



celcuity

EXPANDING TREATMENT OPTIONS

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

Corporate Presentation

September 2023

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 and upcoming clinical trials for gedatolisib, including but not limited to our VIKTORIA-1 Phase 3 clinical trial, the expected results of VIKTORIA-1, including but not limited to the anticipated efficacy of gedatolisib in combination with fulvestrant and with or without palbociclib, the expected timing of funding of tranches under the Company’s debt financing facility, any potential benefits resulting from Breakthrough Therapy designation for gedatolisib, and other expectations with respect to Celcuity’s lead product candidate, gedatolisib, 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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Unlocking the Potential of Treating Cancers That Involve the PI3K/mTOR Pathway

One of the most important oncogenic pathways

PI3K/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 pathways¹

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

PI3K/mTOR	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

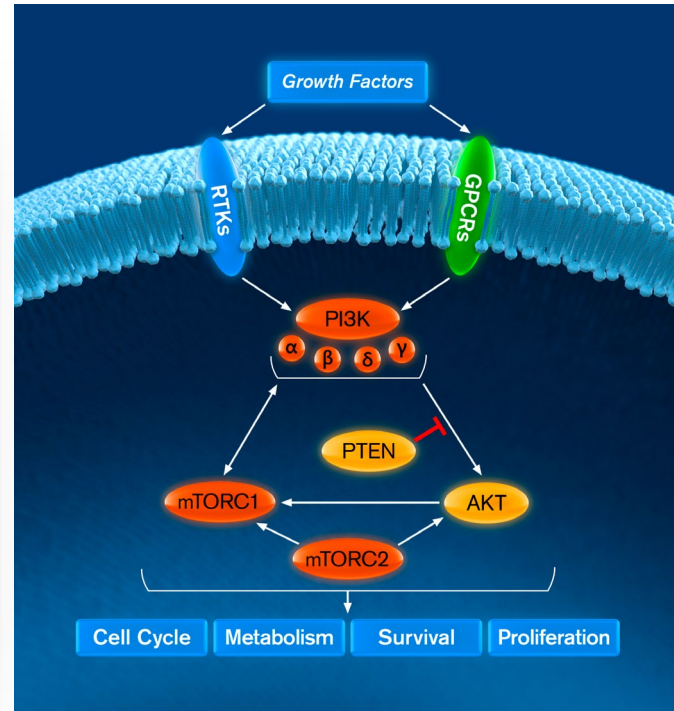
Difficult to Safely and Efficaciously Inhibit PI3K/mTOR

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

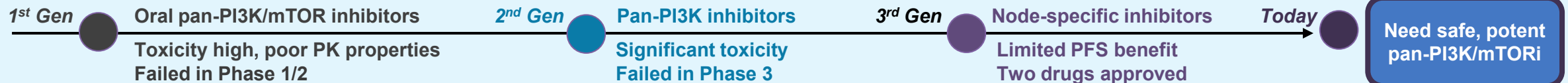
Induces compensatory resistance that reduces efficacy



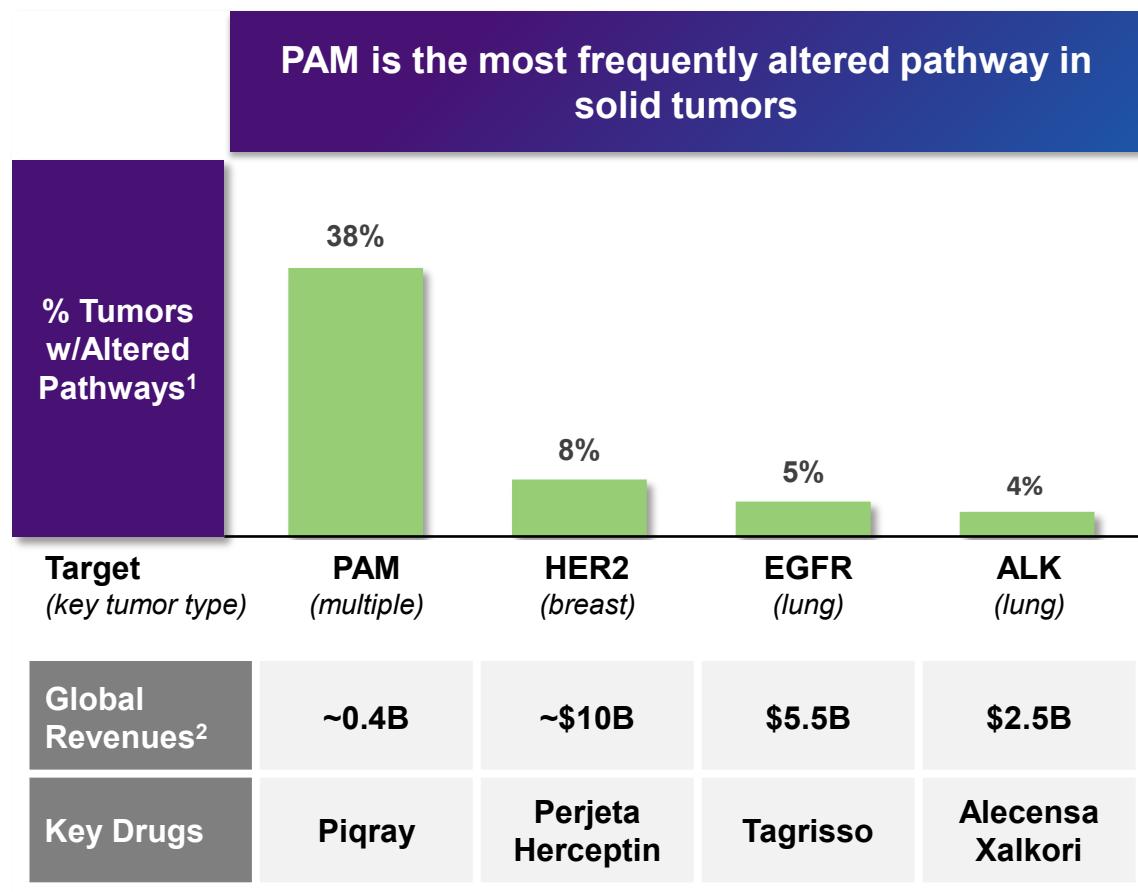
Therapeutic window for oral PI3K/mTOR inhibitors is narrow

Difficult to optimize pathway inhibition without inducing undue toxicity

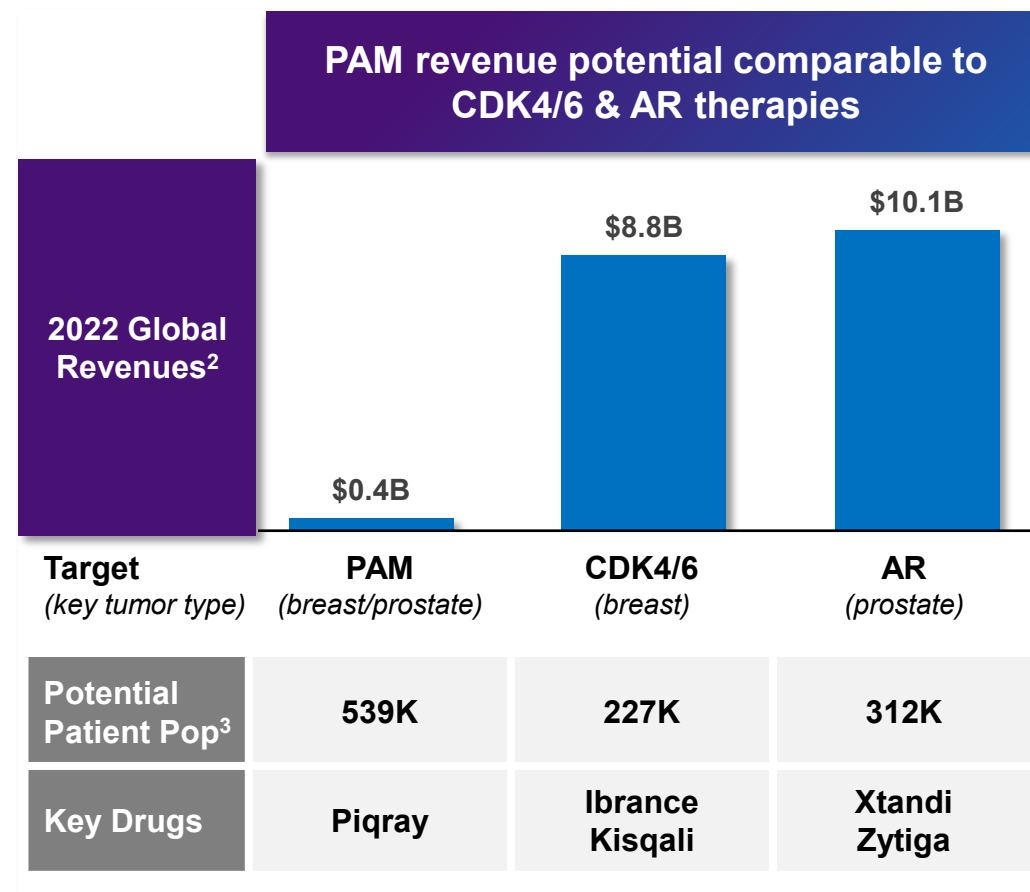
Orally administered pan-PI3K or pan-PI3K/mTOR inhibitors induced unacceptable toxicity



The PAM Pathway is the Most Underdeveloped Target in Solid Tumors



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



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

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 patients¹
- **63% ORR, 12.9 months mPFS** in 2L patients²

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 fulvestrant²

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
- **211,000 1L/2L patients** in US, 5EU, Japan³

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

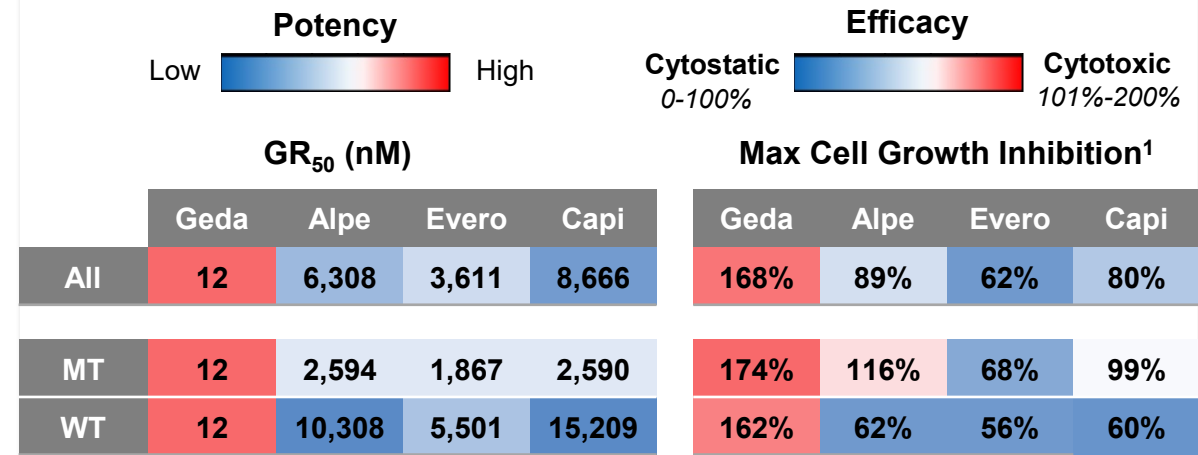
Node	Gedatolisib ²	Alpelisib ³	Everolimus ⁴	Capivasertib ⁵
PI3K- α	0.6	~4.0	-	-
PI3K- β	6.0	1,156	-	-
PI3K- γ	5.4	250	-	-
PI3K- δ	6.0	290	-	-
mTORC1	1.6	-	~2.0	-
mTORC2	1.6	-	-	-
AKT	-	-	-	3.0

