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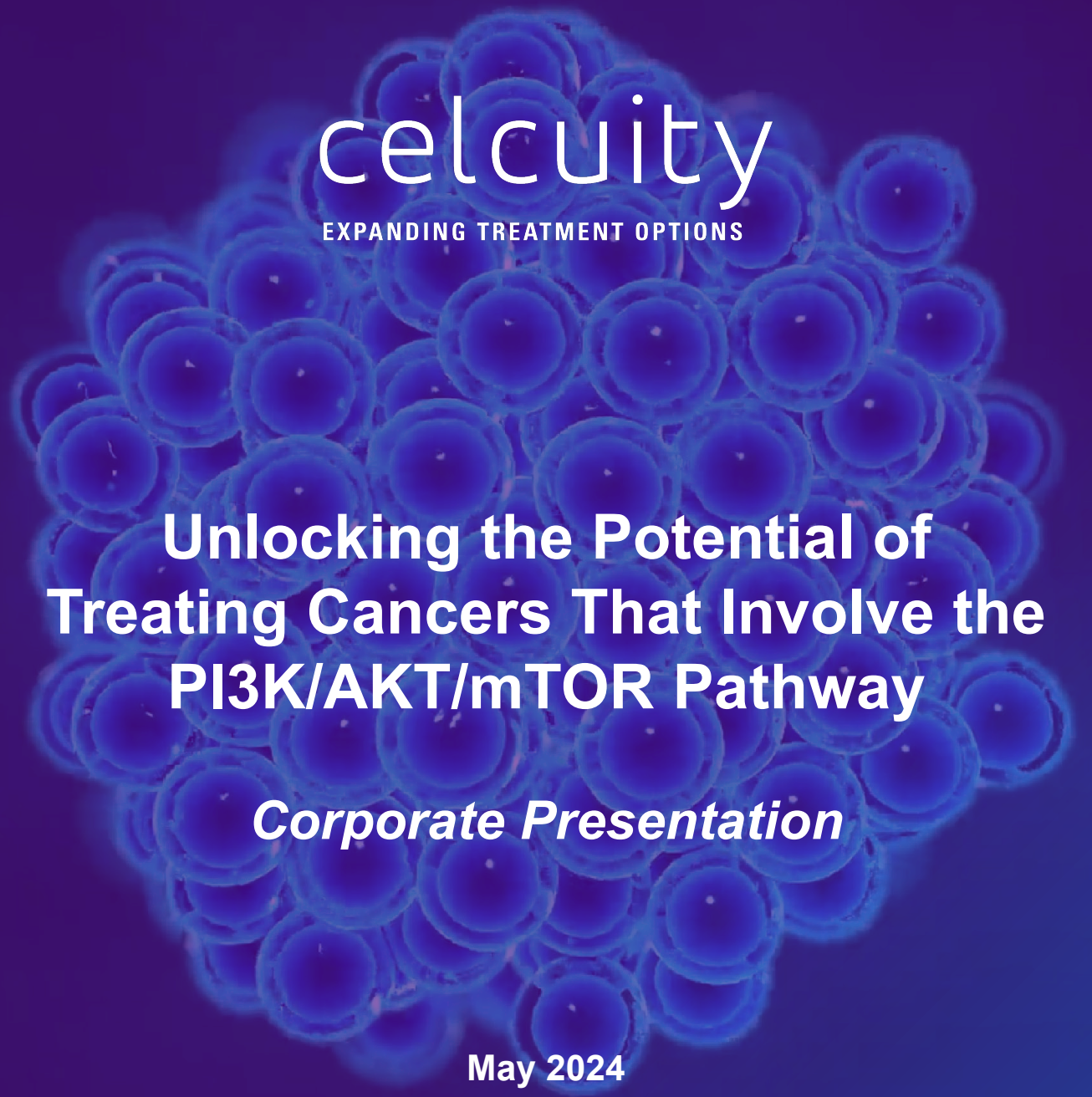
EXPANDING TREATMENT OPTIONS



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

Corporate Presentation

May 2024

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 clinical trial 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, 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**
- Potential to replace currently available standard-of-care

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 of $178M as of Q1 2024 expected to fund operations through data readouts in ABC and mCRPC

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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;

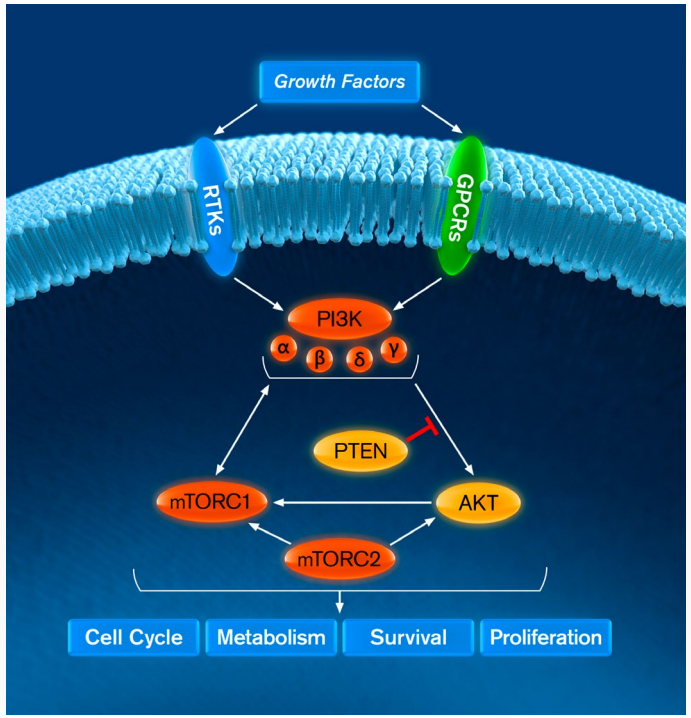
Difficult to Safely and Efficaciously Inhibit PI3K/mTOR

Maximum efficacy requires inhibition of all Class I PI3K isoforms and mTORC1 and mTORC2

Multiple pathway components must be targeted

Feedforward and feedback loops between PI3K isoforms, AKT, and mTOR cross-activates uninhibited sub-units

Induces compensatory resistance that reduces efficacy



Therapeutic window for oral PI3K/mTOR inhibitors is narrow

Difficult to optimize pathway inhibition without inducing undue toxicity

Orally administrated pan-PI3K or pan-PI3K/mTOR inhibitors induced unacceptable toxicity

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 Two drugs approved	*Today*	Need safe, potent pan-PI3K/mTORi

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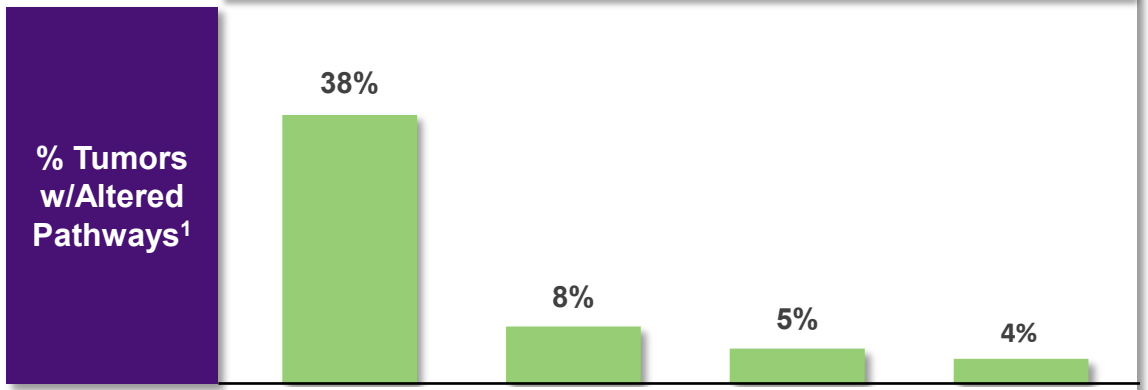
The PAM Pathway is the Most Underdeveloped Target in Solid Tumors

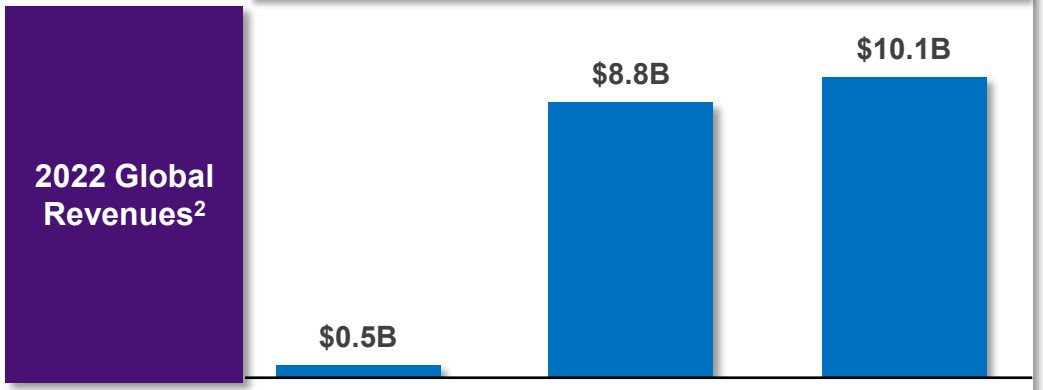
PAM is the most frequently altered pathway in solid tumors



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	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	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 PI3K/mTOR Inhibitor

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

Highly Differentiated Mechanism

- Inhibits all PI3K/mTOR nodes at **low or sub-nanomolar** concentrations
- **More potent & cytotoxic** than other PAM inhibitors being developed for breast or prostate cancer

Compelling Efficacy

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

Well-Tolerated

- Nominal Grade 3, no Gr 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]

Addressing Large Patient Populations

- **Breast Cancer**: Enrolling Phase 3 trial for 2L patients with HR+/HER2- ABC
- **Prostate Cancer**: Phase 1b/2 trial for 2L patients with mCRPC in Q1 '24
- **225,000 1L/2L patients** in US, 5EU, Japan[3]

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(1) Combined data from treatment-naïve patients enrolled in Escalation Arm A and Expansion Arm A of the B2151009 Phase 1b clinical trial (Rugo 2023); (2) Data from Expansion Arm D of the B2151009 clinical trial (Layman, Lancet Oncol, 2024); includes 2 unconfirmed partial responses; (3) 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]

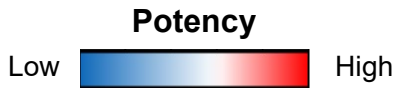
Node	Gedatolisib[2]	Alpelisib[3]	Everolimus[4]	Capivasertib[5]
PI3K-α	**0.6**	~4.0	-	-
PI3K-β	**6.0**	1,156	-	-
PI3K-γ	**5.4**	250	-	-
PI3K-δ	**6.0**	290	-	-
mTORC1	**1.6**	-	~2.0	-
mTORC2	**1.6**	-	-	-
AKT	-[6]	-	-	3.0

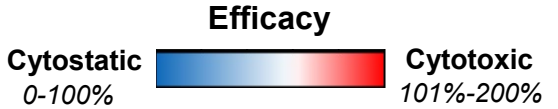
Gedatolisib is potent against all Class I PI3K isoforms & mTORC1/2

- 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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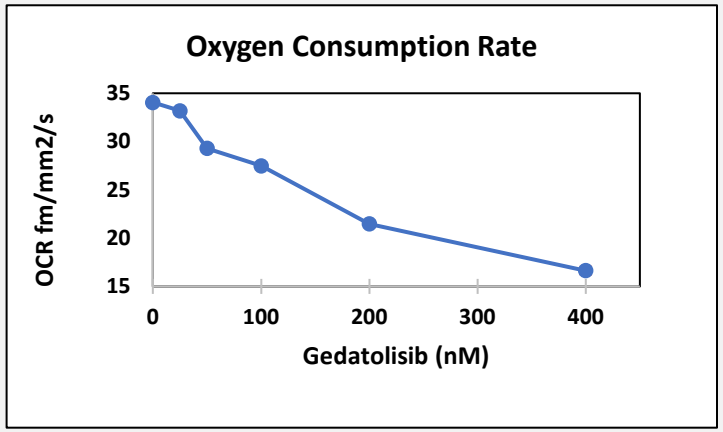
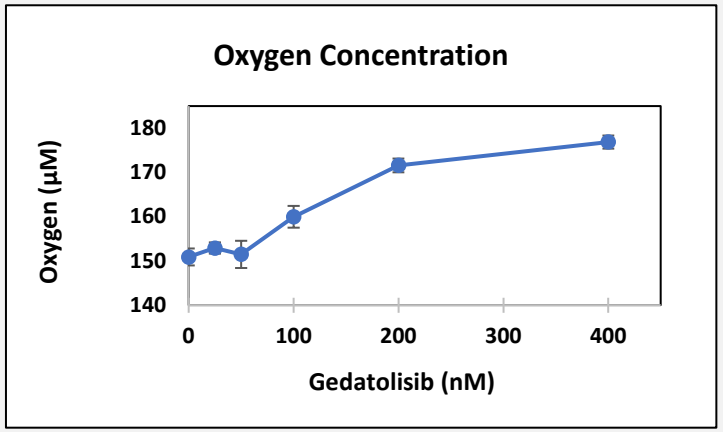
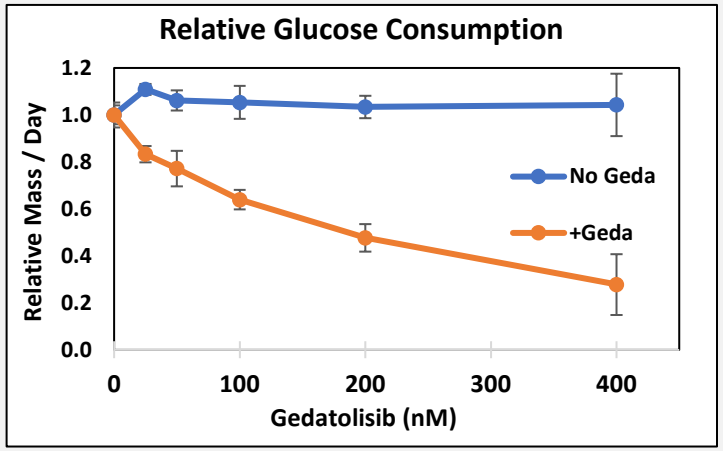
(1) IC50 derived from cell-free biochemical dose response analysis; (2) Venkatesan 2010 J Med Chem 53(6):2636-45. (3) Fritsch 2014, Mol Cancer Ther. 13(5):1117-29. (4) Schuler 1997; Transplantation, 64(1):36-42. (5) Davies 2012, Mol Cancer Ther 11(4):873-87; (6) Mallon 2011, Clin Cancer Res 17(10); (7) 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.

