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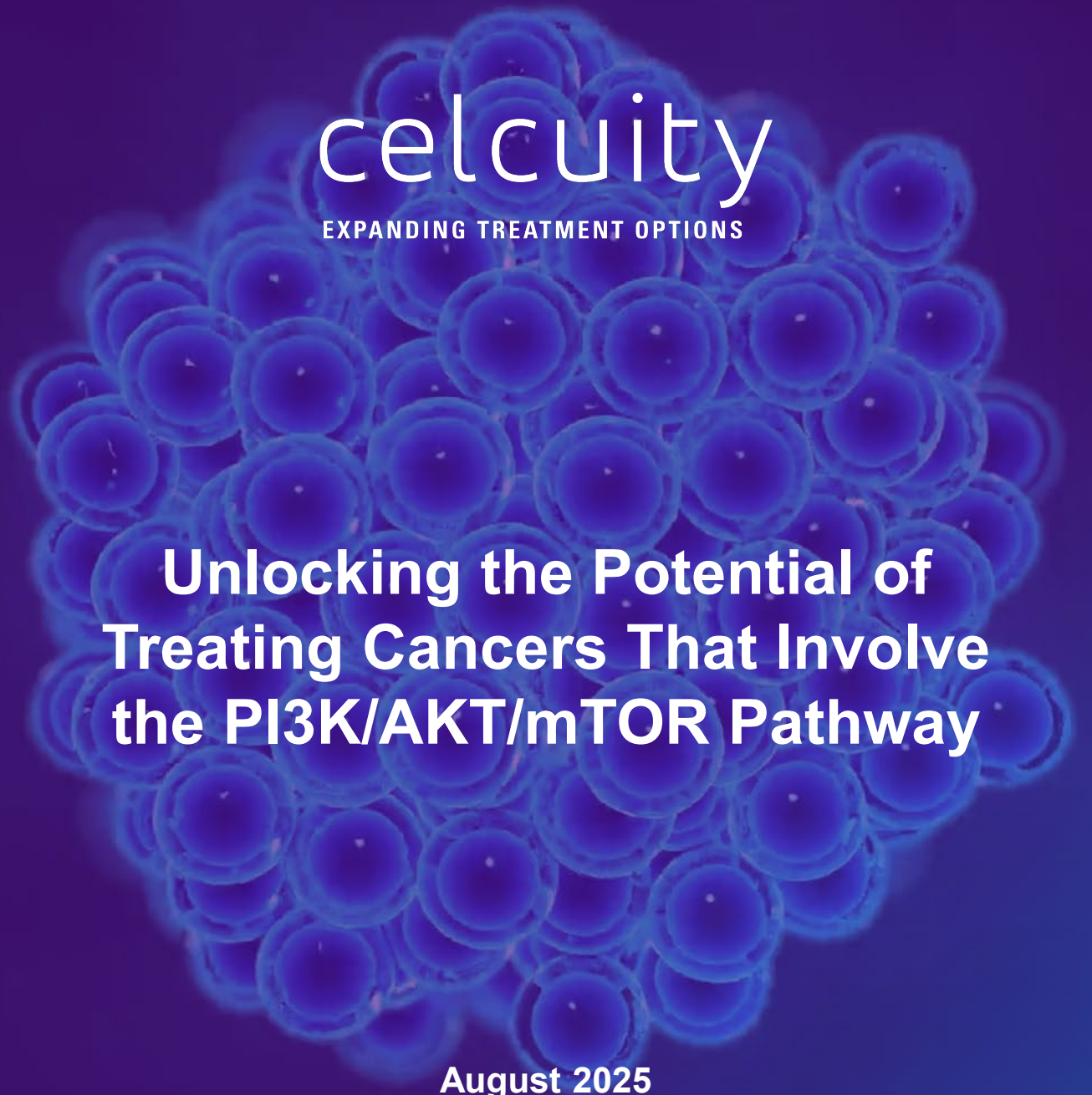

Unlocking the Potential of Treating Cancers That Involve the PI3K/AKT/mTOR Pathway

August 2025

Forward-Looking Statements

This presentation contains statements that constitute “forward-looking statements” within the meaning of the Private Securities Litigation Reform Act of 1995. These statements relate to Celcuity’s business, operations, and financial condition, and include but are not limited to our current beliefs, expectations and assumptions regarding the future of our business and our pipeline, including our lead drug candidate gedatolisib and its potential benefits, that involve substantial risks and uncertainties that could cause actual results to differ materially from those expressed or implied by such statements. These statements include, but are not limited to, (i) our interpretation of topline clinical trial data; (ii) our expectation regarding regulatory interpretations and assessments of our clinical data; (iii) our expectations regarding the timing of and our ability to obtain regulatory approvals for gedatolisib within and outside the U.S.; (iv) our beliefs with respect to the clinical utility of gedatolisib, its market acceptance and the size of the market, as well as the cost to commercialize and our ability to serve that market; (v) our expectations regarding governmental laws and regulations affecting our operations; (vi) our beliefs about our ability to capitalize on the exclusive global development and commercialization rights obtained from our license agreement with Pfizer Inc. (“Pfizer”) with respect to gedatolisib, and payments due to Pfizer thereunder; (vii) our product pricing, coverage, reimbursement and revenue expectations; (viii) our expectations as to the availability of capital and use of proceeds from our financing activities as well as cash on hand; and (ix) our expectations regarding our ability to obtain and maintain intellectual property protection for gedatolisib.

These statements may be affected by underlying assumptions that may prove inaccurate or incomplete and are subject to change. Certain risks, uncertainties and other factors include, but are not limited to: the uncertainties inherent in research and development, including the cost of clinical trials, and the ability to meet anticipated clinical endpoints and commencement and/or completion dates for our clinical trials involving gedatolisib which include our ongoing VIKTORIA-1 and VIKTORIA-2 phase 3 clinical trials, and our ongoing Phase 1b/2 clinical trial; our limited operating history; our potential inability to develop, obtain FDA approval for and commercialize gedatolisib on a timely basis or at all; the reporting of topline results based on a preliminary analysis of key efficacy and safety data prior to a more comprehensive review of the data, and such topline data may not accurately reflect the complete results of a clinical trial; the complexity and difficulty of demonstrating the safety and sufficient magnitude of benefit to support regulatory approval of gedatolisib; the uncertainties and costs associated with commercializing pharmaceuticals; challenges we may face in developing and maintaining relationships with our vendors and partners; the uncertainty regarding market acceptance by physicians, patients, third-party payors and others in the medical community, and with the size of the market opportunity available to us; difficulties we may face in managing growth, such as hiring and retaining a qualified sales force and attracting and retaining key personnel; changes in government regulations; tightening credit markets and limitations on access to capital on favorable terms or at all; the time and expense associated with defending third-party claims of intellectual property infringement, investigations or litigation threatened or initiated against us; and potential changes to economic and trade policy in the U.S. and globally, including tariffs. Actual results may differ materially from past results, future plans and projected future results. As forward-looking statements involve significant risks and uncertainties, caution should be exercised against placing undue reliance on such statements. Additional information regarding these and other factors can be found in Celcuity’s Annual Report on Form 10-K for the fiscal year ended December 31, 2024 and its subsequent Quarterly Reports on Form 10-Q, all of which are filed with the U.S. Securities and Exchange Commission and available at www.sec.gov. The forward-looking statements in this presentation speak only as of the original date of this presentation and we undertake no obligation to update or revise any of these statements, except as required by law.

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Topline VIKTORIA-1
PIK3CA Wild-Type Cohort Data

In patients with HR+/HER2-/*PIK3CA* wild-type (WT) advanced breast cancer (ABC),

- gedatolisib plus fulvestrant and palbociclib (triplet)
- gedatolisib plus fulvestrant (doublet)

MET THE STUDY'S TWO PRIMARY ENDPOINTS

By demonstrating statistically significant and clinically meaningful improvement in progression free survival versus fulvestrant

Gedatolisib Triplet

- mPFS was **9.3 months vs. 2.0** months for fulvestrant
  - **7.3-month incremental improvement** in mPFS
- **HR = 0.24**
  - **4.2x higher** likelihood of survival w/o disease progression

Gedatolisib Doublet

- mPFS was **7.4 months vs. 2.0 months** for fulvestrant
  - **5.4-month incremental improvement** in mPFS
- **HR = 0.33**
  - **3.0x higher** likelihood of survival w/o disease progression

Abbreviations: mPFS - median progression free survival; HR – Hazard Ratio

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The Celcuity Opportunity

Effectively treating PAM pathway driven tumors is one the largest opportunities in oncology

1. • **Gedatolisib's differentiated MOA and PK profile result in a highly potent, cytotoxic, and well tolerated PAM inhibitor**

2. • **Phase 3 VIKTORIA-1 WT cohort efficacy results showed unprecedented reduction in risk of disease progression or death and incremental improvement in progression free survival in patients with HR+/HER2- ABC**

3. • **A Phase 3 study in 1L patients with HR+/HER2- ABC is enrolling**
   • **A Phase 1b/2 trial in 2L patients with mCRPC has reported promising early data and is enrolling additional cohorts**

4. • **Uniquely positioned to advance multiple potential blockbuster indications in breast and prostate cancer**
   • Pro forma cash, cash equivalents, short-term investments of $455M as of Q2 2025 expected to fund operations through 2027[1]

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(1) Includes $287M of net debt and equity capital raised 7/30/25

Unlocking the Potential of Treating Cancers That Involve the PI3K/AKT/mTOR Pathway

One of the most important oncogenic pathways

PI3K/AKT/mTOR (PAM) regulates key metabolic functions

- Plays a key role promoting tumor cell proliferation
- Cross-regulates other oncogenic pathways
- Affects immune response by regulating tumor microenvironment

Most highly altered of all signaling pathways[1]

Proportion of alterations correlates to pathway's role as a cancer driver

PAM	**38%**
RAS	15%
HER2	8%
EGFR	5%

Largest untapped drug development opportunity in solid tumors

Breast and prostate cancers involve PAM pathway

- **>500,000** addressable patient population in US, 5EU, and Japan
- Nominal penetration of PAM drugs in these markets

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

Difficult to Safely and Comprehensively Inhibit the PAM Pathway

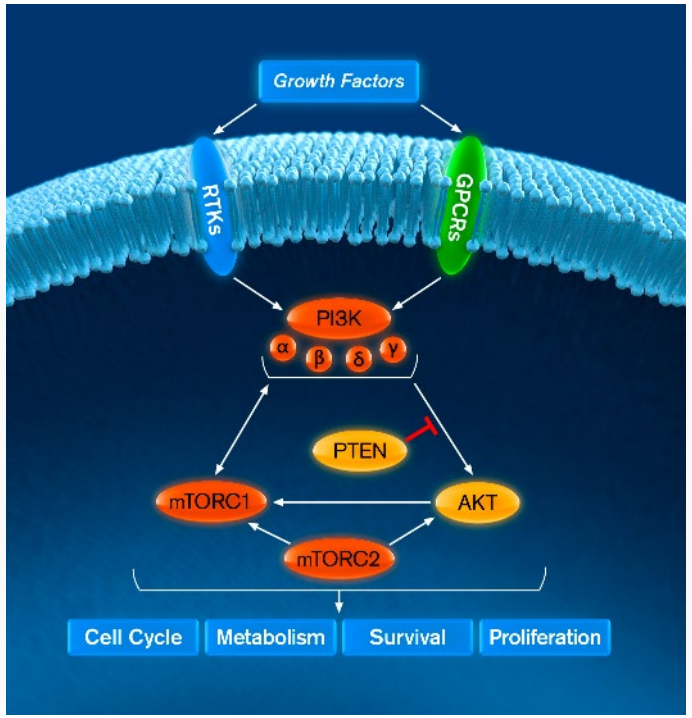
Optimal efficacy may require inhibition of all Class I PI3K isoforms and mTORC1 and mTORC2

Multiple pathway targets provide functional redundancy

If only a single target is inhibited, redundancy ensures pathway function is maintained[1-9]

Feedforward and feedback loops between PI3K isoforms, AKT, and mTOR cross-activates uninhibited targets[1-9]

Explains why 1st generation of PAM inhibitors were pan-PI3K/mTOR inhibitors



Therapeutic window for oral PI3K/mTOR inhibitors is narrow

Difficult to optimize pathway inhibition without inducing undue toxicity

Early generations of orally administrated pan-PI3K or pan-PI3K/mTOR inhibitors induced unacceptable toxicity[10]

Led to focus on development of single-node PAM inhibitors (e.g. PI3Kα, mTORC1, AKT)

1st Gen — Oral pan-PI3K/mTOR inhibitors
Toxicity high, poor PK properties
Failed in Phase 1/2

2nd Gen — Pan-PI3K inhibitors
Significant toxicity
Failed in Phase 3

3rd Gen — Single-target inhibitors
Limited PFS benefit
Four drugs approved

Today → Need safe, potent pan-PI3K/mTORi

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Sources: (1) Juric 2015 Nature. 518:240-244; (2) Castel 2021 Nat Cancer. 2:587-597; (3) Mao 2021 Nat Commun. 12:5053; (4) Schwartz 2015 Cancer Cell. 27:109-122. (5) Chandarlapaty 2011, Cancer Cell. 19:58-71; (6) Bago 2016, EMBO J. 35:1902-1922; (7) Manning 2017, Cell. 169:381-405.; (8) Mukherjee 2021, Mol Cell. 81:708-723 e705 (9) Elkabets 2013, Sci Transl Med; 5(196); (10) Alves 2023, Int. J. Mol. Sci., 24, 4522

Gedatolisib Has a Highly Differentiated Mechanism of Action and Potency

Potential First-in-Class PAM (PI3K, AKT, mTOR) Inhibitor with superior cytotoxicity vs. single node PAM inhibitors

Cell-Free Biochemical Dose Response Analysis

IC_{50} (nM)[1]

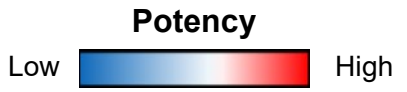
Node	Gedatolisib[2]	Alpelisib[3]	Everolimus[4]	Capivasertib[5]
PI3K-α	**0.4**	~4.0	-	-
PI3K-β	**6.0**	1,156	-	-
PI3K-γ	**5.4**	250	-	-
PI3K-δ	**6.0**	290	-	-
mTORC1	**1.6**	-	~2.0	-
mTORC2	**1.6**	-	-	-
AKT	-[6]	-	-	3.0

Gedatolisib is potent against all Class I PI3K isoforms & mTORC1/2

- Limits cross-activation that occurs with node-specific drugs
- Gedatolisib is more potent against each node than other PAM inhibitors
  - 70-100x more potent than capivasertib against targets downstream of AKT[6]
- Comprehensive pathway blockade can induce anti-tumor activity independent of PIK3CA status

Live Cell Proliferation Rate Dose Response Analysis[7]

