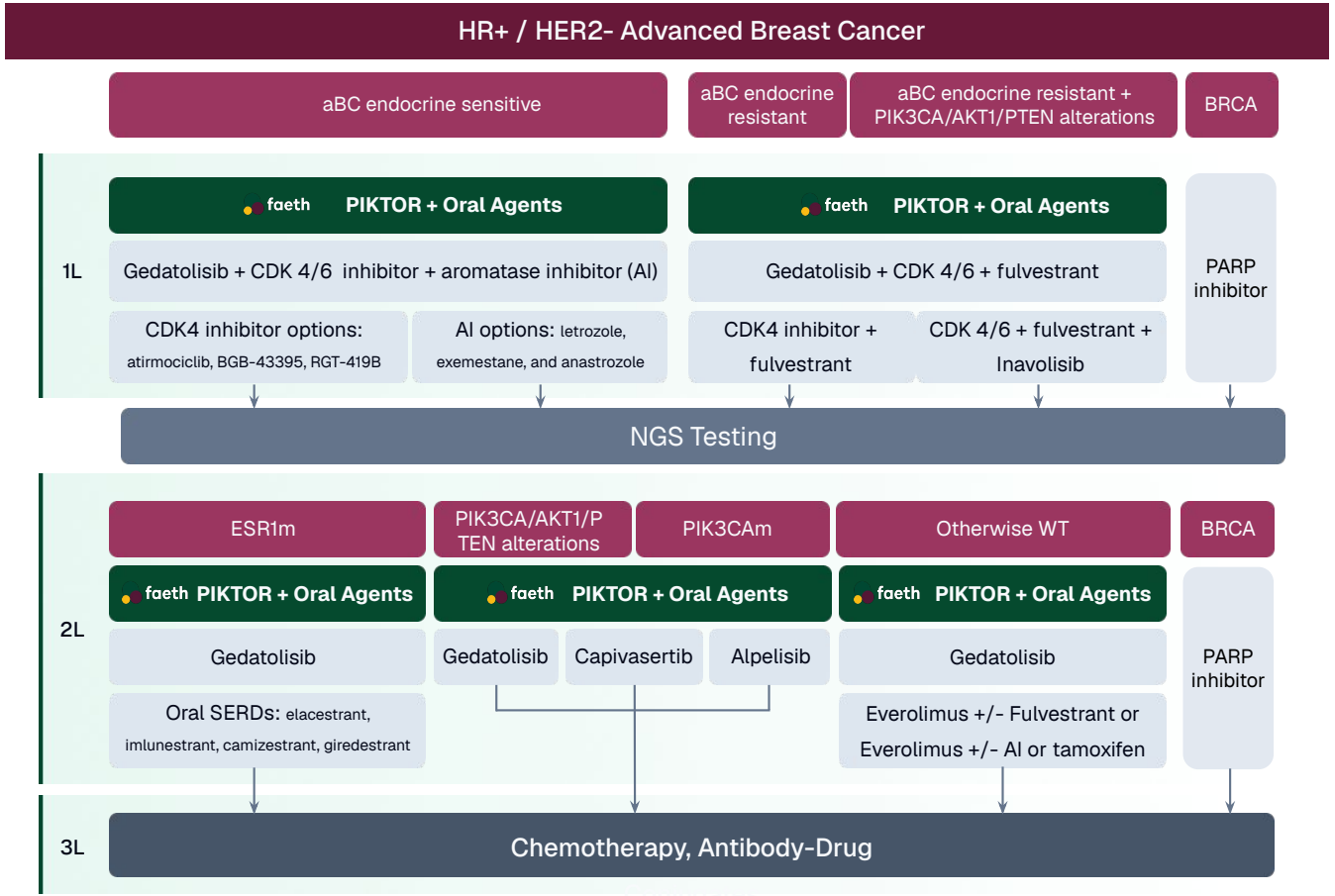
Pivotal development informed by Phase 1b/2 readouts.

Pivotal

TBD

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.



