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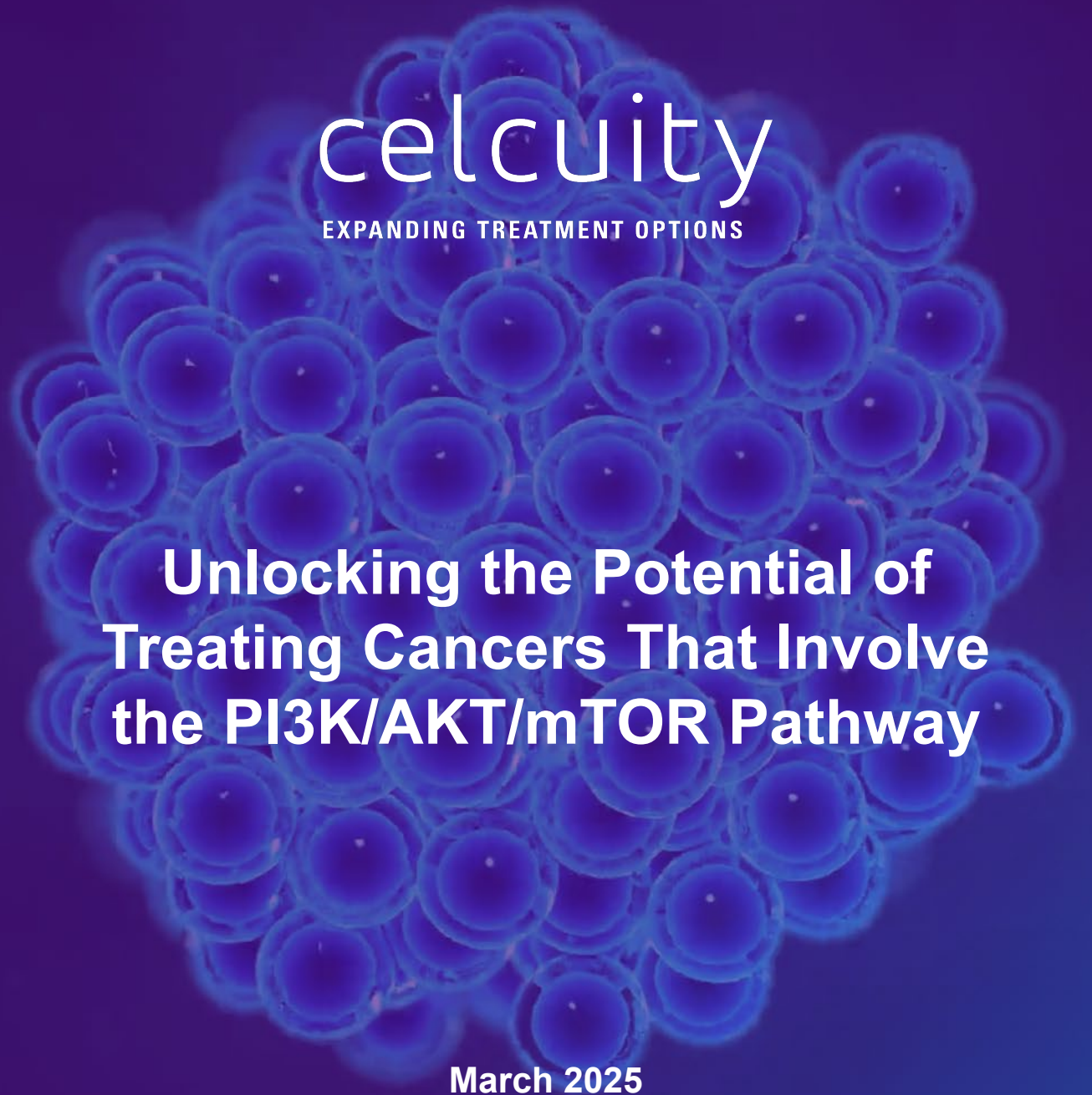
EXPANDING TREATMENT OPTIONS



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

March 2025

Forward-Looking Statements

This presentation contains “forward-looking statements” within the meaning of the Private Securities Litigation Reform Act of 1995 relating to our business, operations, and financial condition, including but not limited to current beliefs, expectations and assumptions regarding the future of our business, future plans and strategies, our development plans, our preclinical and clinical results and expected timing thereof, our plans to develop and commercialize gedatolisib, our first internally developed drug candidate, our plans to research, discover and develop additional product candidates, our planned milestones and timing of achieving such milestones, the focus and design of our clinical development program, our expectations regarding the timeline of patient enrollment, and receiving results and data, from clinical trials, including our existing Phase 3 VIKTORIA-1 and VIKTORIA-2 clinical trials and Phase 1b/2 study and clinical trial for gedatolisib, any potential benefits resulting from Breakthrough Therapy designation for gedatolisib, and other expectations with respect to Celcuity's lead product candidate, gedatolisib, the estimated costs of our clinical trials, our expectations as to the use of proceeds from our recent financing activities and the adequacy of cash to fund operations, and our beliefs related to the perceived advantages of our CELsignia tests compared to traditional molecular or other diagnostic tests and its CELsignia platform. Words such as, but not limited to, “look forward to,” “believe,” “expect,” “anticipate,” “estimate,” “intend,” “plan,” “would,” “should,” and “could,” and similar expressions or words, identify forward-looking statements.

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The Celcuity Opportunity

Significant untapped potential to effectively treat PAM pathway involved cancers

1

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

2

- **Very compelling data in 1L (mPFS 48 months) and 2L (mPFS 12.9 months) patients with HR+/HER2- ABC**
- A Phase 3 study in 2L patients is enrolling and a Phase 3 study in 1L patients is expected to begin enrolling in Q2 2025

3

- **Strong scientific rationale to develop gedatolisib for prostate cancer indications**
- Parallels between breast and prostate cancer – interdependent activity between PAM pathway and hormonal pathways

4

- **Uniquely positioned to advance multiple potential blockbuster indications in breast and prostate cancer**
- Cash, cash equivalents and short-term investments of $235M as of Q4 2024 expected to fund operations through 2026

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

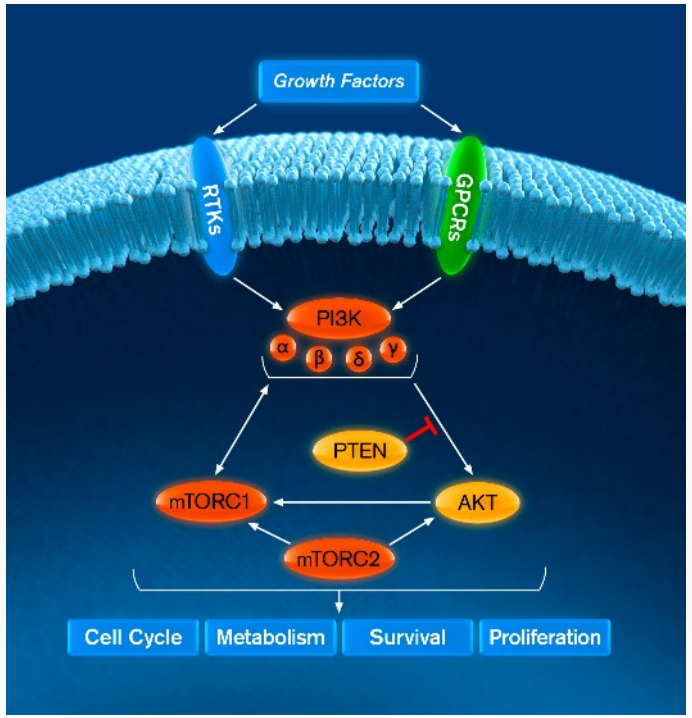
Optimal efficacy may require inhibition of all Class I PI3K isoforms and mTORC1 and mTORC2

Multiple pathway components provide functional redundancy

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

Feedforward and feedback loops between PI3K isoforms, AKT, and mTOR cross-activates uninhibited sub-units[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*	Node-specific inhibitors Limited PFS benefit Four drugs approved	*Today*	Need safe, potent pan-PI3K/mTORi

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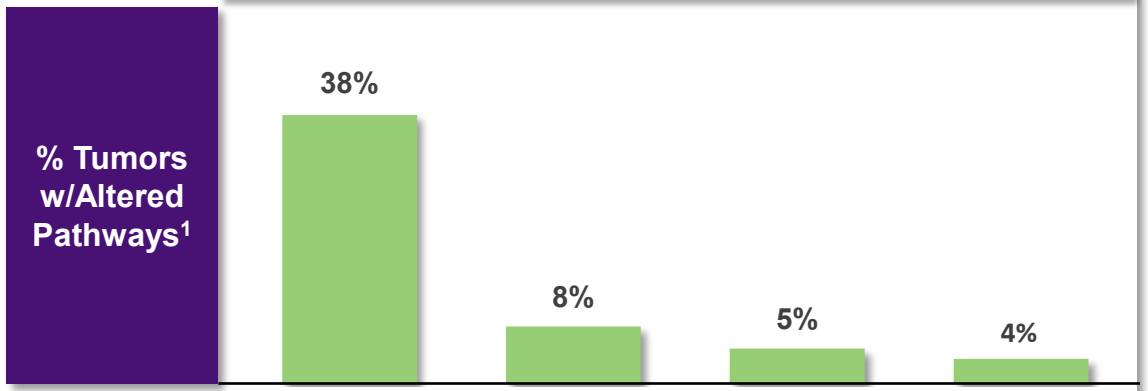
Sources: (1) Juric 2015 Nature. 518:240-244; (2) Castel 2021 Nat Cancer. 2:587-597; (3) Mao 2021 Nat Commun. 12:5053; (4) Schwartz 2015 Cancer Cell. 27:109-122. (5) Chandarlapaty 2011, Cancer Cell. 19:58-71; (6) Bago 2016, EMBO J. 35:1902-1922; (7) Manning 2017, Cell. 169:381-405.; (8) Mukherjee 2021,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

The PAM Pathway is the Most Underdeveloped Target in Solid Tumors

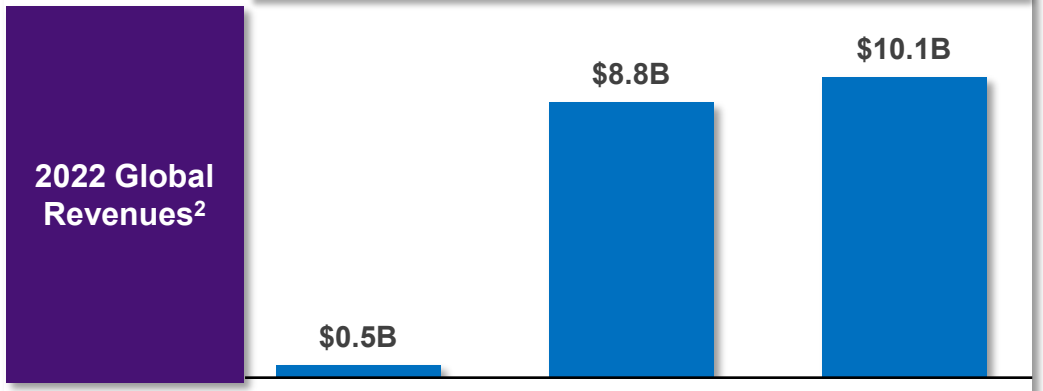
PAM is the most frequently altered pathway in solid tumors



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

Gedatolisib is a Potential First-in-Class PAM (PI3K, AKT, mTOR) Inhibitor

Breakthrough Therapy Designation granted for 2L HR+/HER2- advanced breast cancer indication

Differentiated Mechanism of Action

- Inhibits all PI3K/mTOR nodes at **low or sub-nanomolar** concentrations
- **Nonclinical data suggests more potent & cytotoxic** than the single-node PAM inhibitors approved for breast or prostate cancer

Compelling Preliminary Results

- Gedatolisib + ET + CDK4/6 in HR+/HER2- ABC patients
- **79% ORR, 48.6 months mPFS** in 1L patients (n=41)[1]
- **63% ORR, 12.9 months mPFS** in 2L patients (n=27)[2]

Well-Tolerated

- Nominal Gr 3, no Grade 4 TEAE's as a single agent
- **Only 4% treatment discontinuation** due to AE with Phase 3 dosing in combination with palbociclib and fulvestrant[2]

Potential to Address Large Unmet Need

- **HR+/HER2- ABC**: Enrolling Phase 3 trials for 2L and 1L[3]
- **mCRPC**: Enrolling Phase 1b/2 trial for 1L/2L patients[3]
- **225,000 1L/2L patients** in US, EU5, Japan[4]

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Sources: (1) Rugo ESMO 2023; (2) Data from Expansion Arm D of the B2151009 Phase 1b clinical trial (Layman, Lancet Oncology 2024); includes 2 unconfirmed partial responses; (3) Clinicaltrial.gov (4) Salvi, The Breast, 2021; Globocan 2020; Abbreviations: ORR = objective response rate; mPFS = median progression free survival; 1L = 1st line; 2L = 2nd line; TEAE = Treatment emergent adverse event; AE = adverse events; ABC = advanced breast cancer; mCRPC = metastatic castration resistant prostate cancer; 5EU = France, Germany, Italy, Spain, UK

Gedatolisib Has a Highly Differentiated Mechanism of Action and Potency

Results in superior cytotoxicity vs. single node PAM inhibitors

Cell-Free Biochemical Dose Response Analysis

IC_{50} (nM)[1]

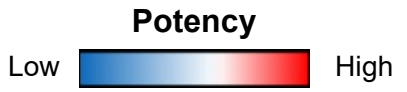
Node	Gedatolisib[2]	Alpelisib[3]	Everolimus[4]	Capivasertib[5]
PI3K-α	**0.4**	~4.0	-	-
PI3K-β	**6.0**	1,156	-	-
PI3K-γ	**5.4**	250	-	-
PI3K-δ	**6.0**	290	-	-
mTORC1	**1.6**	-	~2.0	-
mTORC2	**1.6**	-	-	-
AKT	-[6]	-	-	3.0

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)

	Geda	Alpe	Evero	Capi
All	12	6,308	3,611	8,666
MT	12	2,594	1,867	2,590
WT	12	10,308	5,501	15,209

Max Cell Growth Inhibition[1]

	Geda	Alpe	Evero	Capi
All	168%	89%	62%	80%
MT	174%	116%	68%	99%
WT	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.