Average values for 14 PIK3CA MT and 14 PIK3CA WT breast cancer cell lines



	GR_{50} (nM)				Max Cell Growth Inhibition[1]			
	Geda	Alpe	Evero	Capi	Geda	Alpe	Evero	Capi
All	12	6,308	3,611	8,666	168%	89%	62%	80%
MT	12	2,594	1,867	2,590	174%	116%	68%	99%
WT	12	10,308	5,501	15,209	162%	62%	56%	60%

Gedatolisib is highly potent and cytotoxic in vitro

- Significantly more potent and cytotoxic than other PAM inhibitors in vitro
  - > 300X higher potency
  - 1.5x – 2.8x higher cytotoxicity
- Only PAM inhibitor with similar activity in PIK3CA MT and WT

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(1) IC50 derived from cell-free biochemical dose response analysis; (2) Venkatesan 2010 J Med Chem 53(6):2636-45. (3) Fritsch 2014, Mol Cancer Ther. 13(5):1117-29. (4) Schuler 1997; Transplantation, 64(1):36-42. (5) Davies 2012, Mol Cancer Ther 11(4):873-87; (6) Mallon 2011, Clin Cancer Res 17(10); (7) Rossetti 2023 SABCS. Footnote: Growth rate (GR) was assessed using 28 cell lines by measuring live cells reducing potential with Real Time-Glo MT luciferase assay before and after 72h drug treatment. GR50 (conc required to inhibit growth rate by 50%) is a measure of potency. GR-Max (GR at highest drug conc. tested) is a measure of efficacy. Hafner et al, Nat. Methods, 2016 (Sorger lab, Harvard); NIH LINCS program. No head-to-head trials have been conducted; data collected from different trials, in different patient populations and may not be comparable

Gedatolisib PK Properties and IV Administration Optimize Safety Profile

Lower toxicity vs. approved PI3K inhibitors

	Gedatolisib[1]	Alpelisib[2,3]	Copanlisib[3]	Duvelisib[3]	Idelalisib[3]
Target(s)	Pan-PI3K mTOR	PI3K-α	Pan-PI3K	PI3K-δ	PI3K-δ
Administration	IV	Oral	IV	Oral	Oral
Dosing (mmol/month)	0.88	*19.03*	0.37	*3.22*	*20.22*
Volume of distribution (L)	39	114	*871*	29	23
Hyperglycemia (G 3/4)	1%	*26%*	*41%*	-	-
Treatment related SAE's	2%	*10%*	*26%*	65-73%	50-77%
Treatment related (TR) Discontinuations	0%	*13%*	*16%*	35%	17-53%

Gedatolisib vs. PI3K-α and pan-PI3K drugs

(single-agents)

- >95% lower rate of Grade 3/4 hyperglycemia
  - Due to gedatolisib's lower liver exposure
  - Alpelisib dosage 22x > gedatolisib
  - Copanlisib 50x > retention liver vs plasma
- >80% lower rate of TR discontinuations
- 3x-20x more balanced distribution

Gedatolisib vs. PI3K-δ drugs

(single-agents)

- 73%-97% lower dosage (molar/month)
- No direct GI exposure
- Minimal GI, liver, and infection-related AE's

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(1) Shapiro 2015, internal data on file; 154 mg weekly dose (MTD); all AE refers to related AEs; (2) Juric 2018, hyperglycemia from 300 mg daily dose arms (MTD); SAE and related treatment related discontinuation data from all arms; (3) US Package Insert; Note: No head-to-head trials have been conducted; data collected from different trials, in different patient populations and may not be comparable. Abbreviations: G, Grade; SAE, serious adverse event; mmol = miliimolar; L = liter

Gedatolisib Single Agent Safety Profile

Phase 1 Trial: gedatolisib at maximum tolerated dose (MTD) - 154 mg weekly (IV)[1]

- **Limited incidence of Grade 3 adverse events**
- The most frequent AE, stomatitis, is manageable with prophylactic steroidal mouth rinse
  - Stomatitis was not treated prophylactically in this study
  - **Prophylactic treatment may reduce G2 incidence by 90%; G3 by 100%[2]**
  - Phase 3 studies prescribe prophylaxis
- **Low incidence of Grade 3 hyperglycemia (1%)**
- **No treatment related neutropenia**
- No Grade 4 or 5 adverse events

MTD Arm (n=42)

Related TEAE's > 20%			
	Grade 1	Grade 2	Grade 3/4
Adverse Event	%	%	%
Stomatitis	45	2	7
Nausea	36	2	2
Hyperglycemia	17	7	1
Vomiting	19	2	2
Asthenia	7	12	2
Fatigue	19	2	-
Appetite decrease	14	7	-

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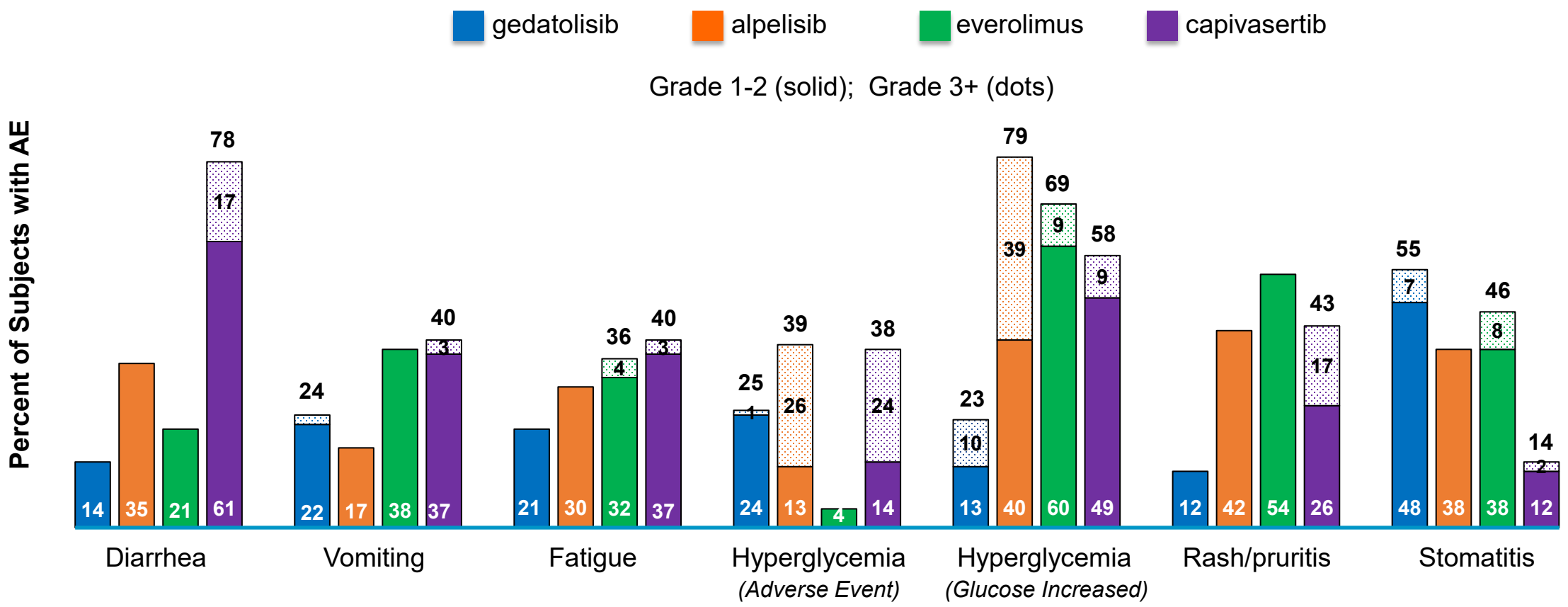
Source: (1) Shapiro 2015; (2) Rugo 2017; TEAE = treatment emergent adverse events; AE = adverse event.

Safety Data for Gedatolisib vs. Single Node PAM Inhibitors

Fewer patients reported AE when treated with gedatolisib compared to other PAM inhibitors



Source for all data except Hyperglycemia (Glucose Increased) from single agent studies: Source: (GED) Shapiro 2015, internal data. (ALP) Juric 2018, 300 mg daily dose; (EVE) Tabernero JCO 2008, 10 mg QD or 50 mg QW; (CAP) Hyman JCO 2017; Source for Hyperglycemia (Glucose Increases) data: ALP, EVE, CAP: US Package Insert. GED: Layman Lancet 2024. Note: Hyperglycemia (Glucose Increased) is a laboratory abnormality graded according to specific fasting glucose values whereas Hyperglycemia (Adverse Event) is graded according to a clinical assessment. No head-to-head trials have been conducted; data collected from different trials, in different patient populations and may not be comparable

Clinical Development Programs
Current

2nd Line HR+/HER2- Advanced Breast Cancer

Phase 3 clinical trial for gedatolisib with fulvestrant +/- palbociclib

- Patients with **HR+/HER2- advanced breast cancer (ABC)** who progressed on CDK4/6 therapy and an AI[1]
- All-comer design (*PIK3CA*+/-) includes separate primary endpoints for mutated and non-mutated *PIK3CA* patients
- **Breakthrough Therapy Designation** was granted by the FDA in July 2022

1st Line HR+/HER2- Advanced Breast Cancer

Phase 3 clinical trial for gedatolisib + CDK4/6i + fulvestrant

- Patients with HR+/HER2- ABC who are **endocrine therapy resistant (ETR)** and treatment naïve for ABC
- All-comer design (*PIK3CA*+/-) includes separate primary endpoints for mutated and non-mutated *PIK3CA* patients
- Significant unmet need – mPFS with SOC is approximately 7 months[2]

2nd Line Metastatic Castration Resistant Prostate Cancer

Phase 1b/2 clinical trial for gedatolisib with darolutamide

- Extensive literature describes androgen pathway linkage to the PAM pathway[3]
- Gedatolisib demonstrated superior potency and efficacy compared to other PAM inhibitors in nonclinical studies[4]
- Promising preliminary clinical activity with an AR inhibitor in Celcuity Phase 1 study[5]

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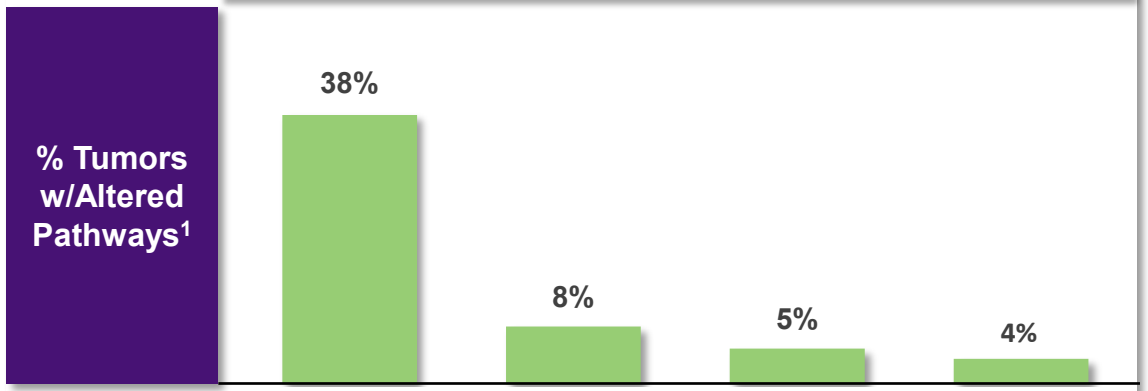
(1) Clinicaltrials.gov (2) Jhaveri, SABCS, 2023 (INAVO120); (3) Carver et al, Cancer Cell 2011; (4) Sen, ASCO-GU, 2023; (5) Celcuity Press Release, June 30, 2025.
Abbreviations: AI – aromatase inhibitor

The PAM Pathway is the Most Underdeveloped Target in Solid Tumors

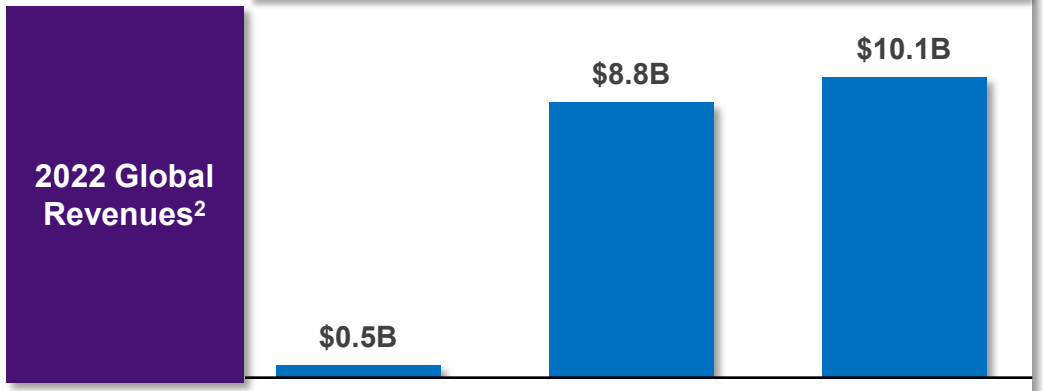
PAM is the most frequently altered pathway in solid tumors



Target *(key tumor type)*	PAM *(multiple)*	HER2 *(breast)*	EGFR *(lung)*	ALK *(lung)*
Global Revenues[2]	~0.5B	~$10B	$5.5B	$2.5B
Key Drugs	Piqray Truqap	Perjeta Herceptin	Tagrisso	Alecensa Xalkori

Drug revenues from PAM inhibitors are a small fraction of other targeted therapy classes

PAM revenue potential comparable to CDK4/6 & AR therapies



Target *(key tumor type)*	PAM *(breast/prostate)*	CDK4/6 *(breast)*	AR *(prostate)*
Potential Patient Pop[3]	552K	240K	312K
Key Drugs	Piqray Truqap	Ibrance Kisqali	Xtandi Zytiga

PAM potential patient population is not tumor specific like CDK4/6 or AR inhibitors