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, predictable toxicity on intermittent dosing to date

Contribution of Components

- Independent monotherapy activity observed for each component
- Prior studies largely established sapanisertib contribution of components. Serabelisib plus paclitaxel CoC in EC to be established for PIKTOR approval

[1]Hurvitz, S. et. al., (2025) ESMO Presentation

Abbreviations: PK/BA - Pharmacokinetics / Bioavailability, EC - endometrial cancer, CoC - contribution of components

CMC infrastructure positioned for registration and scale

Mature DS/DP Materials

- Both DS are highly stable
- DS/DP Batches with 36-60 month stability data
- Proven manufacturing history

Manufacturing Scale-Up

- DS and DP process optimisation being completed
- Demo batches planned for 2026
- Registrational readiness on track

Defined Life-Cycle Strategy

- Formulation work underway

Expect ample DS/DP supply for ongoing and proposed Phase 1b/Phase 2 Studies

Key PIKTOR patents

SUBJECT MATTER	PATENT EXPIRATION DATE	NOTES
Composition of Matter • Serabelisib API • Sapanisertib API	Aug 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 = 2026 (20-year expiry ~2046)	• Development work ongoing

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

February 2026 financing expected to fund PIKTOR program through key anticipated catalysts

PROGRAM	2026	2027	2028
ENDOMETRIAL	2H: Enrollment complete, topline data readout	Last patient dosed + 6 months data	
BREAST	1H: Trial initiation, Ph1b dose escalation	- Interim safety data from dose escalation - Interim efficacy data from dose escalation; expansion cohorts initiated	Expansion data
PROC	Potential FDA interaction regarding future development		
KEY EXTERNAL EVENTS	1H/2H: Potential gedatolisib FDA Approval, VIKTORIA-1 PI3Kmt Readout		Potential gedatolisib VIKTORIA-2 Data Readout

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 clinical development: Ovarian

	ENDOMETRIAL	BREAST	OVARIAN	LUNG
Phase	Phase 2 enrolling	Phase 1b enrolling	**Phase 2 complete (Sapa + Pac); FDA interaction planned in 2026**	Phase 2 protocol drafted LungMAP consortium IIT
Global TAM	~$1.5-2B[1]	~$20-25B[1]	**~$1.5-2B[1]**	~$0.75-1.25B[2]
First indication	2L advanced EC	HR+/HER2- advanced BC	**Advanced platinum-resistant OC**	*PIK3CA*, *NFE2L2*, or *KEAP1* mut adv NSCLC
Description	• Likely first approval if successful • >80% PI3K/AKT/mTOR pathway mutated • Large unmet clinical need in 2L	• Well understood mechanism with potential for broad label • 60% PI3K/AKT/mTOR pathway mutated • Oral dosage form advantage vs. gedatolisib	• **Demonstrated activity in an all-comers population** • **60% PI3K/AKT/mTOR pathway mutated** • **Successful Phase 2 complete**	• Promising single agent activity of sapanisertib in NFE2L2/KEAP1 mutated NSCLC

[1]Deallus' February 12, 2024 report to the Company [2]Faeth internal analysis
Abbreviations: TAM - total addressable market