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
- Comprehensive pathway blockade can induce anti-tumor activity independent of PIK3CA status

Live Cell Proliferation Rate Dose Response Analysis⁶

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

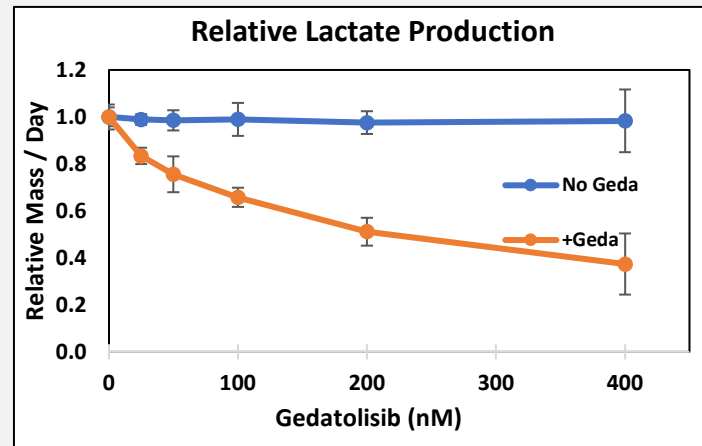
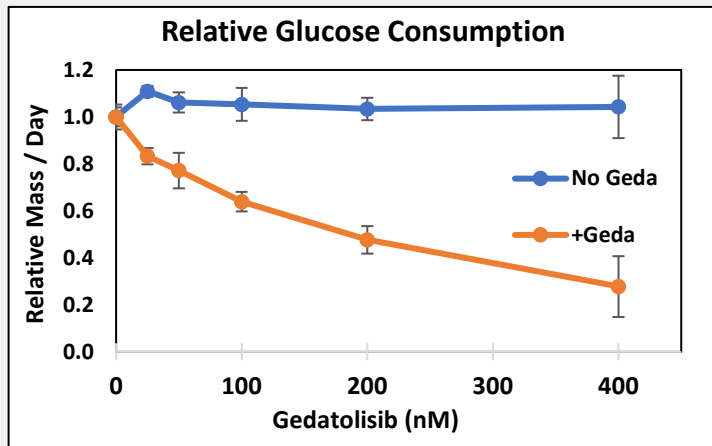
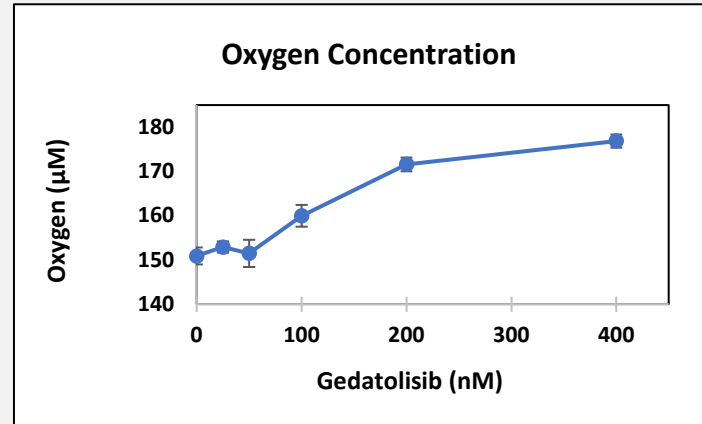
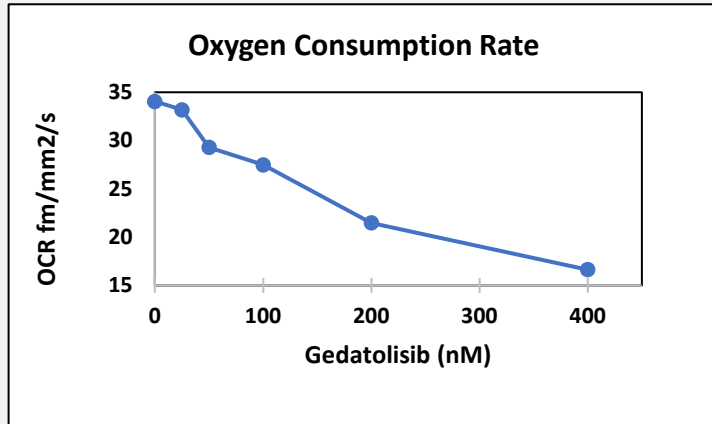


Gedatolisib is highly potent and cytotoxic in vitro

- 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

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 O₂ consumption leads to an increase in O₂ 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

Gedatolisib Increases Immune Cell Tumor Infiltration and Activation

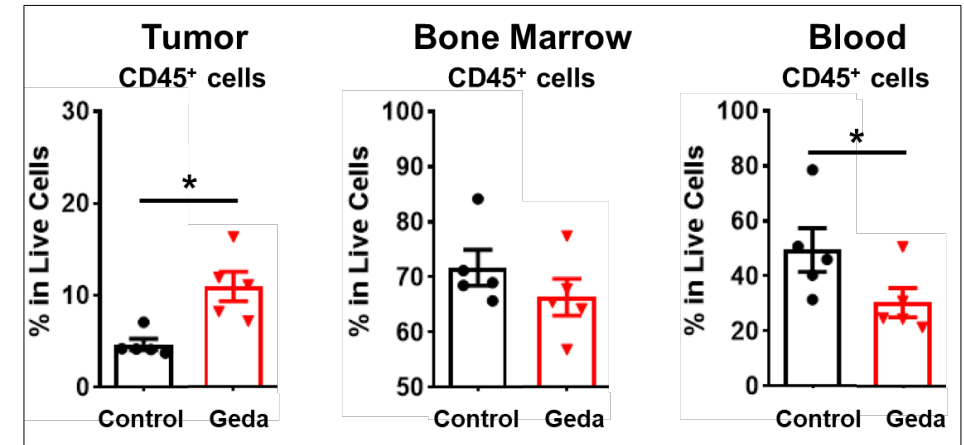
Profiled CD45+ immune cell populations in tumor, bone marrow, peripheral blood

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

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+



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

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

Gedatolisib PK Properties and IV Administration Optimize Safety Profile

Lower toxicity vs. approved PI3K inhibitors

	Gedatolisib¹	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

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 include prophylaxis
- **Low incidence of Grade 3 hyperglycemia (1%)**
- **No treatment related neutropenia**
- No Grade 4 or 5 adverse events

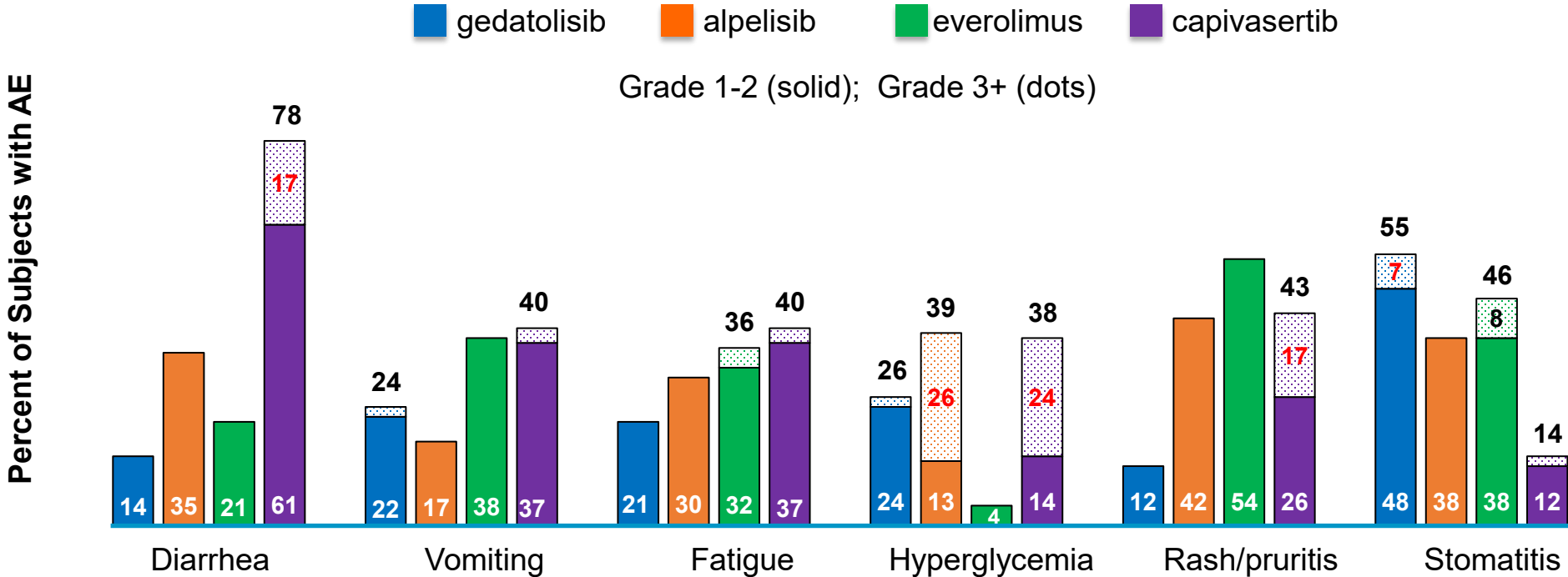
MTD Arm (n=42)

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	-

Safety Data for Single-Agent Gedatolisib vs. Single Node PAM Inhibitors

Fewer patients reported AE when treated with gedatolisib as single agent compared to other PAMi