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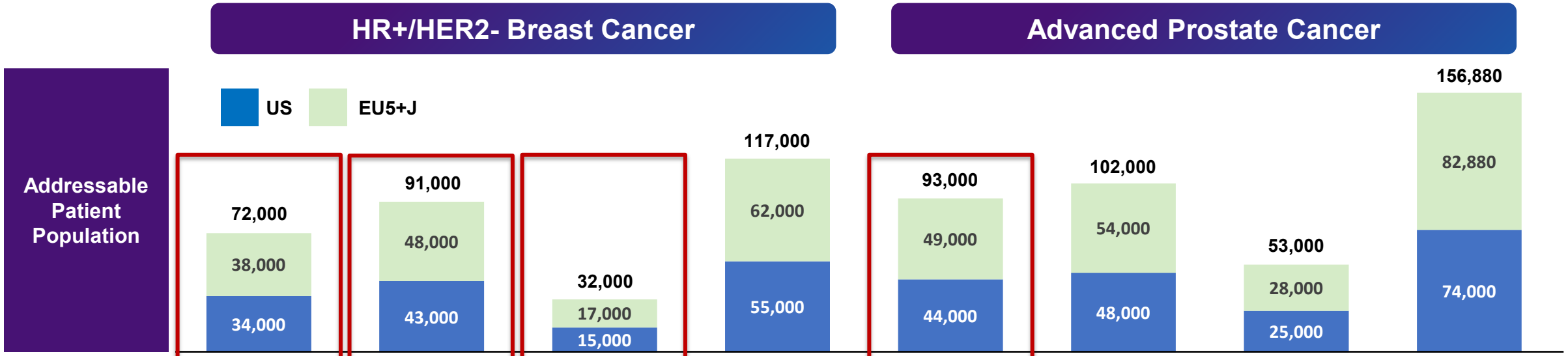
(1) cBioPortal References:Cerami et al., Cancer Discov. 2012, and Gao et al., Sci. Signal, 2013; (2) Annual Reports for Novartis, Pfizer, Astellas, Roche, AstraZeneca, Johnson & Johnson; (3) American Cancer Society, Breast Cancer Statistics 2022; American Cancer Society Facts and Figures 2019-2020; Salvo, E. M. et al. (2021); Scher, et al. 2015; Datamonitor Healthcare; Leith, A. et al. 2022; George, D. J. et al. 2022; EU5 calculated using 112% EU + Japan; scale up factor

Multiple potential blockbuster indications in both tumor types



Indication	2L ABC Post-CDKi + AI	1L ABC ET Sensitive	1L ABC ET Resistant	High Risk EBC Adjuvant	1L/2L mCRPC Post-ARi	1L mCRPC	nmCRPC	mHSPC
Duration of Therapy (DoT)	~12 months	~45 months	~15 months	~12 months	~15 months	~24 months	~24 months	~20 months
Basis for DoT assumption	Geda Ph 1b mPFS	Geda Ph 1b mPFS	Ph 3 data with other PAMi	SOC treatment window	Ph 2/3 data w/other PAMi	SOC DoT + 50%	SOC DoT + 50%	SOC DoT + 50%
US Market Opportunity	~$5-$6B	~$10B+	~$3B	$6-$8B	$8B+	$10B+	$6-$8B	$10B+

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Sources: Internal estimates using data from American Cancer Society, Breast Cancer Statistics 2022; American Cancer Society Facts and Figures 2019-2020; Dowsett, M 2009; Salvo, E. M. et al. 2021; Scher, et al. 2015; Datamonitor Healthcare; Leith, A. et al. 2022; George, D. J. et al. 2022; EU5+Japan calculated using 112% scale up factor from Globocan 2020 data; Celcuity internal estimates
Abbreviations: HR, hormone receptor; ABC, advanced breast cancer, EBC, early breast cancer; CRPC, castration resistant prostate cancer; nm, non-metastatic; HSPC, hormone sensitive prostate cancer; ET, endocrine therapy; PAMi, PI3K/AKT/mTOR inhibitor

Key Gedatolisib Patents

Loss of exclusivity now expected to occur in 2042; expect new formulations to extend this period

Subject Matter	Patent Expiration Date	Note
Composition of Matter (API) (generic and species)	Dec 2034	▪ Includes 209 days of patent term adjustment (PTA), and expected 5 years of patent term extension (PTE)
Cyclodextrin Formulations	Jan 2041	▪ Includes 578 days of PTA ▪ Drug product formulation used in current Phase 3 trials ▪ Since Cyclodextrin is a functional excipient, this patent extends patent exclusivity period for gedatolisib
Dosage Regimens	August 2042	▪ Patent issued July 8, 2025 ▪ Treatment schedule would be on product label, extending patent exclusivity period for gedatolisib
Method of Treatment for Diseases	Pending	▪ Filed December 2023 ▪ Covers non-oncology indication
Method of Treatment for Cancer	Pending	▪ Filed August 2024 ▪ Covers oncology indications

Gedatolisib for Advanced Breast Cancer (ABC)

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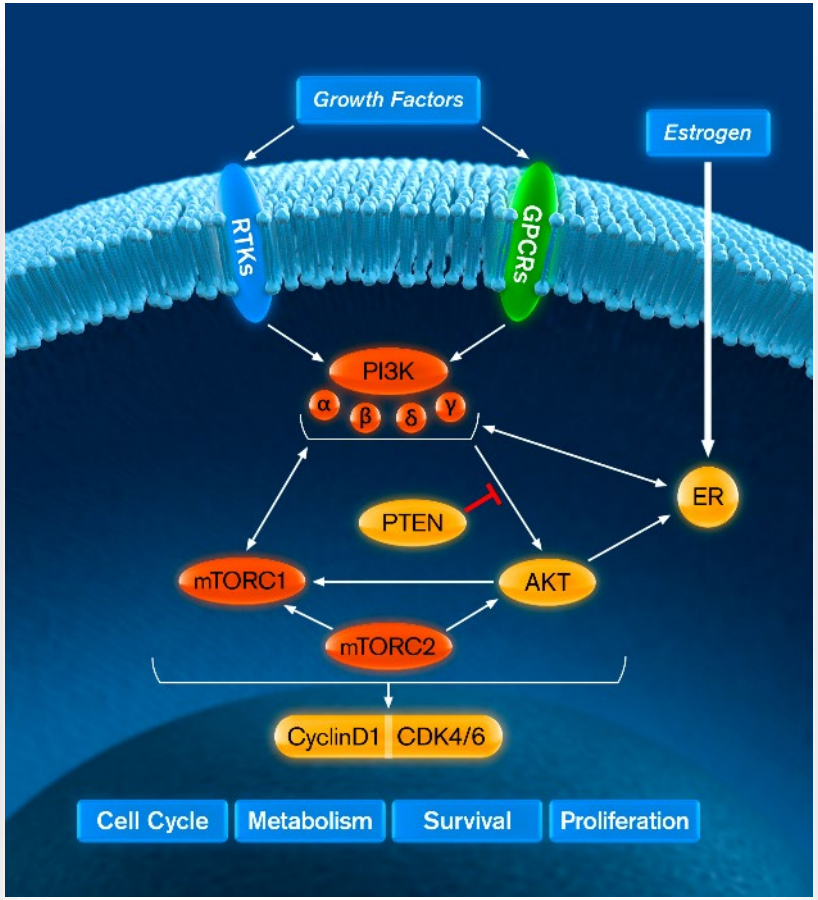
ER, CDK4/6, & PI3K/mTOR are Interdependent Drivers of HR+/HER2- ABC

Dysregulation of these pathways promotes excessive cell proliferation and resistance to apoptosis

ER and PI3K/mTOR

- Activation of the PAM pathway induces estrogen independent ER transcriptional activity ERα phosphorylation[1,2]
- Conversely, ER target gene expression activates upstream effectors of the PI3K/mTOR pathway[3]
- ER also activates the PI3K/mTOR pathway by direct binding to PI3K[4]
- **PI3K/mTOR inhibition can increase ER activity and sensitivity to endocrine therapy[5]**



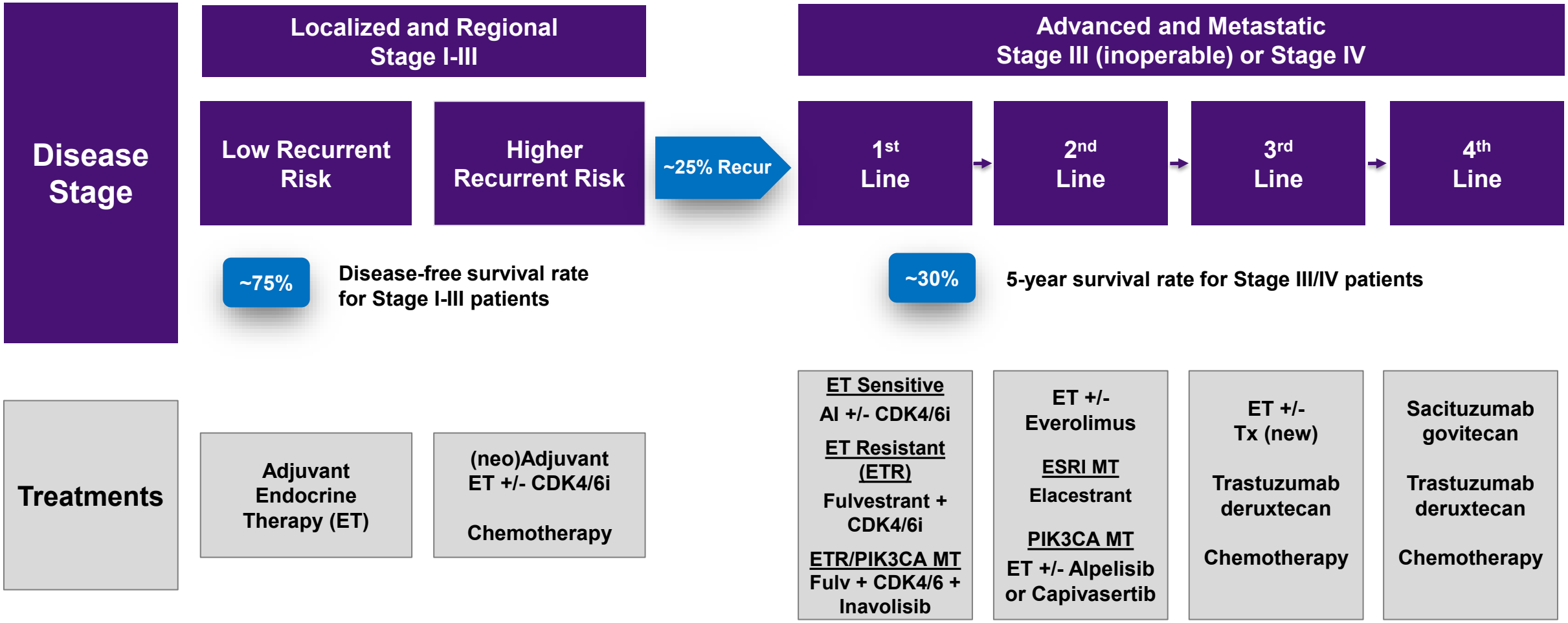
CDK4/6, ER and PI3K/mTOR[6-10]

- Estrogen promotes cyclin D1 transcription and cyclin D1 can cause estrogen independent transcription
- Provides rationale for simultaneously inhibiting ER and CDK4/6
- CDK4/6 inhibition causes incomplete cell cycle arrest – addition of PI3K/mTOR inhibition enables more complete arrest
- **PI3K/mTOR inhibition increases cyclin D1 activity which increases sensitivity to CDK4/6 inhibition**

Sources: (1) Campbell 2001, J Biol Chem 276(13):9817-24; (2) Yamnik 2009, J Biol Chem 6;284(10):6361-9) (3) Alves, Int J Mol. Sci. 2023; 24, 4522;(4) Simoncini 2000, Nature 407(6803):538–541; (5) Bosch 2015, Sci Transl Med. 7(283):283ra51; (6) Alves 2021, Nature Com, 12:5112; (7) Cai 2022, Sci China Life Sci 65; (8) O'Brien 2020, Breast Cancer Research, 22:89; (9) Karimi 2023, Cancer Communications, 43; (10) Jansen 2017, Cancer Res; 77(9).
Abbreviations: ER = estrogen receptor; ABC = advanced breast cancer

HR+/HER2- Breast Cancer Treatment Landscape[1]

~30,000 women in US and ~33,000 women in 5EU and Japan die from breast cancer annually[2]



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(1) NCCN Guidelines for Breast Cancer 2023; (2) American Cancer Society, Breast Cancer Statistics 2022; American Cancer Society Facts and Figures 2019-2020; Note: EU5 + Japan calculated using 112% EU + Japan scale up factor; Abbreviations: HR, hormone receptor; ET, endocrine therapy; AI, aromatase inhibitor; i, inhibitor; Tx, targeted therapy

Phase 3 VIKTORIA-1
2nd Line HR+, HER2- ABC

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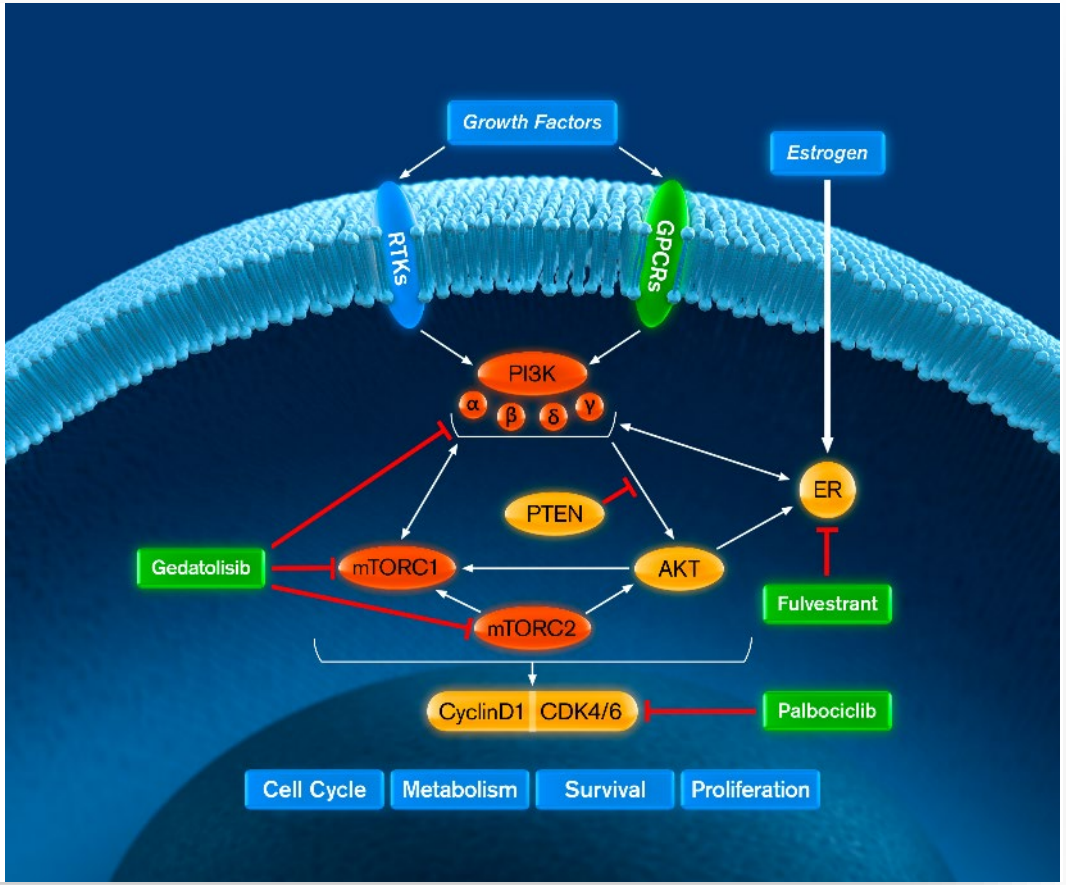
Clinical Strategy: Simultaneous Blockade of PAM, ER, & CDK4/6 Pathways