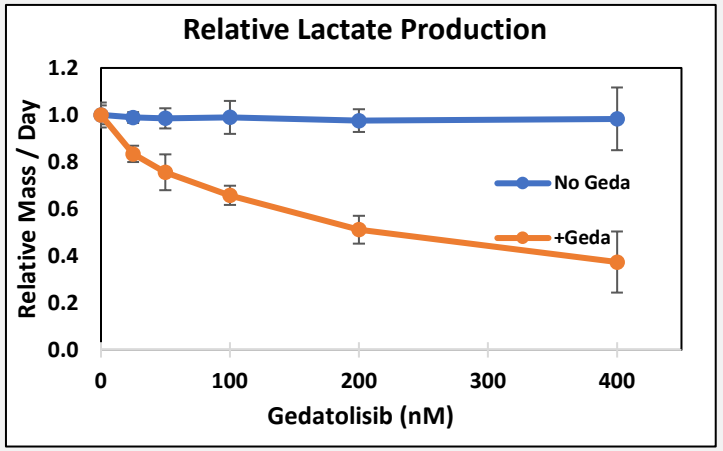
Gedatolisib Favorably Impacts Tumor Microenvironment

PAM inhibition decreases O2 and glucose consumption and lactate production









A TME with low O2, low glucose, and high lactate is correlated with immuno-suppression, low anti-tumor activity[1, 2,3,4]

Gedatolisib's dose-dependent reduction of O_2 consumption leads to an increase in O_2 available

Gedatolisib's dose-dependent reduction in glucose consumption leads to decreased lactate production

Data demonstrates Gedatolisib improves TME factors that enable anti-tumor immune function

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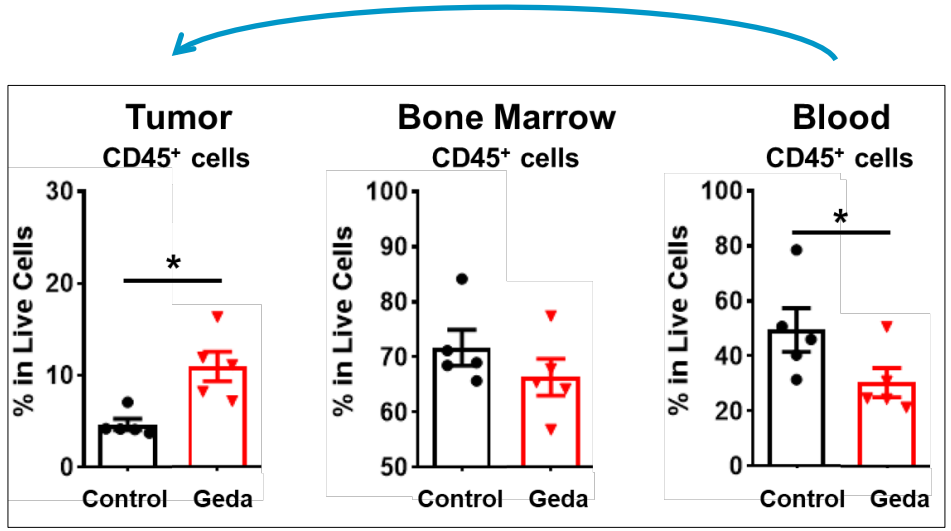
(1) DePeaux 2021, Nat Rev Immunol 21(12):785:797. (2) Jayaprakash 2018, J Clin Invest.128(11):5137-5149. (3) Semenza 2021 PHYSIOLOGY 36: 73-83. (4) Thomas 2023, Front. Oncol. 13:1063051

Gedatolisib Increases Immune Cell Tumor Infiltration and Activation

Profiled CD45+ immune cell populations in tumor, bone marrow, peripheral blood

Proportions of CD45+ anti-tumor immune cell subsets in tumor

	Day 10			Day 17		
	Control	Geda	P-Value	Control	Geda	P-Value
% CD45+	4.7	10.9	0.03	-	-	-
% DC (in CD45+)	9.0	15.4	0.0002	2.9	4.0	NA
% CD4+ (in CD45+)	8.6	19.6	0.0002	7.4	19.2	0.014
% CD8+ (in CD45+)	1.7	4.8	NA	13.6	24.5	0.02



Tumor infiltration likely resulted from recruitment of leukocytes from blood circulation into the TME

Desired immune cell types infiltrated into the tumor

- Gedatolisib increased CD45+ cells in tumors 2.3 fold vs control
- Gedatolisib induced durable infiltration of key anti-tumor immune cell types - DC, CD4+, CD8+

Immune cells that infiltrated are activated

- Gedatolisib induced a 1.5-2 fold increase of activated CD8+ cytotoxic T cells (CD69+) and activated NK cells (CD69+) in tumors at day 10 and day 17

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Source: Yan, C.et al. Int. J. Mol. Sci. 2021, 22, 5207.
Abbreviations; DC = dendritic cell; NA = not available

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 study will prescribe prophylaxis
- **Low incidence of Grade 3 hyperglycemia (2%)**
- **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	2
Vomiting	19	2	2
Asthenia	7	12	2
Fatigue	19	2	-
Appetite decrease	14	7	-

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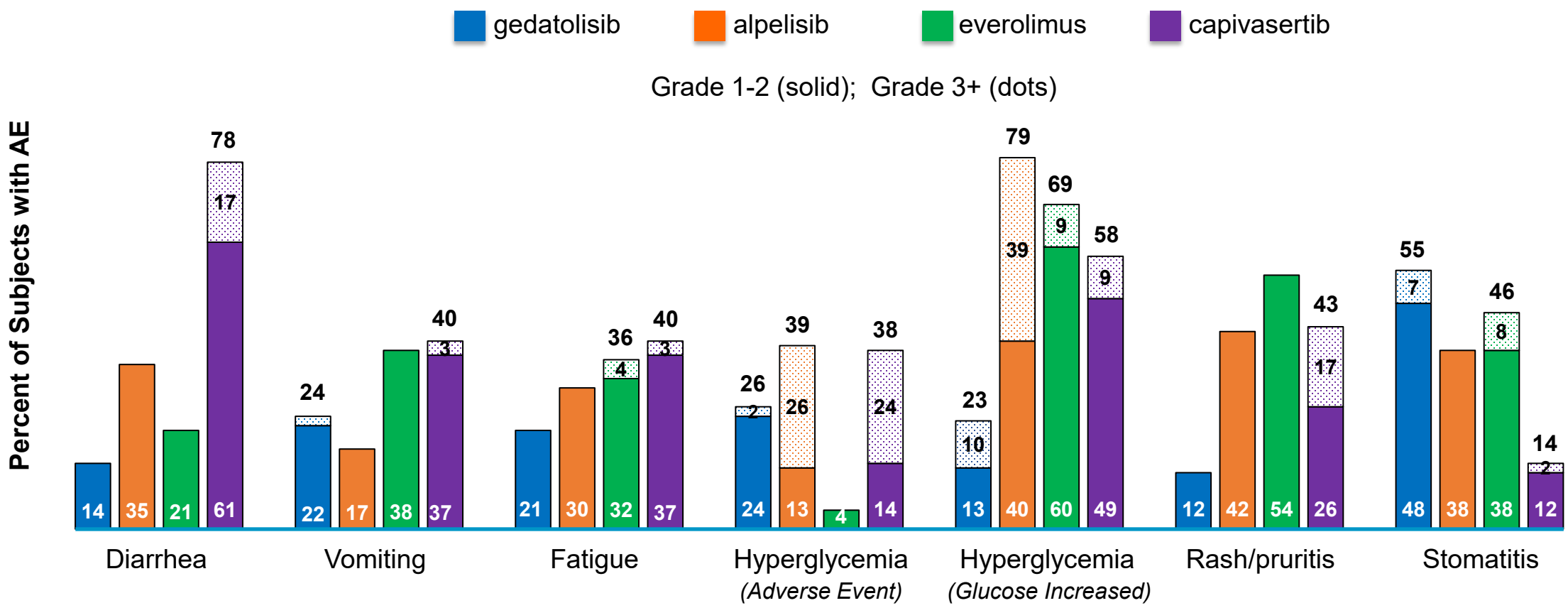
Source: (1) Shapiro 2015; (2) Rugo 2017; TEAE = treatment emergent adverse events; AE = adverse event.

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

2nd Line HR+/HER2- Advanced Breast Cancer

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

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

2nd Line Metastatic Castration Resistant Prostate Cancer

Phase 1b/2 clinical trial for gedatolisib with darolutamide is enrolling

- Extensive literature describes androgen pathway linkage to the PI3K/AKT/mTOR (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 has been reported in prostate cancer trials[3]

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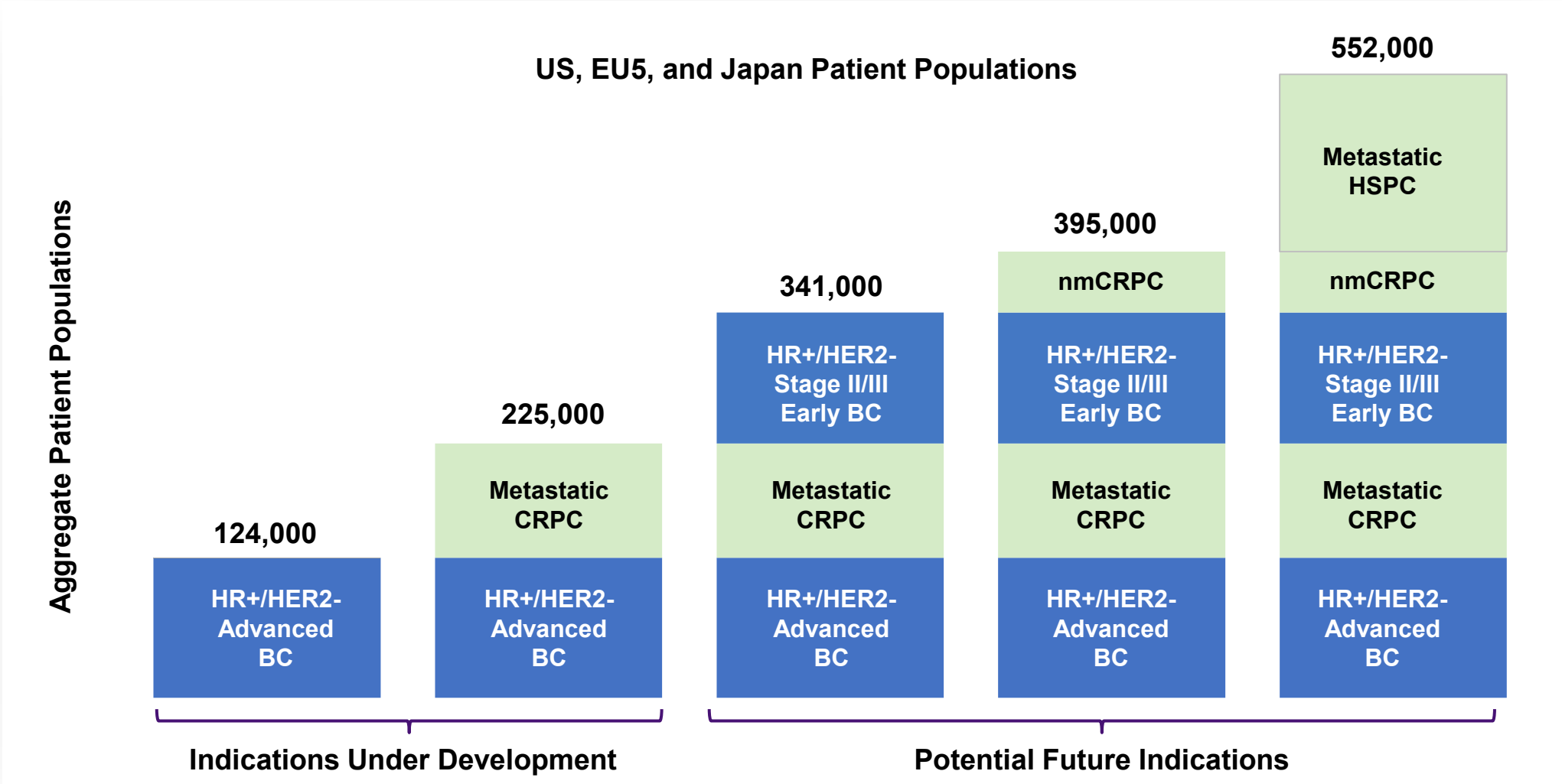
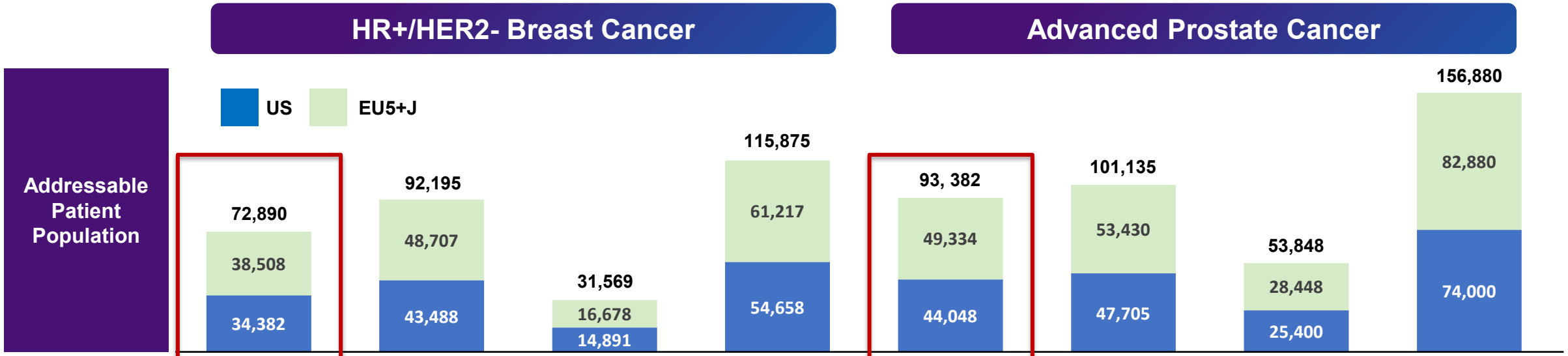
(1) NCT05501886; (2) Sen et al., Therapeutic effect of gedatolisib on prostate cancer models differing in PI3K or PTEN mutational status, ASCO GU 2023; (3) Sweeny et al., Phase Ib/II study of enzalutamide with samotolisib in mCRPC, CCR 2022