Clinical Development Programs

2nd Line HR+/HER2- Advanced Breast Cancer

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

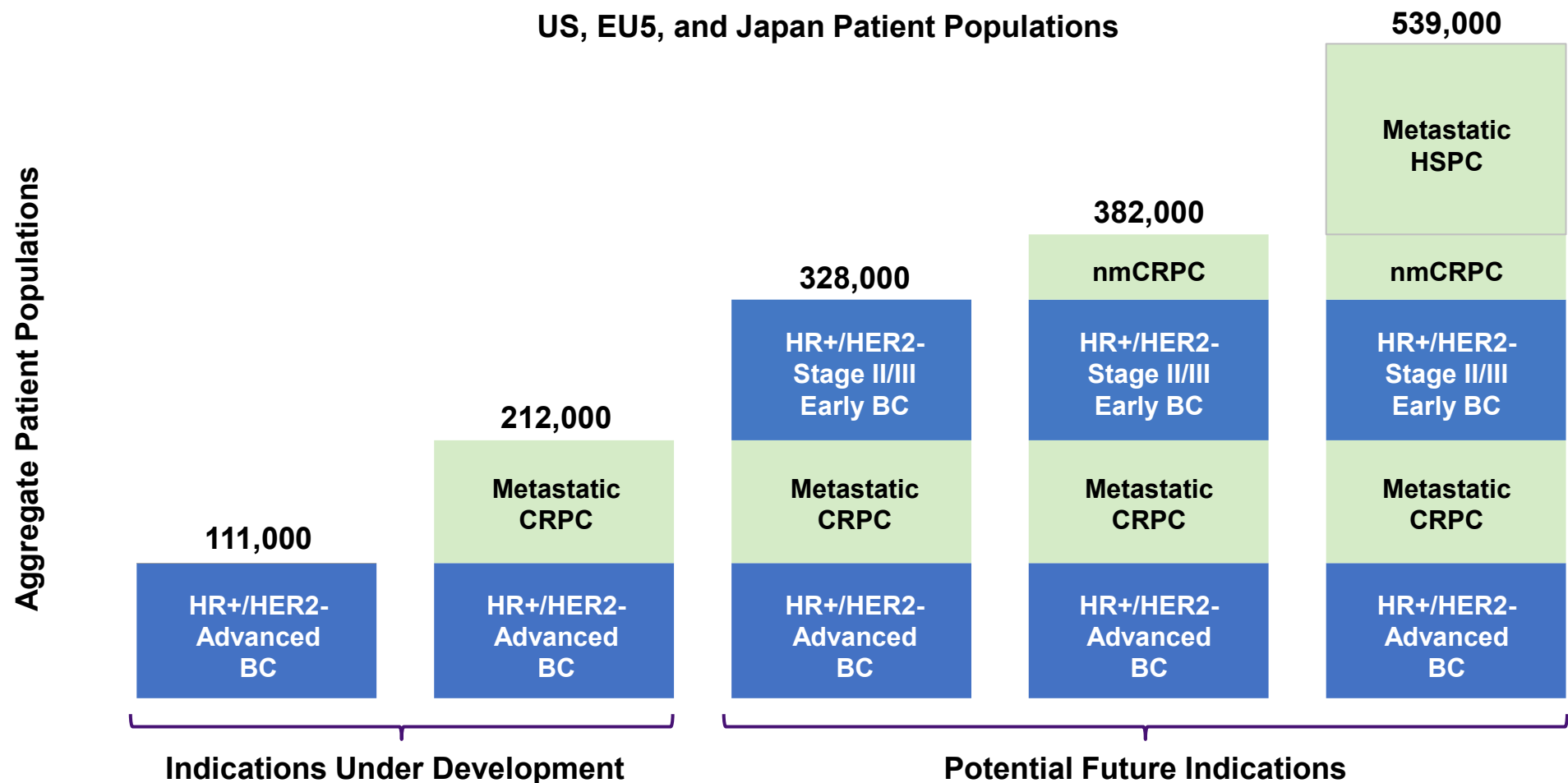
- Enrolling patients with **HR+/HER2- advanced breast cancer** who progressed on CDK4/6 therapy¹
- All-comer design (*PIK3CA*+/-) includes separate primary endpoints for mutated and non-mutated *PIK3CA* patients
- Breakthrough Therapy Designation for this indication was granted by the FDA in July 2022

2nd Line Metastatic Castration Resistant Prostate Cancer

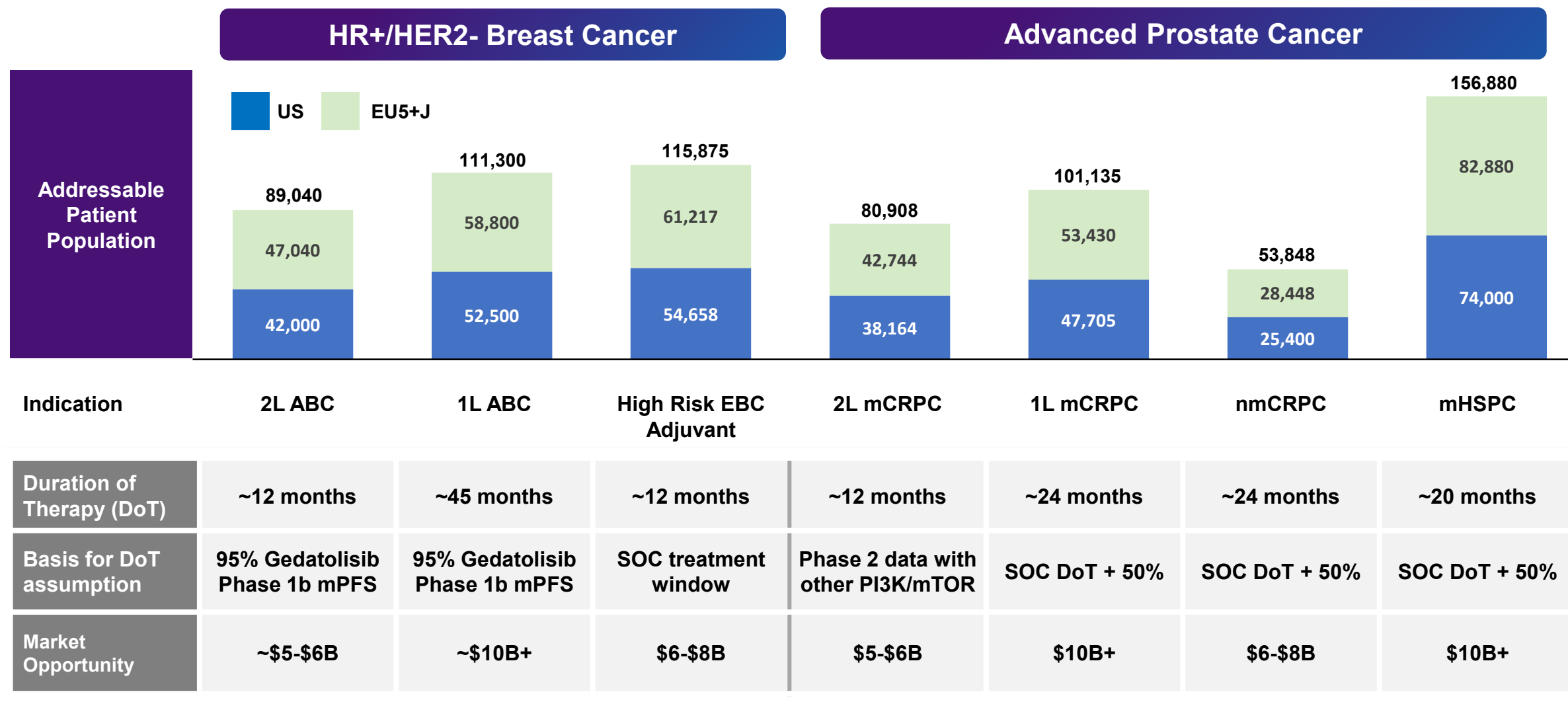
Phase 1b/2 clinical trial for gedatolisib with darolutamide planned to begin Q1 2024

- 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 studies²
- Promising clinical activity with an AR inhibitor when combined with less active PAM inhibitors than gedatolisib has been reported in prostate cancer trials³

Addressable Patient Population in Breast and Prostate Cancer



Multiple potential blockbuster indications in both tumor types





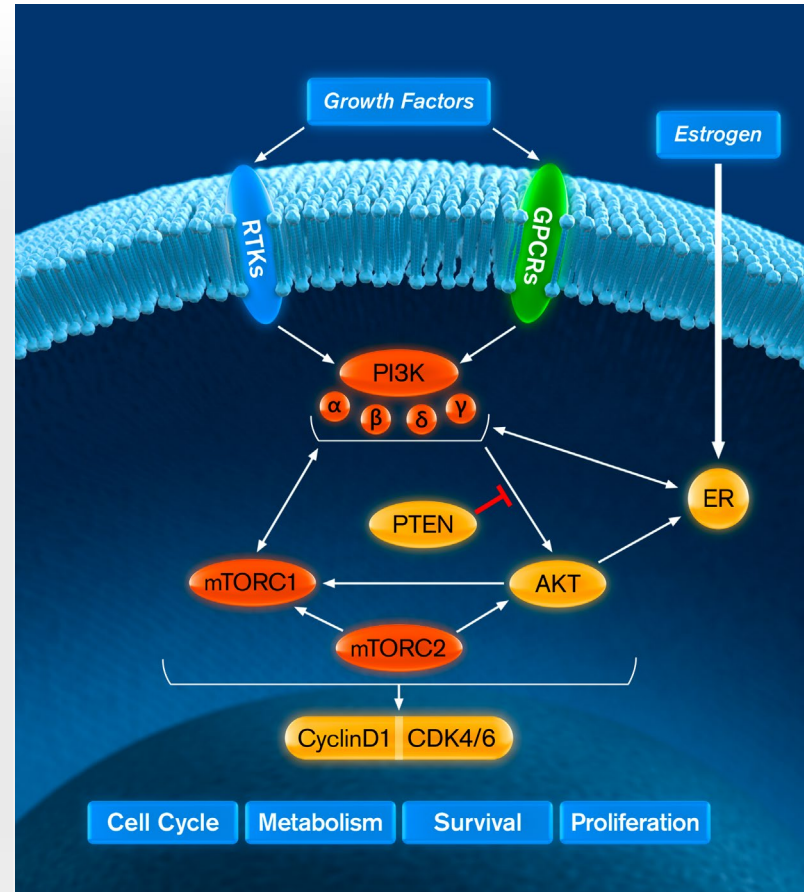
Gedatolisib for Advanced Breast Cancer (ABC)

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 PI3K α
- **PI3K/mTOR inhibition increases ER activity which increases sensitivity to endocrine therapy**