Clinical Hypothesis

- Blockade of interdependent ER, PI3K, mTOR & CDK signaling pathways is required to optimize anti-tumor control
- PAM inhibition:[1-4]
  - Blockades PAM pathway and limits cross-activation when ER or CDK4/6 is inhibited
  - Increases ER activity which increases sensitivity to endocrine therapy
  - Increases cyclin D1 activity which increases sensitivity to CDK4/6 inhibition



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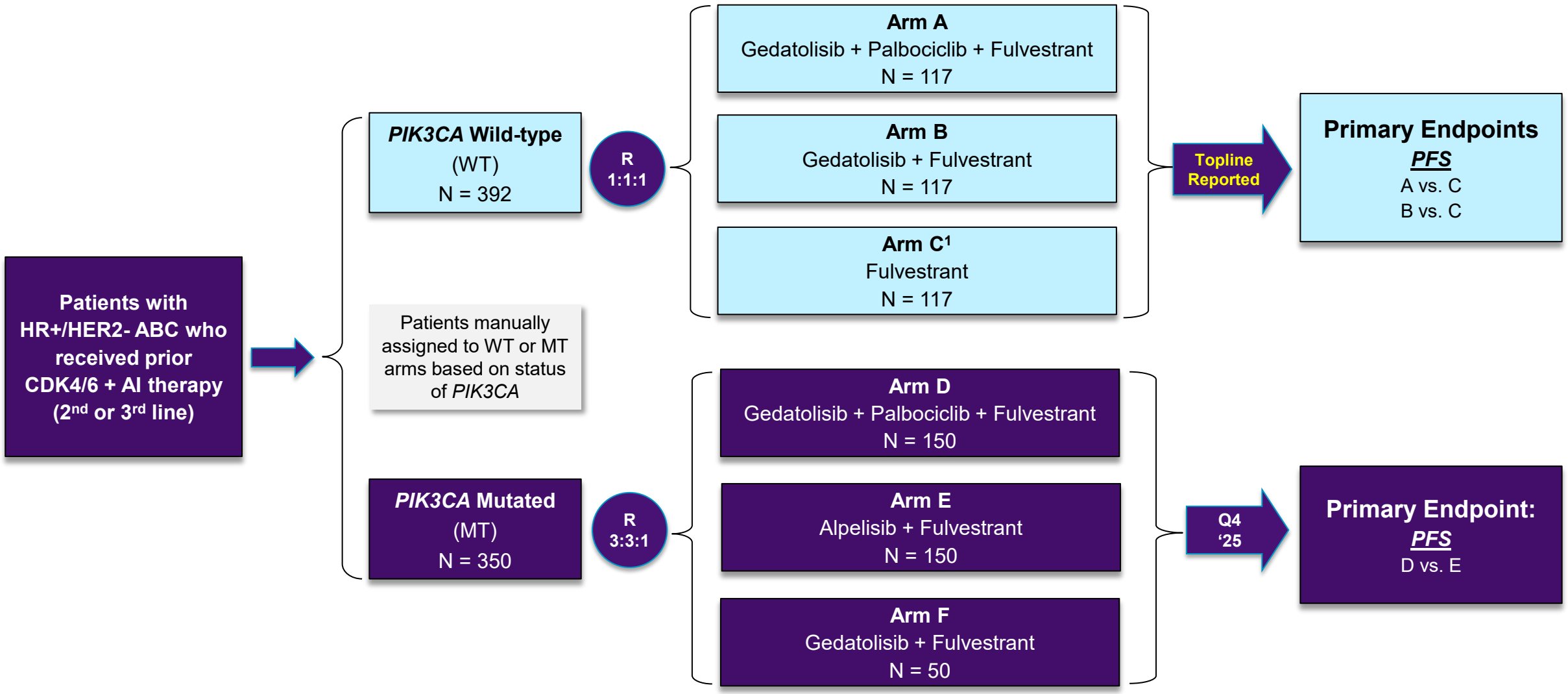
Sources: (1) Alves, Int J Mol. Sci. 2023; 24, 4522; (2) Cai 2022, Sci China Life Sci 65; (3) Karimi 2023, Cancer Communications, 43; (4) Jansen 2017, Cancer Res; 77(9); Abbreviations: ER = estrogen receptor; ABC = advanced breast cancer

VIKTORIA-1: Phase 3 Trial Design Overview for 2L HR+/HER2- ABC



1) Optional Cross-over to Arm A or Arm B upon progressive disease; WT = wild type; MT = mutant; PFS = progression free survival

VIKTORIA-1: Topline Efficacy Data

Both primary endpoints met; HR and incremental mPFS established new milestones in HR+/HER- ABC

Topline Efficacy

Gedatolisib Triplet

- mPFS was **9.3 months vs. 2.0** months for fulvestrant[1]
  - **7.3-month incremental improvement** in mPFS
- **HR = 0.24** (95% CI: 0.24-0.48[2], P<0.0001[3])
  - **4.2x higher** likelihood of survival w/o disease progression

Gedatolisib Doublet

- mPFS was **7.4 months vs. 2.0 months** for fulvestrant[1]
  - **5.4-month incremental improvement** in mPFS
- **HR = 0.33** (95% CI: 0.24-0.48[2], P<0.0001[3])
  - **3.0x higher** likelihood of survival w/o disease progression

Milestones Achieved

- Most favorable hazard ratio ever reported by any Phase 3 trial in HR+/HER2- ABC
- Highest incremental improvement in mPFS ever reported by any Phase 3 trial in 2nd line HR+/HER2- ABC
- First PI3K/AKT/mTOR (PAM) inhibitor to achieve positive Phase 3 data in PIK3CA WT patients post-CDK4/6 inhibitor

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Note: Full data will be released at a major medical conference later this year. At the request of conference organizers, the Kaplan-Meier curves have been removed from Celcuity presentations to comply with conference data embargo policies.

CI, confidence interval; HR, hazard ratio; m, Median; PFS, Progression-Free Survival. 1. Kaplan Meier method; 2. Stratified Cox regression model; 3. Stratified log-rank test

VIKTORIA-1: Additional Findings and Next Steps

Discontinuation rates and safety profile better than observed in Phase 1b study

Additional Findings

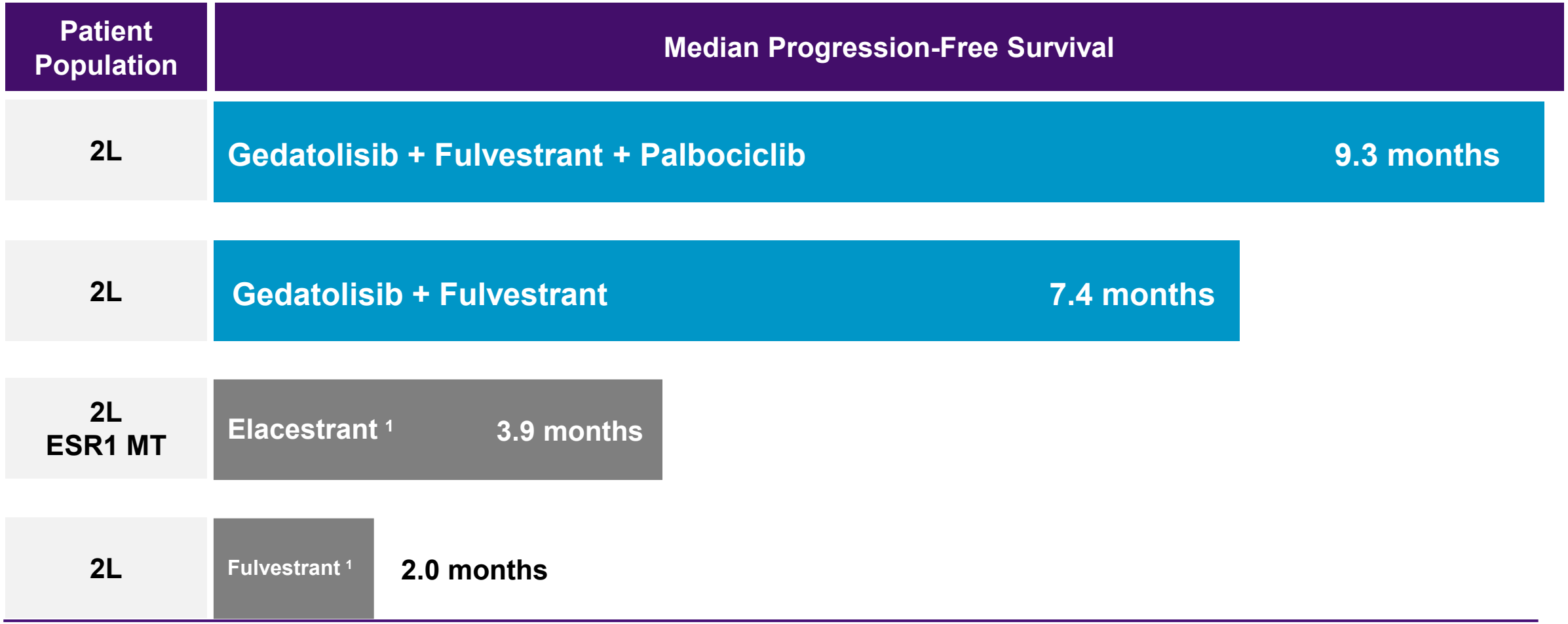
- The **treatment discontinuation rates** due to a TRAE for the gedatolisib triplet and doublet were **lower than observed in Arm D** of the Phase 1b trial in ABC patients
  - Additionally, they were lower than was observed in any Phase 3 trials for currently approved drug combinations in HR+/HER2- ABC.
- The **safety profile** of the gedatolisib triplet and gedatolisib doublet was **better than observed in the Phase 1b trial in ABC patients**, including lower rates of hyperglycemia and stomatitis
- **Favorable overall survival trend** for both the gedatolisib triplet and the gedatolisib doublet, although the data is immature

Next Steps

- Full results for VIKTORIA-1 *PIK3CA* Wild-Type cohort will be presented at an upcoming medical conference later in 2025
- Anticipate filing NDA submission in Q4 2025

How Does the Gedatolisib Regimen Potentially Fit in the 2L Landscape?

Gedatolisib and approved regimens with Phase 3 data for 2L HR+/HER2-/*PIK3CA* WT post-CDK4/6i



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(1) Bidard F, JCO 2022; (2) Campone M, NEJM 2025; (3) Jhaveri KL, NEJM 2024; (4) Neven P, ESMO Breast Poster FPN-306P , 2025 (5) Kalinsky K, ASCO Presentation, 2024. Note: Vepdegestrant and Imlunestrant are investigational therapies and do not have FDA approval. Abemaciclib + imlunestrant is an investigational therapeutic regime and does not have FDA approval. Abbreviations: ET – endocrine therapy; WT – wild-type; MT – mutant. To-date, no head-to-head comparisons of any other products to any of our product candidates in any clinical trial have been completed; results have been obtained from different trials with different designs, endpoints and patient populations; results may not be comparable

How Does the Gedatolisib Regimen Potentially Fit in the 2L Landscape?

Gedatolisib regimens showed highest incremental mPFS improvement versus endocrine therapy

Patient Population	Incremental mPFS improvement relative to endocrine therapy
2L	Gedatolisib + Fulvestrant + Palbociclib — Δ 7.3 months
2L	Gedatolisib + Fulvestrant — Δ 5.4 months
1L/2L	Imlunestrant + Abemaciclib [3] — Δ 3.9 months — *Not FDA approved*
2L ESR1 MT	Vepdegestrant [2] — Δ 2.9 months — *Not FDA approved*
2L ESRI MT	Elacestrant [1] — Δ 1.9 months
1L/2L ESR1 MT	Imlunestrant [3,4] — Δ 1.7 months — *Not FDA approved*
2L	Abema + Fulv[5] — Δ 0.7 months

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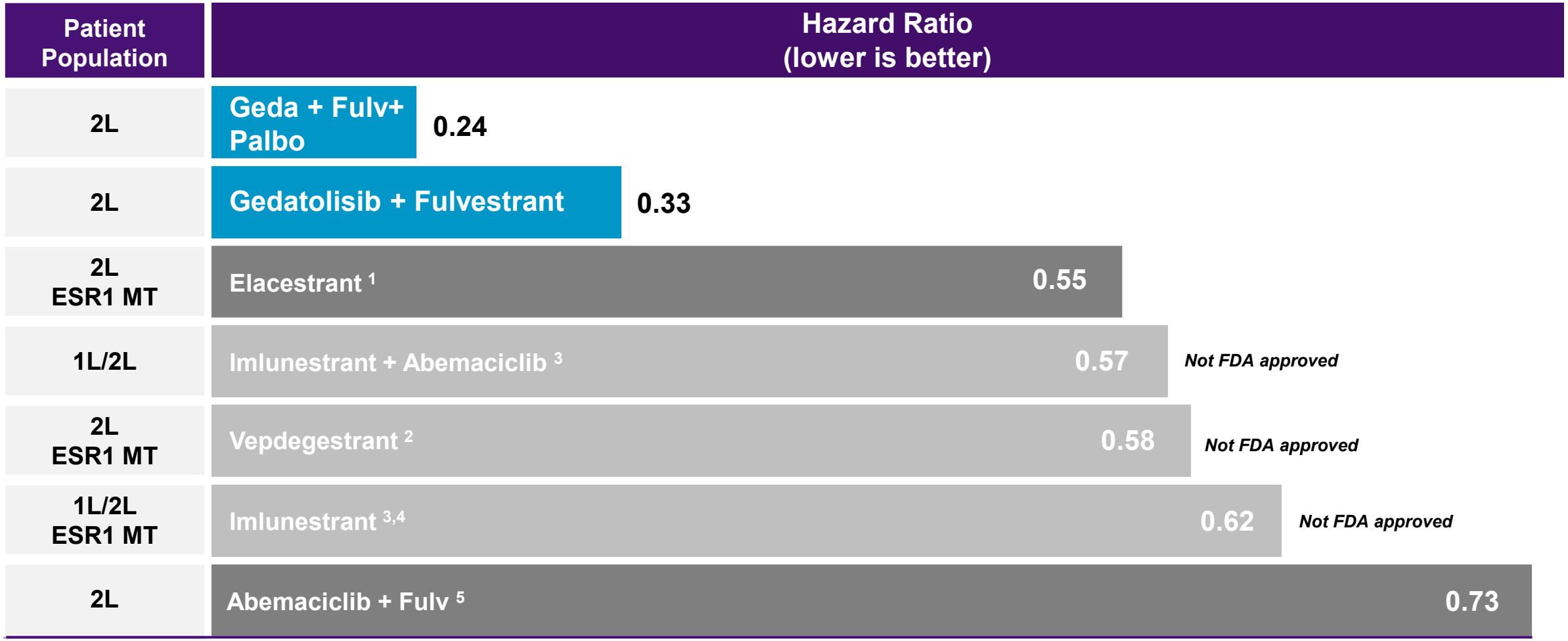
(1) Bidard F, JCO 2022; (2) Campone M, NEJM 2025; (3) Jhaveri KL, NEJM 2024; (4) Neven P, ESMO Breast Poster FPN-306P , 2025 (5) Kalinsky K, ASCO Presentation, 2024. Note: Vepdegestrant and Imlunestrant are investigational therapies and do not have FDA approval. Abemaciclib + imlunestrant is an investigational therapeutic regime and does not have FDA approval. Abbreviations: ET – endocrine therapy; WT – wild-type; MT – mutant. To-date, no head-to-head comparisons of any other products to any of our product candidates in any clinical trial have been completed; results have been obtained from different trials with different designs, endpoints and patient populations; results may not be comparable

How Does the Gedatolisib Regimen Potentially Fit in the 2L Landscape?