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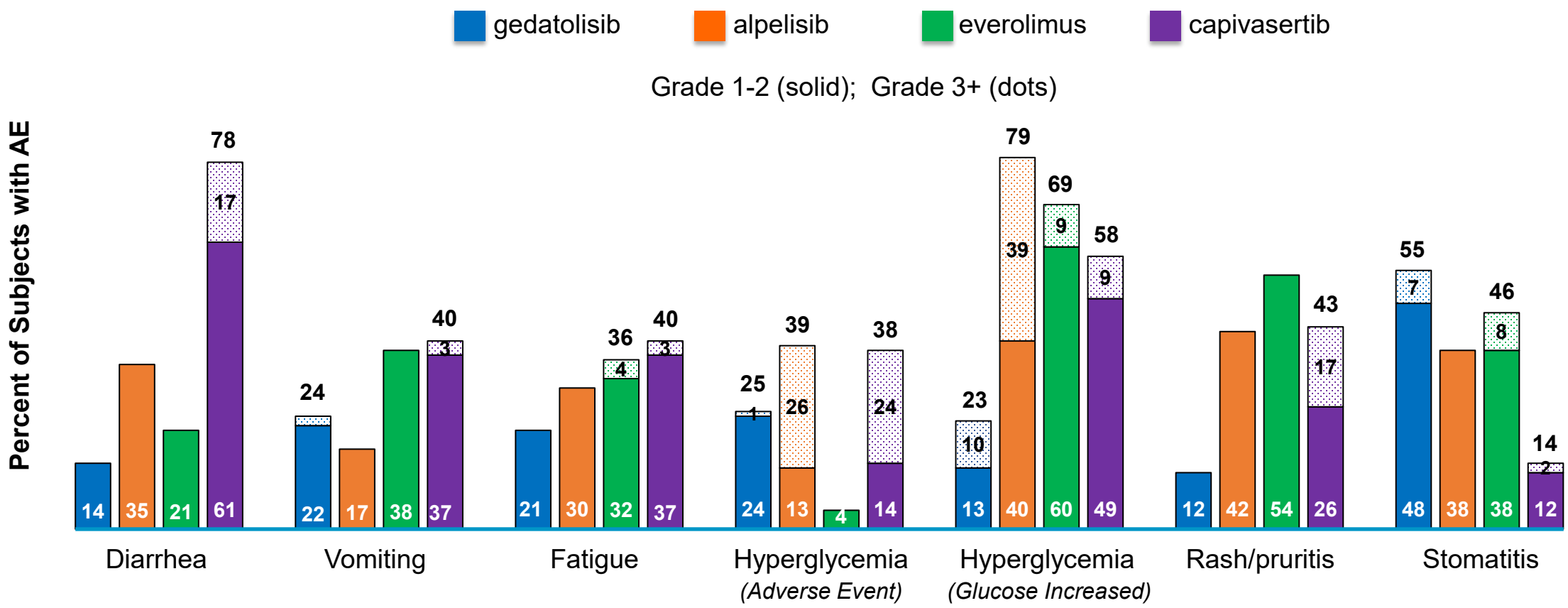
Source: (1) Shapiro 2015; (2) Rugo 2017; TEAE = treatment emergent adverse events; AE = adverse event.

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

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[1]

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
- Gedatolisib demonstrated superior potency and efficacy compared to other PAM inhibitors in nonclinical studies[2]
- Promising clinical activity with an AR inhibitor when combined with less active PAM inhibitors than gedatolisib[3]

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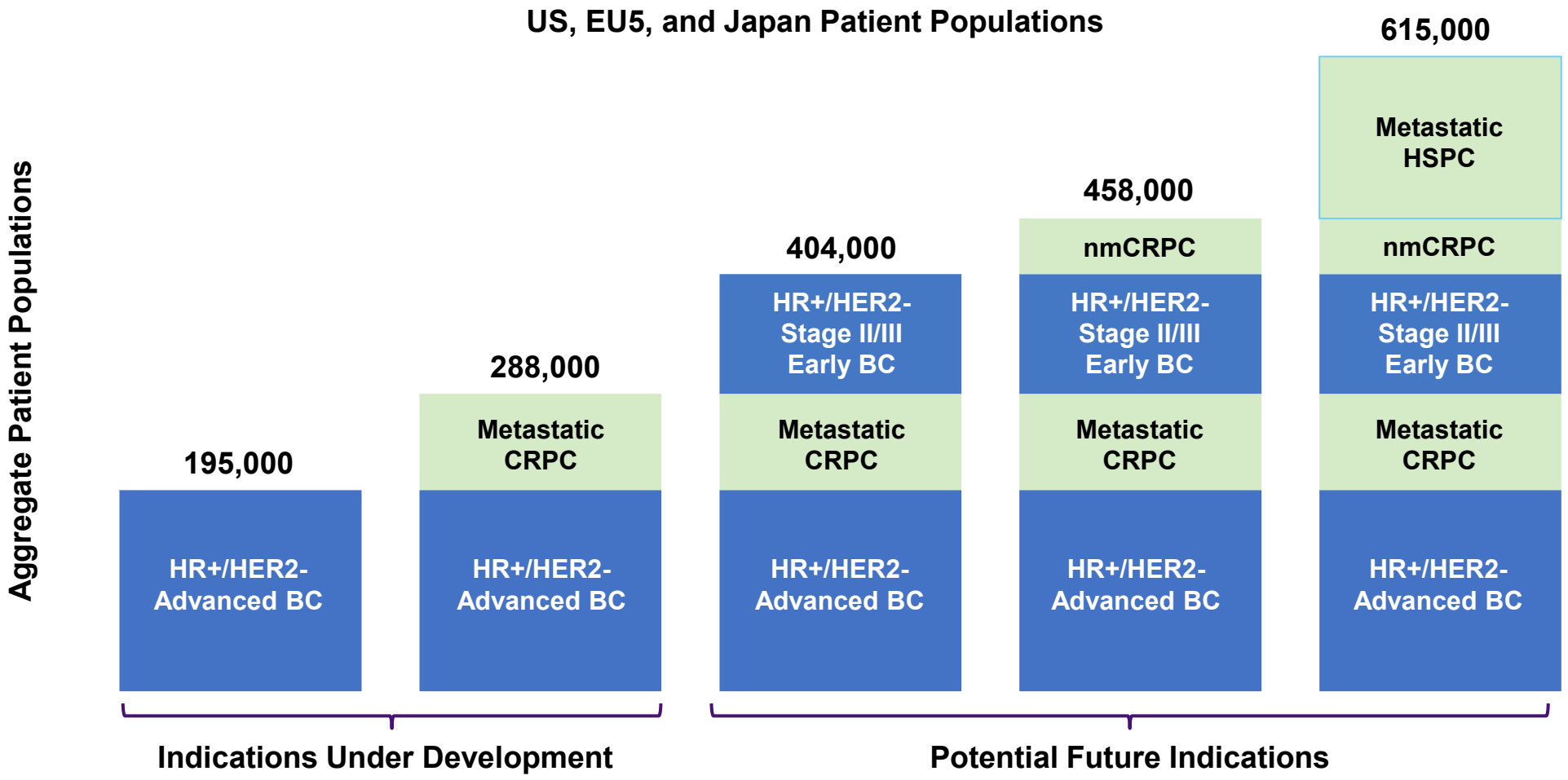
(1) Clinicaltrials.gov (2) Jhaveri, SABCS, 2023 (INAVO120); (2) Carver et al, Cancer Cell 2011; (3) Sen, ASCO-GU, 2023; (4) Sweeney Clin Cancer Res 2022; (5) De Bono, Lancet, 2021; Abbreviations: AI – aromatase inhibitor

Addressable Patient Population in Breast and Prostate Cancer



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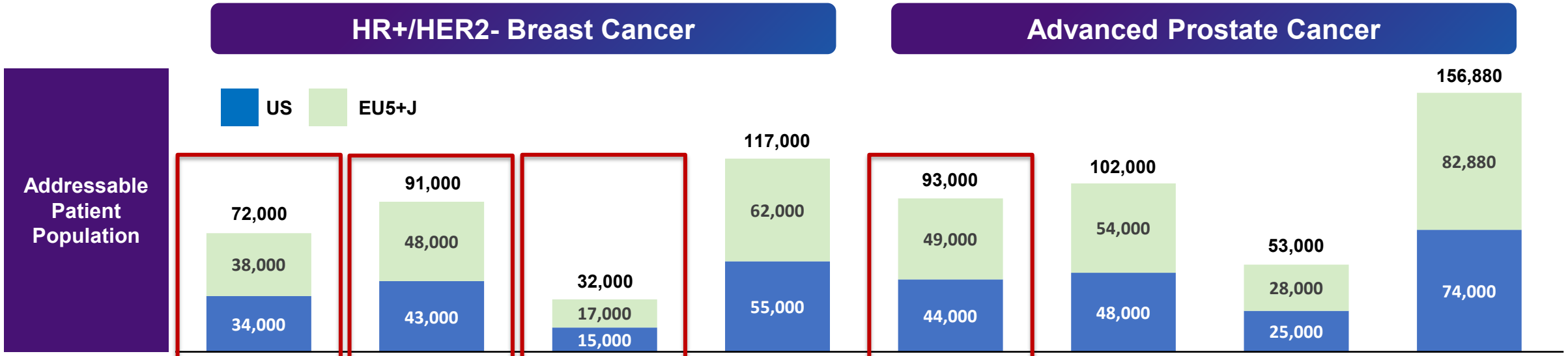
Indications under development include 2L ETS, 1L ETR,1L ETS, 1L/2L mCRPC. Sources: Internal estimates using data from American Cancer Society, Breast Cancer Statistics 2022; American Cancer Society Facts and Figures 2019-2020; Salvo, E. M. et al. (2021); Scher, et al. 2015; Datamonitor Healthcare; Leith, A. et al. 2022; George, D. J. et al. 2022; EU5 calculated using 112% EU + Japan from Globocan 2020 data; scale up factor Abbreviations: HR, hormone receptor; BC, breast cancer; CRPC, castration resistant prostate cancer; nm, non-metastatic; HSPC, hormone sensitive prostate cancer

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

Gedatolisib for Advanced Breast Cancer (ABC)

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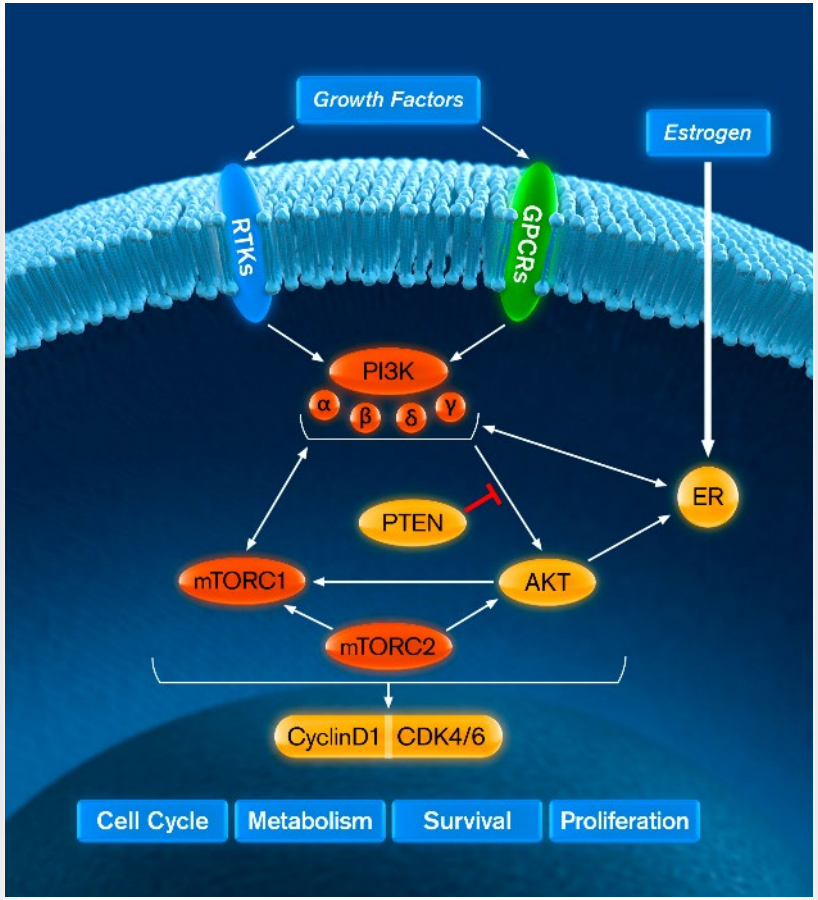
ER, CDK4/6, & PI3K/mTOR are Interdependent Drivers of HR+/HER2- ABC