CDK4/6, ER and PI3K/mTOR

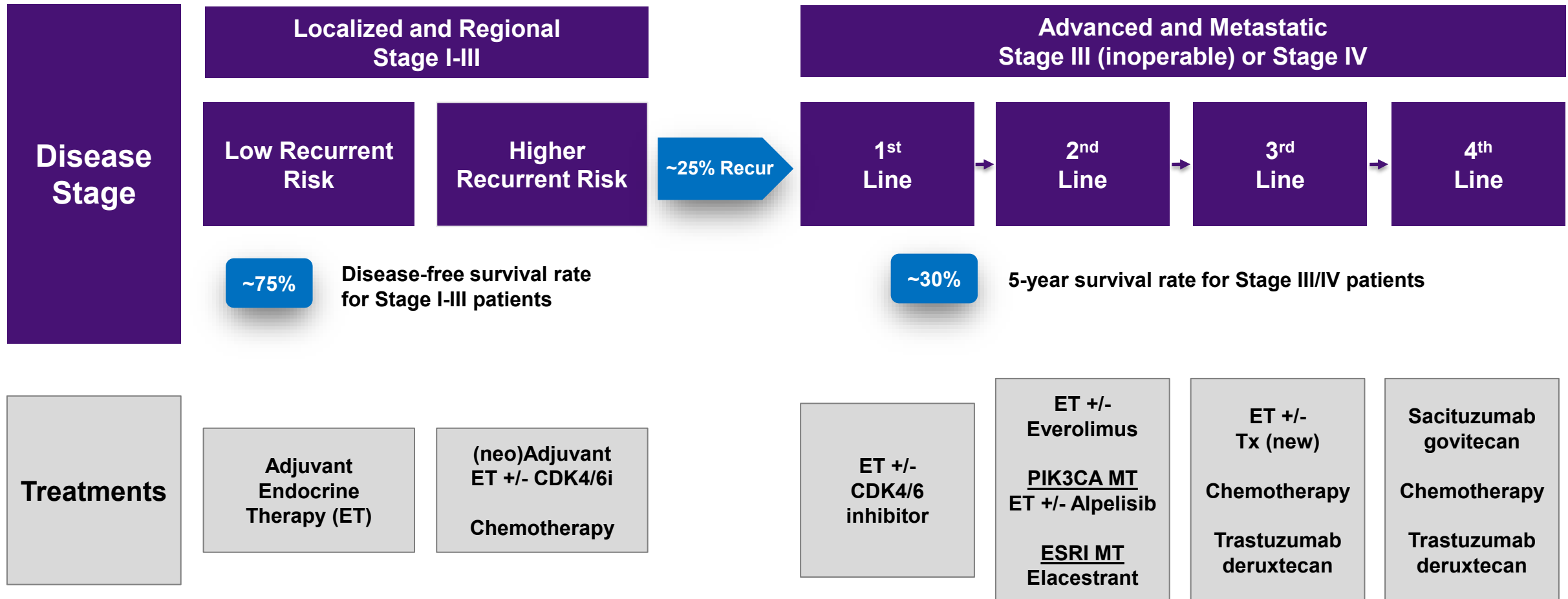
- Estrogen promotes cyclin D1 transcription and cyclin D1 can cause estrogen independent transcription
- Provides rationale for simultaneously inhibiting ER and CDK4/6
- CDK4/6 inhibition causes incomplete cell cycle arrest – addition of PI3K/mTOR inhibition enables more complete arrest
- **PI3K/mTOR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

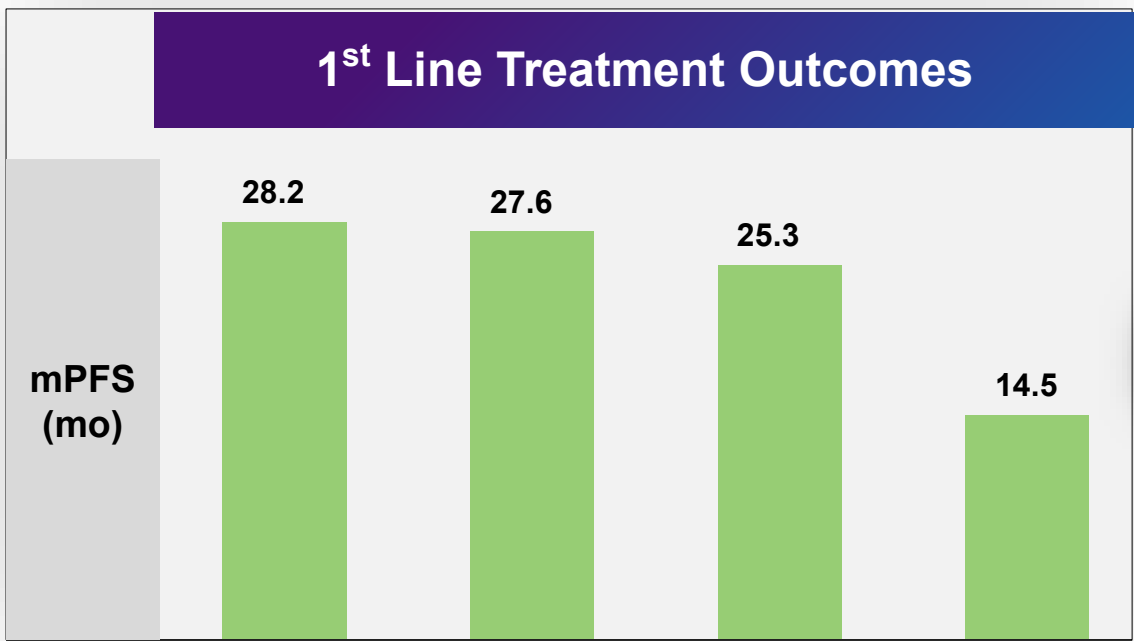
HR+/HER2- Breast Cancer Treatment Landscape¹

~30,000 women in US and ~33,000 women in 5EU and Japan die from breast cancer annually²

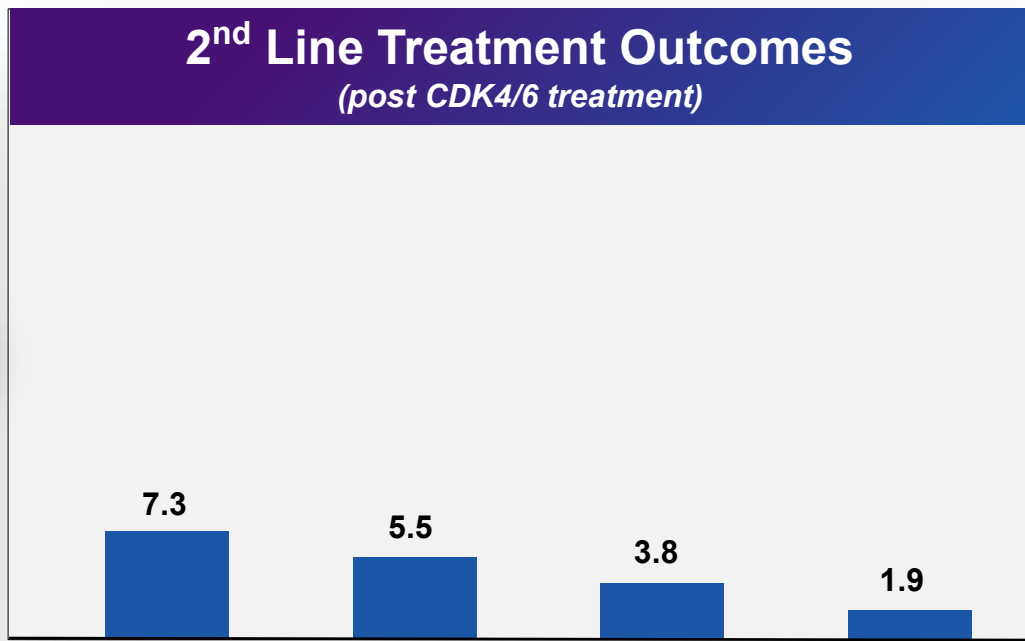


Limited Benefit for 2nd Line HR+/HER2- ABC Patients Post-CDK4/6 Treatment

Significant need for better therapeutic options



Drugs	Abemaciclib + letrozole ¹	Palbociclib + letrozole ²	Ribociclib + letrozole ³	Letrozole ²
MOA	CDK4/6 + AI	CDK4/6 + AI	CDK4/6 + AI	AI
Pat Pop	All	All	All	All
mPFS	28.2	27.6	25.3	14.5
ORR	55%	55%	53%	44%



Alpelisib + fulvestrant ⁴	Capivasertib + fulvestrant ⁵	Elacestrant ⁶	Fulvestrant ⁶
PI3Kα + SERD	AKT + SERD	SERD	SERD
PIK3CA MT	All	ESR1 MT	All
7.3	5.5	3.8	1.9
21%	23%	7%	6%

(1) Goetz JCO 2017; Johnson S, et al. npj Breast Cancer 2019; (2) Finn NEJM 2016; Rugo H, et al. Breast Cancer Res Treat, 2019; (3) Hortobagyi NEJM 2016; Hortobagyi Ann Oncol 2018; USPI; (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, except for capivasertib