Hazard ratios for gedatolisib regimens are unprecedented



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EXPANDING TREATMENT OPTIONS

(1) Bidard F, JCO 2022; (2) Campone M, NEJM 2025; (3) Jhaveri KL, NEJM 2024; (4) Neven P, ESMO Breast Poster FPN-306P, 2025 (5) Kalinsky K, ASCO Presentation, 2024. Note: Vepdegestrant and Imlunestrant are investigational therapies and do not have FDA approval. Abemaciclib + imlunestrant is an investigational therapeutic regime and does not have FDA approval. Abbreviations: ET – endocrine therapy; WT – wild-type; MT – mutant. To-date, no head-to-head comparisons of any other products to any of our product candidates in any clinical trial have been completed; results have been obtained from different trials with different designs, endpoints and patient populations; results may not be comparable

Phase 1B Data: Geda-Triplet & SOC for 2L HR+/HER2-/PIK3CA MT ABC Post-CDKi

Data from PIK3CA MT Cohort of VIKTORIA-1 expected in late 2025

Patient Population	2nd Line ER+/HER2-/*PIK3CA* Mutant ABC	
2L *PIK3CA* WT/MT	Gedatolisib + Fulvestrant + Palbociclib[1]	mPFS 12.9 months ORR 63%
2L *PIK3CA* MT	Alpelisib + Fulvestrant [2]	mPFS 8.0 months ORR 19%
2L *PIK3CA* MT	Alpelisib + Fulvestrant [3]	mPFS 5.6 months ORR 24%
2L *PIK3CA*, AKT, PTEN MT	Capivasertib + Fulvestrant [4]	mPFS 5.5 months ORR 23%

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(1) Layman 2024, Arm D; (2) Rugo, Lancet Onco, 2024; (3) Rugo, SABCS, 2021;(4) Oliveira, ESMO Breast, 2023, CDK4/6 prior treated patients (5) Bidard, JCO, 2022 and FDA Note: All drugs listed are FDA approved. No head-to-head trials have been conducted; data collected from different trials, in different patient populations and may not be comparable.

Phase 3 VIKTORIA-2
1st Line HR+, HER2- ABC

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VIKTORIA-2: Phase 3 Study Features for 1L HR+/HER2- ABC

Global open-label randomized study (~200 sites)

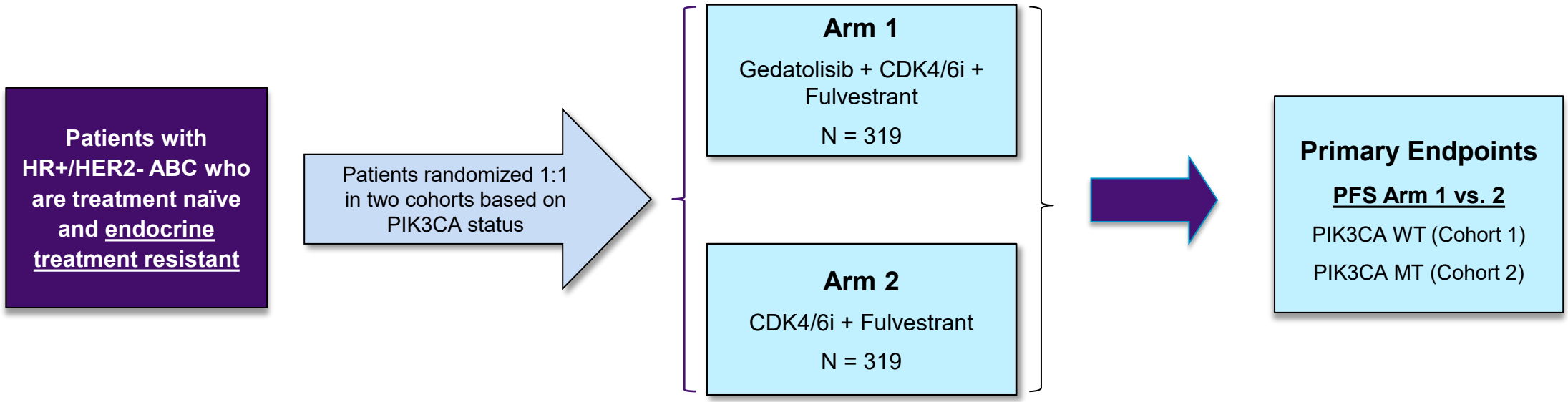
- **Key eligibility criteria:**
  - ER+/HER2- advanced or metastatic breast cancer
  - No prior treatment for advanced or metastatic breast cancer
  - Progression or relapse of disease during or within 12 months of completing adjuvant endocrine treatment
  - Pre-diabetic or patients with controlled diabetes allowed
- Investigator's choice of CDK4/6 inhibitor (ribociclib or palbociclib) for investigational and control arm
- Randomizing patients to cohorts based on PIK3CA status (MT or WT); primary analysis for each cohort is independent
- Stratification by primary vs secondary endocrine treatment resistance, site of metastases (bone-only vs other), geographical area (US vs other)

Key Considerations

- 1L endocrine treatment resistant patients receive limited benefit from CDK4/6 + fulvestrant
  - mPFS = 7.3M in recent study
- Supports potential indication allowing use of either ribociclib or palbociclib
- Minimizes exclusion of patients based on fasting glucose or HbA_{1c} levels
- Independent primary analyses of PIK3CA WT and MT provides two potential opportunities to obtain approval

VIKTORIA-2: Phase 3 Trial Design Overview for 1L HR+/HER2- ABC

Will conduct small safety run-in with gedatolisib plus ribociclib plus fulvestrant prior to Phase 3



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Phase 1B: Gedatolisib + Palbociclib + Letrozole in 1L HR+/HER2- ABC (N=41)[1]

mPFS of 48.6 months, mDOR of 46.9 months, and ORR of 79%

Treatment-Naïve Patients who are Endocrine Treatment Sensitive (ETS) (N=41)			
	Escalation Arm A	**Expansion Arm A**	**Total Treatment Naïve**
Progression-Free Survival (full analysis set)	n = 11	n = 30	n = 41
Median PFS, mos (95% CI)	**45.8 (32.3, NR)**	**48.6 (11.6, NR)**	**48.6 (30.4, NR)**
Responses (evaluable, measurable disease) [1], n (%)	n = 7	n = 26	n = 33
CR	0	1 (3.8)	1 (3.0)
PR	4 (57.1)	21 (80.8)	25 (75.8)
SD	3 (42.9)	3 (11.5)	6 (18.2)
Unconfirmed PR	0	0	0
Durable SD (≥24 weeks)	1 (14.3)	2 (7.7)	3 (9.1)
PD	0	1 (3.8)	1 (3.0)
ORR [1]	**4 (57.1)**	**22 (84.6)**	**26 (78.8)**
Median DOR, mos (95% CI) [2]	**39.7 (30.5, NR)**	**46.9 (11.3, NR)**	**46.9 (24.6, 49.5)**

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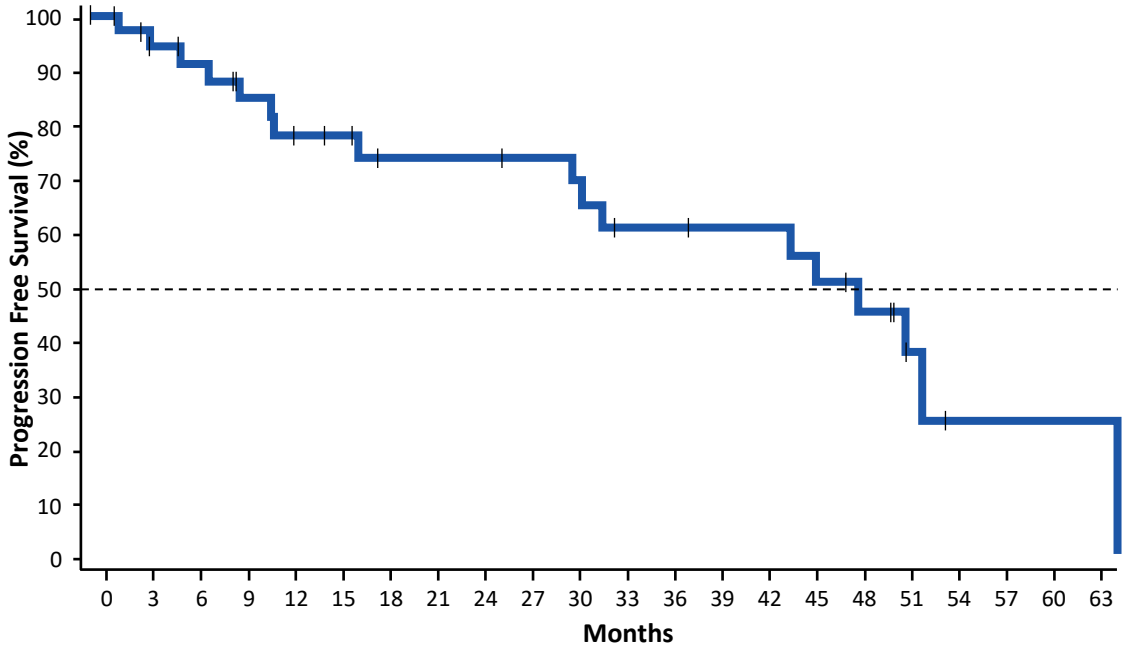
Source: Rugo 2023 ESMO Breast. (1) Subjects with measurable disease in response evaluable analysis set per RECIST v1.1;(2) Confirmed responders in the full analysis set. Abbreviations: CR, complete response; DOR, duration of response; mos, months; NR, Not Reached; ORR, objective response rate; PD, progressive disease; PFS, progression free survival; PR, partial response; SD, stable disease

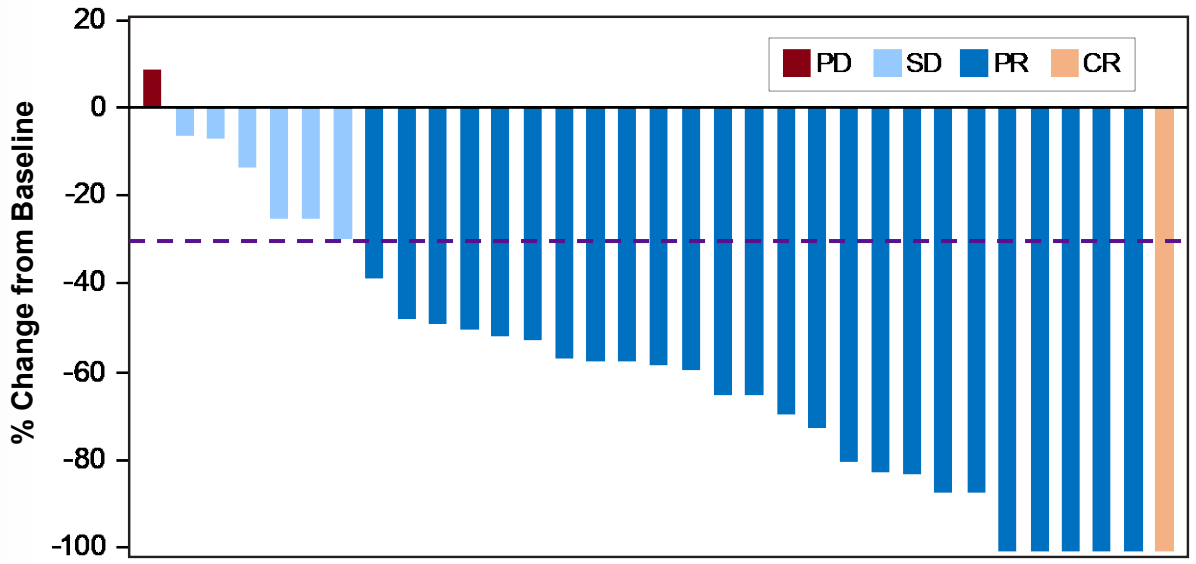
Phase 1B: Gedatolisib + Palbociclib + Letrozole in 1L HR+/HER2- ABC (N=41)1

mPFS and ORR for treatment-naïve ETS patients compares favorably to published data for SOC palbociclib + letrozole[2]