Addressable Patient Population in Breast and Prostate Cancer



Sources: 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 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	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%
Market Opportunity	~$5-$6B	~$10B+	~$3B	$6-$8B	$8B+	$10B+	$6-$8B	$10B+

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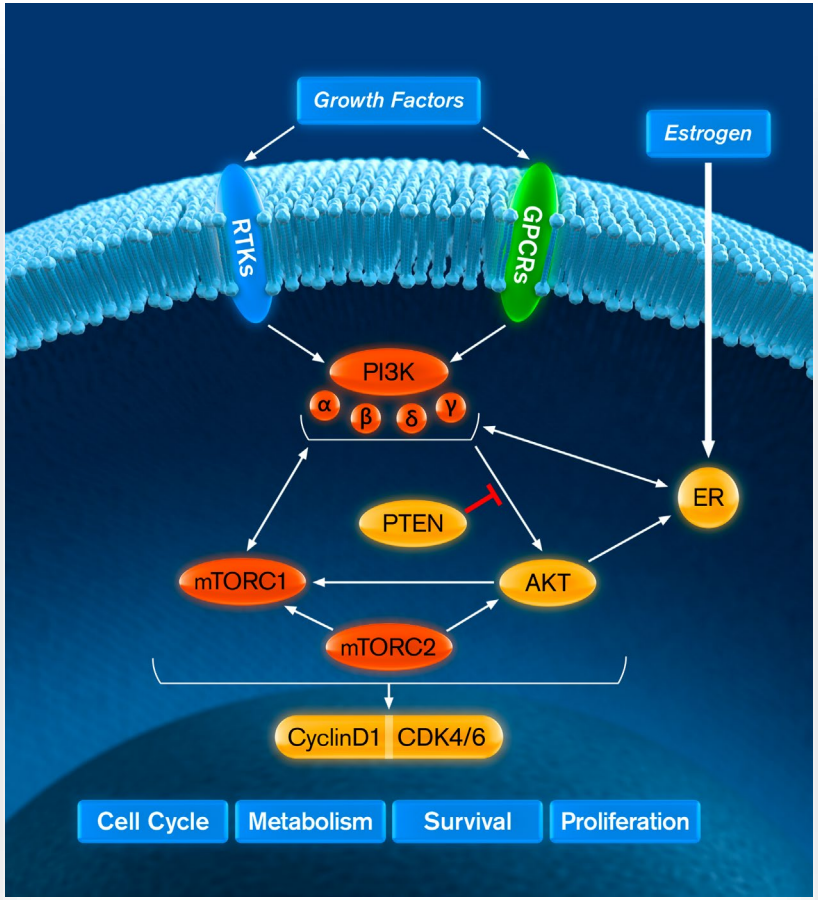
ER, CDK4/6, & PI3K/mTOR are Interdependent Drivers of HR+/HER2- ABC

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

ER and PI3K/mTOR

- Activation of the PI3K/mTOR pathway induces estrogen independent ER transcriptional activity by mTOR
- Conversely, ER target gene expression activates upstream effectors of the PI3K/mTOR pathway
- ER also activates the PI3K/mTOR pathway by direct binding to PI3Kα
- **PI3K/mTOR inhibition increases ER activity which increases sensitivity to endocrine therapy**



CDK4/6, ER and PI3K/mTOR

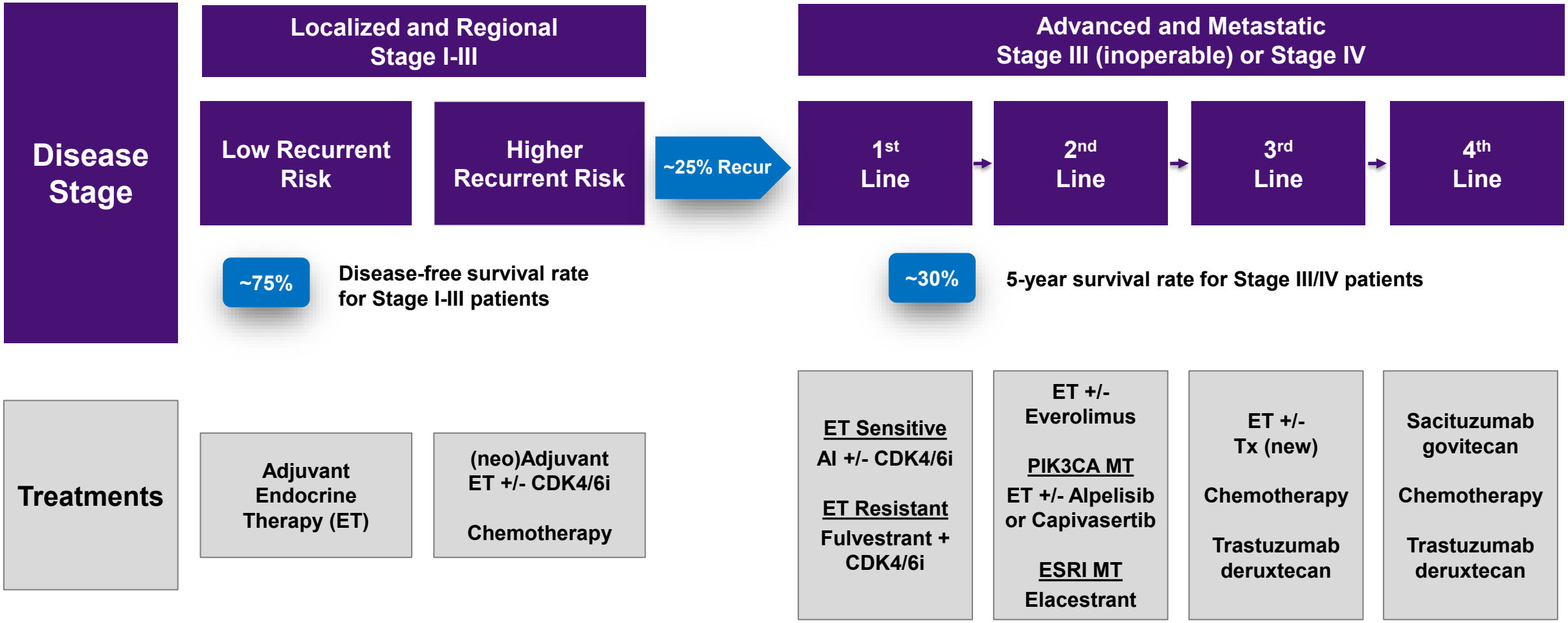
- Estrogen promotes cyclin D1 transcription and cyclin D1 can cause estrogen independent transcription
- Provides rationale for simultaneously inhibiting ER and CDK4/6
- CDK4/6 inhibition causes incomplete cell cycle arrest – addition of PI3K/mTOR inhibition enables more complete arrest
- **PI3K/mTOR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

Alves, Int J Mol. Sci. 2023

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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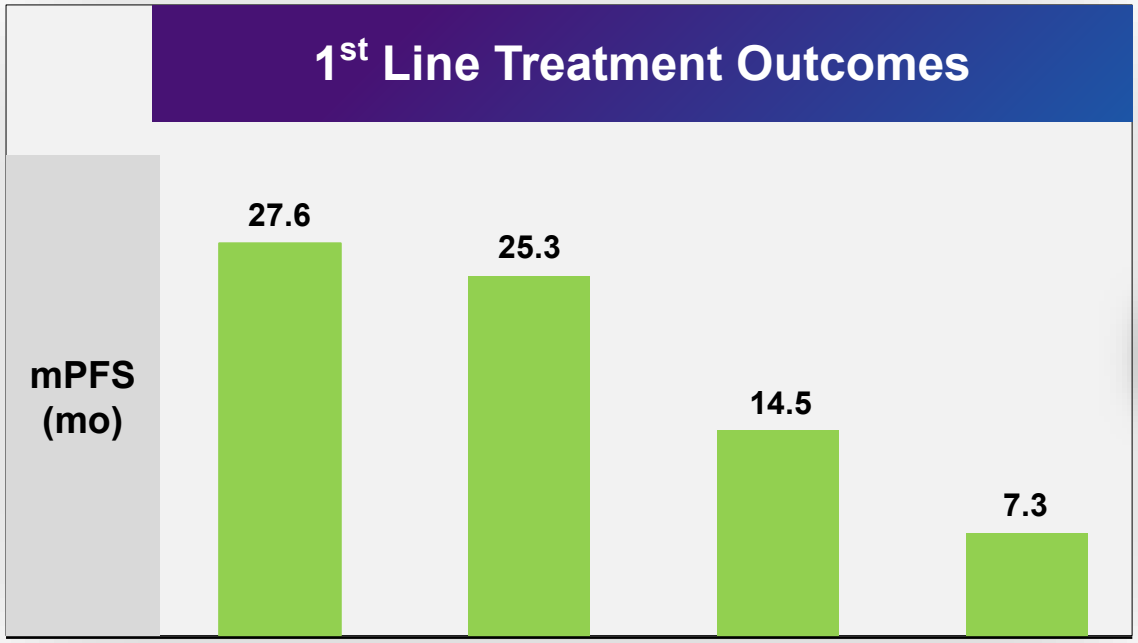
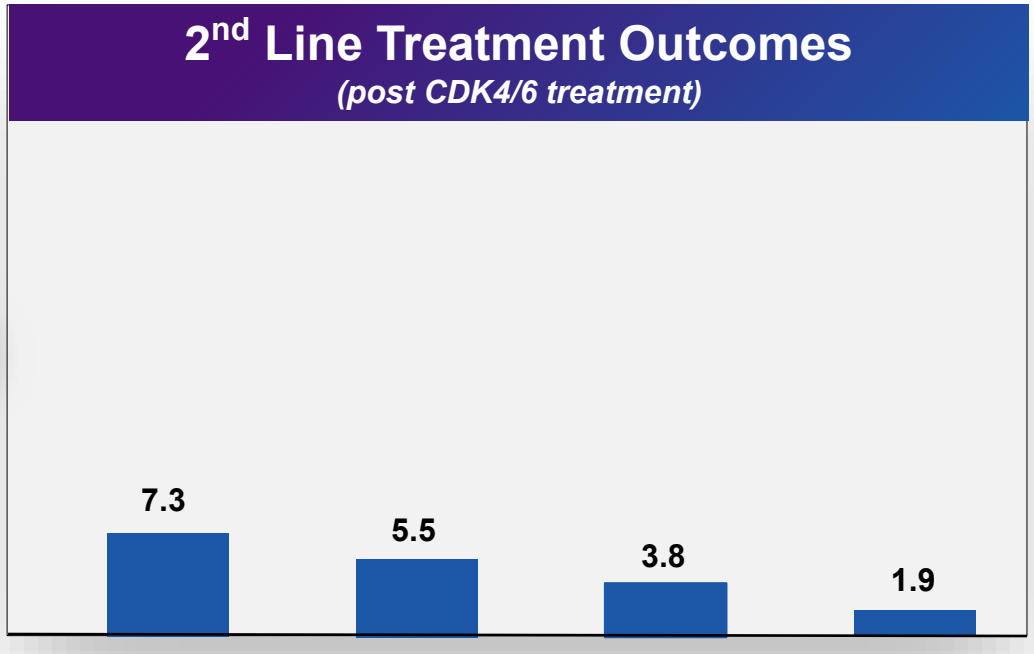
EXPANDING TREATMENT OPTIONS