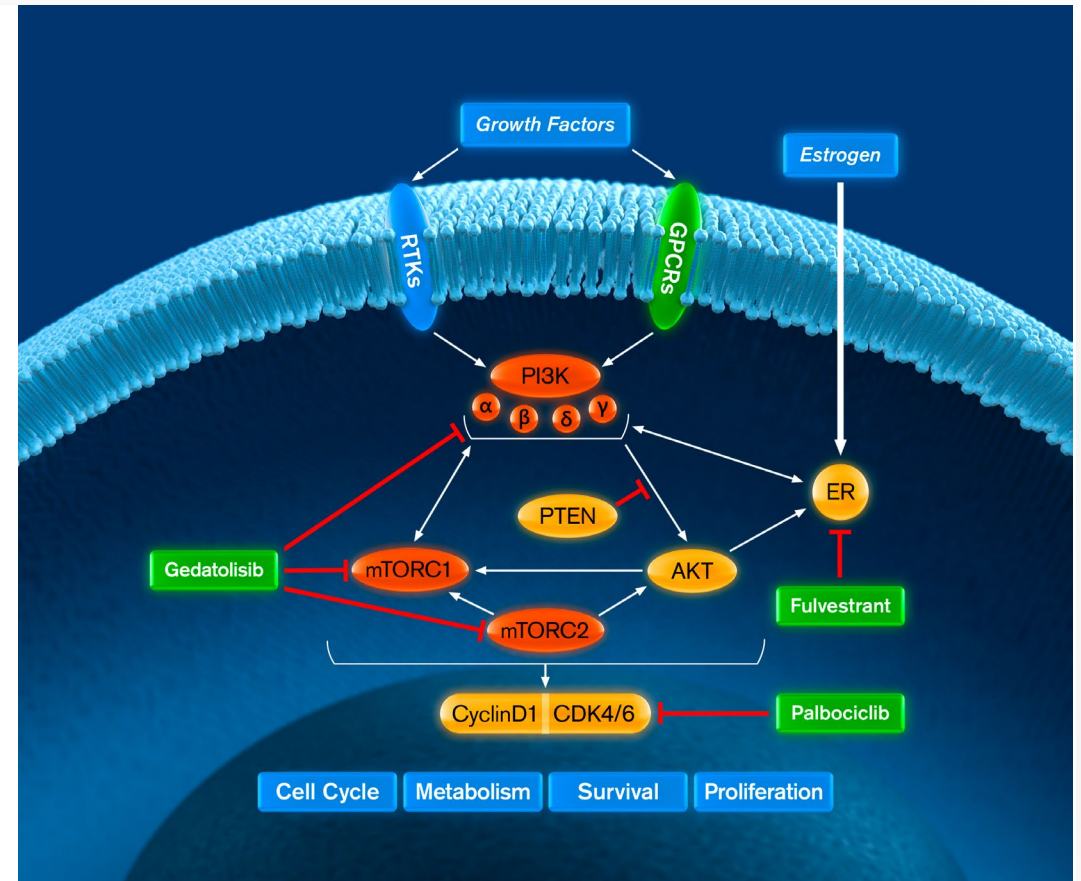
Review of Phase 1b Data

Gedatolisib + Palbociclib + Fulvestrant/Letrozole

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

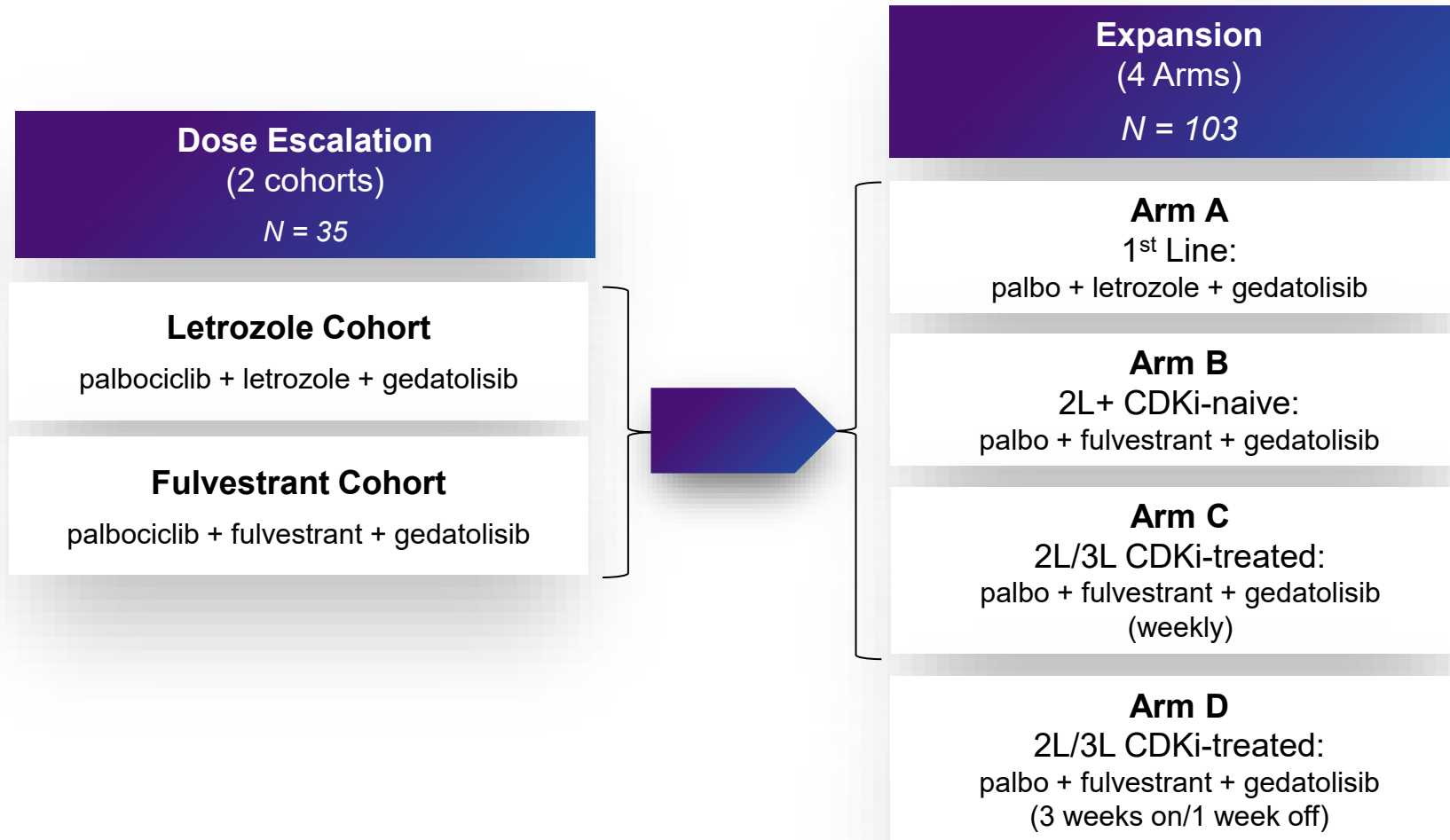
Treatment Rationale

- 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



B2151009: Phase 1b Study (138 patients)

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



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 Therapy¹	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

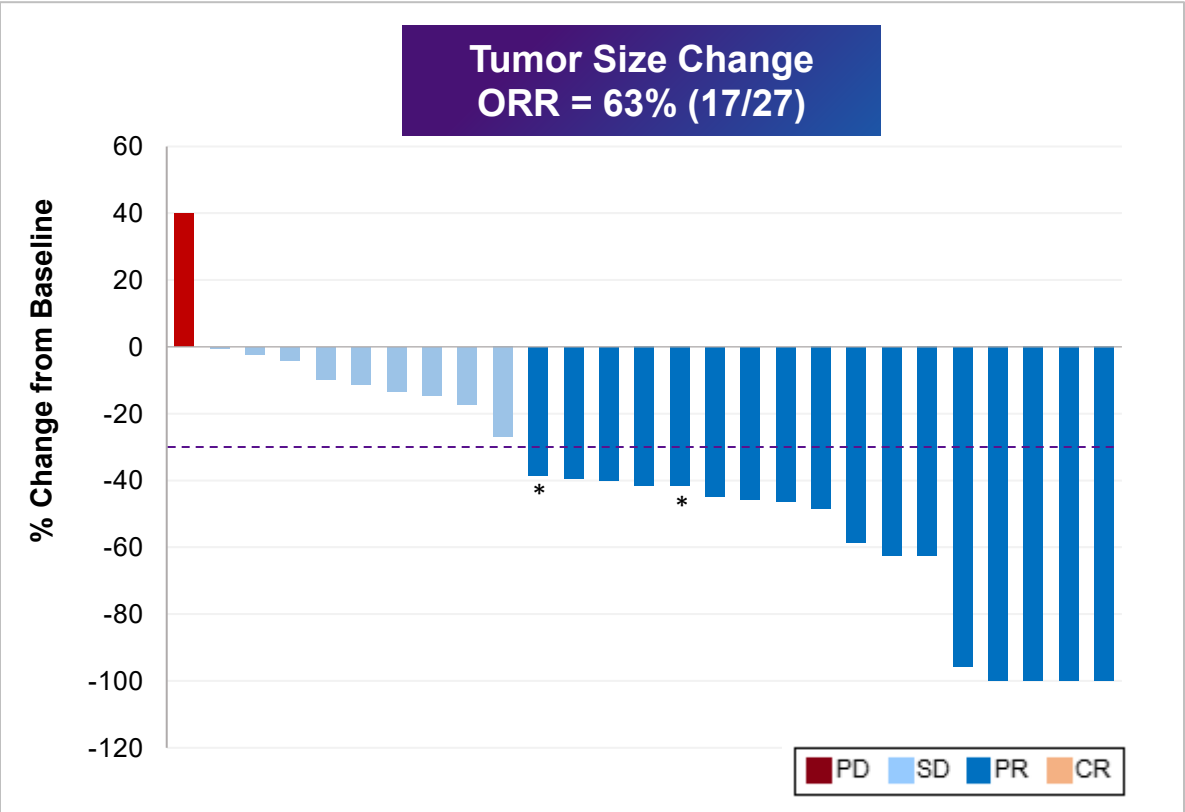
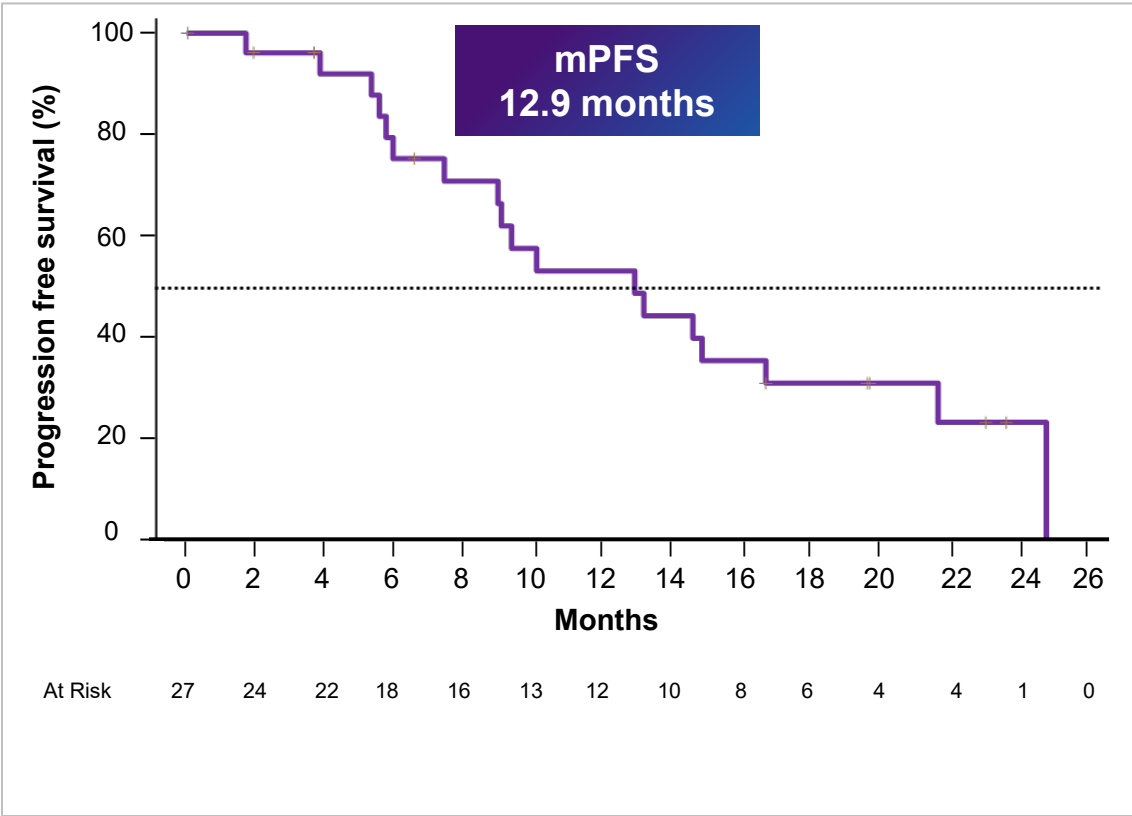
ORR and PFS in Each Expansion Arm Was Superior to SOC