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]**



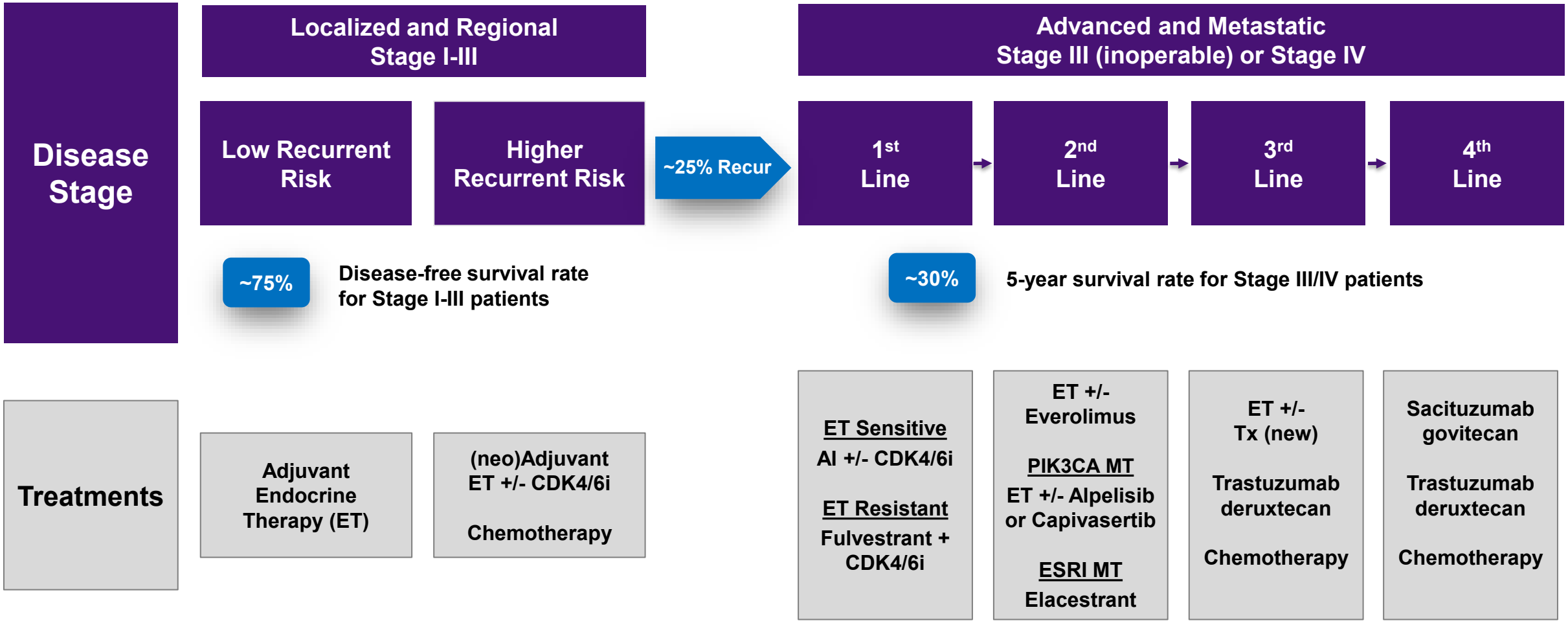
CDK4/6, ER and PI3K/mTOR[6-10]

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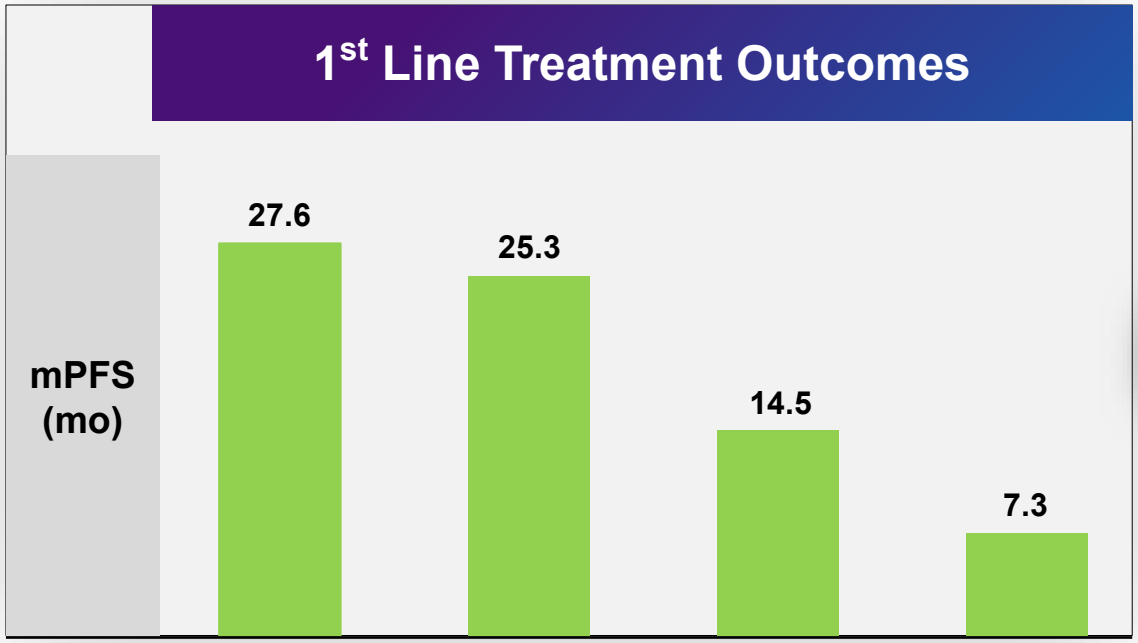
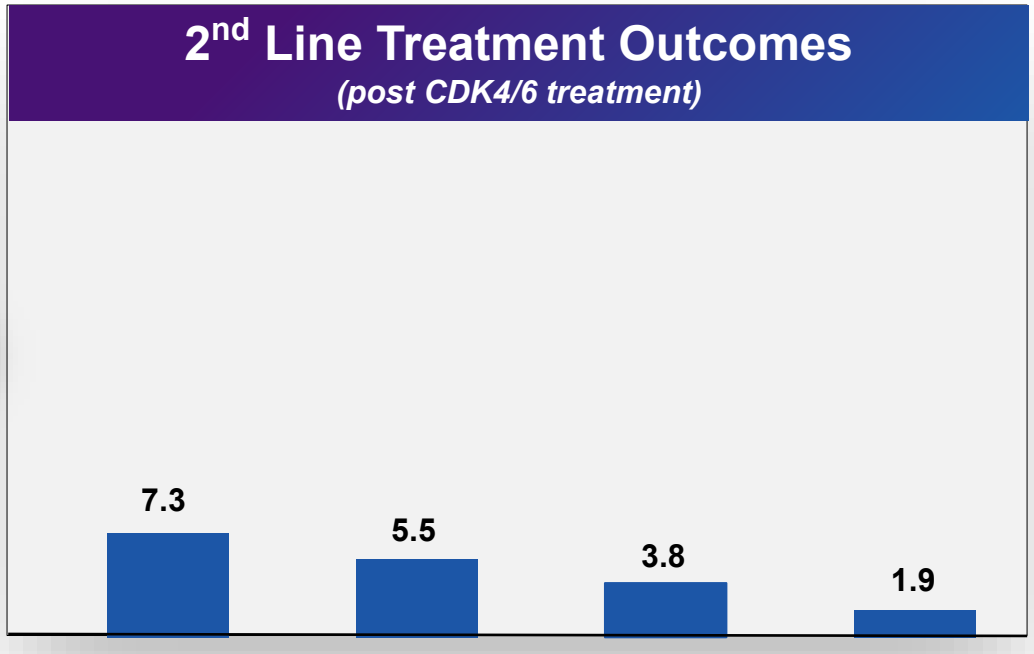
(1) NCCN Guidelines for Breast Cancer 2023; (2) American Cancer Society, Breast Cancer Statistics 2022; American Cancer Society Facts and Figures 2019-2020; Note: EU5 + Japan calculated using 112% EU + Japan scale up factor; Abbreviations: HR, hormone receptor; ET, endocrine therapy; AI, aromatase inhibitor;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

Limited Benefit for 1st Line ET Resistant or 2nd Line HR+/HER2- ABC Patients

Significant need for better therapeutic options





Drugs	Palbociclib + letrozole [1]	Ribociclib + letrozole [2]	Letrozole [1]	Palbociclib + Fulvestrant [3]
MOA	CDK4/6 + AI	CDK4/6 + AI	AI	AI
Pat Pop	ET Sensitive	ET Sensitive	ET Sensitive	ET Resistant
mPFS	27.6	25.3	14.5	7.3
ORR	55%	53%	44%	25%

Alpelisib + fulvestrant [4]	Capivasertib + fulvestrant [5]	Elacestrant [6]	Fulvestrant [6]
PI3Kα + SERD	AKT + SERD	SERD	SERD
PIK3CA+	PIK3CA/AKT/PTEN+	ESR1+	All
7.3	5.5	3.8	2-4
21%	23%	7%	6%

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(1) Finn NEJM 2016; Rugo H, et al. Breast Cancer Res Treat, 2019; (2) Hortobagyi NEJM 2016; Hortobagyi Ann Oncol 2018; USPI; (3) Jhaveri SABCS 2023 (4) Rugo Lancet Onco 2021; (5) Oliveira, ESMO Breast, 2023, CDK4/6 prior treated patients (6) Bidard, JCO, 2022 and FDA. Note: All drugs listed are FDA approved

Review of Phase 1b Data

Gedatolisib + Palbociclib + Fulvestrant/Letrozole

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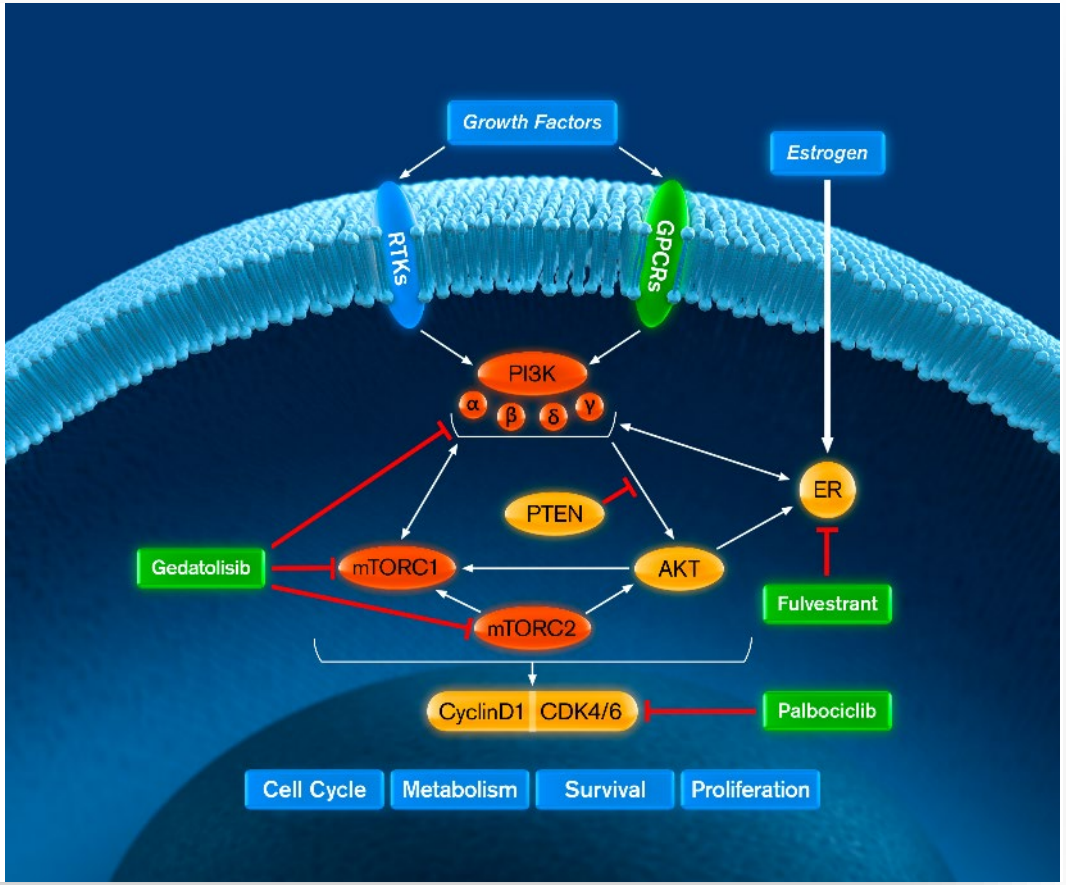
Clinical Strategy: Simultaneous Blockade of PAM, ER, & CDK4/6 Pathways