(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	1.9
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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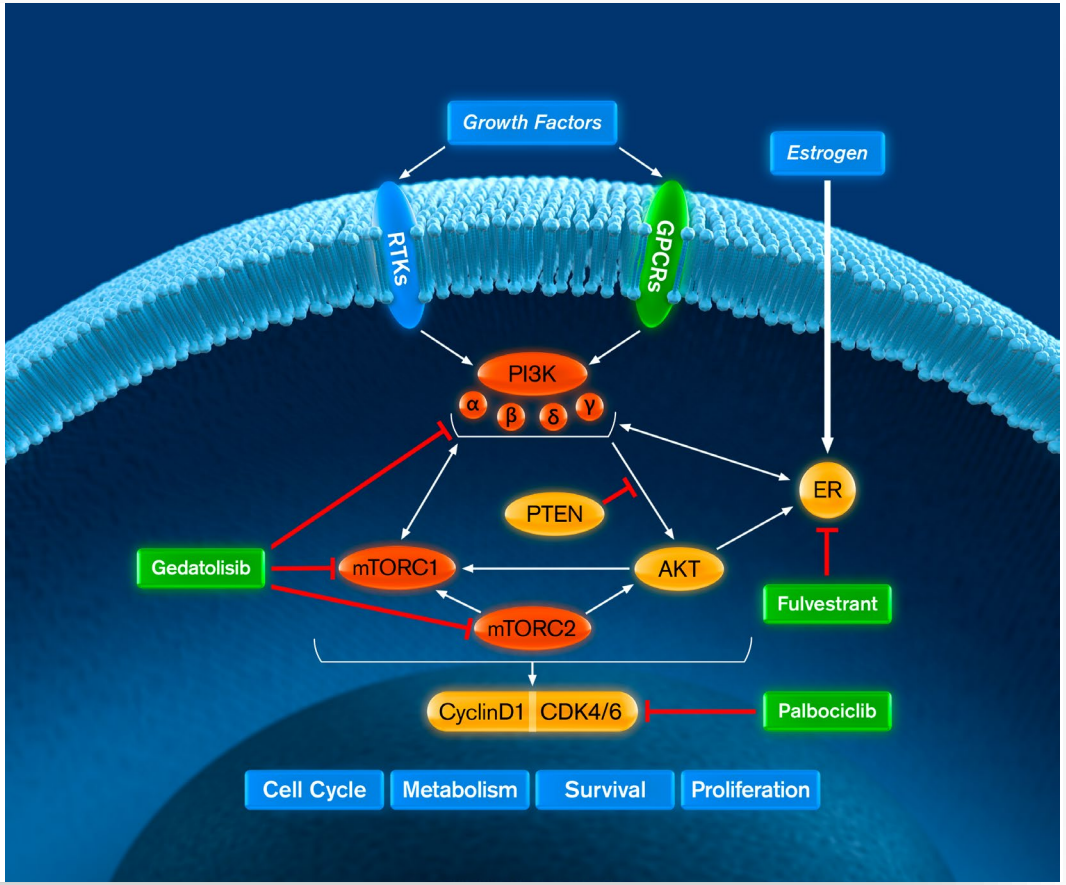
Treatment Strategy: Simultaneous Blockade of PAM, ER, & CDK4/6 Pathways

Treatment Rationale

- Blockade of interdependent ER, PI3K, mTOR & CDK signaling pathways is required to optimize anti-tumor control
- PAM inhibition:
  - Blockades pathway and limits 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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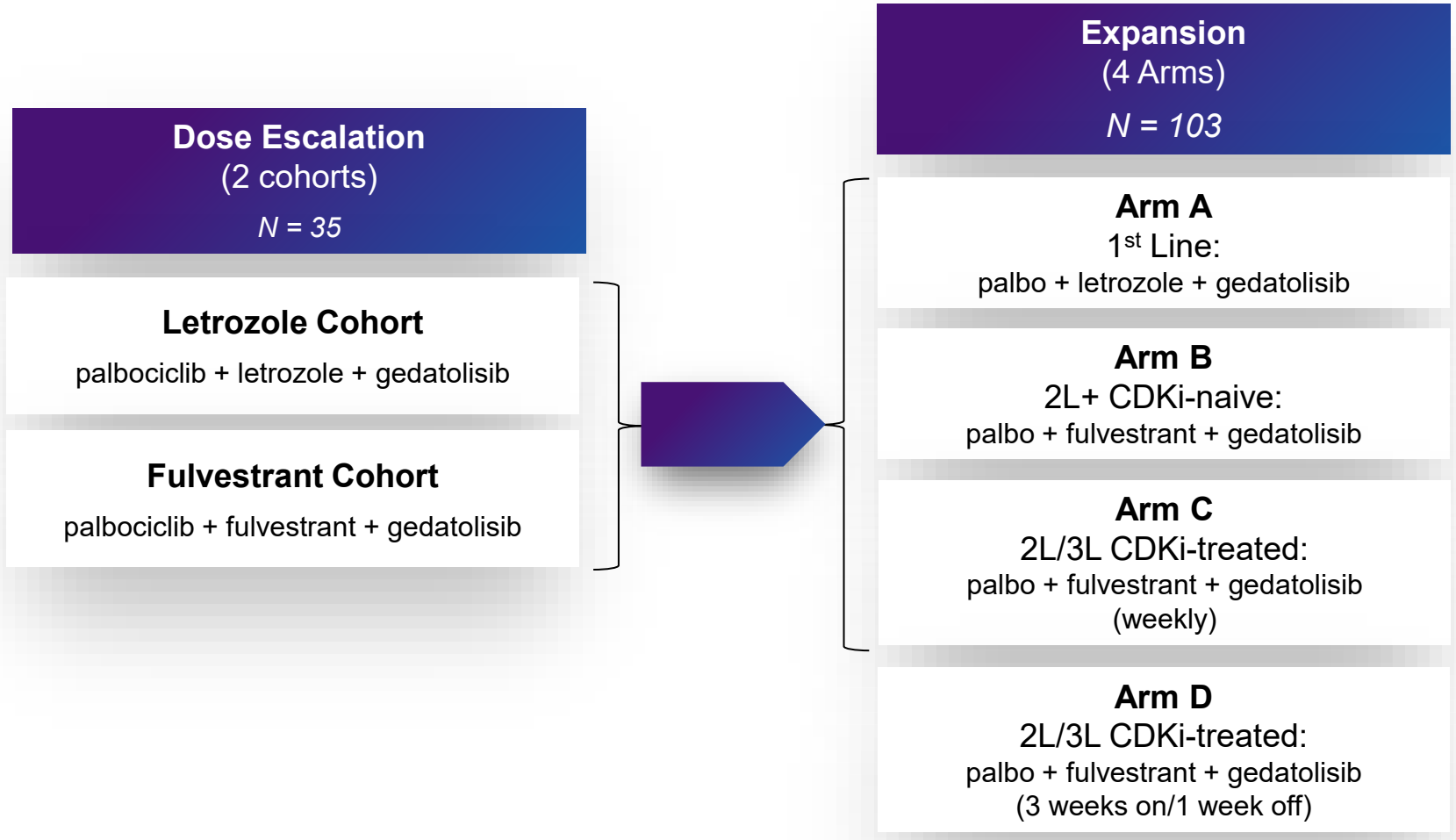
B2151009: Phase 1b Study (138 patients)

Provided Data in Treatment Naïve and Prior CDK4/6 Treated Patients with HR+/HER2- ABC



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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. Source: Layman, Lancet, 2024

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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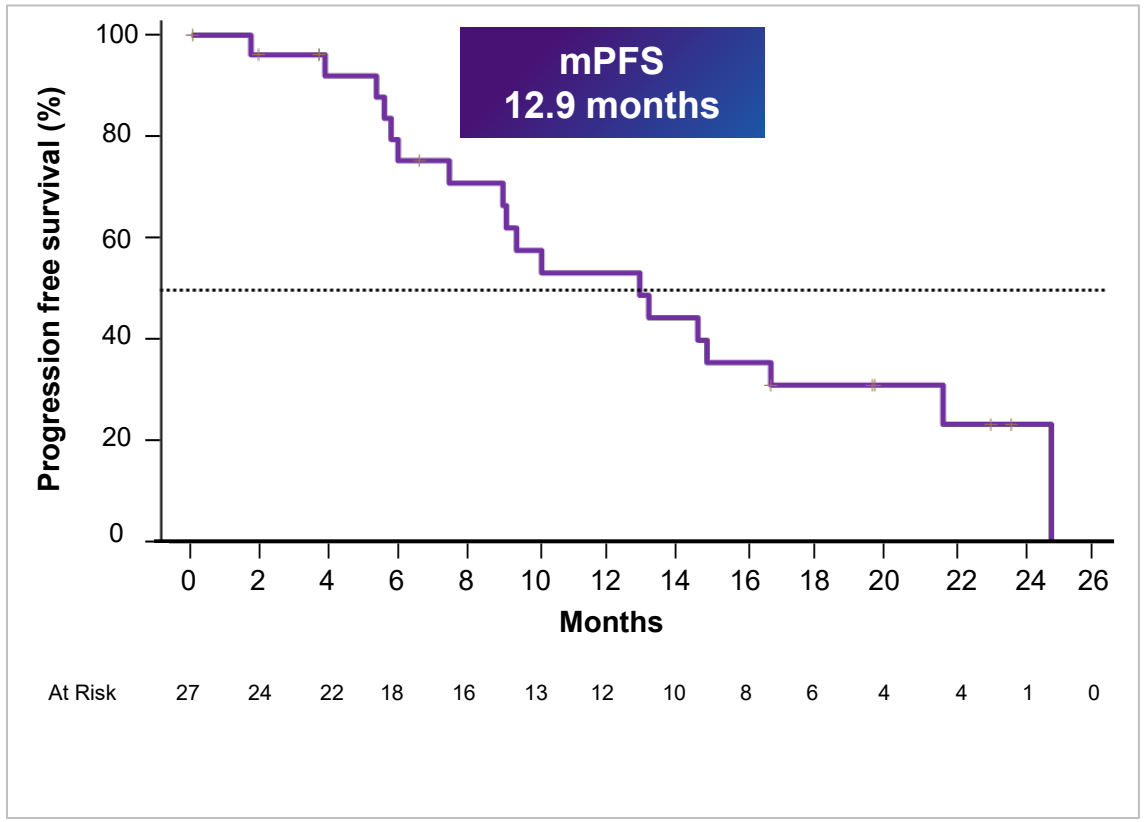
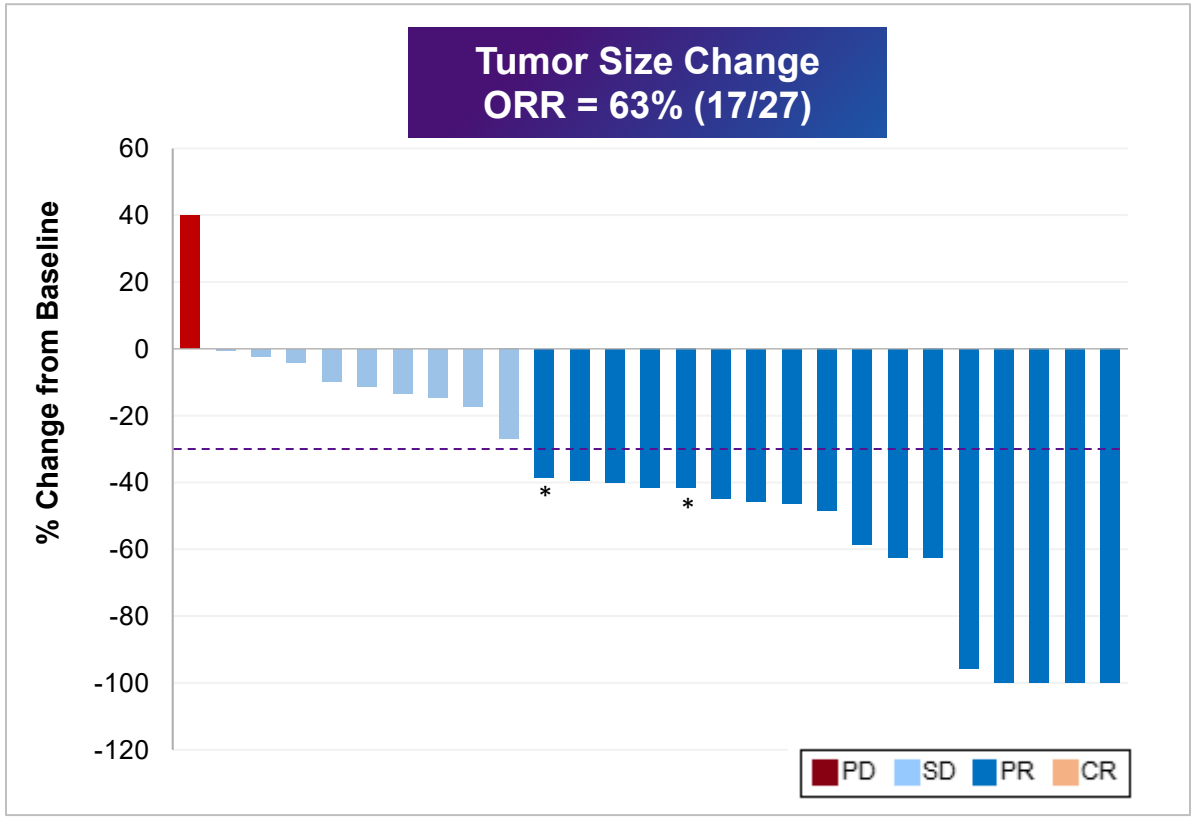
Source: Layman, Lancet, 2024; Rugo 2023 ESMO-Breast. Footnotes: (1) Response evaluable analysis set per RECIST v1.1 including uPR (n=2, Arm B; n=3, Arm C; n=2, Arm D); (2) full analysis set, mPFS updated with data cutoff 29-May-2023; (3) Baseline *PIK3CA* mutation status missing for one patient. Abbreviations: 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

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





Source: Layman, Lancet, 2024, Arm D data from B2151009 study. ORR includes 2 unconfirmed PRs; *unconfirmed PR. Data presented is from a data analysis cutoff as of June 29, 2022

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)[9]
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 (SOLAR-1 study); (2) USPI Everolimus (BOLERO-2 study); (3) USPI Capivasertib (CAPItello-291 study); (4) Rugo 2017 (SWISH study); (5) Stomatitis category includes mucositis; (6) Neutropenia includes neutrophil count decrease; (7) Leukopenia includes white blood cell decrease; (8) Anemia includes hemoglobin decrease; (9) Layman, Lancet, 2024. Abbreviations: G = gedatolisib; P = palbociclib; F = fulvestrant; TEAE = treatment emergent adverse events; AE = adverse event