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

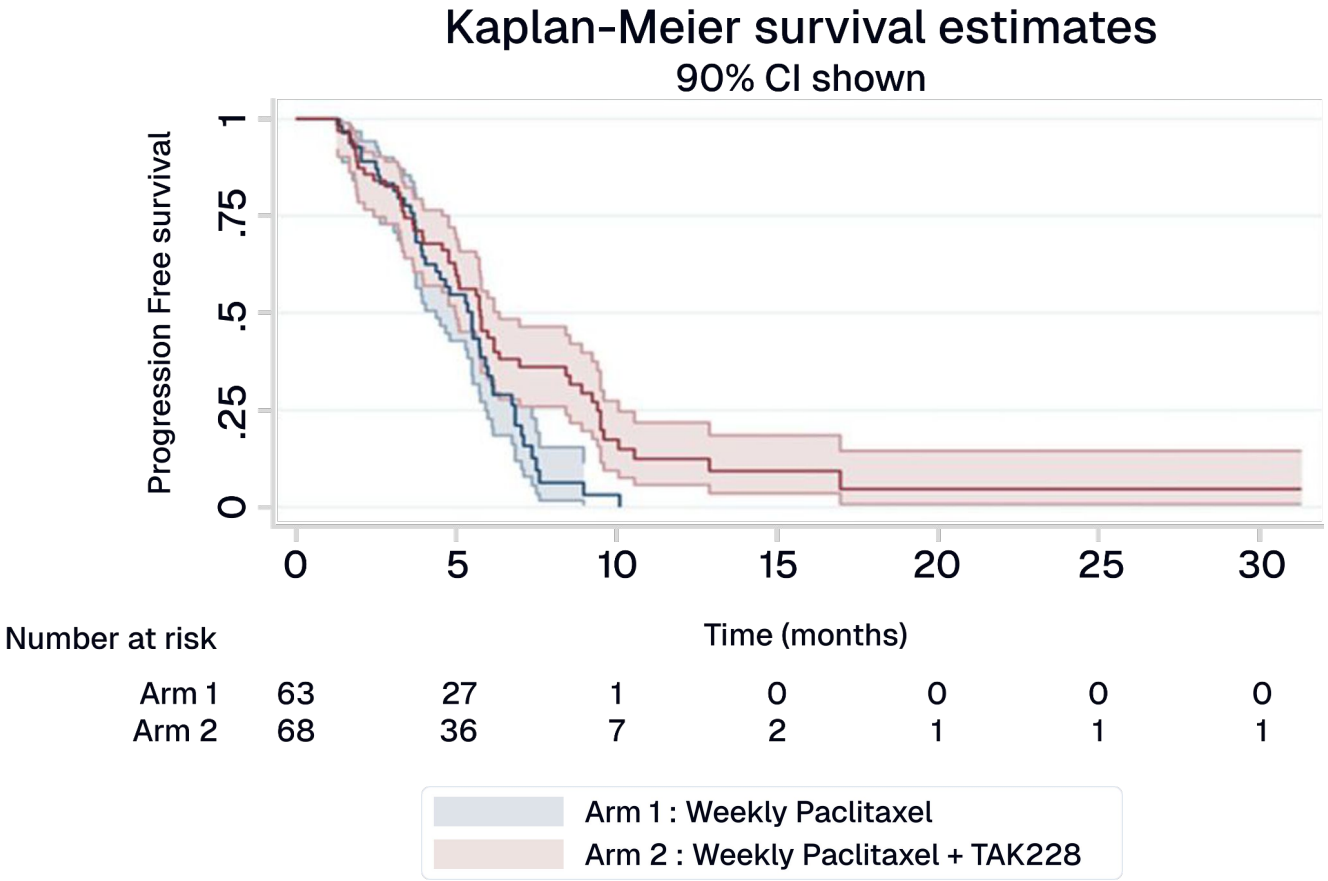
- OS, ORR and detailed safety data to come
- Potential FDA interaction in 2026 regarding future development, including potential registrational trial