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-naïve		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%

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



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 – 25% discontinued ¹
- Most TRAE's were Grade 1 or 2
- Few hyperglycemia adverse events
 - 26% all Grades, 7% Grade 3/4
 - Alpelisib (65% all, 37% Grade 3/4) ²
- Stomatitis prophylaxis was not utilized in this study
 - **Swish-and-Spit dexamethasone prophylactic mouth rinse reduced Grade 2-4 stomatitis by 90%** ³
 - Phase 3 study will include 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 ⁴	11	56	22
Neutropenia ⁵	-	15	67
Nausea	44	30	-
Fatigue	22	37	7
Dysgeusia	44	7	-
Diarrhea	37	-	4
Rash	19	15	7
Leukopenia ⁶	-	19	23
Constipation	30	4	4
Vomiting	22	11	4
Anemia ⁷	4	15	15
Hyperglycemia	15	4	7

Gedatolisib Combo vs. SOC for 2L HR+ / HER2- ABC Post-CDKi

Gedatolisib Combo Offers Potential for Superior Efficacy Compared to Alternatives

Patient Population	2 nd 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%
All	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%

Efficacy in Treatment-Naïve Population Superior to SOC

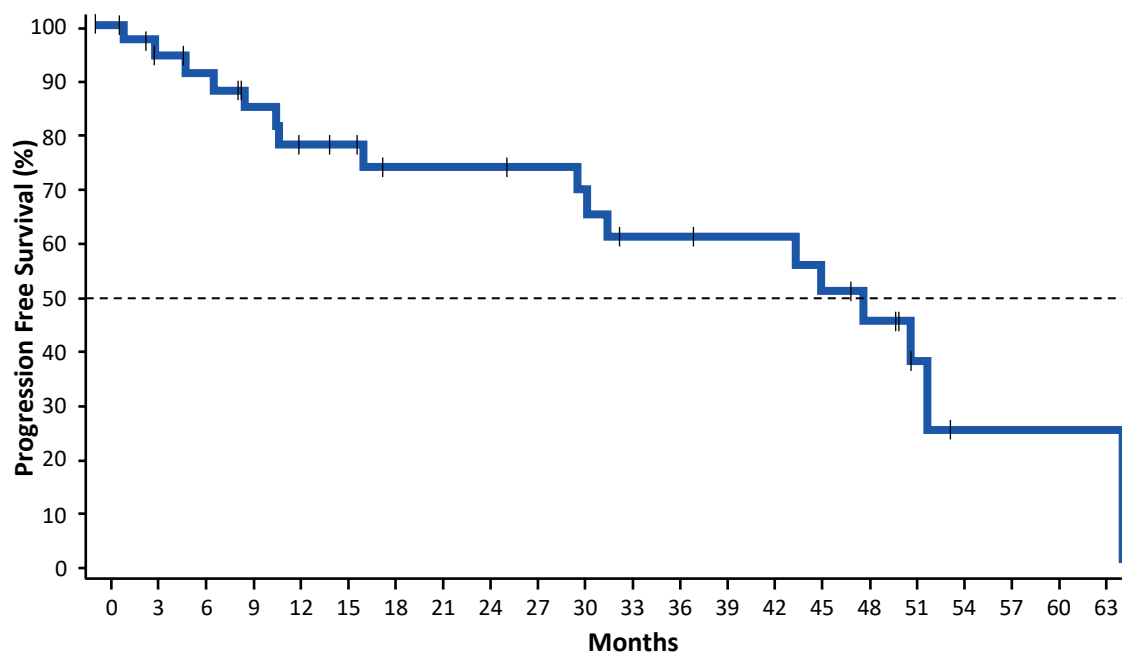
mPFS of 48.6 months, mDOR of 46.9 months, and ORR of 79%

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)

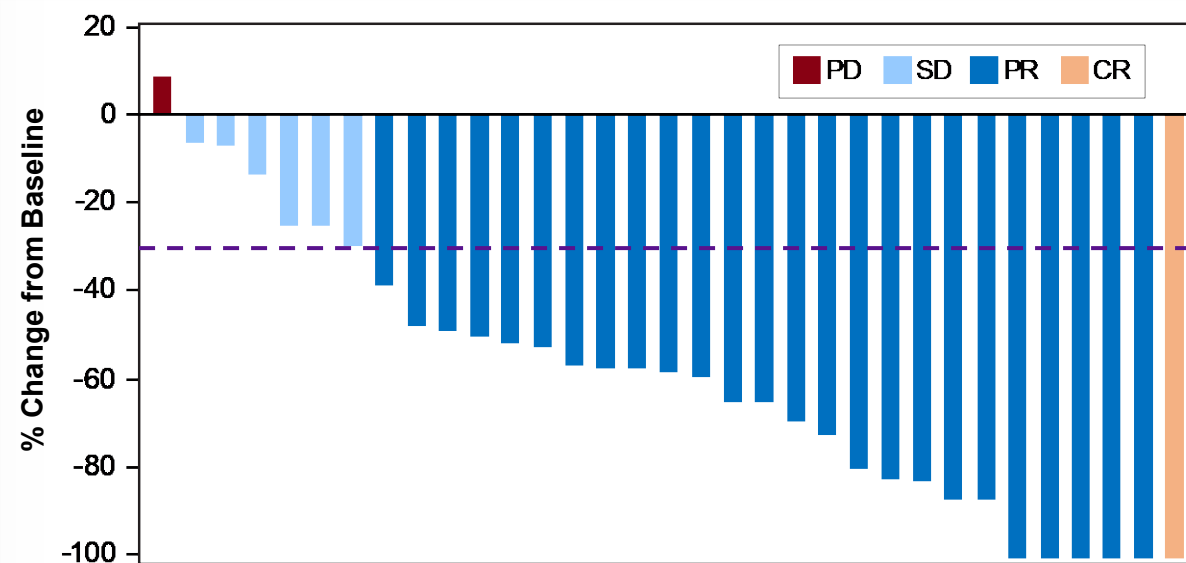
Gedatolisib + Palbociclib + Letrozole in 1st Line HR+/HER2- ABC (N=41)¹

Combined 1L data from Esc Arm A + Exp Arm A compares favorably to published data for SOC palbociclib + letrozole²

mPFS
48.6 Months

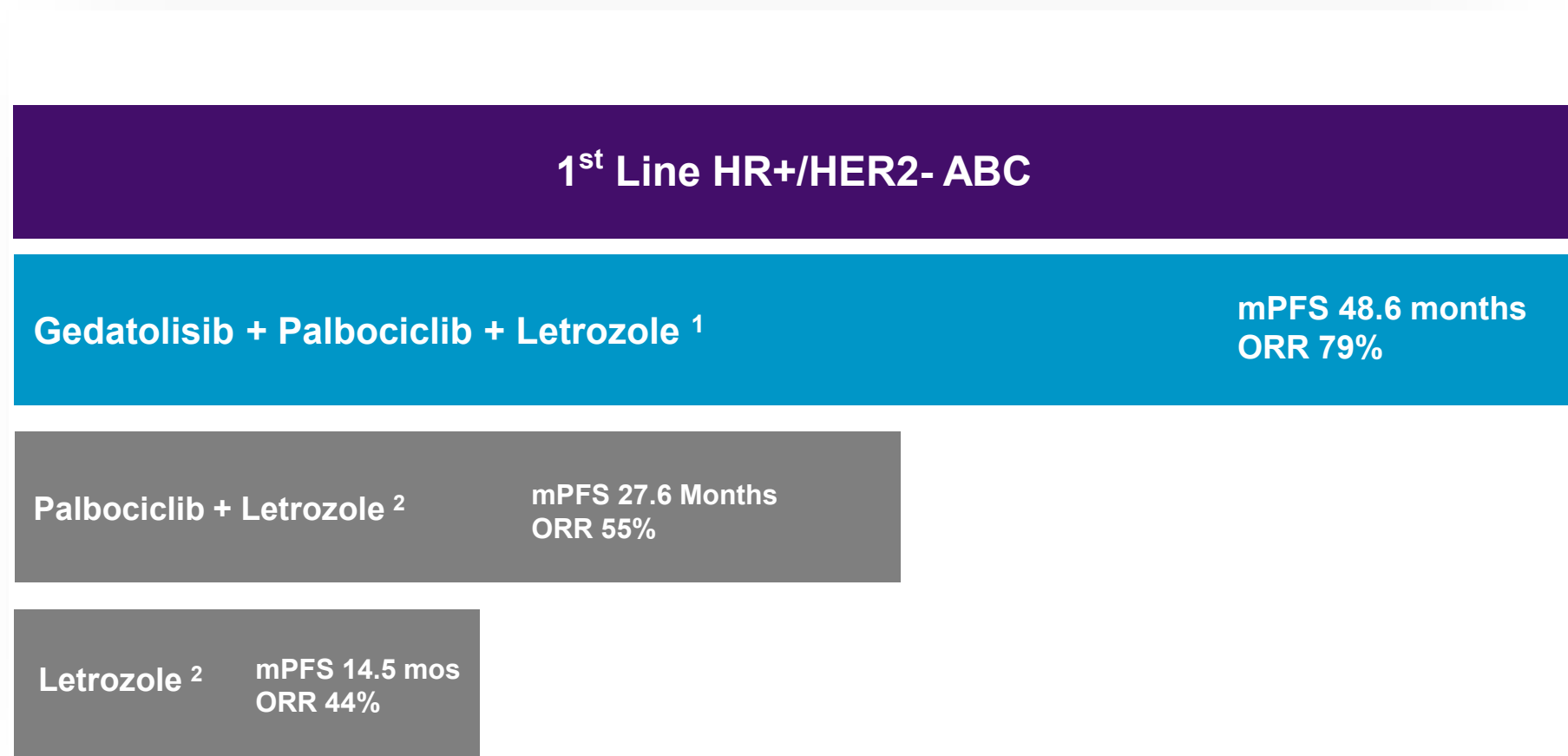


Tumor Size Change
ORR = 79% (26/33)