Gedatolisib Combo vs. SOC 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 7.3 months ORR 17%
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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(1) Layman, Lancet, 2024, Arm D; (2) Rugo, Lancet Onco, 2021; (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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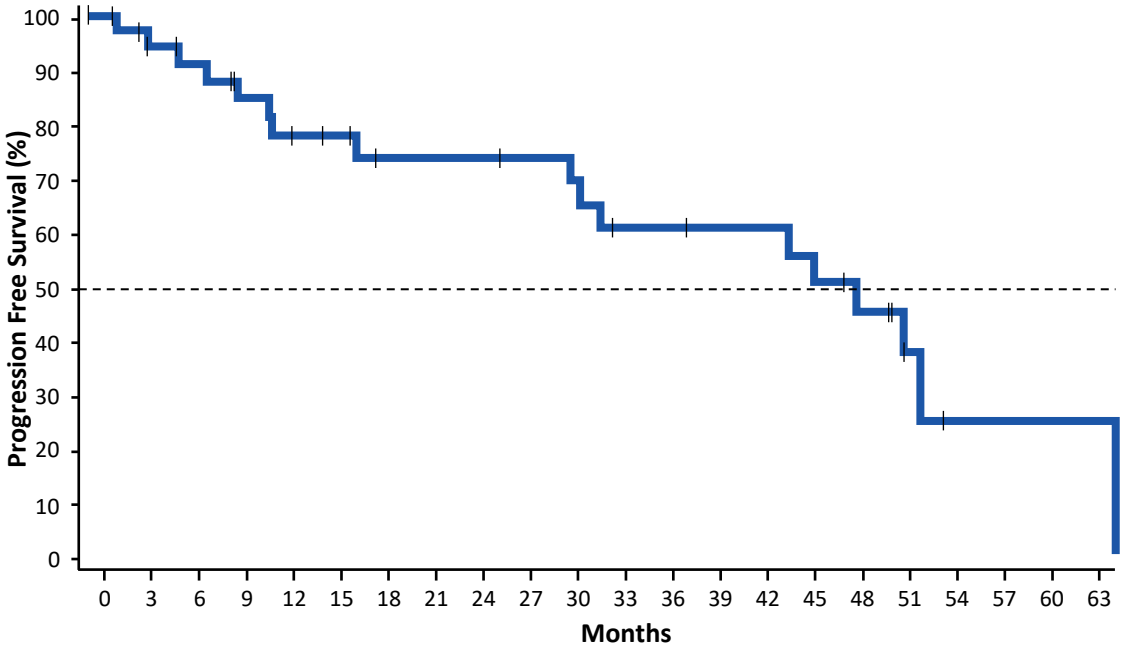
Source: Rugo 2023 ESMO Breast. (1) Subjects with measurable disease in response evaluable analysis set per RECIST v1.1;(2) Confirmed responders in the full analysis set. Abbreviations: CR, complete response; DOR, duration of response; mos, months; NR, Not Reached; ORR, objective response rate; PD, progressive disease; PFS, progression free survival; PR, partial response; SD, stable disease

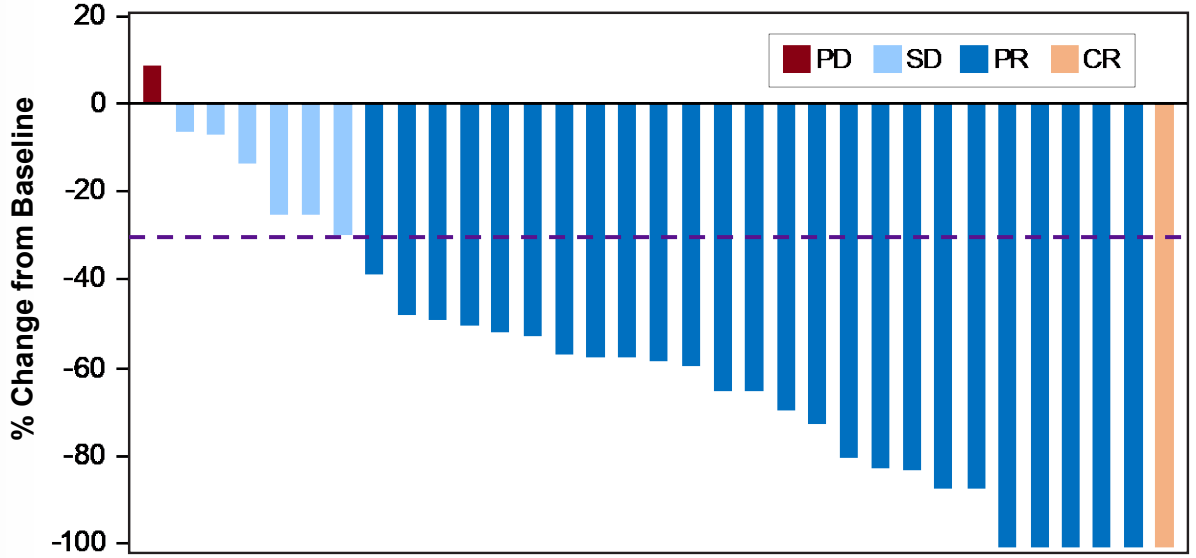
Gedatolisib + Palbociclib + Letrozole in 1st Line HR+/HER2- ABC (N=41)[1]

Combined 1L data from Esc Arm A + Exp Arm A compares favorably to published data for SOC palbociclib + letrozole[2]

mPFS
48.6 Months



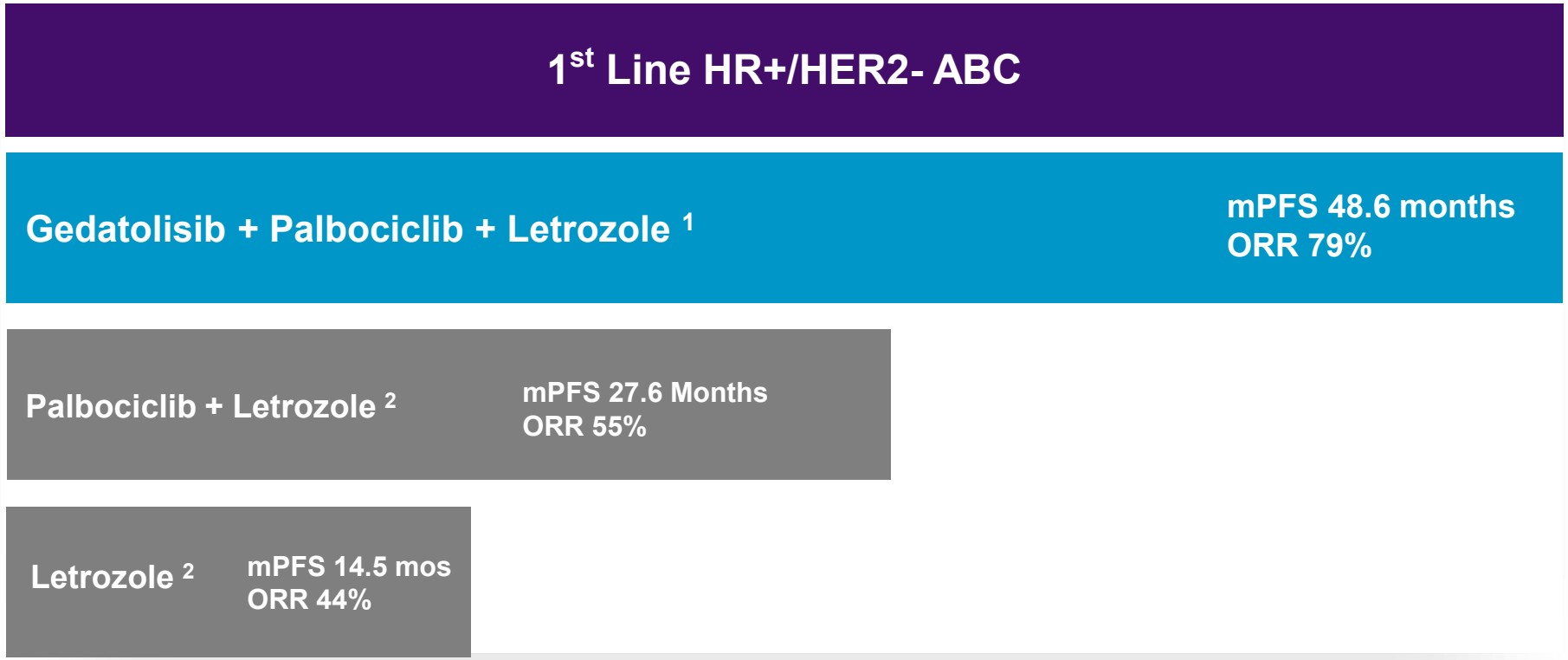
Tumor Size Change
ORR = 79% (26/33)



(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. (c) Data presented is from data analysis as of a cutoff date of June 29, 2022.

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

Gedatolisib Combo Offers Potential for Superior Efficacy 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.

Phase 3 Study Design
VIKTORIA-1

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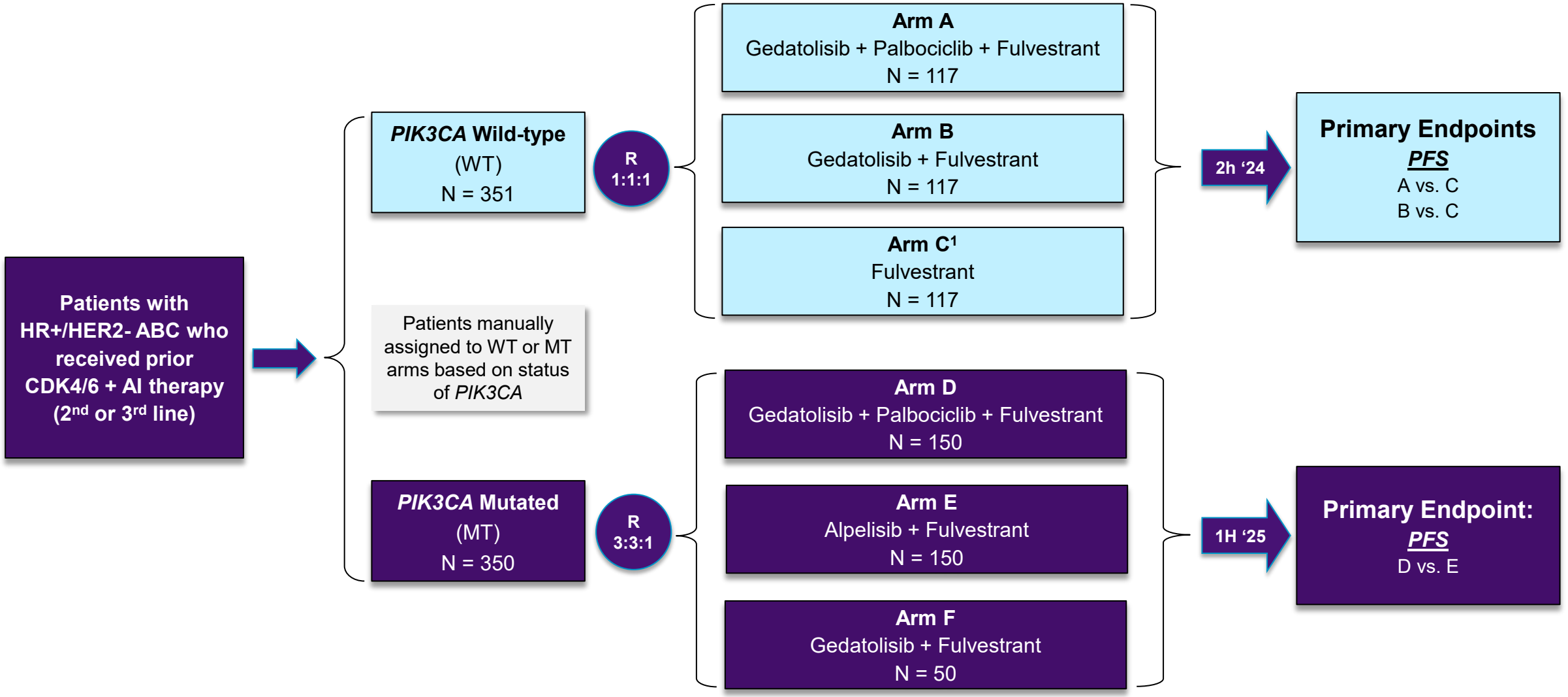
Pivotal Trial Design Considerations for 2nd Line HR+/HER2- ABC

- Standard-of-care 2nd line treatment is based on *PIK3CA* status
- ~35% 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 Pivotal Phase 3 Trial Design Overview



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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 prior CDK4/6i

	Gedatolisib + Palbociclib + Fulvestrant N=27[1,2]	Fulvestrant N=165[3]	Fulvestrant N=52[5]	Alpelisib + Fulvestrant N=126[6]	Alpelisib + Fulvestrant N=121[7]
***PIK3CA* Status**	WT / M (56% / 41%)	WT	WT / MT (70% / 30%)	M	M
Line of Therapy (% by line)	2L / 3L+ (67% / 33%)	2L / 3L+ (73%/27%)[4]	2L / 3L+ (83% / 17%)	2L / 3L+ (37%/ 63%)	1L / 2L/ 3L+ (12% / 70% / 19%)
mPFS (months)	12.9	1.9	1.9	5.6	7.3
ORR	63% (overall)[2] WT 60% M 73%	NR	6%	22%	17%
PFS % at 12 months	53% (overall) WT 49% M 60%	10%	12%	22%	27%

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Sources: (1) Wesolowski, SABCS, 2022; (2) Includes 2 unconfirmed PR.(3) Bidard 2022 – 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) Lindeman 2021, VERONICA trial; (6) Rugo 2021 SABCS (7) Rugo 2021 Lancet. Note: No head-to-head trials have been conducted; data collected from different trials, in different patient populations and may not be comparable. Data presented for gedatolisib is from a preliminary data analysis as of a cutoff date of June 29, 2022.