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

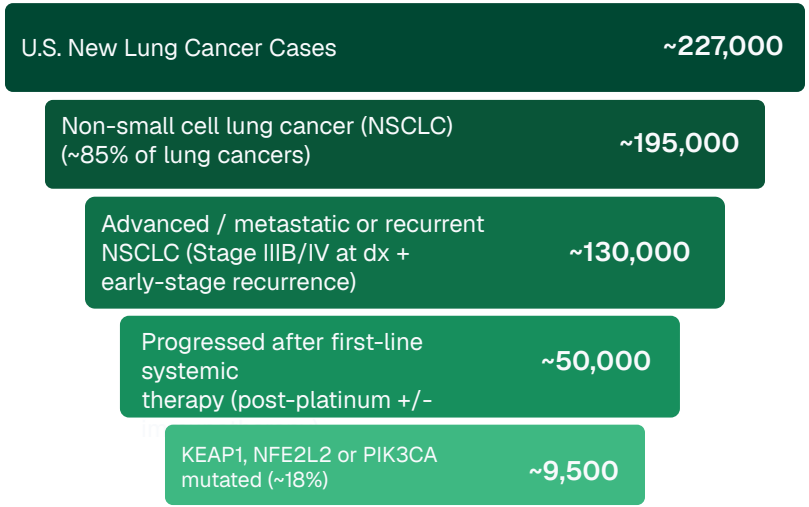
PIKTOR clinical development: Lung

	ENDOMETRIAL	BREAST	OVARIAN	LUNG
Phase	Phase 2 enrolling	Phase 1b enrolling	Phase 2 complete (Sapa + Pac); FDA interaction planned in 2026	**Phase 2 protocol drafted** **LungMAP consortium IIT**
Est. Market size	~$1.5-2B[1]	~$20-25B[1]	~$1.5-2B[1]	**~$0.75-1.25B[2]**
First indication	2L advanced EC	HR+/HER2- advanced BC	Advanced platinum-resistant OC	***PIK3CA, NFE2L2*, or *KEAP1* mut adv NSCLC**
Description	• Likely first approval if successful • >80% PI3K/AKT/mTOR pathway mutated • Large unmet clinical need in 2L	• Well understood mechanism with potential for broad label • 60% PI3K/AKT/mTOR pathway mutated • Oral dosage form advantage vs. gedatolisib	• Demonstrated activity in an all-comers population • 60% PI3K/AKT/mTOR pathway mutated • Successful Phase 2 complete	• **Promising single agent activity of sapanisertib in NFE2L2/KEAP1 mutated NSCLC**

[1]Deallus' February 12, 2024 report to the Company [2]Faeth internal analysis
Abbreviations: TAM - total addressable market

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-stageentrants/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 before2L 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 astable 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 (Franket 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

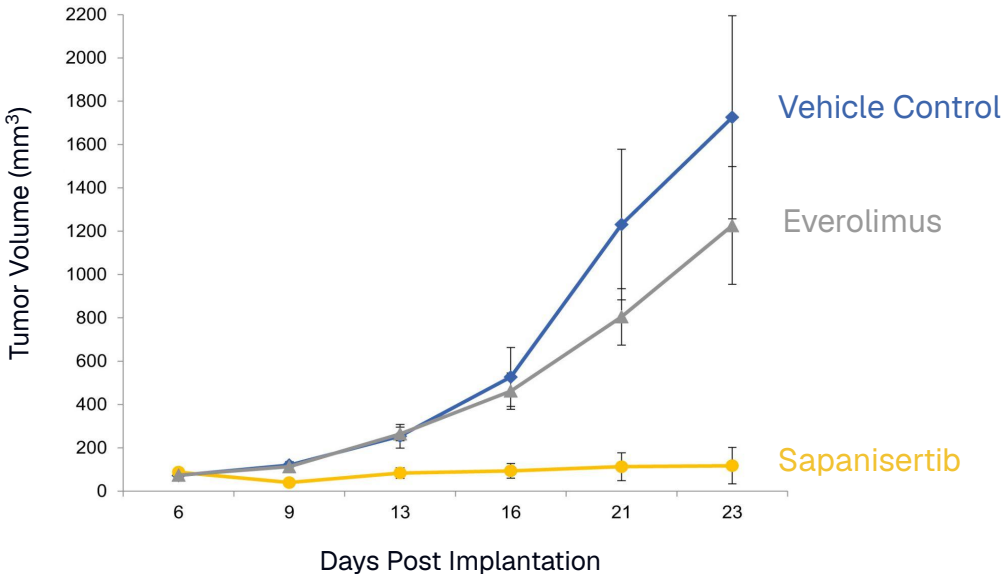
Sapanisertib shows preclinical efficacy in lung cancer models

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

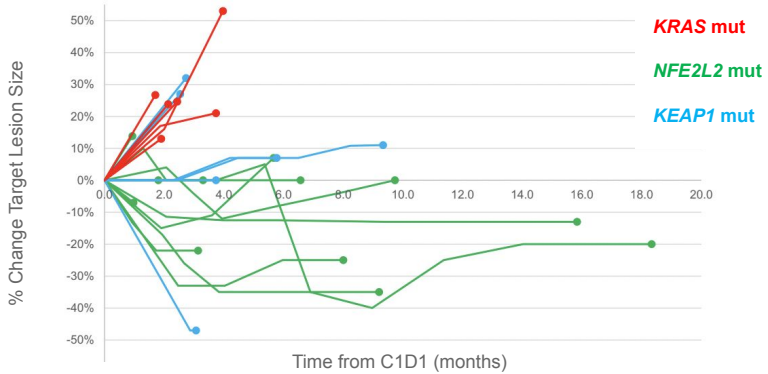


 Paik PK et al. *J Thorac Oncol.* 2023;18(4):516–526

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

Thank you