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

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



Phase 3 Study Design

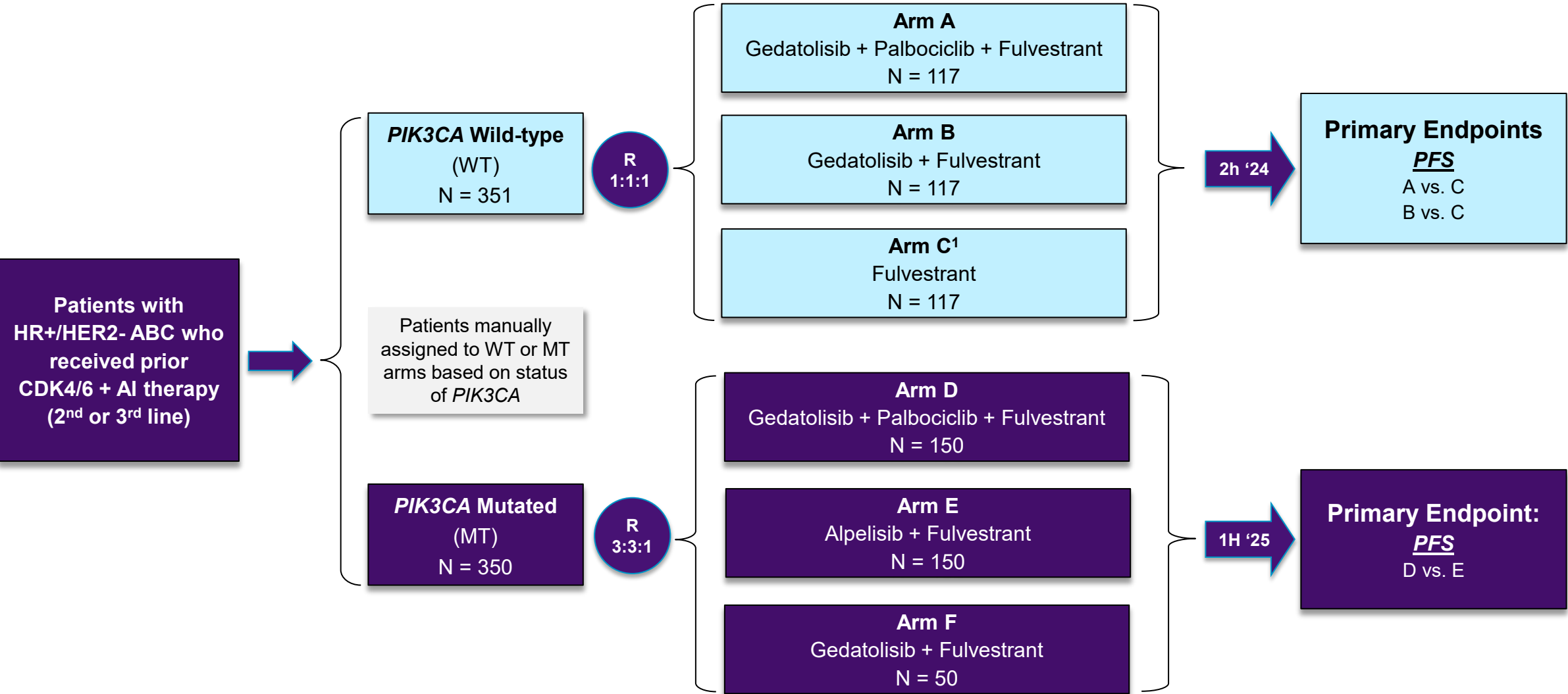
VIKTORIA-1

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



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

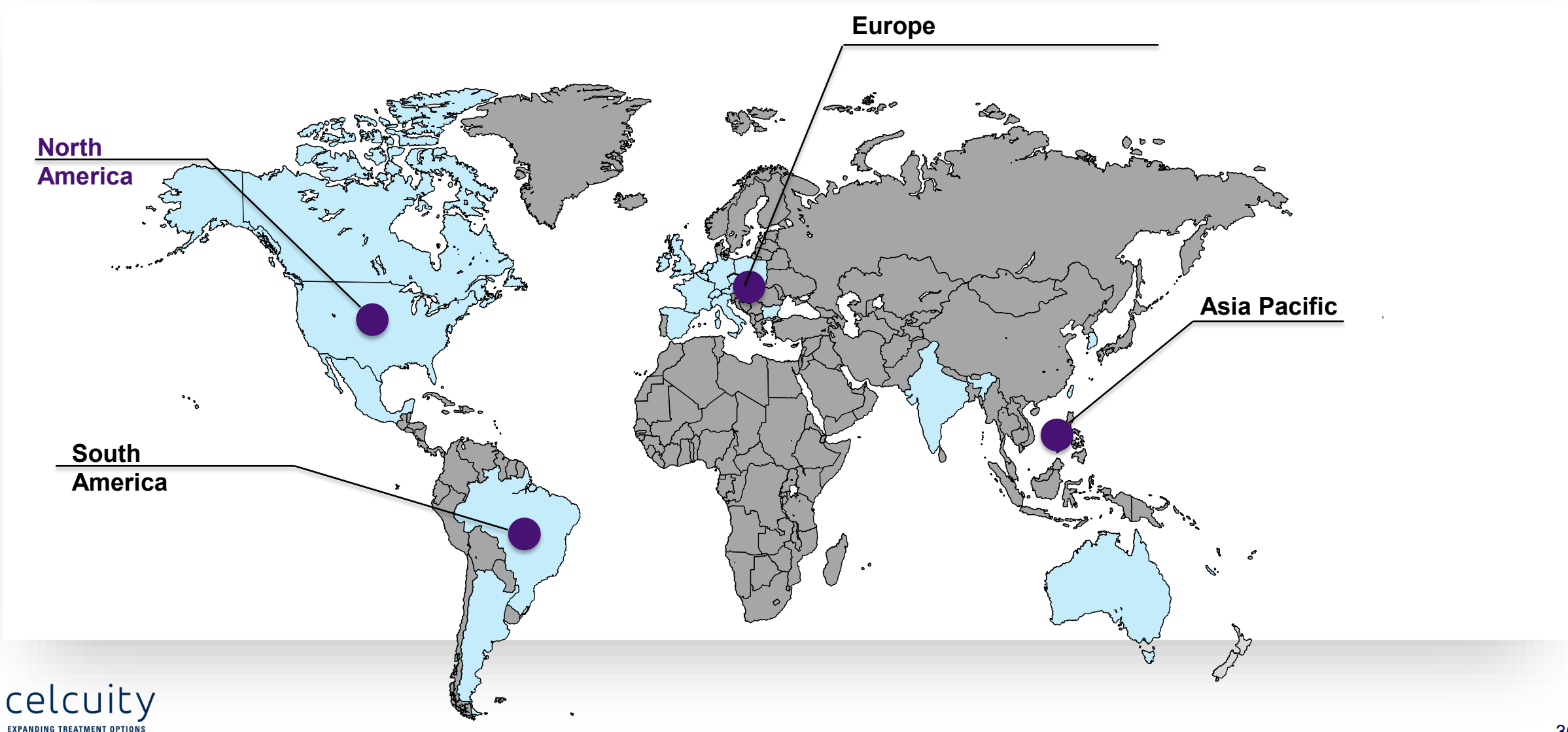
	Gedatolisib + Palbociclib + Fulvestrant N=27 ^{1,2}	Fulvestrant N=165 ³	Fulvestrant N=52 ⁵	Alpelisib + Fulvestrant N=126 ⁶	Alpelisib + Fulvestrant N=121 ⁷
PIK3CA Status	WT / M (56% / 41%)	WT	WT / MT (70% / 30%)	M	M
Line of Therapy (% by line)	2L / 3L+ (67% / 33%)	2L / 3L+ (73%/27%) ⁴	2L / 3L+ (83% / 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 <u>WT</u> 60% <u>M</u> 73%	NR	6%	22%	17%
PFS % at 12 months	53% (overall) <u>WT</u> 49% <u>M</u> 60%	10%	12%	22%	27%

VIKTORIA-1 Pivotal Study Features

- **Global open-label randomized study**
- **Key eligibility criteria:**
 - Any *PIK3CA* status
 - Prior CDK4/6i + NSAI
 - Any menopausal status
 - ≤ 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 and shows contribution of gedatolisib**
- **Stratification by geography, prior treatment response (\leq 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