VIKTORIA-1 Pivotal Study Features

- **Global open-label randomized study**
- **Key eligibility criteria:**
  - Any *PIK3CA* status
  - Prior CDK4/6i + NSAI
  - ≤ 2 prior endocrine therapy
  - No prior chemotherapy for ABC
  - Bone only patients with measurable lytic lesion
- **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 and shows contribution of gedatolisib**
- **Stratification by geography, prior treatment response (≤ or > 6 months), presence of liver or lung metastasis (yes/no)**

Supports indications for **gedatolisib and fulvestrant with or without palbociclib as second or third treatment for patients with HR+/HER2- advanced or metastatic breast cancer** who have progressed on prior treatment with a CDK4/6 therapy in combination with AI

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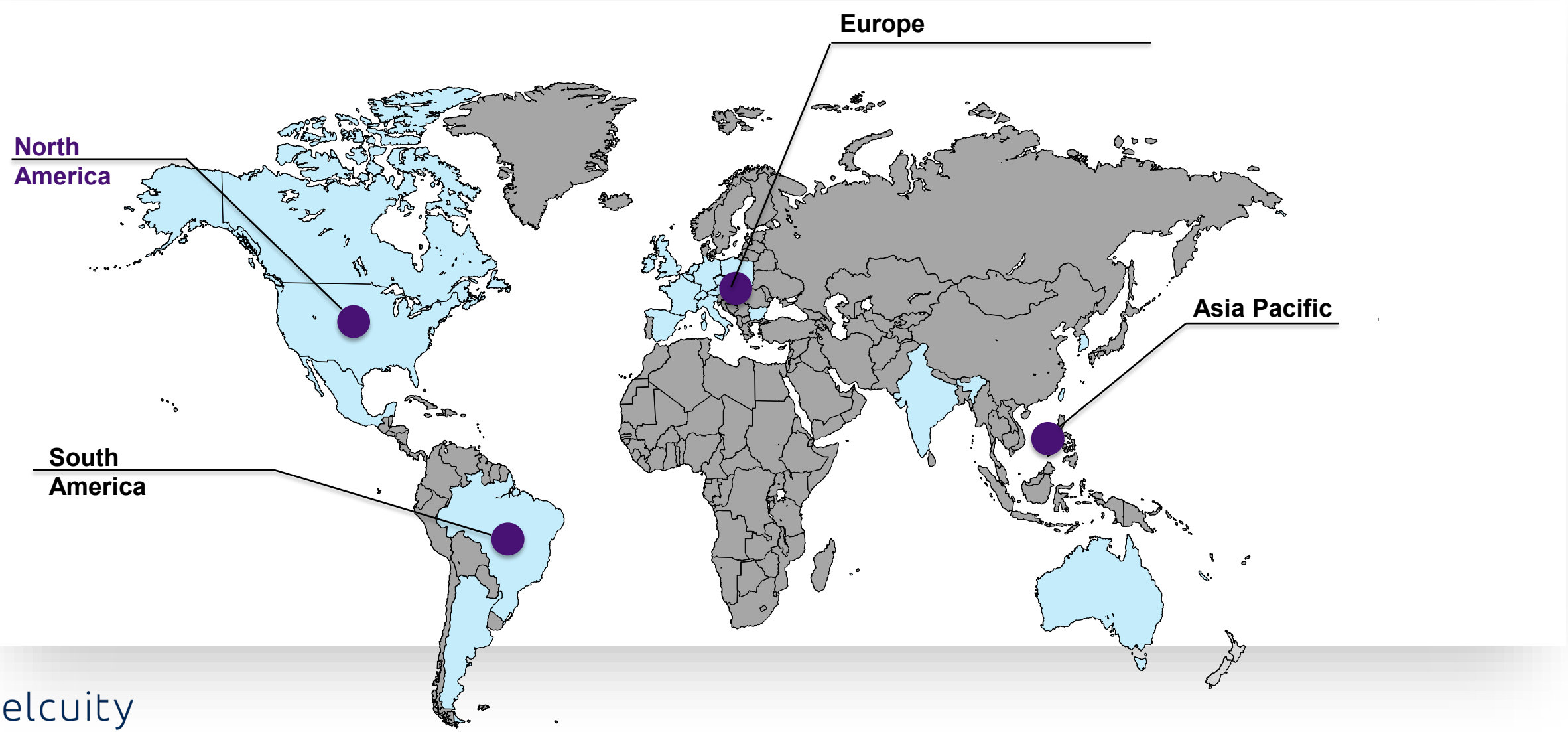
200+ Sites Across 20 Countries



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Gedatolisib for Prostate Cancer

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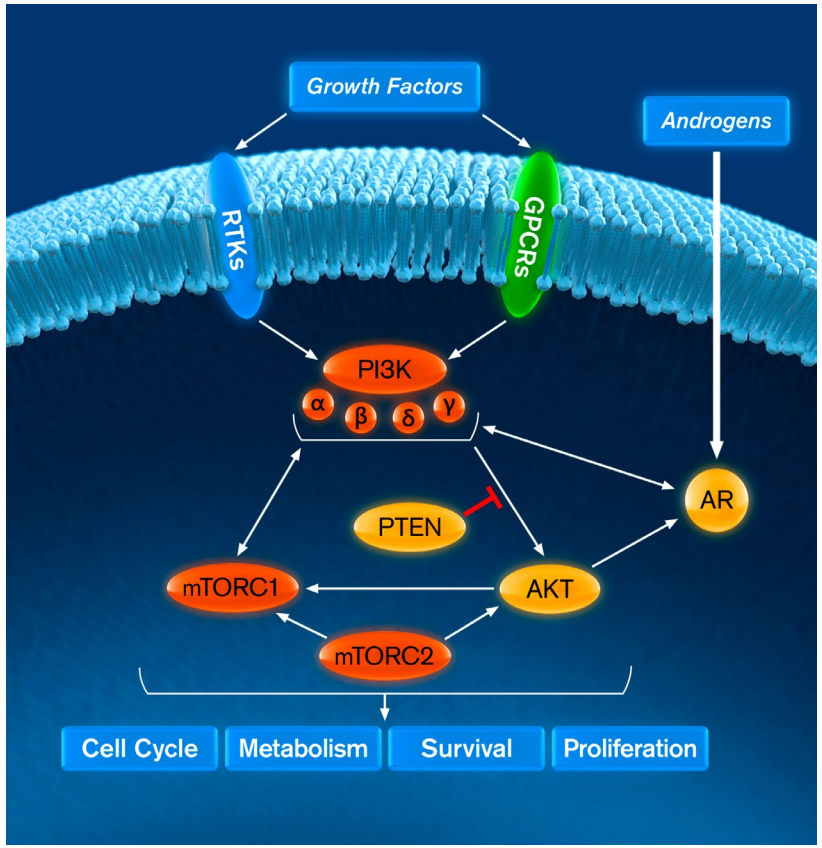
Androgen Signaling is the Key Driver of Prostate Cancer

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



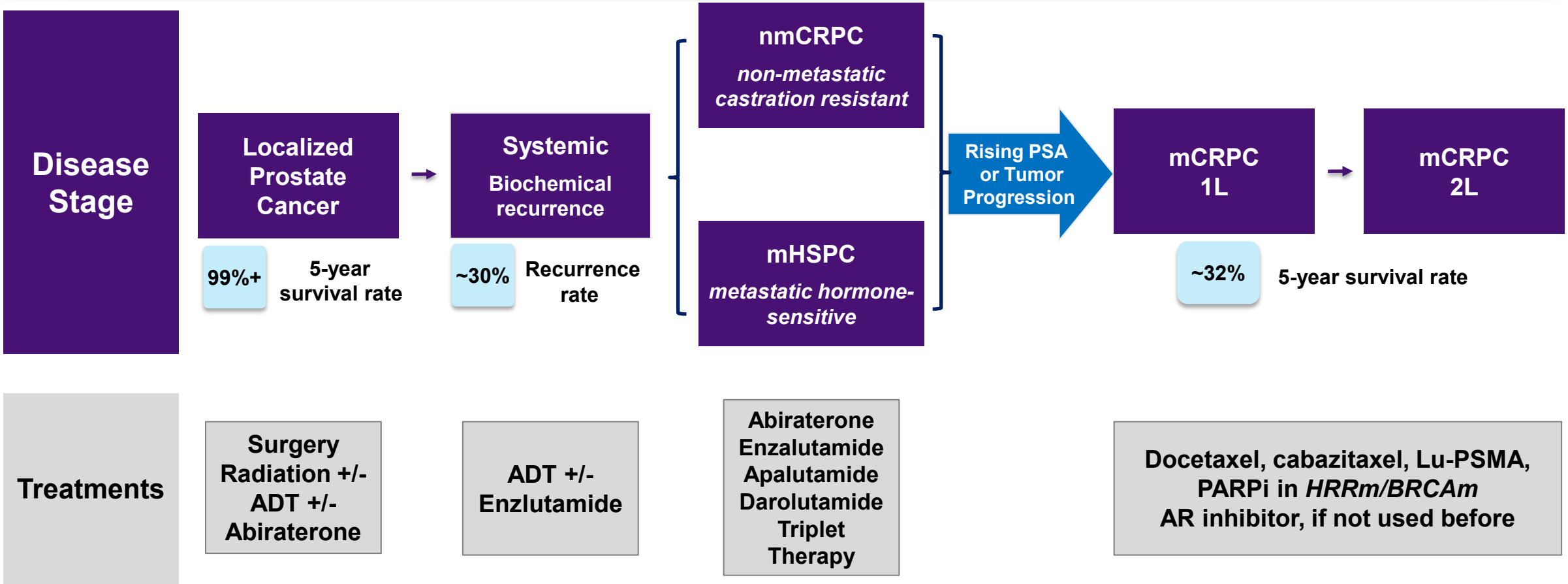
The PAM Pathway Plays a Key Role in mCRPC

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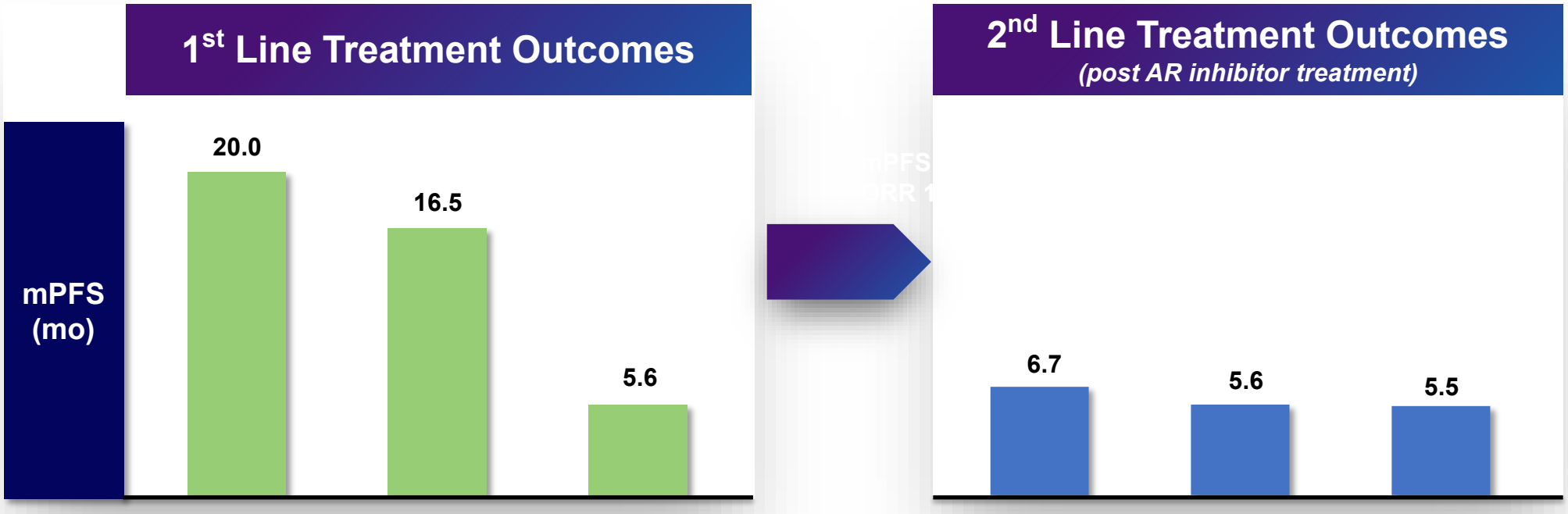
(1) Saad, Prostate Cancer Prostatic Dis. 2021; (2) Scher, Plos One 2015; Leith, A. et al. 2022; George, D. J. et al. 2022; NCCN Guidelines for Prostate Cancer Version 1.2023; (3) American Cancer Society, Cancer Facts & Figures 2023; (4) Wang, Front. Public Health, 2022; Abbreviations: mCRPC = metastatic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



Drugs	Xtandi [1]	Zytiga [2]	Docetaxel [3]	Docetaxel [4]	Zytiga [5]	Xtandi [6]
MOA	ARi	ARi	Chemotherapy	Chemotherapy	ARi	ARi
Pat Pop	All	All	All	Prior ARi	Prior Xtandi	Prior Zytiga
mPFS	20.0	16.5	5.6	6.7	5.6	5.5
OS	35.3	34.7	19.5	20.0	-	-