Median Progression Free Survival
48.6 Months



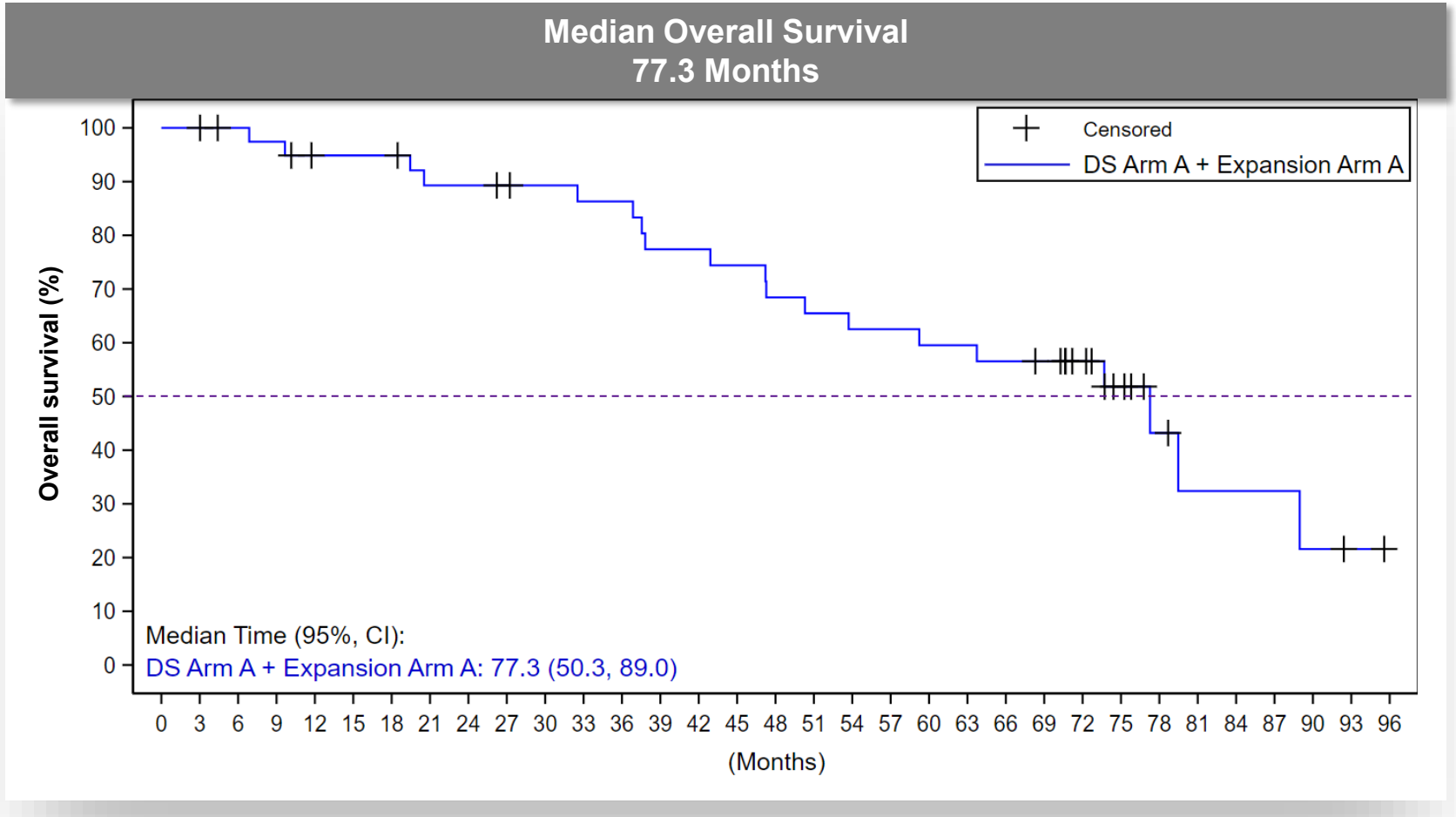
Tumor Size Change
ORR = 79% (26/33)



(1) Rugo 2023 ESMO-Breast; Escalation Arm A & Expansion Arm A data from B2151009 study; (2) Finn 2016 NEJM – PALOMA-2; (3). Note: (a) ORR reported is for patients with measurable disease of a target lesion. (b) No head-to-head trials have been conducted; data collected from different trials, in different patient populations and may not be comparable.

Phase 1B: Gedatolisib + Palbociclib + Letrozole in 1L HR+/HER2- ABC (N=41)1

mOS data for treatment-naïve patients ETS compares favorably to published data for current SOC



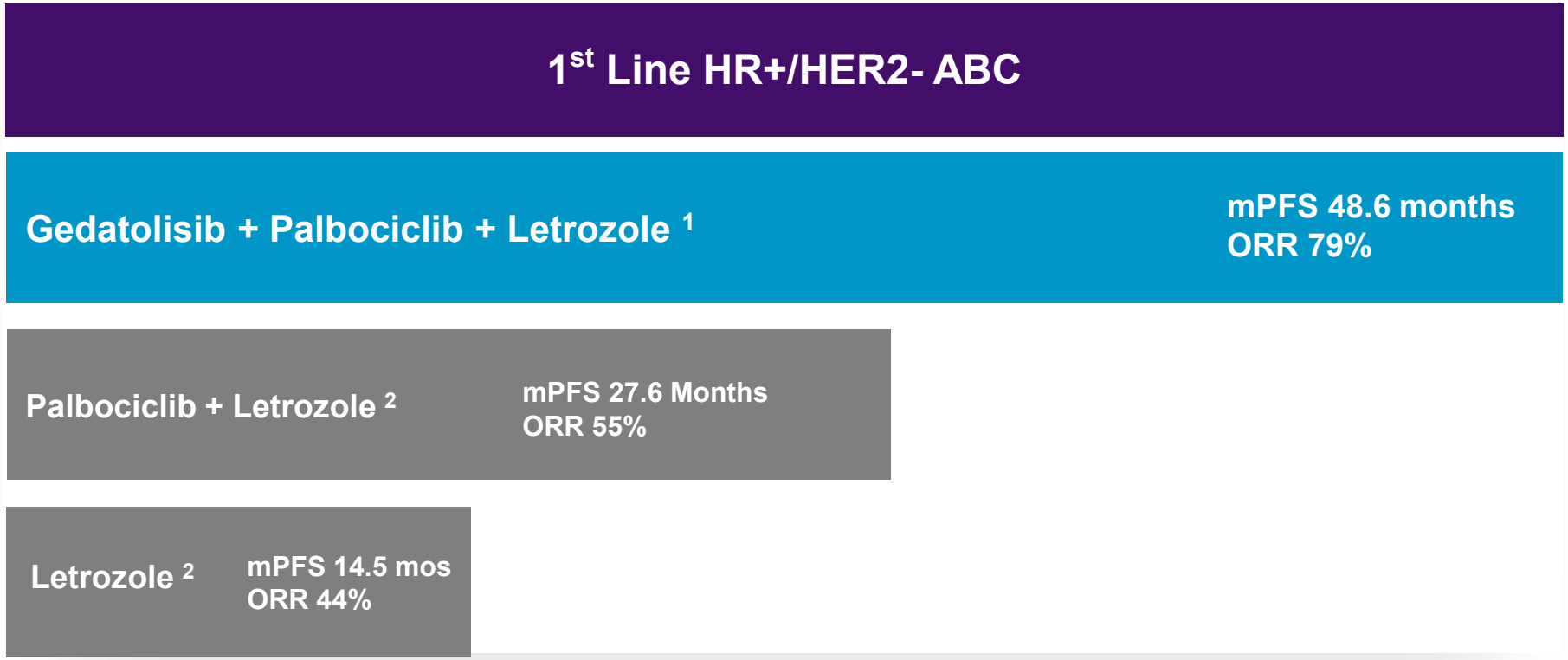
Relevant OS data in 1L setting

- **Palbociclib + letrozole: 53.8 months[2]**
  - **PALOMA-2 study**

1) Rugo H. et al., SABCS, 2024; Escalation Arm A & Expansion Arm A data from B2151009 study; 2) Slamon D. et al., J Clin Oncol 2024

Gedatolisib Combo vs. SOC for 1L HR+ / HER2- ABC (Endocrine Sensitive)

Gedatolisib Combo Offers Potential for Superior mPFS Compared to 1L SOC



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Sources: (1) Rugo 2023 ESMO-Breast. (2) Rugo H, et al. Breast Cancer Res Treat, 2019; Finn 2016. Abbreviations: mPFS = median progression free survival; ORR = objective response rate. SOC = standard of care. Note: No head-to-head trials have been conducted; data collected from different trials, in different patient populations and may not be comparable.

Relevant Comparisons to VIKTORIA-2 Control

B2151009 study results for 1L patients compares favorably to published data for 1L ETS patients

	Gedatolisib + Palbociclib + Letrozole N=41 [1]	Palbociclib + Letrozole N=441 [2]	Palbociclib + Fulvestrant N=164 [3]
***PIK3CA* Status**	WT / MT (76% / 22%)	NR	MT (100%)
Endocrine Therapy Sensitivity	Sensitive (ETS)	Sensitive (ETS)	Resistant (ETR)
mPFS (months)	**48.6**	**27.6**	**7.3**
ORR	**79%**	**55%**	**25%**

Sources: (1) Rugo, ESMO-Breast, 2023; (2) Rugo, Palbociclib plus letrozole as 1st Line therapy in ER+/HER2- ABC – PALOMA-2; (3) Jhaveri, SABCS 2023.
Note: No head-to-head trials have been conducted; data collected from different trials, in different patient populations and may not be comparable.

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B2151009 Arm D: Safety Summary for Phase 3 Dosing

G + P + F was well tolerated overall; < 4% discontinuation rate

- **Discontinuation of gedatolisib due to AE - <4%**
  - Alpelisib – 26% discontinued [1]
  - Everolimus – 24% discontinued [2]
  - Capivasertib – 10% discontinued [3]
- Most TRAE's were Grade 1 or 2
- **Few hyperglycemia adverse events**
  - Gedatolisib - 7% Grade 3/4
  - Alpelisib - 37% Grade 3/4 [1]
- Stomatitis prophylaxis was not utilized in this study
  - **Swish-and-Spit dexamethasone prophylactic mouth rinse reduced Grade 2-4 stomatitis by 90% [4]**
  - Phase 3 study prescribes prophylaxis
- Neutropenia, leukopenia, and anemia AE incidence is nearly identical to PALOMA-3 (palbociclib + fulvestrant)

Arm D (n=27)
Gedatolisib + Palbociclib + Fulvestrant
(180 mg IV, 3 weeks on, one week off)

Related TEAE's > 30%			
	Grade 1	Grade 2	Grade 3/4
Adverse Event	%	%	%
Stomatitis[5]	11	56	22
Neutropenia[6]	-	15	67
Nausea	44	30	-
Fatigue	22	37	7
Dysgeusia	44	7	-
Diarrhea	37	-	4
Rash	19	15	7
Leukopenia[7]	-	19	23
Constipation	30	4	4
Vomiting	22	11	4
Anemia[8]	4	15	15
Hyperglycemia	15	4	7

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Source: (1) USPI Alpelisib; (2) USPI Everolimus; (3) USPI Capivasertib; (4) Rugo 2017; (5) Stomatitis category includes mucositis; (6) Neutropenia includes neutrophil count decrease; (7) Leukopenia includes white blood cell decrease; (8) Anemia includes hemoglobin decrease; Abbreviations: G = gedatolisib; P = palbociclib; F = fulvestrant; TEAE = treatment emergent adverse events; AE = adverse event

Gedatolisib for Prostate Cancer

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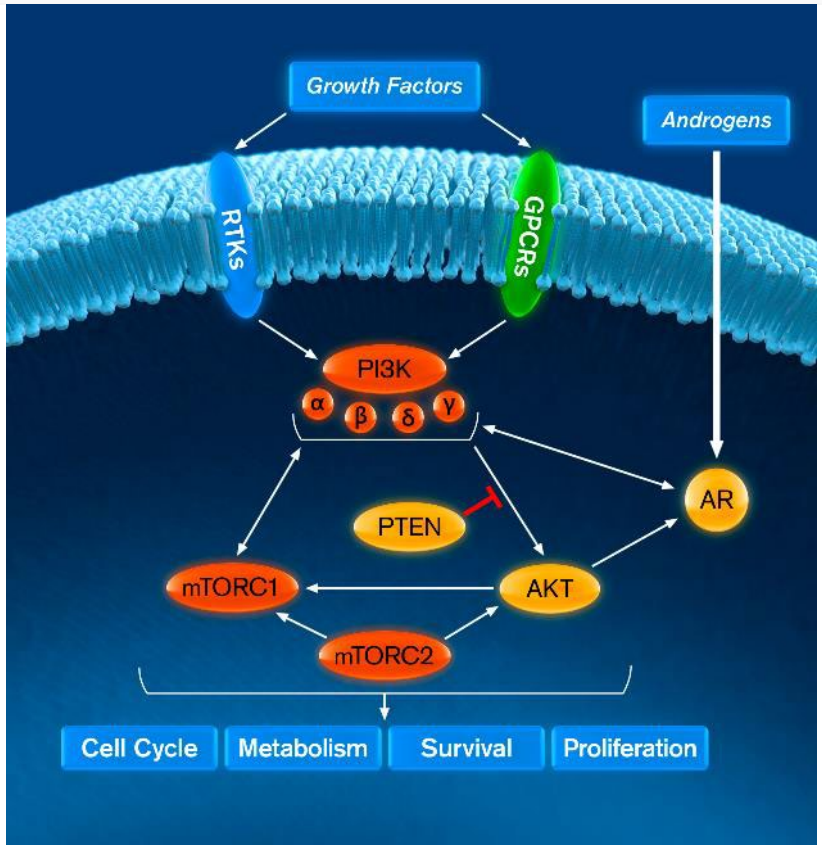
Androgen Signaling is the Key Driver of Prostate Cancer

The PI3K/AKT/mTOR (PAM) pathway helps promote excessive cell proliferation and resistance to apoptosis

The AR Pathway is the Primary Therapeutic Target

- The androgen receptor (AR) drives the expression of target genes which promote cancer cell survival and growth
- The androgen signaling pathway is the primary therapeutic target for prostate cancer at all stages of disease
- Androgen deprivation therapies (ADT) are used primarily for localized disease
- Second generation AR inhibitors are used for advanced disease



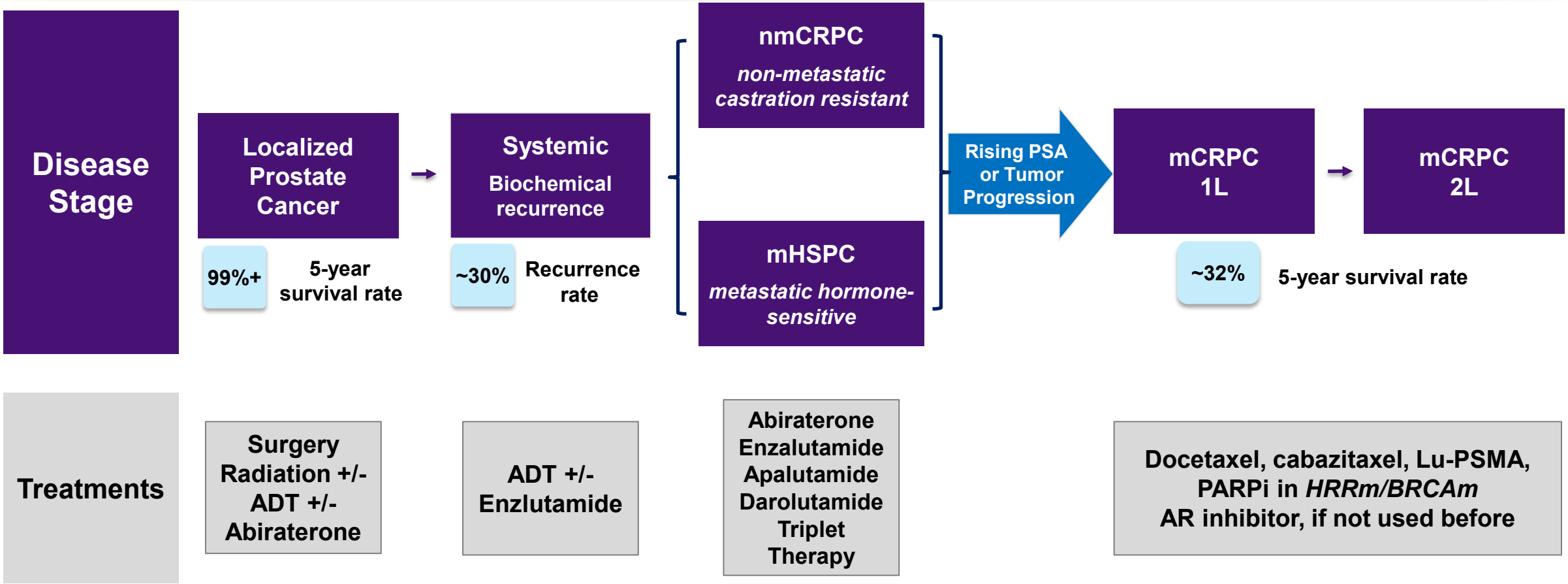
The PAM Pathway Plays a Key Role in mCRPC