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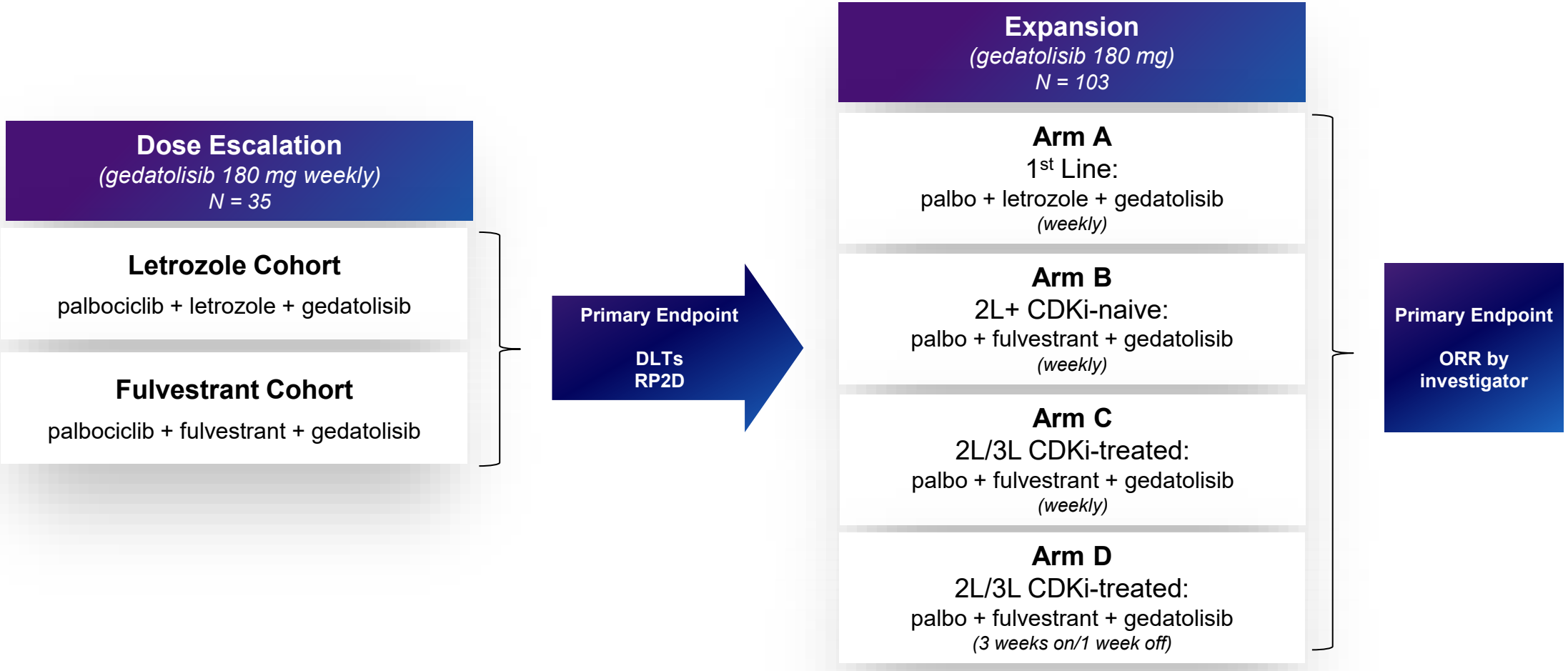
Sources: (1) Alves, Int J Mol. Sci. 2023; 24, 4522; (2) Cai 2022, Sci China Life Sci 65; (3) Karimi 2023, Cancer Communications, 43; (4) Jansen 2017, Cancer Res; 77(9); Abbreviations: ER = estrogen receptor; ABC = advanced breast cancer

Phase 1b Dose Escalation and Expansion Study (B2151009)

Key eligibility criteria: patients with HR+, HER2-, advanced breast cancer



Arm A subjects did not receive prior ET for ABC; Arm B subjects had 1-2 lines prior ET and no prior CDK4/6i for ABC; Arms C and D subjects had prior ET and CDK4/6i for ABC; Dose Escalation start date: May 2016; Dose Expansion Arms A, B, C start date: December 2017; Dose Expansion Arm D start date: October 2018: Database lock date: June 29, 2022
Reference: Layman RM, et al. *Lancet Oncol*. 2024; 25: 474-487.Abbreviations: DLTs – dose limiting toxicities; RP2D – recommended phase 2 dose; ORR - objective response rate

B2151009 Expansion Arms: Baseline Characteristics

	Arm A (N=31)	Arm B (N=13)	Arm C (N=32)	Arm D (N=27)
Tumor, Node, Metastasis (TNM) Current Stage, n (%)				
Stage IV	31 (100)	13 (100)	32 (100)	27 (100)
Prior therapies for ABC, n (%)				
Prior Chemotherapy	1 (3.2)	4 (30.8)	15 (46.9)	5 (18.5)
Prior Endocrine Therapy[1]	0	11 (84.6)	31 (96.9)	26 (96.3)
Prior CDK4/6 inhibitor	0	0	32 (100)	26 (96.3)
Number of prior systemic therapies ABC, n (%)				
0	30 (96.8)	2 (15.4)	0	0
1	1 (3.2)	9 (69.2)	15 (46.9)	18 (66.7)
≥2	0	2 (15.4)	17 (53.2)	9 (33.3)
Metastatic disease site involved				
Liver or Lung	20 (64.5)	12 (92.3)	23 (71.9)	22 (81.5)
Liver	14 (45.2)	10 (76.9)	20 (62.5)	17 (63.0)
Lung	7 (22.6)	3 (23.1)	7 (21.9)	6 (22.2)
Bone	18 (58.1)	11 (84.6)	25 (78.1)	18 (66.7)
Bone only	0	0	0	0

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(1) SERD, SERM, or aromatase inhibitor

ORR and PFS in Each Expansion Arm Was Superior to SOC

Results from Arm D - 63% ORR and 12.9 months PFS – provide basis for Phase 3 clinical trial

B2151009 Expansion Arms Efficacy Summary (N=103)

	Arm A		Arm B		Arm C		Arm D	
Prior Therapy	1L		2L+ CDKi-naive		2L/3L CDKi-pretreated		2L/3L CDKi-pretreated	
n (Full, response evaluable)	31, 27		13, 13		32, 28		27, 27	
Study Treatment (gedatolisib dosing schedule)	P + L + G (weekly)		P + F + G (weekly)		P + F + G (weekly)		P + F + G (3 weeks on / 1 week off)	
ORR[1] (evaluable)	85%		77%		36%		63%	
mPFS [2], months (range)	48.4 (16.9, NR)		12.9 (7.6, 38.3)		5.1 (3.3, 7.5)		12.9 (7.4, 16.7)	
PFS % at 12 mos [2]	72%		55%		24%		53%	
PIK3CA Status	WT	MT	WT	MT	WT	MT	WT	MT
	81%[3]	16%	69%	31%	75%	25%	56%[3]	41%
ORR [1] (evaluable)	81%	100%	78%	75%	25%	63%	60%	73%
PFS % at 12 mos [2]	74%	60%	50%	67%	22%	29%	49%	60%

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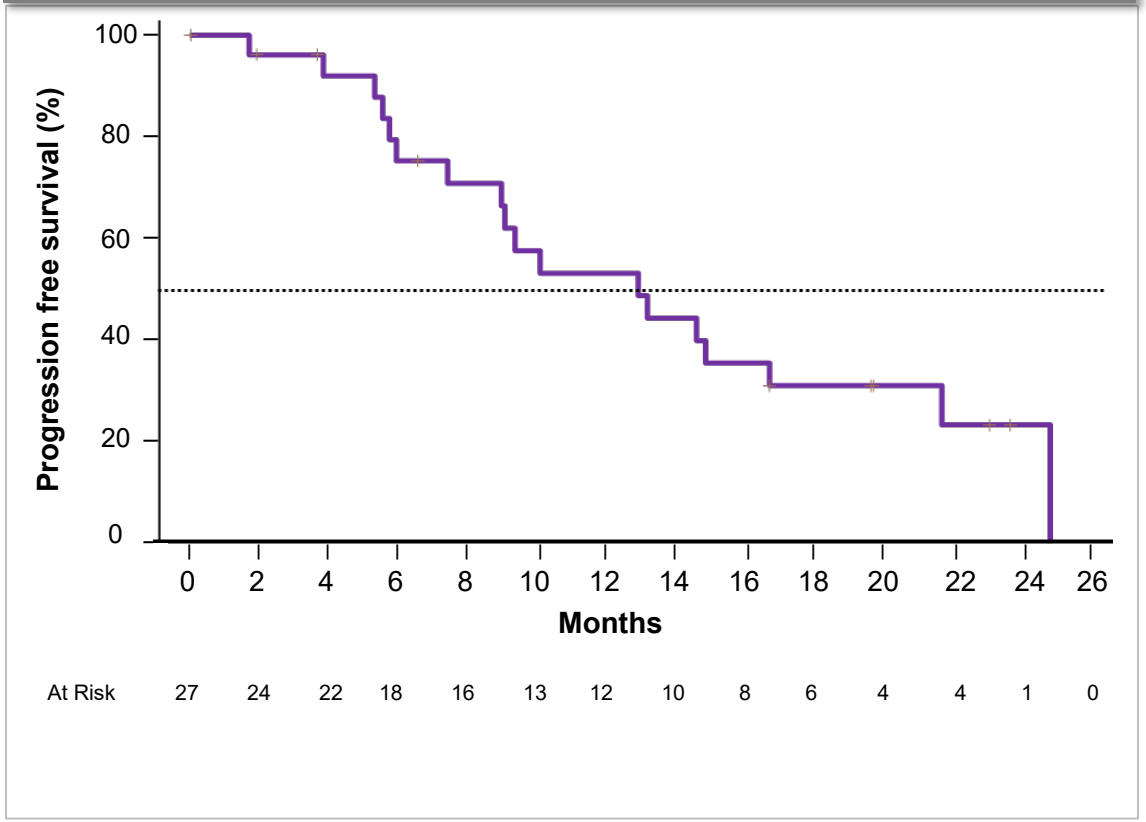
Source: Layman, Lancet 2024; Rugo 2023 ESMO-Breast. Footnotes: (1) Response evaluable analysis set per RECIST v1.1 including uPR (n=2, Arm B; n=3, Arm C; n=2, Arm D); (2) full analysis set, (3) Baseline *PIK3CA* mutation status missing for one patient. Abbreviations: 1L, first line, 2L, second line; mos, months; MT, *PIK3CA* mutation; NR, Not reached; ORR, objective response rate; mPFS, median progression free survival; SOC, standard of care; WT, wild type

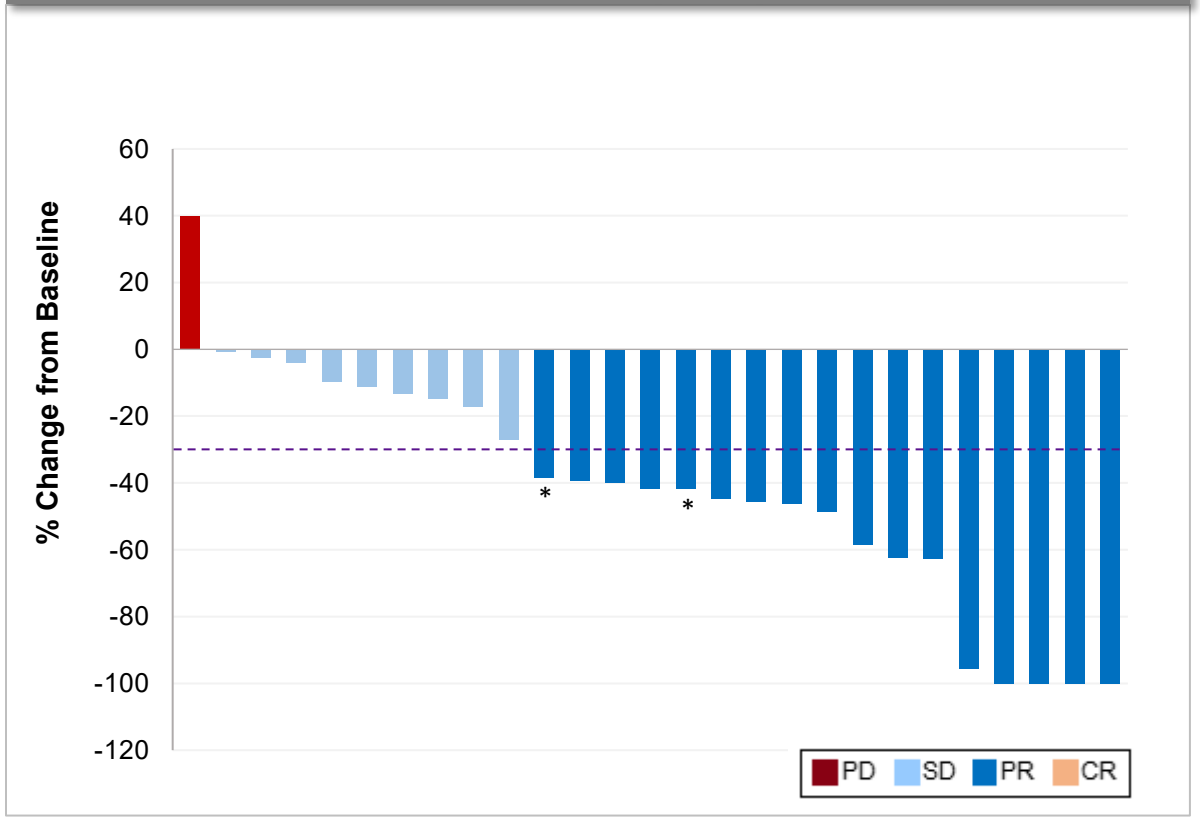
Gedatolisib + Palbociclib + Fulvestrant in 2nd/3rd Line HR+/HER2- ABC Patients

mPFS and ORR from Arm D with Phase 3 regimen compares favorably to published data for current SOC

Median Progression Free Survival
12.9 Months



Tumor Size Change
ORR = 63% (17/27)