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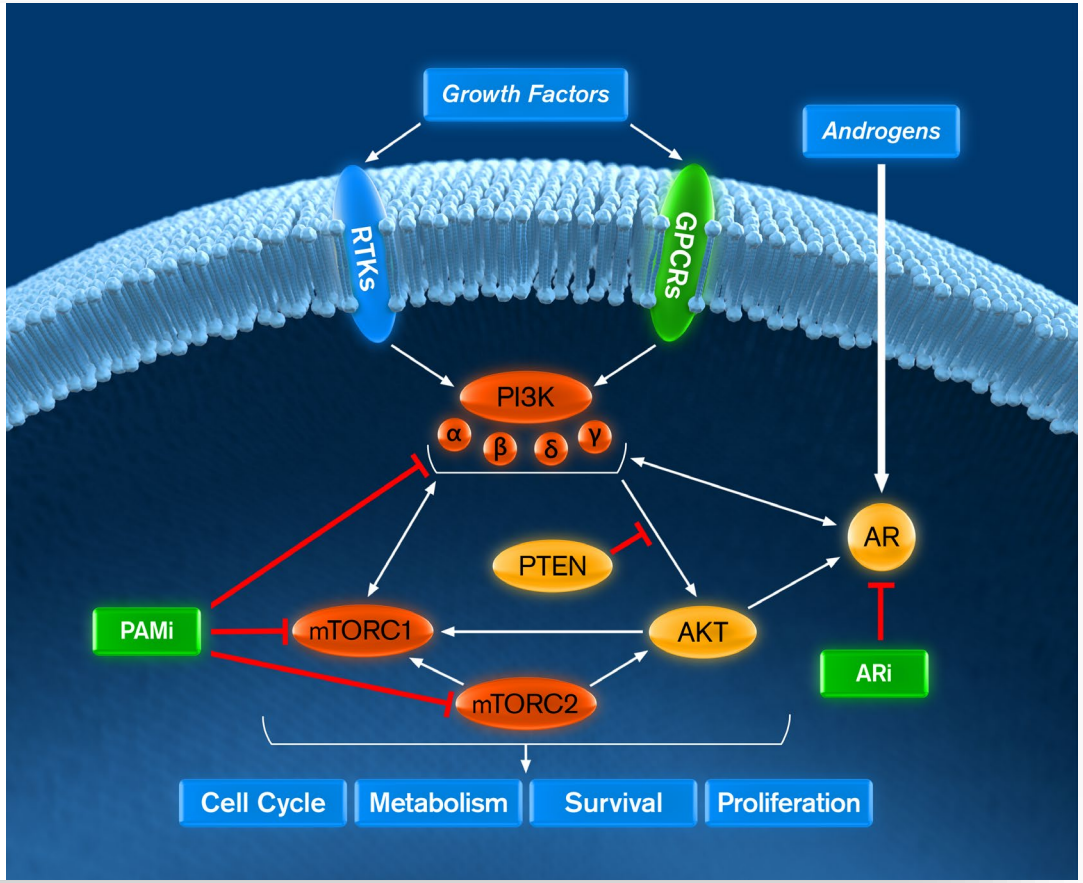
(1) Beer Eur Urol. 2017; (2) Ryan NEJM 2013; Ryan Lancet Oncol 2015 (3) Kellokumpa-Lehtinen Lancet Oncol. 2013, time-to-treatment failure reported; (4) Crabb J Clin Oncol 2021; (5) Attard J Clin Oncol 2018; (6) Sweeny Clin Cancer Res 2022. Abbreviations: HRR = homologous recombination repair; AR = androgen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

Clinical Trial Results for PAM Inhibitors After Progression on AR Therapy

Evidence of PAM pathways involvement and sensitivity to PAM inhibitors in mCRPC

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

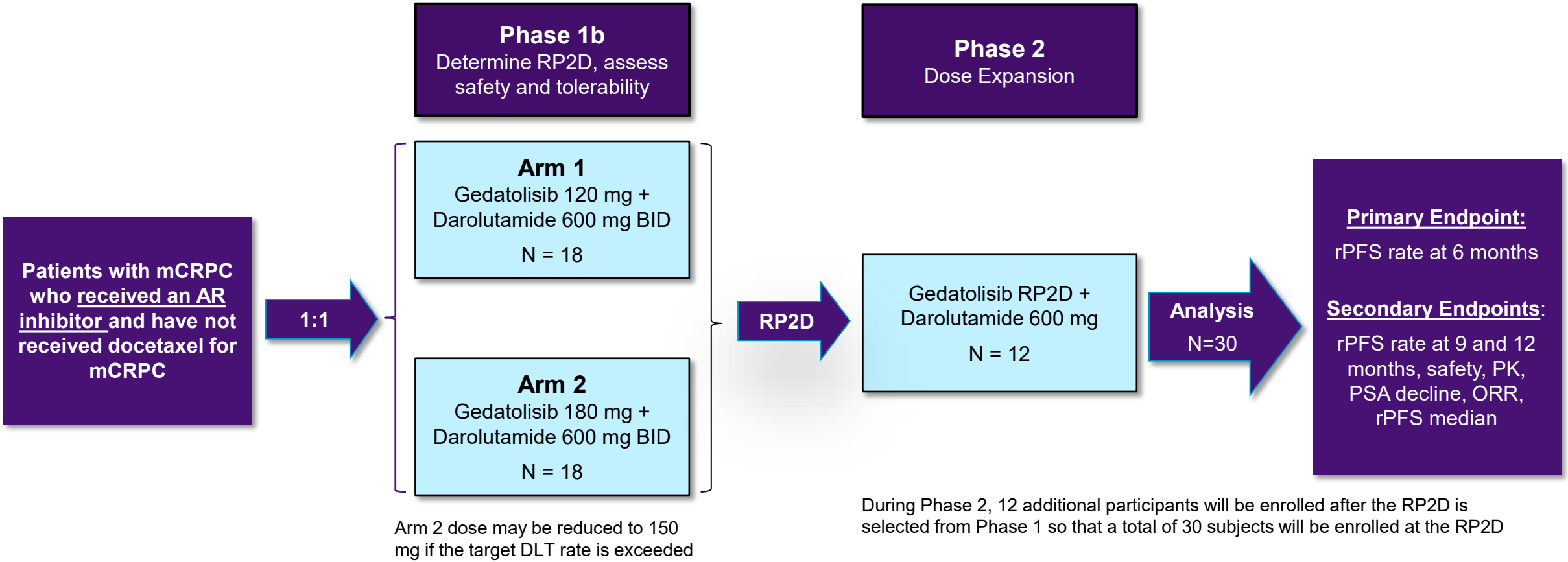
		AR-v7-negative	103	**13.2 vs. 5.3 months** *(HR = 0.52; P = 0.03)*	
		PTEN wild-type	60	**13.2 vs. 3.6 months** *(HR = 0.49; P = 0.07)*	
Ipatasertib (AKT) + Abiraterone vs. Abiraterone [2]	1st Line	All	1101	**19.2 vs. 16.6 months** *(HR = 0.84; P = 0.04)*	▪ Efficacy limited to PTEN loss patients ▪ Limited 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

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

Evaluating gedatolisib combined with darolutamide, a potent next generation androgen receptor inhibitor



Arm 2 dose may be reduced to 150 mg if the target DLT rate is exceeded

During Phase 2, 12 additional participants will be enrolled after the RP2D is selected from Phase 1 so that a total of 30 subjects will be enrolled at the RP2D

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

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Darolutamide is More Potent and Better Tolerated than SOC 1L AR Inhibitors

Bayer is collaborating with Celcuity and will supply darolutamide for the trial

	Darolutamide		Abiraterone		Enzalutamide	
Approved Indications	**nmCRPC, mHSPC**		**mCRPC, mHSPC**		**mCRPC, nmCRPC, mHSPC**	
IC_{50} [1]	**11 nM [2]**		**72 nM [3]**		**86 nM [2]**	
Most Common AE's (%) [4]	**All Grades**	**Grade 3/4**	**All Grades**	**Grade 3/4**	**All Grades**	**Grade 3/4**
Fatigue	16	1	39	2	51	9
Pain in extremities	6	0	30	2	21	3
Edema	<2	0	25	0.4	15	1
Constipation	<2	0	23	0.4	<2	0
Diarrhea	<2	0	23	1	22	2
Hot Flush	<2	0	22	0.2	20	0
Hypertension	<2	0	22	4	<2	1
Back Pain	<2	0	<5	0	26	5

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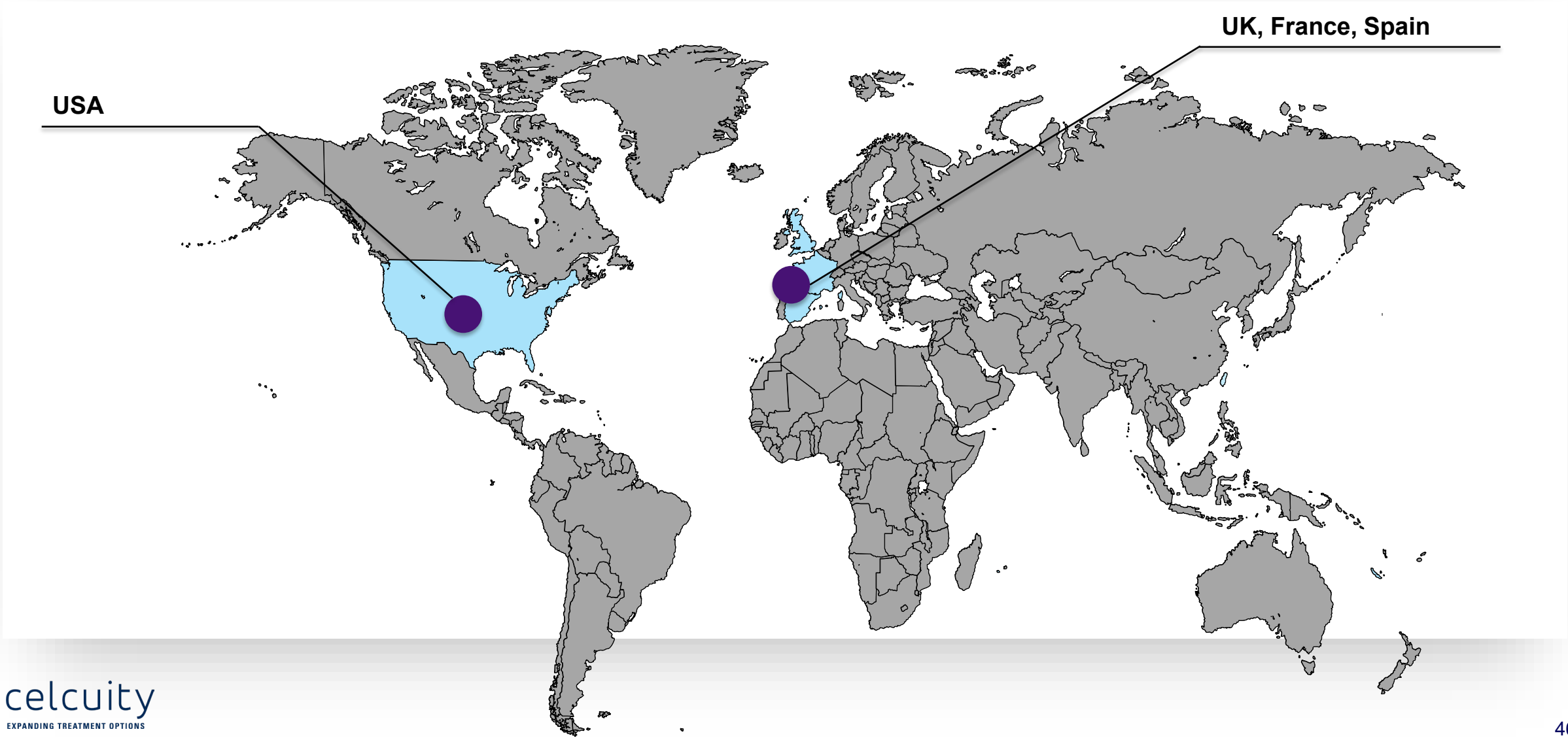
(1) IC50 derived from cell-free biochemical dose response analysis; (2) Moilanen, et al. Sci Rep 2015; (3) Pinto-Bazurco Mendieta et al. J Med Chem 2008. (4) US Package Inserts. Abbreviations: mCRPC = metastatic castration resistant prostate cancer; nm = non-metastatic; HSPC = hormone sensitive prostate cancer

~12 Sites Across US and Europe

Expect to enroll first patient Q1 2024 and announce initial data 1H 2025



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Additional Early Phase Clinical Data