- AR and PI3K-AKT-mTOR pathways cross-regulate each other.
- 70% - 100% of mCRPC tumors have PI3K/AKT/mTOR related pathway alterations.
- Mutations dispersed across PTEN, PI3K, AKT, and mTOR sub-units

Source: Alves, Int J Mol. Sci. 2023

Prostate Cancer Disease and Treatment Landscape[1,2]

34,700 men in US and 62,400 men in 5EU and Japan die from prostate cancer annually[3,4]



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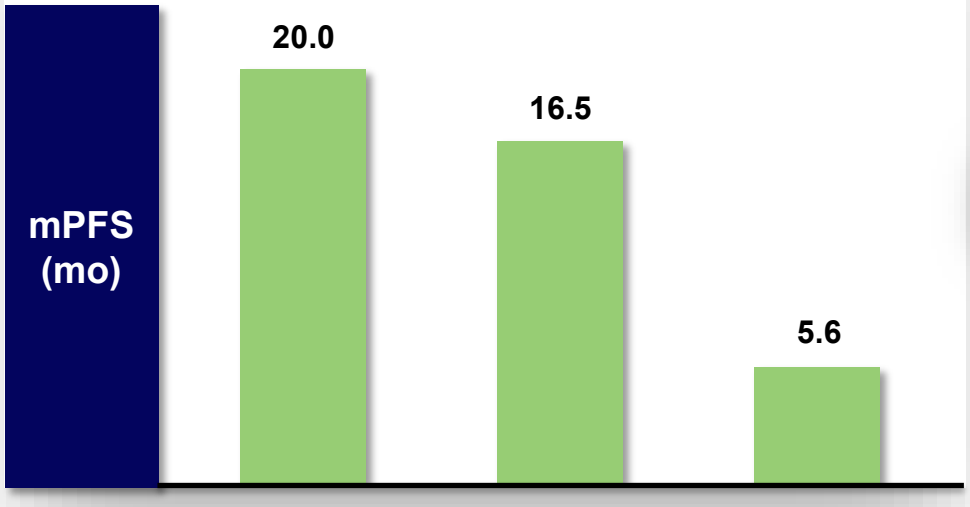
(1) Saad, Prostate Cancer Prostatic Dis. 2021; (2) Scher, Plos One 2015; Leith, A. et al. 2022; George, D. J. et al. 2022; NCCN Guidelines for Prostate Cancer Version 1.2023; (3) American Cancer Society, Cancer Facts & Figures 2023; (4) Wang, Front. Public Health, 2022; Abbreviations: mCRPC = metastatic castration resistant prostate cancer; HRR = homologous recombination repair 1L = first line of therapy; 2L = second line of therapy; ADT = androgen deprivation therapy; AR = androgen receptor

Limited Benefit for 2L HRR- mCRPC Patients After Treatment with AR Inhibitor

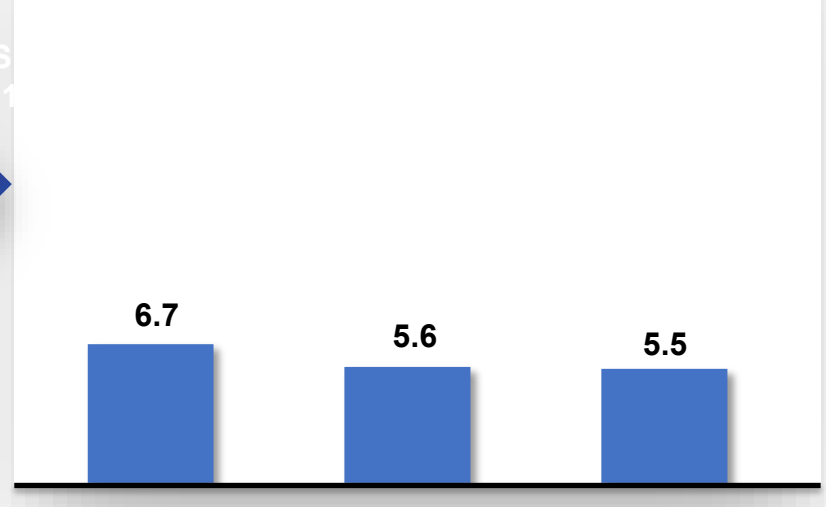
Significant need for better therapeutic options

1st Line Treatment Outcomes



Drugs	Xtandi [1]	Zytiga [2]	Docetaxel [3]
MOA	ARi	ARi	Chemotherapy
Pat Pop	All	All	All
mPFS	20.0	16.5	5.6
OS	35.3	34.7	19.5

2nd Line Treatment Outcomes *(post AR inhibitor treatment)*



Docetaxel [4]	Zytiga [5]	Xtandi [6]
Chemotherapy	ARi	ARi
Prior ARi	Prior Xtandi	Prior Zytiga
6.7	5.6	5.5
20.0	-	-

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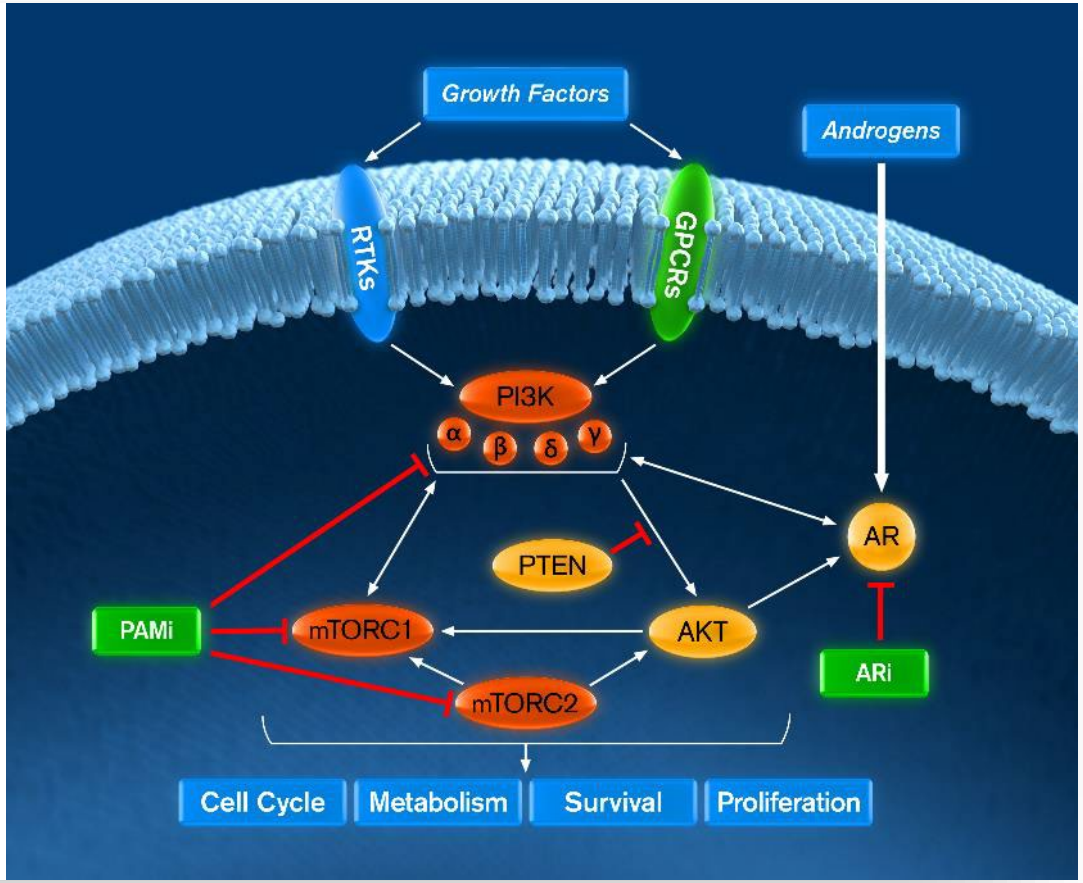
(1) Beer Eur Urol. 2017; (2) Ryan NEJM 2013; Ryan Lancet Oncol 2015 (3) Kellokumpa-Lehtinen Lancet Oncol. 2013, time-to-treatment failure reported; (4) Crabb J Clin Oncol 2021; (5) Attard J Clin Oncol 2018; (6) Sweeny Clin Cancer Res 2022. Abbreviations: HRR = homologous recombination repair; AR = androgen receptor

Combining a PAM Inhibitor with an AR Inhibitor has Strong Scientific Rationale

Biological parallels between mCRPC and HR+ ABC – PAM and hormonal pathway drive progression [1]

PI3K/mTOR + AR Inhibition
Treatment Rationale

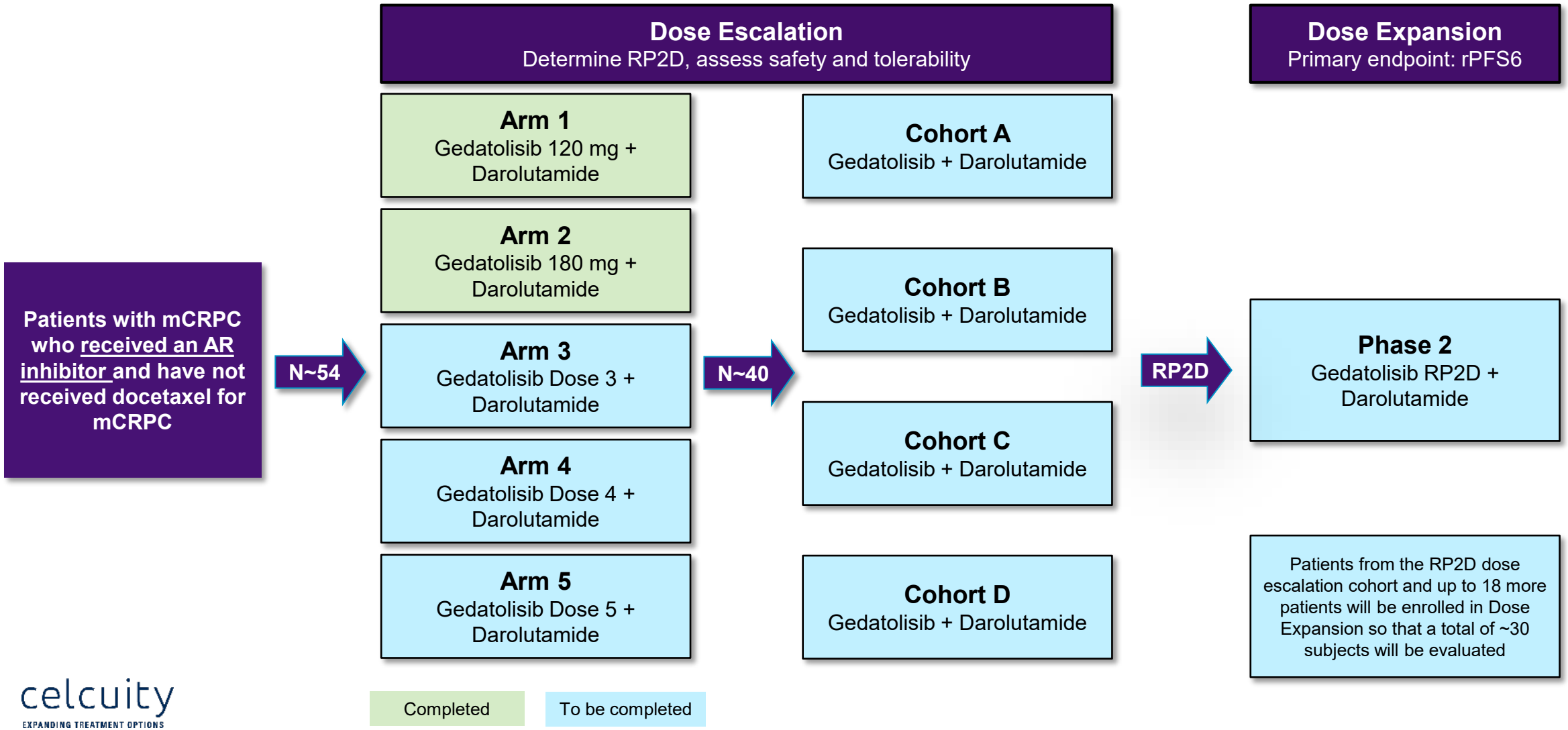
- Favorable clinical data in mCRPC with PAM inhibitors provides "proof-of-concept" of benefit of combining a PAM and AR inhibitor in 2L setting
- Gedatolisib's clinical results in breast cancer correlated with strong activity in nonclinical tumor models
- Gedatolisib exhibits similar potency and efficacy in prostate cancer cell lines as those reported in breast cancer cell lines
- Xenograft data in PR models is consistent with in vivo data – gedatolisib exhibits anti-tumor effects independent of PTEN or AR status



(1) Carver et al, Cancer Cell 2011; (2) Mulholland et al, Cancer Cell 2011; (3) Crumbaker et al, Cancers 2017

CELC-G-201: Phase 1b/2 Trial Design Overview

Evaluating gedatolisib plus darolutamide to determine preliminary safety and efficacyRP2D