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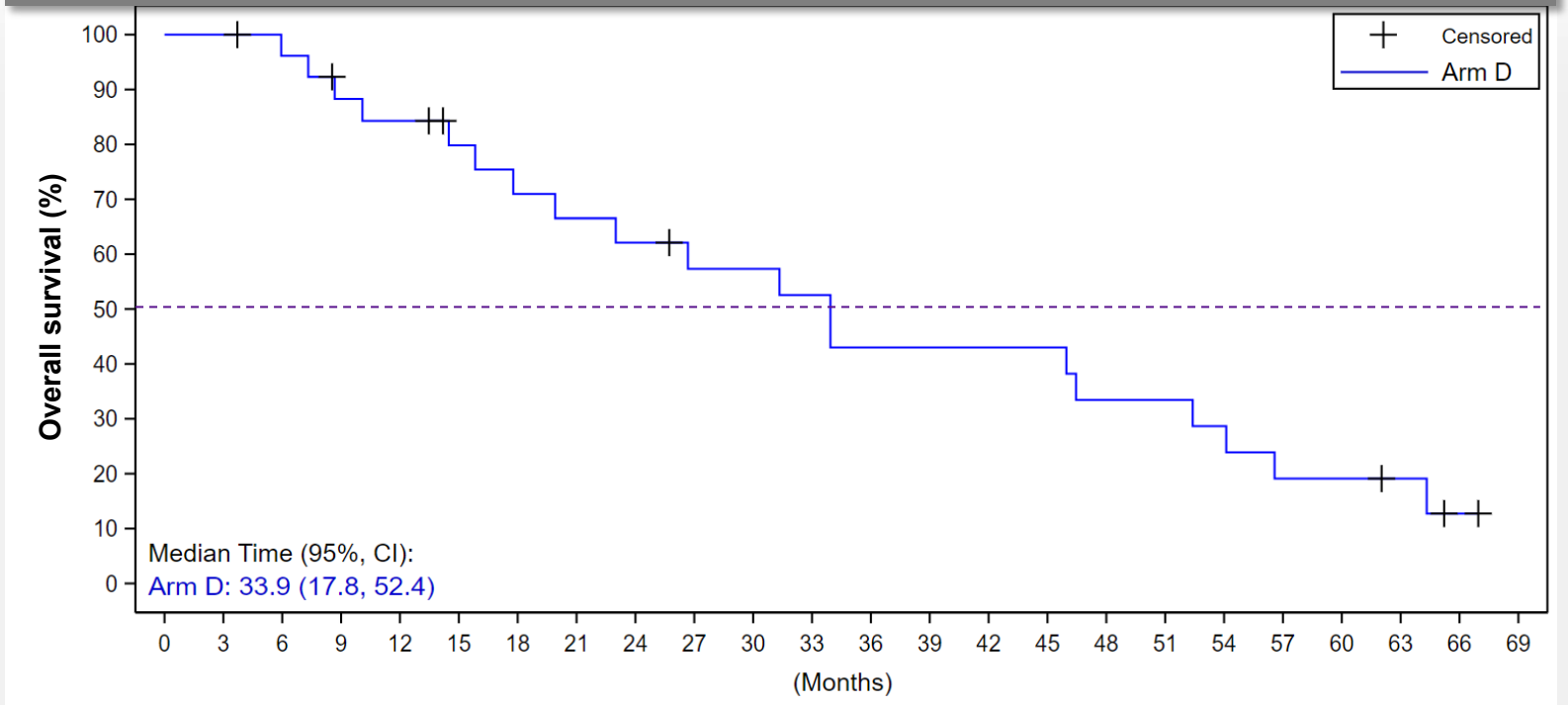
Source: Layman, Lancet, 2024, Wesolowski 2022 SABCS - Arm D data from B2151009 study. ORR includes 2 unconfirmed PRs; *unconfirmed PR.

Gedatolisib + Palbociclib + Fulvestrant in 2nd/3rd Line HR+/HER2- ABC Patients

mOS from Arm D with Phase 3 regimen compares favorably to published data for current SOC

Median Overall Survival
33.9 months[1]



Relevant OS data in 2L post-CDK4/6 setting

- **Alpelisib + fulvestrant: 27.3 months[2]**
  - **BYLieve Cohort A study**
  - **PIK3CA MT patients**
- **Endocrine monotherapy: 17.0 months[3]**
  - **EMERALD study**
  - **ESR1 MT patients**

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1) Rugo H. et al., SABCS, 2024; 2) Rugo H. et al., Lancet Onco, 2024; 3) Bardia A. et al., Clin Cancer Res, 2024

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 Combo and SOC Data for 2L HR+ / HER2- ABC Post-CDKi

Gedatolisib Combo Offers Potential for Superior Efficacy Compared to Alternatives

Patient Population	2nd Line ER+/HER2- ABC	
All	Gedatolisib + Fulvestrant + Palbociclib [1]	mPFS 12.9 months ORR 63%
PIK3CA+	Alpelisib + Fulvestrant [2]	mPFS 8.0 months ORR 19%
PIK3CA+	Alpelisib + Fulvestrant [3]	mPFS 5.6 months ORR 24%
PIK3CA/AKT1/ PTEN+	Capivasertib + Fulvestrant [4]	mPFS 5.5 months ORR 23%
ESR1+	Elacestrant [5]	3.8 months ORR 4%
All	Fulvestrant [5]	mPFS 1.9 months ORR 6%

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EXPANDING TREATMENT OPTIONS

(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.

Efficacy in Treatment-Naïve Population Superior to SOC

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

B2151009 Treatment-Naïve Patients (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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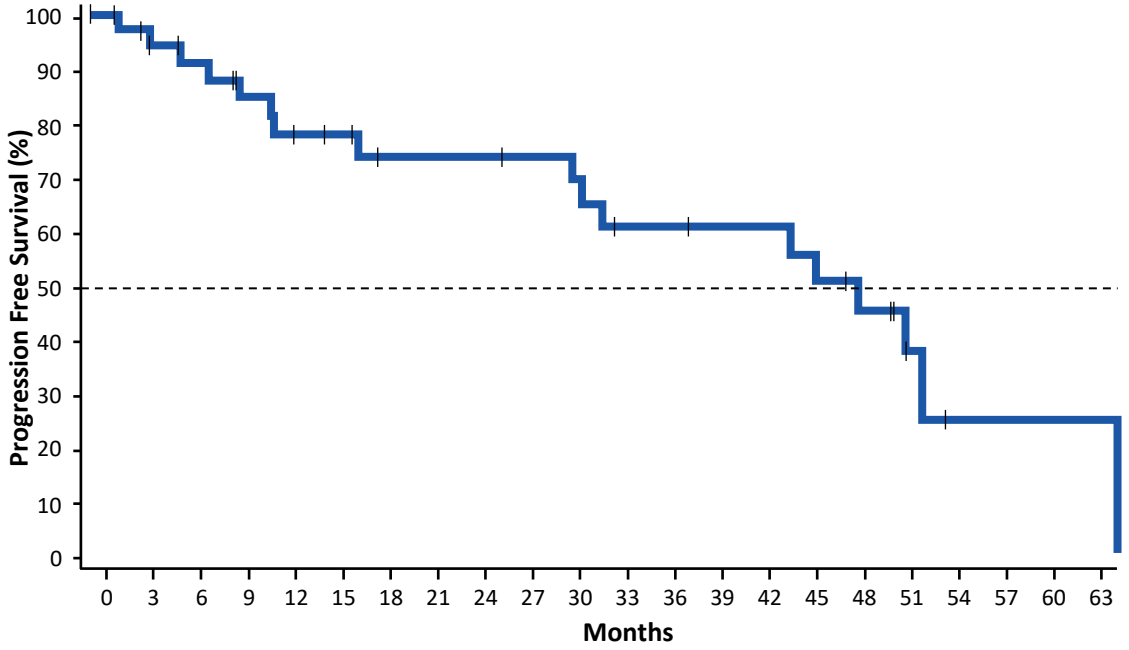
Source: Rugo 2023 ESMO Breast. (1) Subjects with measurable disease in response evaluable analysis set per RECIST v1.1;(2) Confirmed responders in the full analysis set. Abbreviations: CR, complete response; DOR, duration of response; mos, months; NR, Not Reached; ORR, objective response rate; PD, progressive disease; PFS, progression free survival; PR, partial response; SD, stable disease

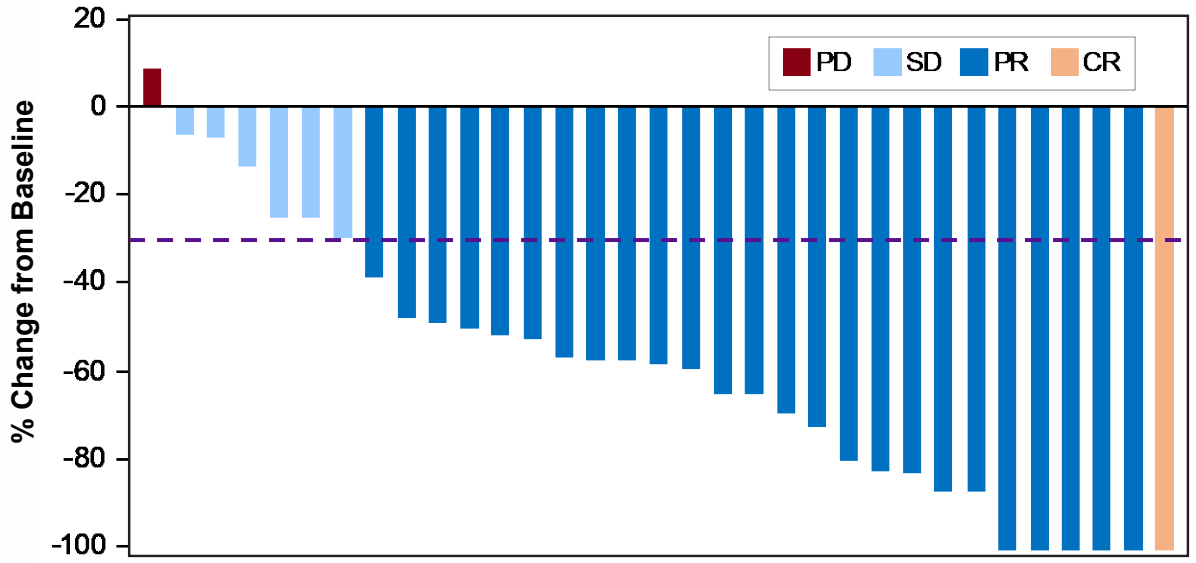
Gedatolisib + Palbociclib + Letrozole in 1st Line HR+/HER2- ABC (N=41)[1]

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

Median Progression Free Survival
48.6 Months



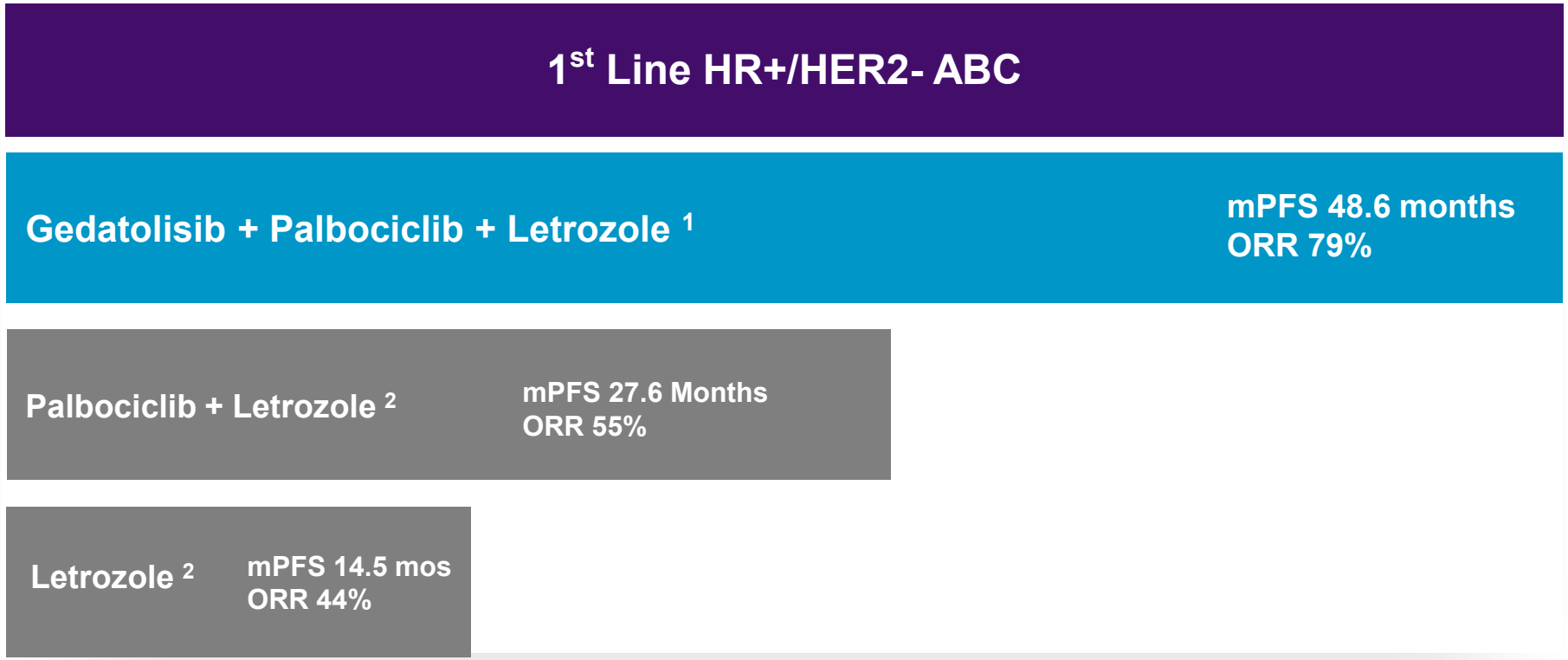
Tumor Size Change
ORR = 79% (26/33)



(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.