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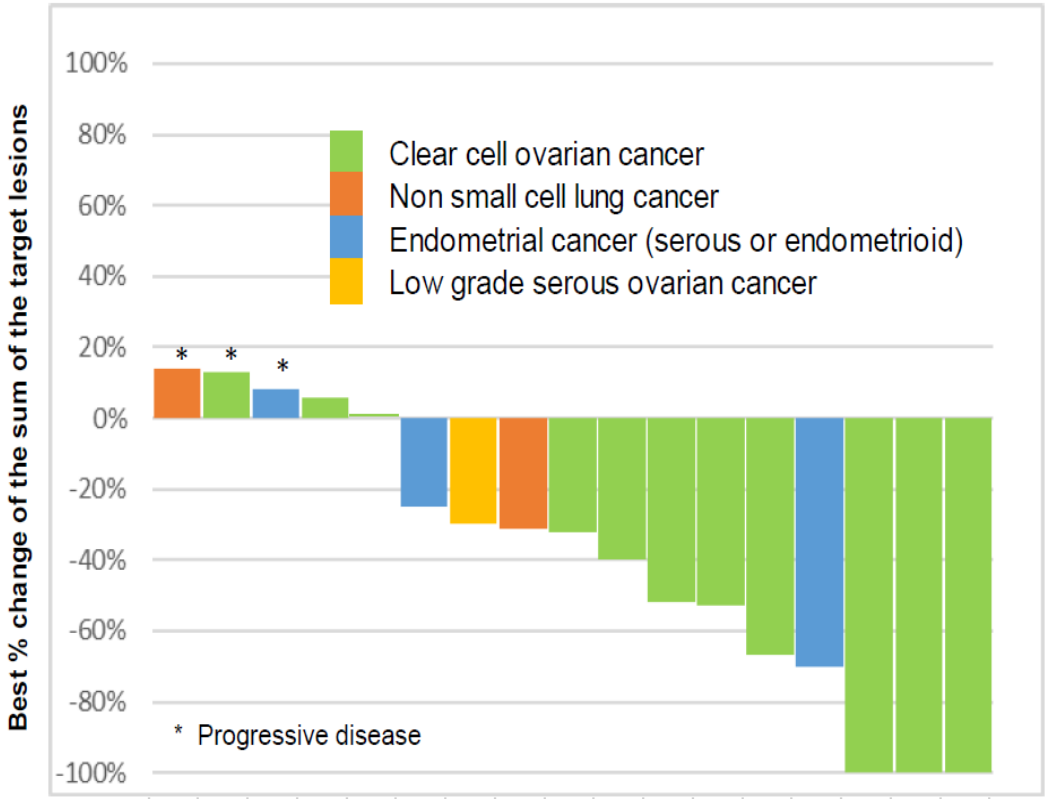
Gedatolisib + Paclitaxel + Carboplatin in Patients with Solid Tumors (N=17)[1]

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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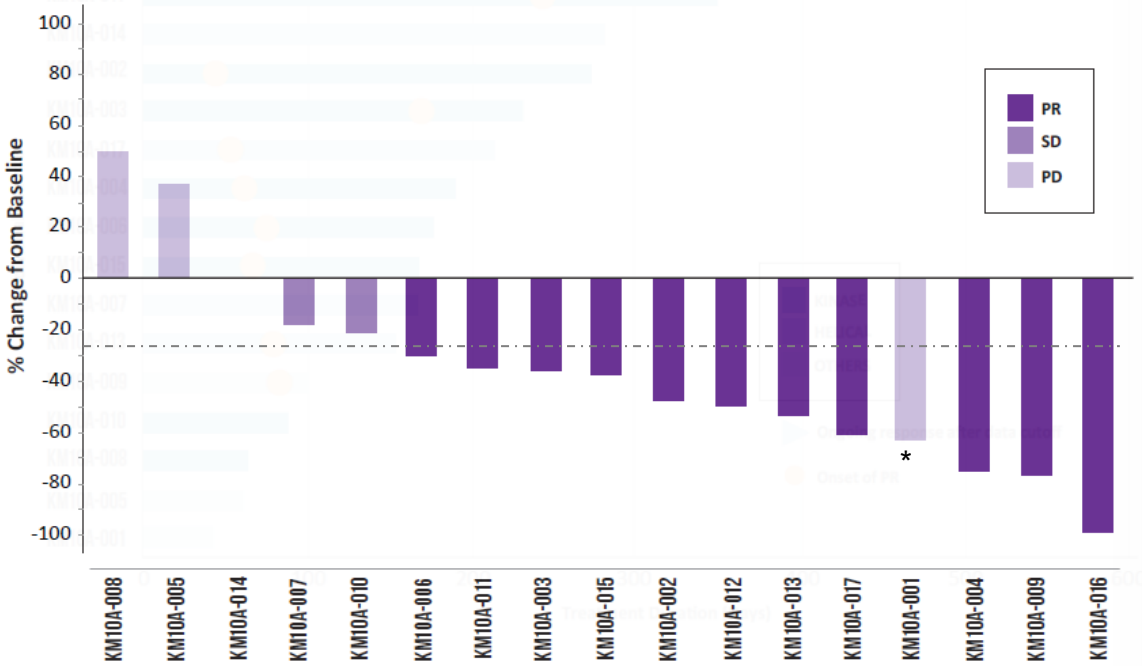
(1) Columbo 2021 CCR

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

59% ORR and 83% clinical benefit rate

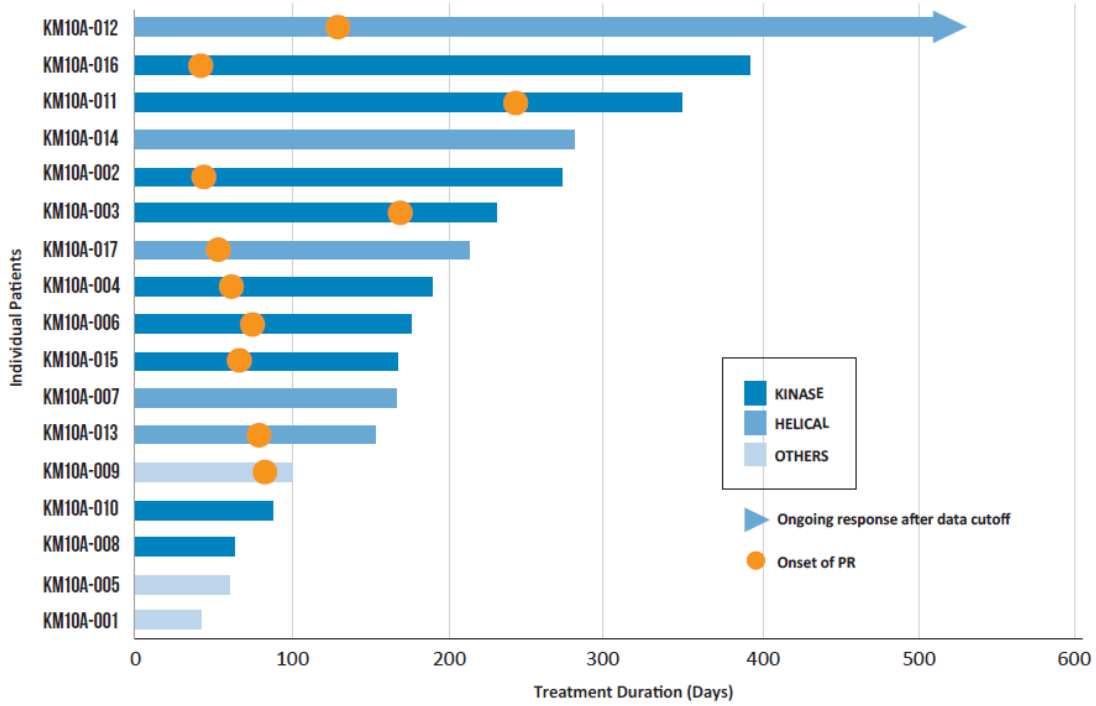
Best Response



* 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

Clinical Advisory Board

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

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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**
- Potential to replace currently available standard-of-care

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 of $178M as of Q1 2024 expected to fund operations through data readouts in ABC and mCRPC

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Live tumor cells contain infinitely more data than the fragmented cells current cancer diagnostics use

CELsignia

The CELsignia platform captures this data

Researchers recognize need for alternatives to genomic analysis

Complexity of signaling pathway networks requires much greater data to characterize than genomics can provide

"It is becoming increasingly clear that pathways rather than individual genes govern the course of tumorigenesis."

Kornelia Polyak, MD, PhD
Professor of Medicine
Harvard Medical School

HARVARD UNIVERSITY

"In order to fully understand aberrant signaling, the systematic perturbation of the entire network is required."

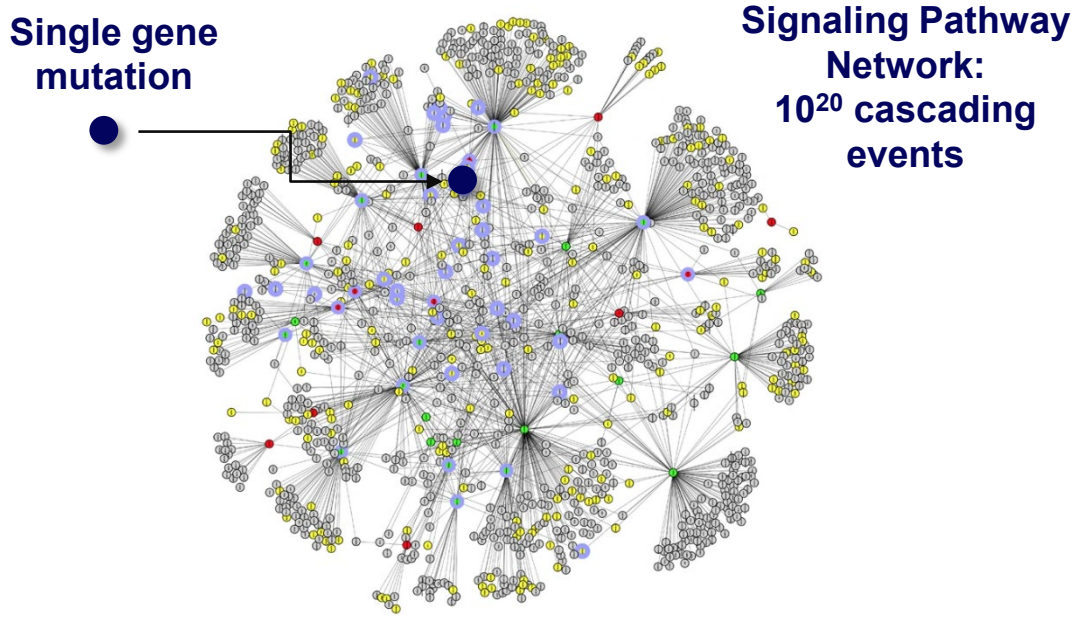
Neal Rosen, MD, PhD
Director, Center for Mechanism-Based Therapy
Memorial Sloan Kettering Cancer Institute

Memorial Sloan Kettering Cancer Center

"Sequencing alone cannot definitively determine whether a specific gene actually contributes to tumor formation."

Ben Ho Park, MD, PhD
Co-Leader Breast Cancer Research Program
Vanderbilt University Medical Center

VANDERBILT UNIVERSITY MEDICAL CENTER



CELsignia – the first 3rd generation diagnostic

Measures dynamic cell signaling activity to identify cancer drivers genomic tests cannot detect

Live Tumor Cells Isolated

>100,000 patient tumor cells are isolated in a **proprietary cell microenvironment**

Cell Signaling Quantified

Cell pathways are activated to generate **data from >10^{20} cellular events** at 240 time points to create a "movie" of the signaling activity[1]

Algorithmic Analysis

A **proprietary algorithm analyzes this "big data"** set to identify signaling activity 5 standard deviations from normal

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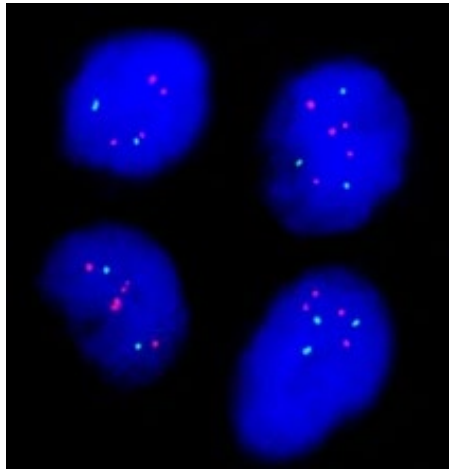
Source: 1) Internal Celcuity Studies

Current Molecular Diagnostics vs. CELsignia – HER2 Example

CELsignia identifies new sub-group of patients with HER2 driven cancer

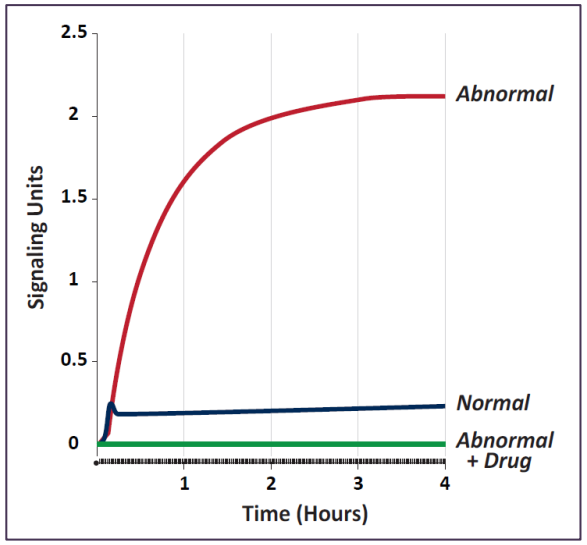
FISH HER2 Dx
(1 pathway gene)



FISH+
15%

CELsignia+
15%-20%

CELsignia HER2 Activity
(4 hours of pathway signaling events)



$9 billion
anti-HER2 drug annual revenue[1]

CELsignia identifies new
patients for anti-HER2 drugs

$Billions additional
anti-HER2 drug revenue potential

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Source: 1) Roche 2020 Annual Report

Key research discoveries drive test development

CELsignia platform provides powerful tool to discover new cancer sub-types and mechanisms

Specific target mutations (e.g. HER2+) not required for oncogenic signaling

- Discovered 16 cancer sub-types that genomic tests cannot detect
- Confirms mutational status is not sufficiently specific

Implications

- May miss 50% of HER2, EGFR, PI3K, c-Met driven cancers

Mutations often don't lead to oncogenic signaling

- Demonstrated that target specific mutations often do not drive aberrant signaling
- Further confirms mutational status is not sufficiently specific

Implications

- Explains low response rates of many targeted therapies

Drug resistance mechanisms characterized

- Linkages identified between:
  - c-Met, HER3, HER2, & EGFR
  - LPA, S1PA, PI3K, MEK
- Untreated cooperative pathways drive drug resistance

Implications

- May miss 50% of HER2, EGFR, PI3K, c-Met driven cancers

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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