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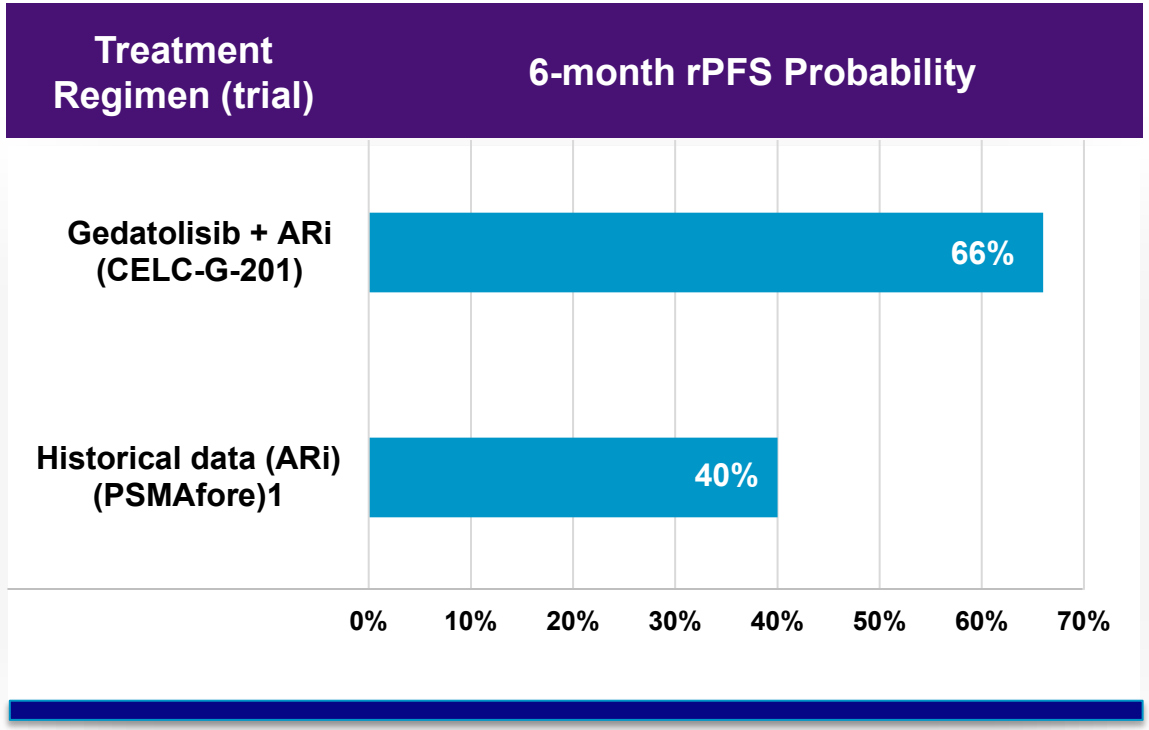
CELC-G-201: Preliminary Topline Data for Gedatolisib + Darolutamide

rPFS6 for G + ARi (darolutamide) compares favorably to historical data for ARi monotherapy



	CELC-G-201 Arms 1 & 2 (N=38)
rPFS6	66%
Discontinuation Rate due to AE	0%
Grade 3 hyperglycemia	0%
Grade 2 stomatitis	7.9%
Grade 3 stomatitis	2.6%

(1) Morris NEJM 2024; Abbreviations: rPFS6 – six-month radiographic progression free survival probability

Additional Early Phase Clinical Data

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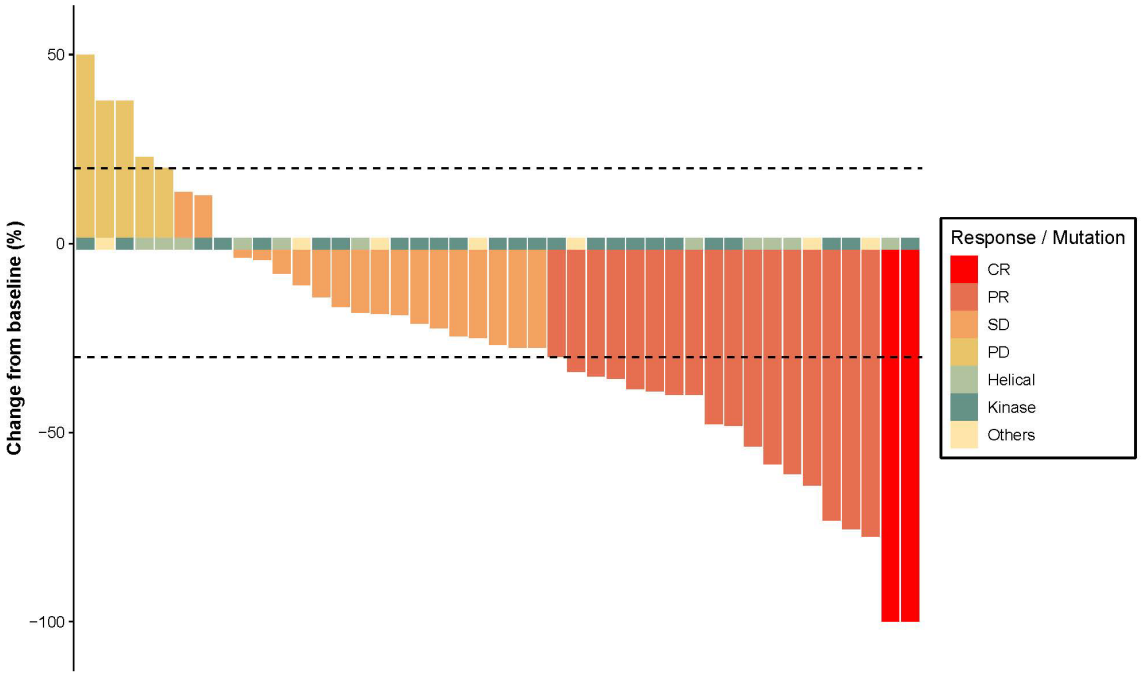
Gedatolisib + Trastuzumab Biosimilar in 3L+ HER2+ ABC Patients (N=44)

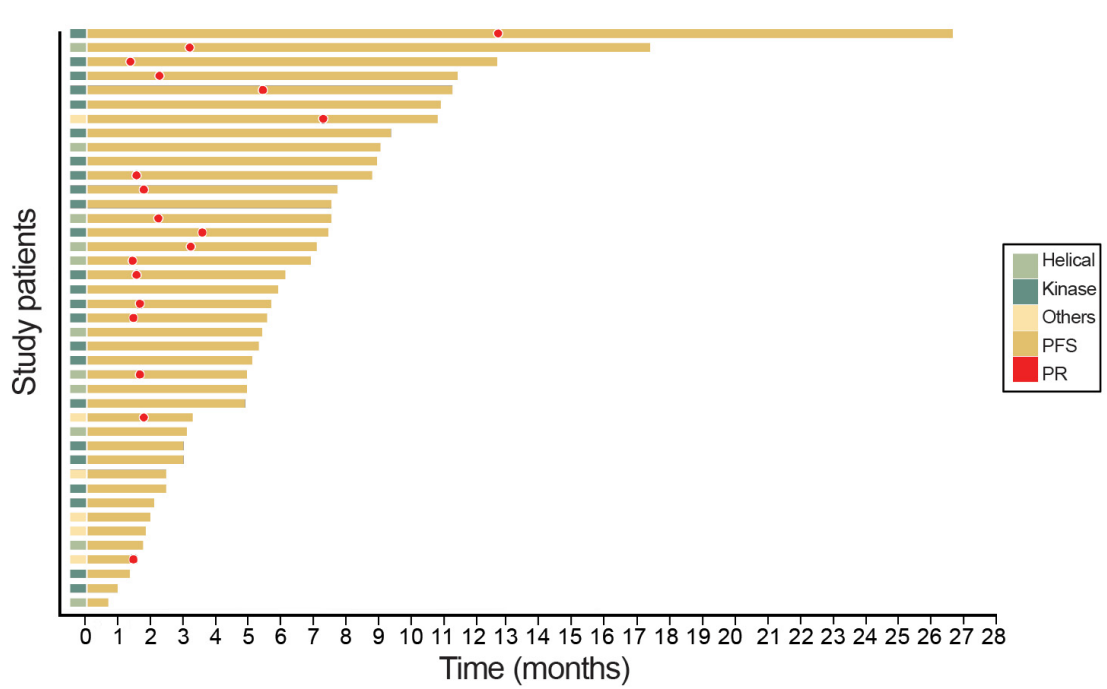
43% objective response rate

Best Response



- 2 of 44 best response was a complete response (CR)
- 17 of 44 best response was a partial response (PR)

Duration of Response



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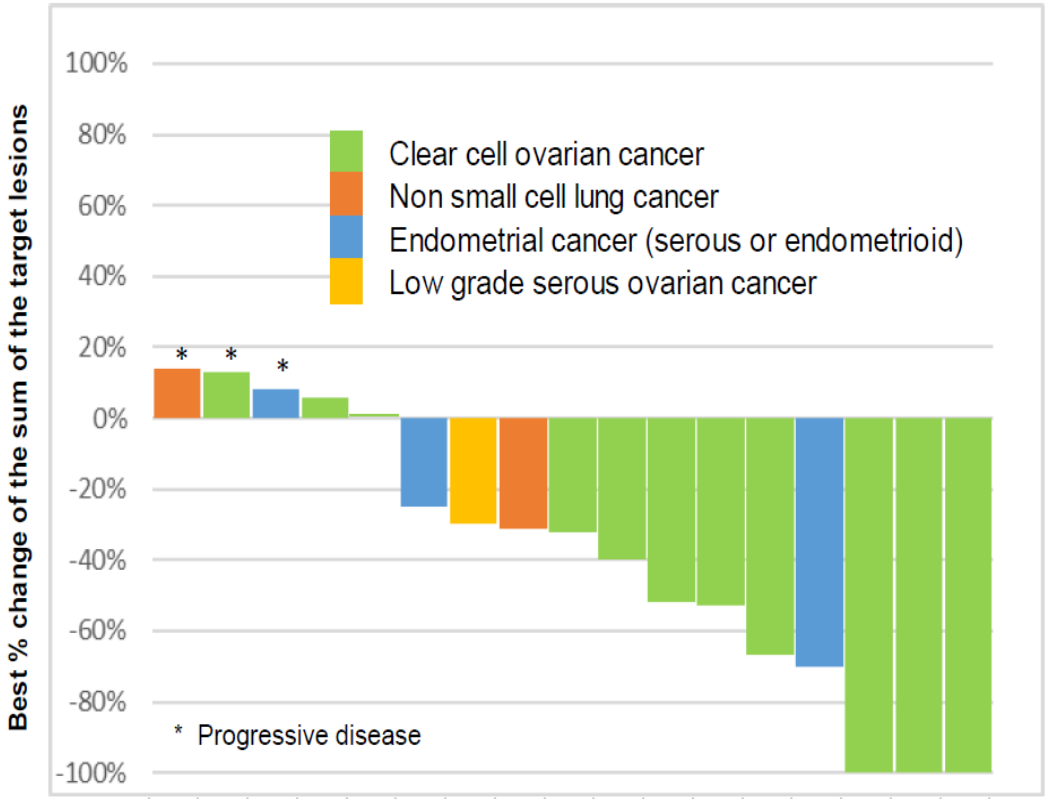
Source: Kim ASCO 2025. Note: Data presented is as of a cutoff date of February 10, 2025, representing a database snapshot, and may change based on ongoing routine data monitoring and enrollment.

Gedatolisib + Paclitaxel + Carboplatin in Patients with Solid Tumors (N=17)[1]

65% ORR in all patients, 82% ORR in patients with ovarian cancer



- Ovarian Cancer (N=11)
  - ORR: 82%
    - Clear cell ovarian cancer (CCOC) (N = 10)
      - ORR: 80% - 5/10 PR, 3/10 CR
    - Low grade serous ovarian (N=1)
      - 1/1 PR
- Other solid tumors (N= 6)
  - ORR = 33%
- Median PFS = 6.35 months (95% CI 4.6-11.11)
- Median duration of response = 7.6 months (95% CI 1.9-13.4)

- The CCOC data compares very favorably to ORR for platinum therapy reported in platinum-naïve CCOC patients - 25%-50%
- CCCO accounts for ~15% ovarian cancers in Asia
- Will assess likelihood other ovarian sub-types may benefit from gedatolisib + platinum therapy

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(1) Columbo 2021 CCR

Leading cancer KOLs are participating in our research

Clinical Advisory Board

Mark Pegram M.D. Ph.D.

Stanford University

Sara Hurvitz M.D.

University of Washington

Ben Ho Park M.D., Ph.D.

Vanderbilt University

Adam Brufsky M.D., Ph.D.

UPMC Life Changing Medicine

Lee Schwartzberg M.D.

The West Clinic Advancing Cancer Care. Together.

Hung Khong M.D.

Moffitt Cancer Center

Bora Lim M.D.

Baylor College of Medicine

Mothaffar Rimawi M.D.

Baylor College of Medicine

Alberto Montero M.D.

University Hospitals
The Science of Health. The Art of Compassion.

Scientific Advisory Board

Carol Lange Ph.D.

University of Minnesota

Manfred Auer Ph.D.

The University of Edinburgh

John Katzenellenbogen Ph.D.

University of Illinois Urbana Champaign

Ron McGlennen M.D.

access genetics

Benita Katzenellenbogen Ph.D.

University of Illinois Urbana Champaign

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Leadership Team: Track Record of Developing Approved Therapies and Building Companies

Brian Sullivan

Chief Executive Officer
Co-Founder

Lance Laing, PhD

Chief Scientific Officer
Co-Founder

Vicky Hahne

Chief Financial Office

Igor Gorbatchevsky, MD

Chief Medical Officer

Eldon Mayer

Chief Commercial Officer

Brent Eilefson

General Counsel

Bernhard Lampert, PhD

VP, Pharmaceutical Development

David Bridge

VP, Quality Assurance and Process Development

Fred Kerwood

VP, Program Management

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Upcoming Milestones

- Present full data for VIKTORIA-1 *PIK3CA* wild-type cohort at a major medical conference later this year
- Submit New Drug Application for VIKTORIA-1 *PIK3CA* wild-type cohort indication in Q4 2025
- Report topline data for VIKTORIA-1 *PIK3CA* mutation cohort by end of 2025

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The Celcuity Opportunity

Effectively treating PAM pathway driven tumors is one the largest opportunities in oncology

1. • **Gedatolisib's differentiated MOA and PK profile result in a highly potent, cytotoxic, and well tolerated PAM inhibitor**

2. • **Phase 3 VIKTORIA-1 WT cohort efficacy results showed unprecedented reduction in risk of disease progression or death and incremental improvement in progression free survival in patients with HR+/HER2- ABC**

3. • **A Phase 3 study in 1L patients with HR+/HER2- ABC is enrolling**
   • **A Phase 1b/2 trial in 2L patients with mCRPC has reported promising early data and is enrolling additional cohorts**

4. • **Uniquely positioned to advance multiple potential blockbuster indications in breast and prostate cancer**
   • Pro forma cash, cash equivalents, short-term investments of $455M as of Q2 2025 expected to fund operations through 2027[1]

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(1) Includes $287M of net debt and equity capital raised 7/30/25

Celcuity is focused on unlocking the potential of treating cancers that involve the PI3K/AKT/mTOR pathway

Our third-generation cellular analysis platform unravels complex oncogenic activity molecular tests can’t detect.

We harvest these insights to develop new targeted therapies for cancer patients

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