Gedatolisib Combo vs. SOC for 1L HR+ / HER2- ABC

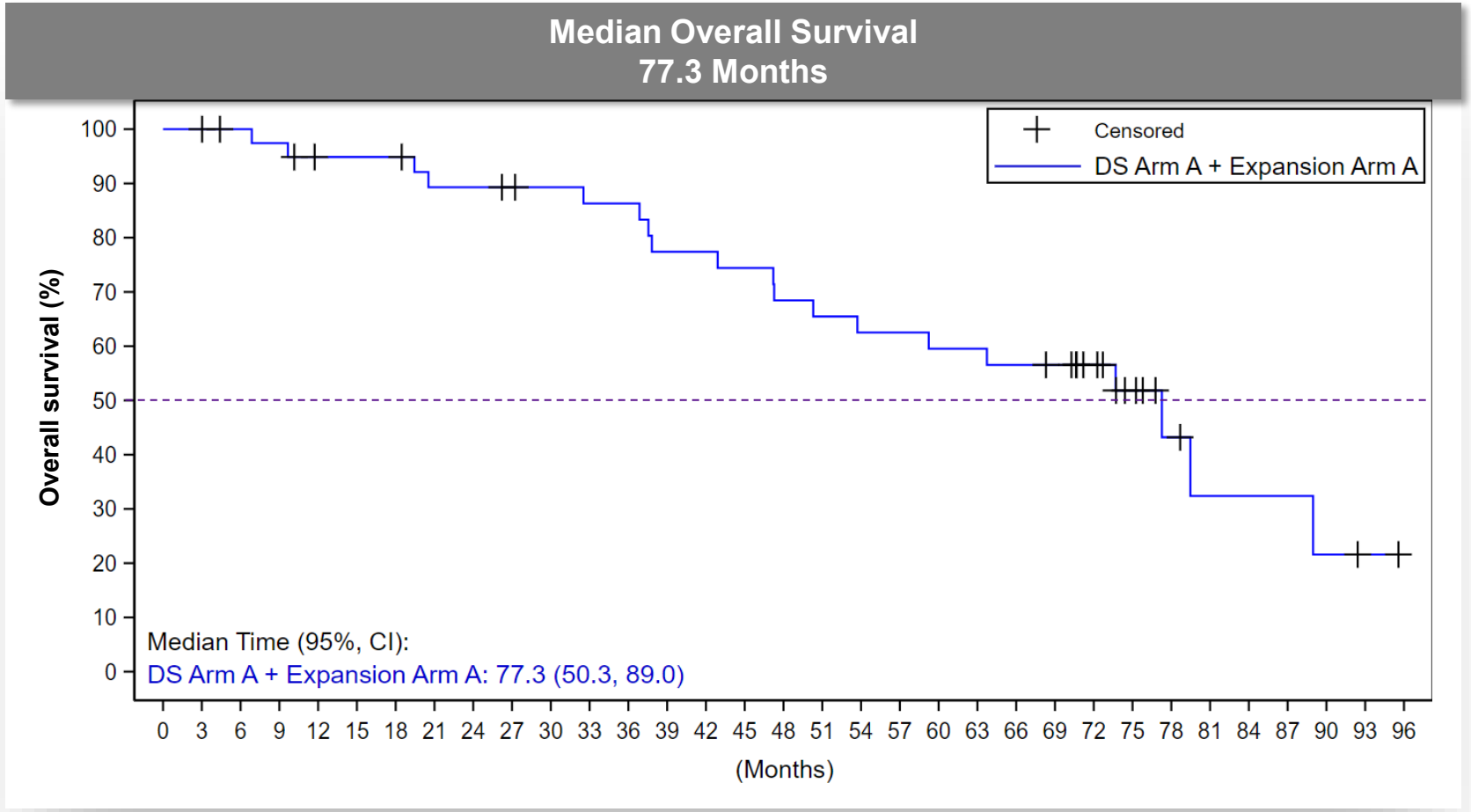
Gedatolisib Combo Offers Potential for Superior mPFS Compared to 1L SOC



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.

Gedatolisib + Palbociclib + Letrozole in 1st Line HR+/HER2- ABC Patients

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



Relevant OS data in 1L setting

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

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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

Phase 3 Study Designs
VIKTORIA-1 and VIKTORIA-2

VIKTORIA-1: Trial Design Considerations for 2nd Line HR+/HER2- ABC

- Standard-of-care 2nd line treatment is based on *PIK3CA* status
- ~35-40% of patients have disease with *PIK3CA* mutations
- PFS is accepted primary end point for randomized studies in ABC

Supports design with multiple primary endpoints in different sub-groups

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

Global open-label randomized study (>200 sites)

- **Key eligibility criteria:**
  - ER+/HER2- advanced or metastatic breast cancer
  - Prior CDK4/6i + NSAI
  - Bone-only with measurable lesions
  - ≤ 2 prior endocrine therapy
  - No prior chemotherapy for ABC
- **Three primary endpoints could support three separate indications**
  - Two co-primary endpoints (PFS) in *PIK3CA* WT patients
  - One primary endpoint (PFS) in *PIK3CA* MT patients
- **Three-arm design for *PIK3CA* WT and MT patients enables evaluation of two different regimens**
- **Stratification by geography, prior treatment response (≤ or > 6 months), presence of liver or lung metastasis (yes/no)**

Phase 3 vs. Phase 1b Arm D
Key Eligibility Criteria Differences

- **Prior chemotherapy for ABC**
  - Phase 3: 0% (not eligible)
  - Arm D: 19% had prior chemo
- **Bone-only with measurable lesions**
  - Phase 3: Typically, 15%-20% ABC
  - Arm D: 0% (not eligible)
- **Implications**
  - Bone only and chemo naïve patients typically have better prognosis than those with visceral disease and prior chemo

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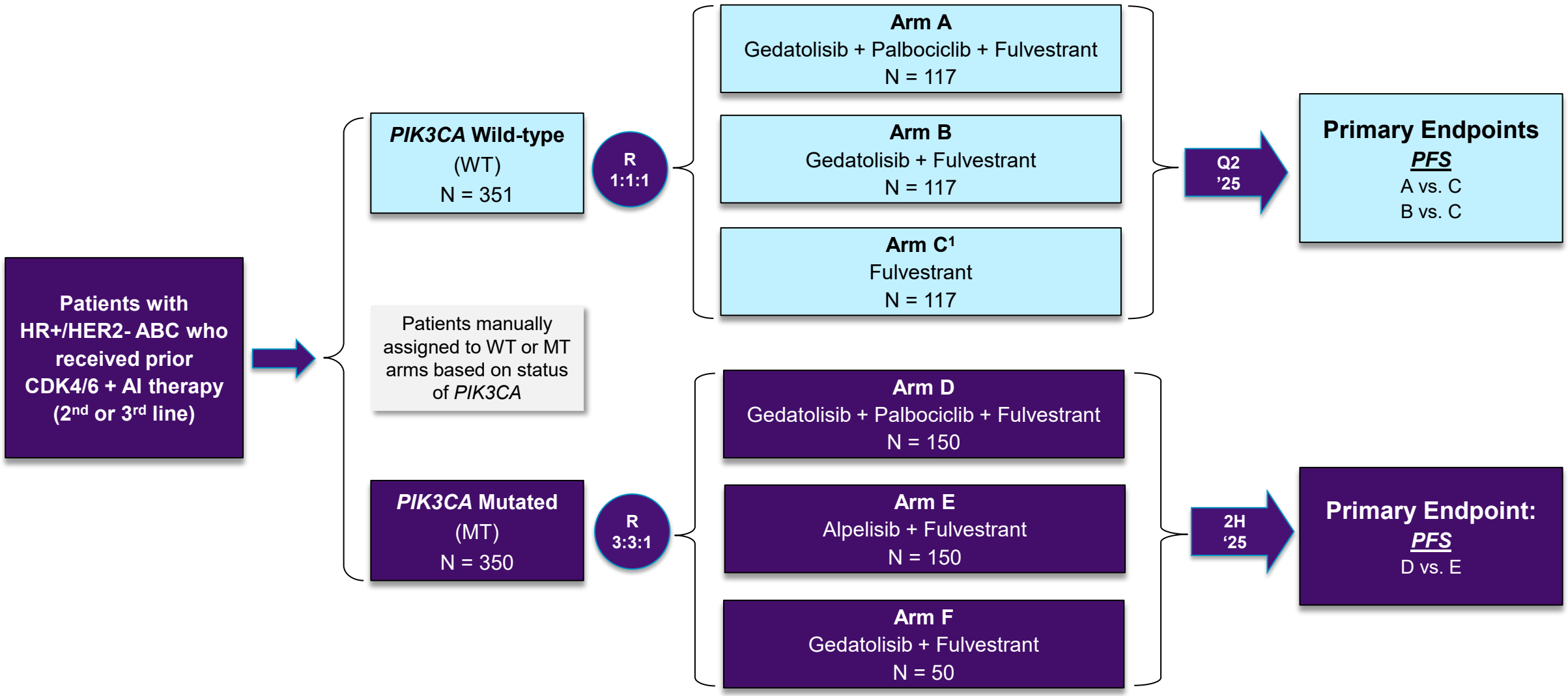
VIKTORIA-1: Phase 3 Trial Design Overview for 2L HR+/HER2- ABC



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

Relevant Comparisons to VIKTORIA-1 Controls

B2151009 study results compared to published data for patients who received a prior CDK4/6i

	Gedatolisib + Palbo + Fulvestrant N=27[1,2]	Fulvestrant N=165[3]	Fulvestrant N=37[5]	Fulvestrant N=121[6]	Alpelisib + Fulvestrant N=126[8]	Alpelisib + Fulvestrant N=121[9]
Trial	B2151009 – D	EMERALD	SERENA-2	CAPItello-291	BYLieve – C	BYLieve - A
***PIK3CA* Status**	WT / M (56% / 41%)	WT / M (NR)	WT / M (NR)	WT / M	M	M
Line of Therapy (% by line)	2L / 3L+ (67% / 33%)	2L / 3L+ (73% / 27%)[4]	2L / 3L (NR)	2L / 3L (NR)	2L / 3L+ (37%/ 63%)	1L / 2L/ 3L+ (2% / 80% / 18%)
mPFS (months)	**12.9**	**1.9**	**2.1**	**2.6**	**5.6**	**8.0**
ORR	63% (overall)[2] WT 60% M 73%	NR	12%	14%[7]	22%	19%
PFS % at 12 months	53% (overall) WT 49% M 60%	10%	10%	12%	22%	27%

Sources: (1) Layman, Lancet Oncol, 2024; (2) Includes 2 unconfirmed PR.(3) Bidard 2022 NEJM – EMERALD trial; (4) 73% of patients had 1 prior line of endocrine therapy and 80% of patients had no prior chemotherapy in the advance setting; (5) Oliveria, Lancet Oncol, 2024, SERENA-2 trial; (6) Turner, NEJM, 2023, CAPItello-291 trial, mPFS only includes WT patients who had prior CDK4/6 treatment; PFS % at 12 months includes all patients who had prior CDK4/6 treatment; (7) ORR includes unconfirmed responses from all patients treated with fulvestrant, including those who had prior CDK4/6i and those who didn't; (8) Rugo 2021 SABCS (9) Rugo Lancet Oncol, 2024. 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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VIKTORIA-2: Phase 3 Study Features for 1L HR+/HER2- ABC

Global open-label randomized study (~200 sites)

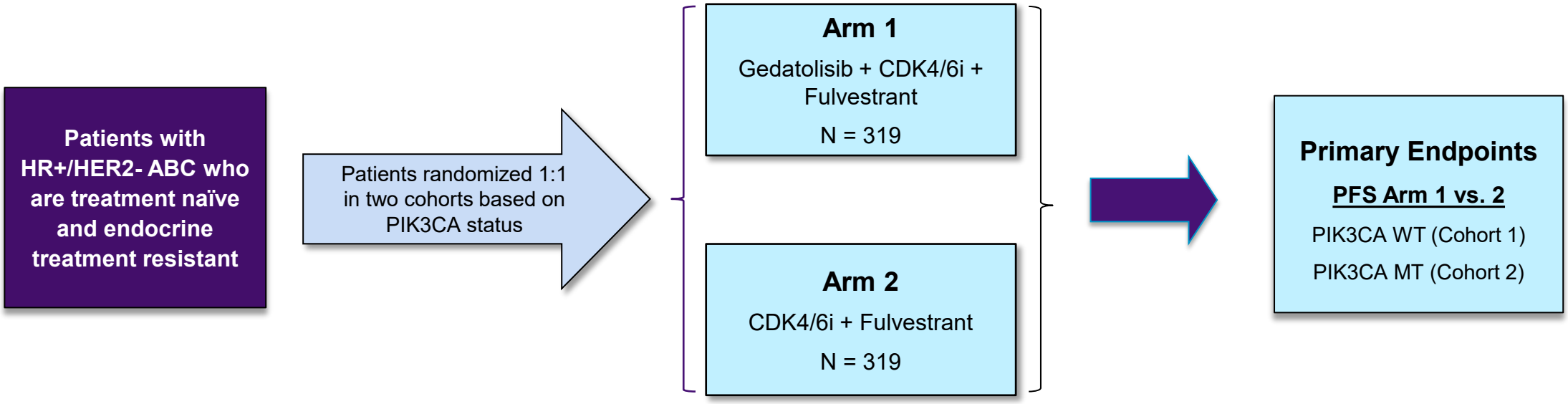
- **Key eligibility criteria:**
  - ER+/HER2- advanced or metastatic breast cancer
  - No prior treatment for advanced or metastatic breast cancer
  - Progression or relapse of disease during or within 12 months of completing adjuvant endocrine treatment
  - Pre-diabetic or patients with controlled diabetes 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



Plan to enroll first patient Q2 2025

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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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Clinical Trial Results Provide POC in this 1L ABC Patient Population[1]

Results for a less potent PAM inhibitor in small fraction of population highlights opportunity for gedatolisib

Study Regimens	Line of Therapy	Patient Population	N	Overall Results (Months rPFS)	Comments

Inavolisib (PI3Kα) + Palbociclib + Fulvestrant vs. Palbociclib + Fulvestrant [1]	1st Line	PIK3CA MT+
		
		Progressed on prior adjuvant ET w/in 12 months after last treatment
		
		Fasting glucose <126 mg/dL and HbA_{1C} <6.0%
			325	**15.0 vs. 7.3 months** *(HR = 0.43; P<0.0001)*	▪ Inavolisib shows clinical activity despite only targeting PI3Kα
					▪ Gedatolisib 5X-10X more potent in vitro than inavolisib[2]
					▪ Indication excludes ~80% of eligible patients
					  ▪ No PIK3CA WT (60%-65% of total ABC)
					  ▪ No pre-diabetics or controlled diabetics (40% of PIK3CA MT)
					▪ Gedatolisib has reported favorable preliminary results in total eligible population in both 1L and 2L patients

(1) Jhaveri SABCS (INAVO120), 2023; (2) Khan AACR, 2021. Note: No head-to-head trials have been conducted; data collected from different trials, in different patient populations and may not be comparable.

Gedatolisib for Prostate Cancer

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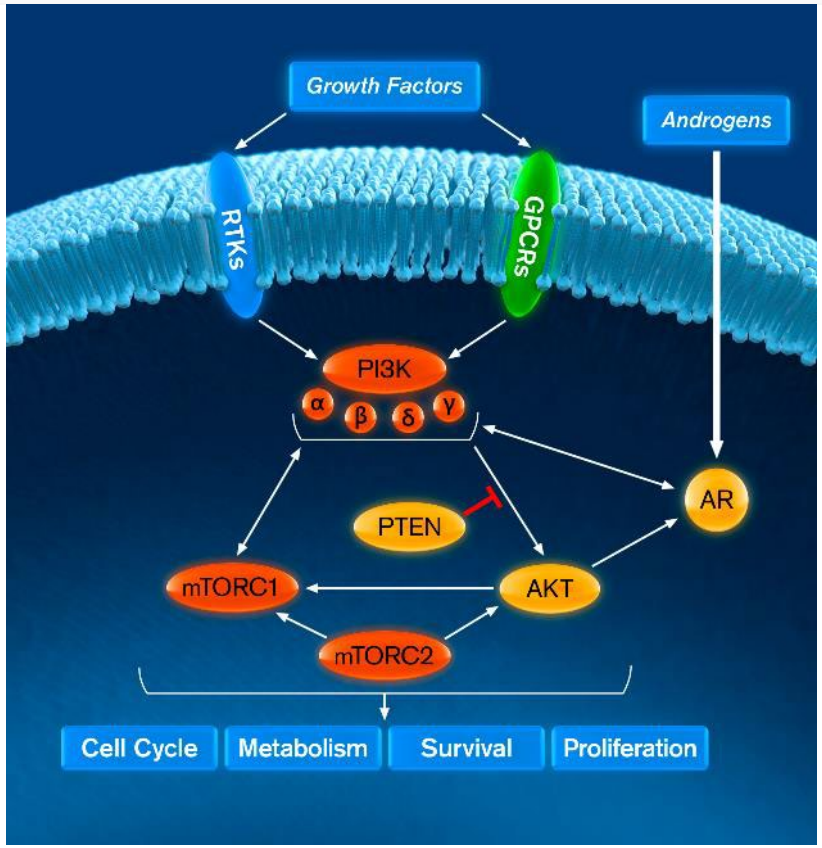
Androgen Signaling is the Key Driver of Prostate Cancer

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

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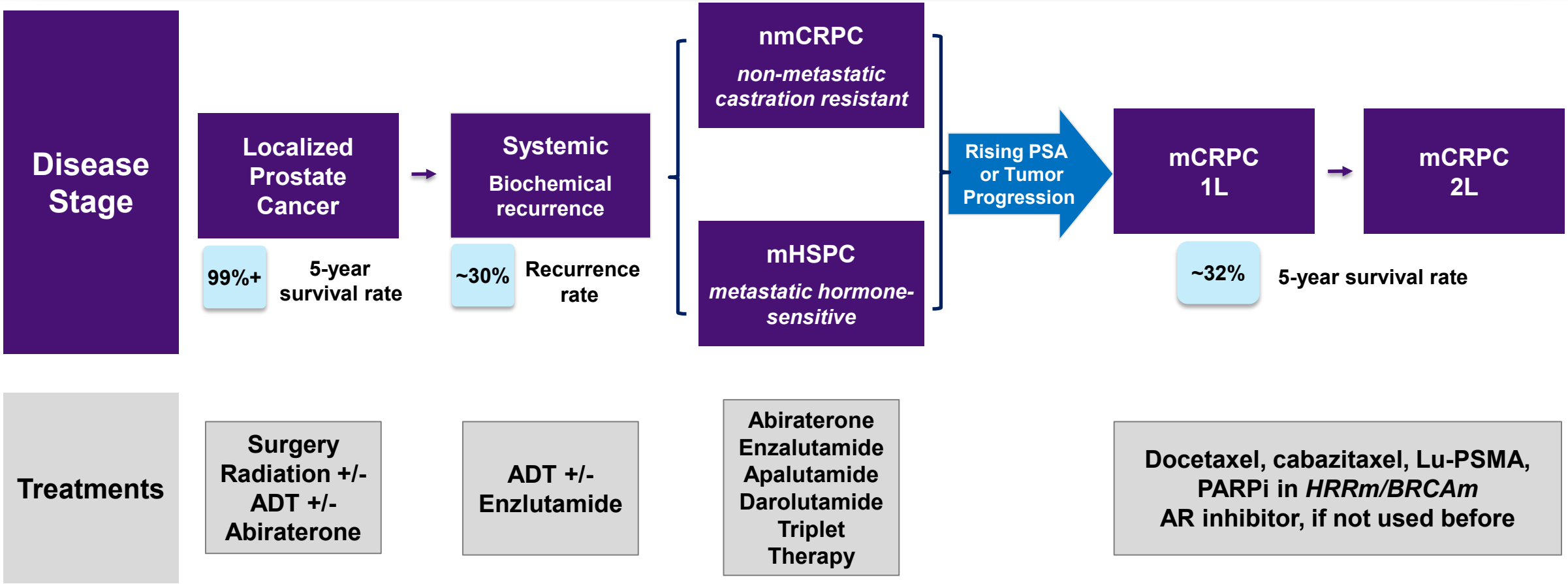
EXPANDING TREATMENT OPTIONS

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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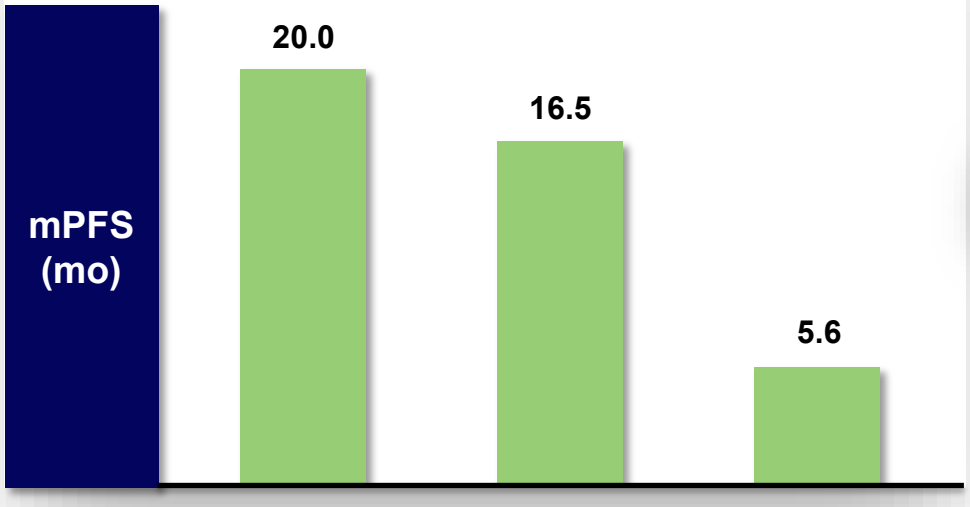
(1) Saad, Prostate Cancer Prostatic Dis. 2021; (2) Scher, Plos One 2015; Leith, A. et al. 2022; George, D. J. et al. 2022; NCCN Guidelines for Prostate Cancer Version 1.2023; (3) American Cancer Society, Cancer Facts & Figures 2023; (4) Wang, Front. Public Health, 2022; Abbreviations: mCRPC = metastatic 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

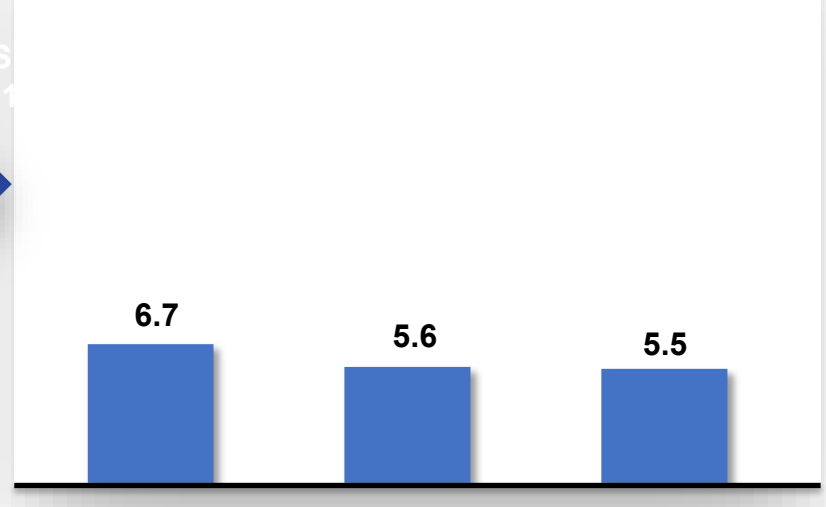
Significant need for better therapeutic options

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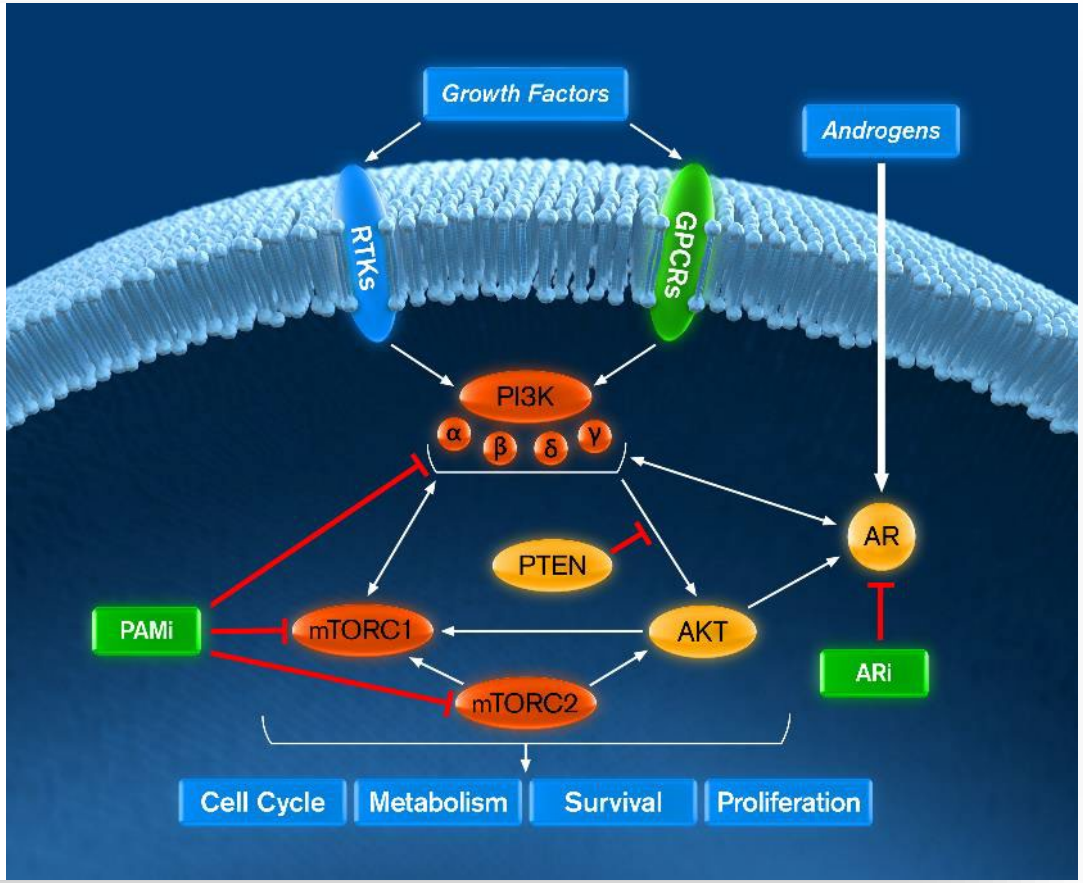
(1) Beer Eur Urol. 2017; (2) Ryan NEJM 2013; Ryan Lancet Oncol 2015 (3) Kellokumpa-Lehtinen Lancet Oncol. 2013, time-to-treatment failure reported; (4) Crabb J Clin Oncol 2021; (5) Attard J Clin Oncol 2018; (6) Sweeny Clin Cancer Res 2022. Abbreviations: HRR = homologous recombination repair; AR = androgen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

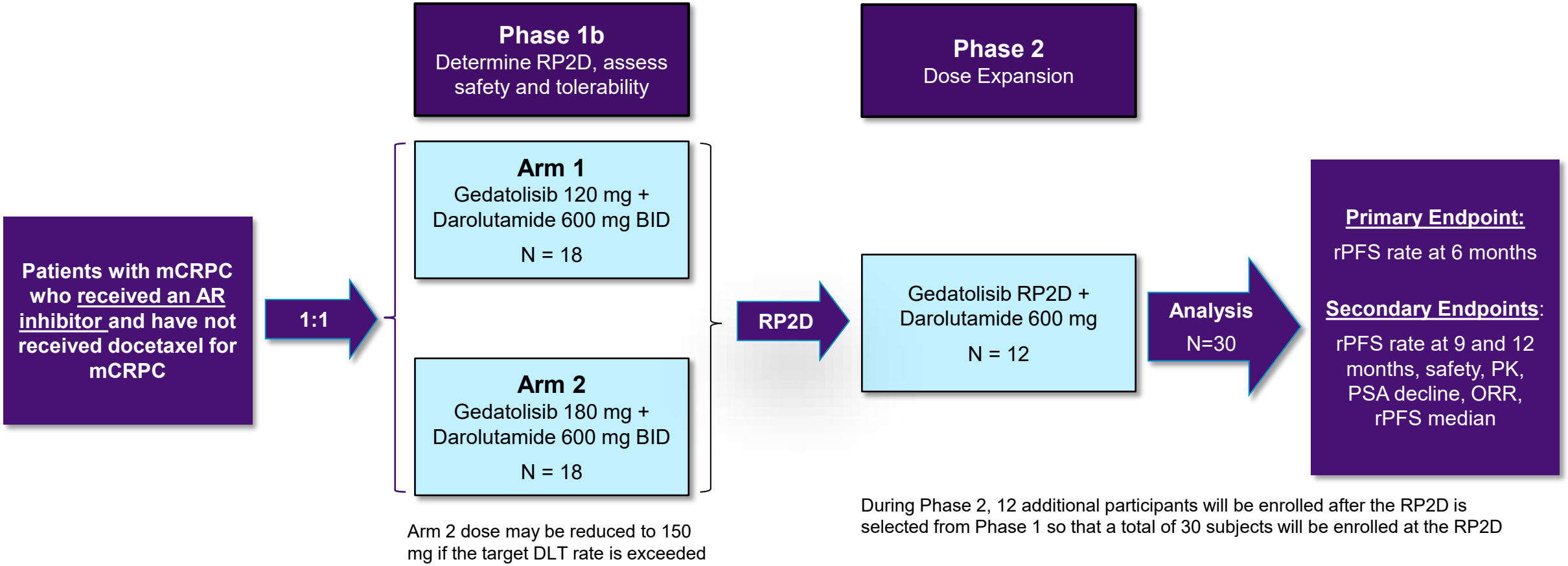
- Favorable clinical data in mCRPC with PAM inhibitors provides "proof-of-concept" of benefit of combining a PAM and AR inhibitor in 2L setting
- Gedatolisib's clinical results in breast cancer correlated with strong activity in nonclinical tumor models
- Gedatolisib exhibits similar potency and efficacy 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 combined with darolutamide, a potent next generation androgen receptor inhibitor



Enrolled first patient Q1 2024 and expect to announce initial data 1H 2025

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Clinical Trial Results Provide POC for PAM Inhibitors in 2L mCRPC post ARi

Less potent PAM inhibitors combined with AR an inhibitor reported favorable results

Study Regimens	Line of Therapy	Patient Population	N	Overall Results (Months rPFS)	Comments

Samotolisib (PI3K/mTOR) + Enzalutamide vs. Enzalutamide [1]	2nd Line prior abiraterone	All	129	**10.5 vs. 5.5 months** *(HR = 0.64; P = 0.03)*	▪ Samotolisib efficacious despite only modest PI3K-α and mTOR potency
					▪ Results in PTEN wild-type patients reflect benefit of mTOR inhibition
					▪ Gedatolisib vs. samotolisib [3]
					  ▪ 7X more potent overall; 100x for mTOR
					  ▪ More cytotoxic
					▪ Drug is not under active development

		AR-v7-negative	103	**13.2 vs. 5.3 months** *(HR = 0.52; P = 0.03)*	
		PTEN wild-type	60	**13.2 vs. 3.6 months** *(HR = 0.49; P = 0.07)*	
Ipatasertib (AKT) + Abiraterone vs. Abiraterone [2]	1st Line	All	1101	**19.2 vs. 16.6 months** *(HR = 0.84; P = 0.04)*	▪ Efficacy limited to PTEN loss patients ▪ Limited response in PTEN functional patients demonstrates role mTOR plays as resistance mechanism to AKT inhibition
		PTEN loss by NGS	209	**19.1 vs. 14.2 months** *(HR = 0.65; P = 0.02)*	

Karim Fizazi, MD, PhD
Gustave Roussy

(1) Sweeney Clin Cancer Res 2022; (2) De Bono, Lancet, 2021; (3) Sen, ASCO-GU, 2023

Additional Early Phase Clinical Data

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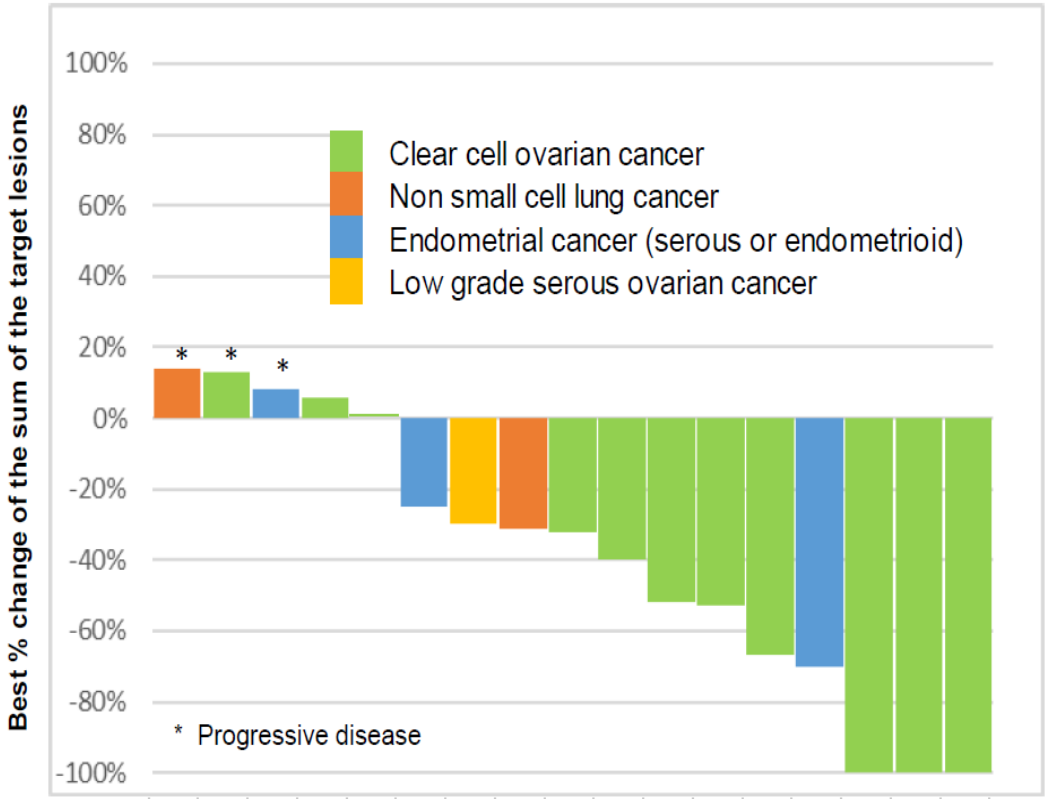
Gedatolisib + Paclitaxel + Carboplatin in Patients with Solid Tumors (N=17)[1]

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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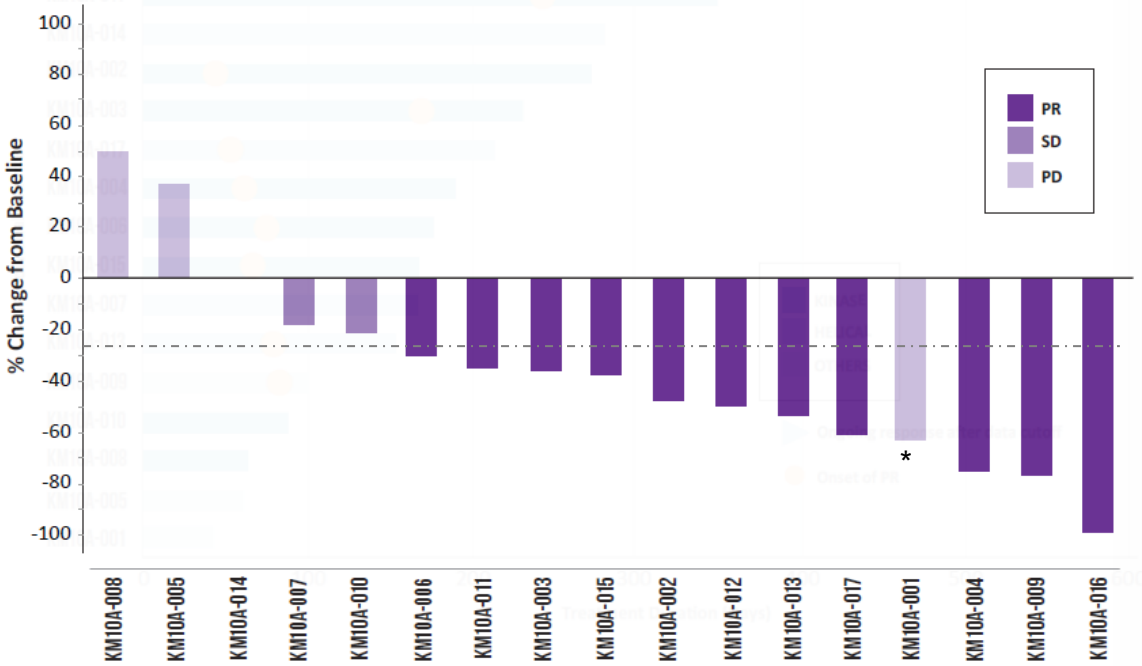
(1) Columbo 2021 CCR

Gedatolisib + Trastuzumab Biosimilar in 3L+ HER2+ ABC Patients (N=17)

59% ORR and 83% clinical benefit rate

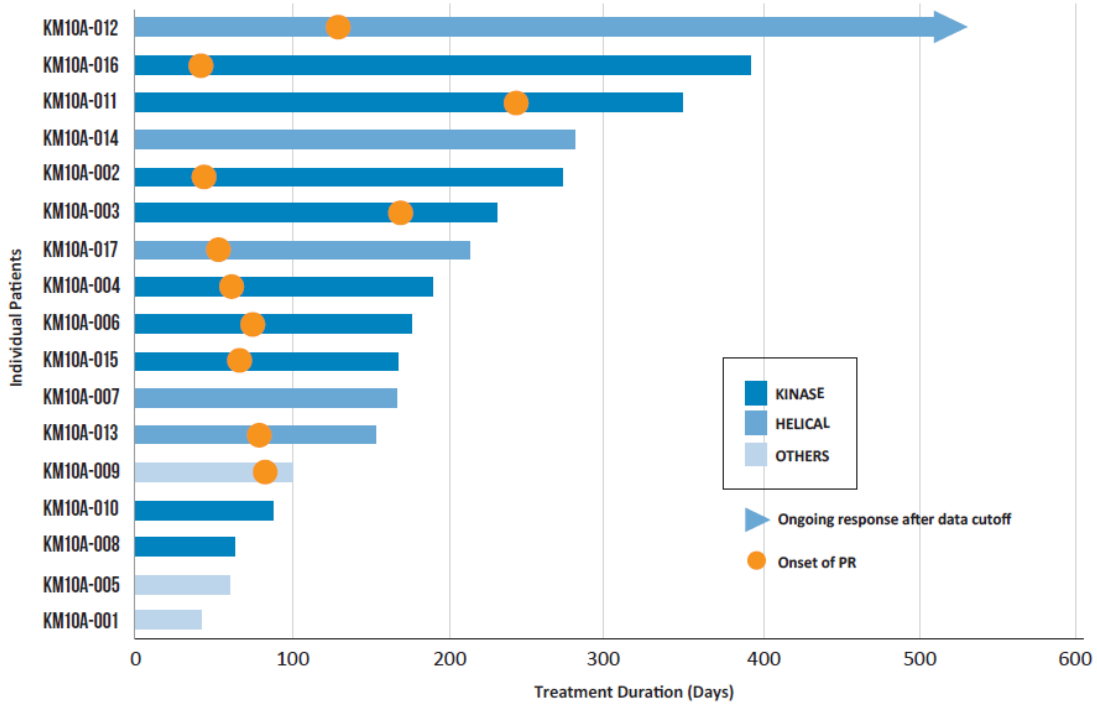
Best Response



* Target lesion decreased by 63% but a new leptomeningeal seeding occurred.

- 10 of 17 (59%) showed partial response (PR)
- 4 of 17 (24%) had stable disease (SD)

Duration of Response



- Median duration of response 7.1 months

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Kim 2022 SABCS. Note: Data presented is from an interim analysis of data as of a cutoff date of October 30, 2022, representing a database snapshot, and may change based on ongoing routine data monitoring and enrollment.

Leading cancer KOLs are participating in our research

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Co-Founder

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The Celcuity Opportunity

Significant untapped potential to effectively treat PAM pathway involved cancers

1

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

2

- **Very compelling data in 1L (mPFS 48 months) and 2L (mPFS 12.9 months) patients with HR+/HER2- ABC**
- A Phase 3 study in 2L patients is enrolling and a Phase 3 study in 1L patients is expected to begin enrolling in Q2 2025

3

- **Strong scientific rationale to develop gedatolisib for prostate cancer indications**
- Parallels between breast and prostate cancer – interdependent activity between PAM pathway and hormonal pathways

4

- **Uniquely positioned to advance multiple potential blockbuster indications in breast and prostate cancer**
- Cash, cash equivalents, and short-term investments of $235M as of Q4 2024 expected to fund operations through 2026

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