200+ Sites Across 20 Countries





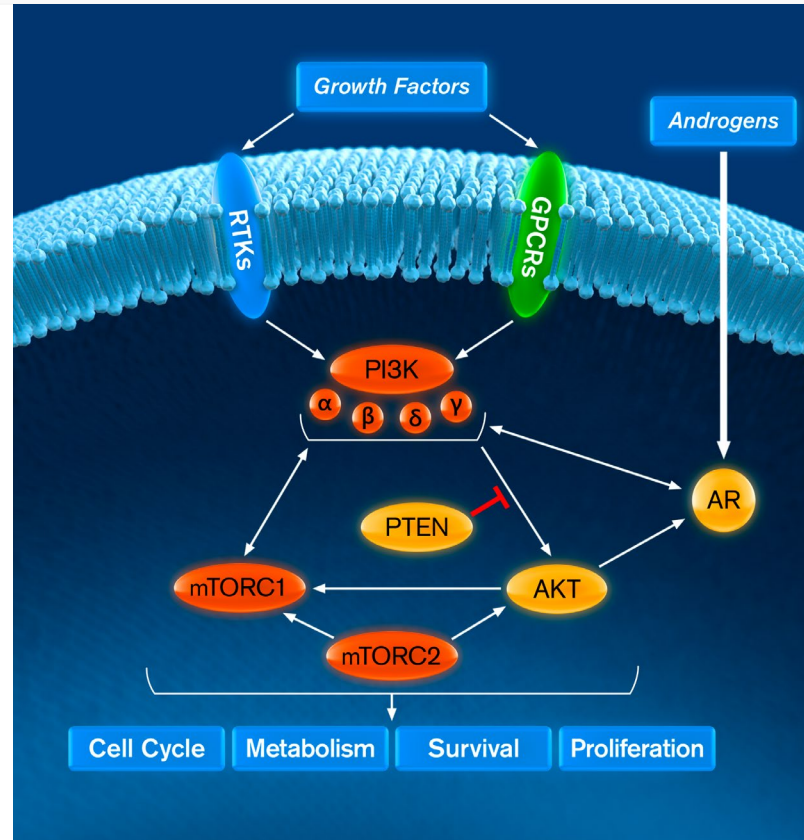
Gedatolisib for Prostate Cancer

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

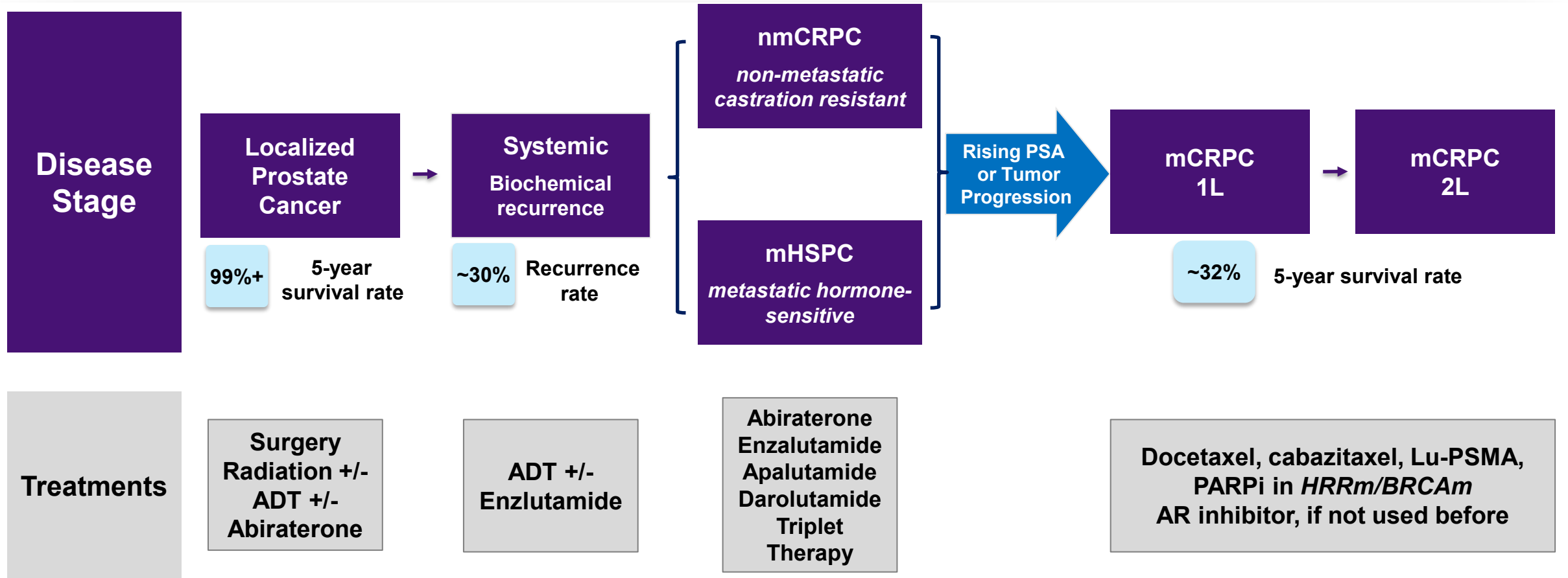


The PAM Pathway Plays a Key Role in mCRPC

- 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

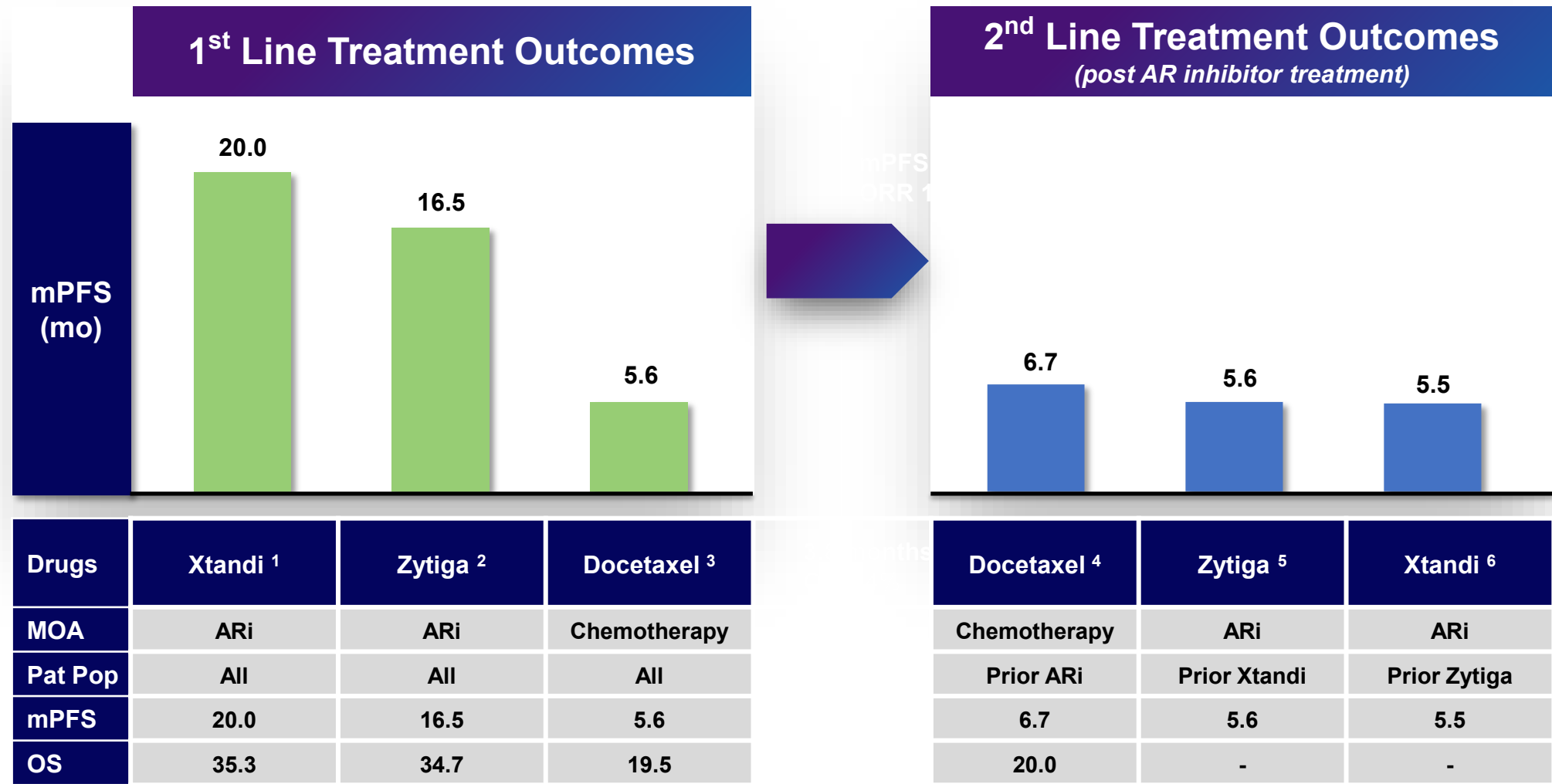
Prostate Cancer Disease and Treatment Landscape^{1,2}

34,700 men in US and 62,400 men in 5EU and Japan die from prostate cancer annually^{3,4}



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

Significant need for better therapeutic options



Karim Fizazi, MD, PhD
Gustave Roussy

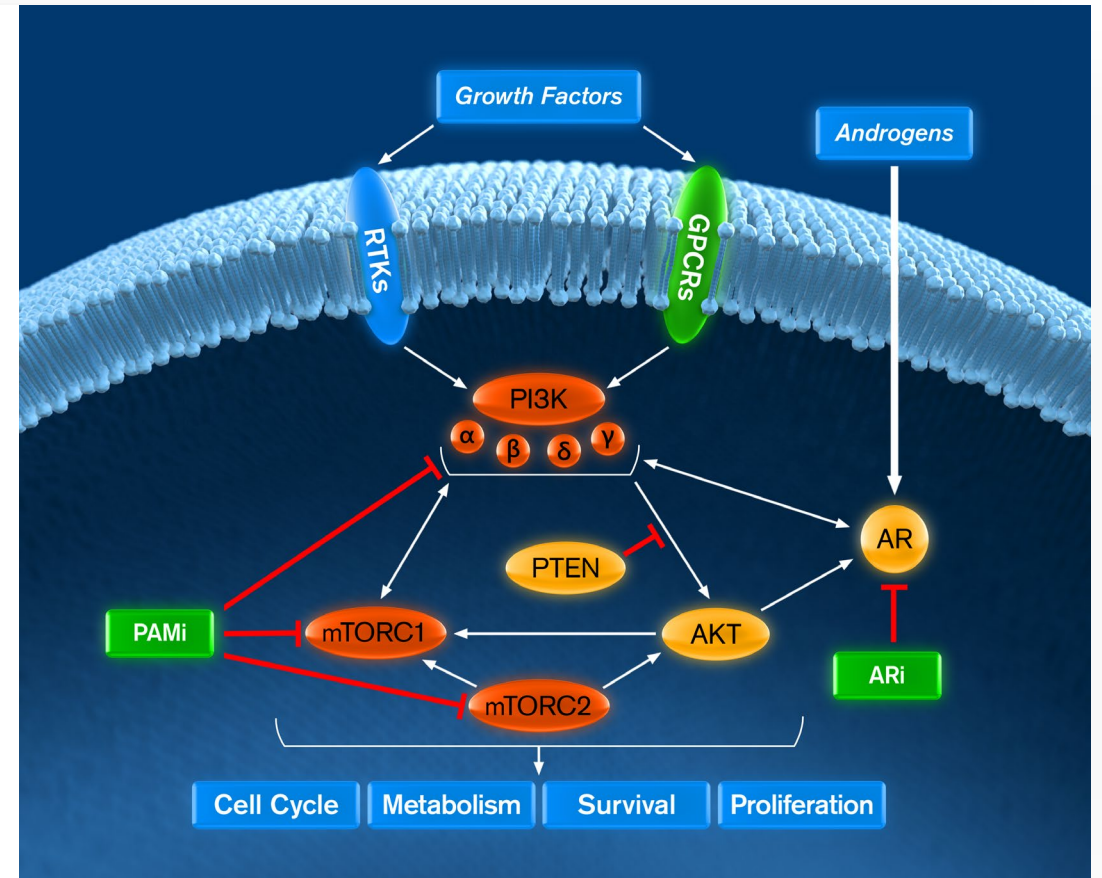
(1) Beer Eur Urol. 2017; (2) Ryan NEJM 2013; Ryan Lancet Oncol 2015 (3) Kellokumpu-Lehtinen Lancet Oncol. 2013, time-to-treatment failure reported; (4) Crabb J Clin Oncol 2021; (5) Attard J Clin Oncol 2018; (6) Sweeny Clin Cancer Res 2022. Abbreviations: HRR = homologous recombination repair; AR = androgen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

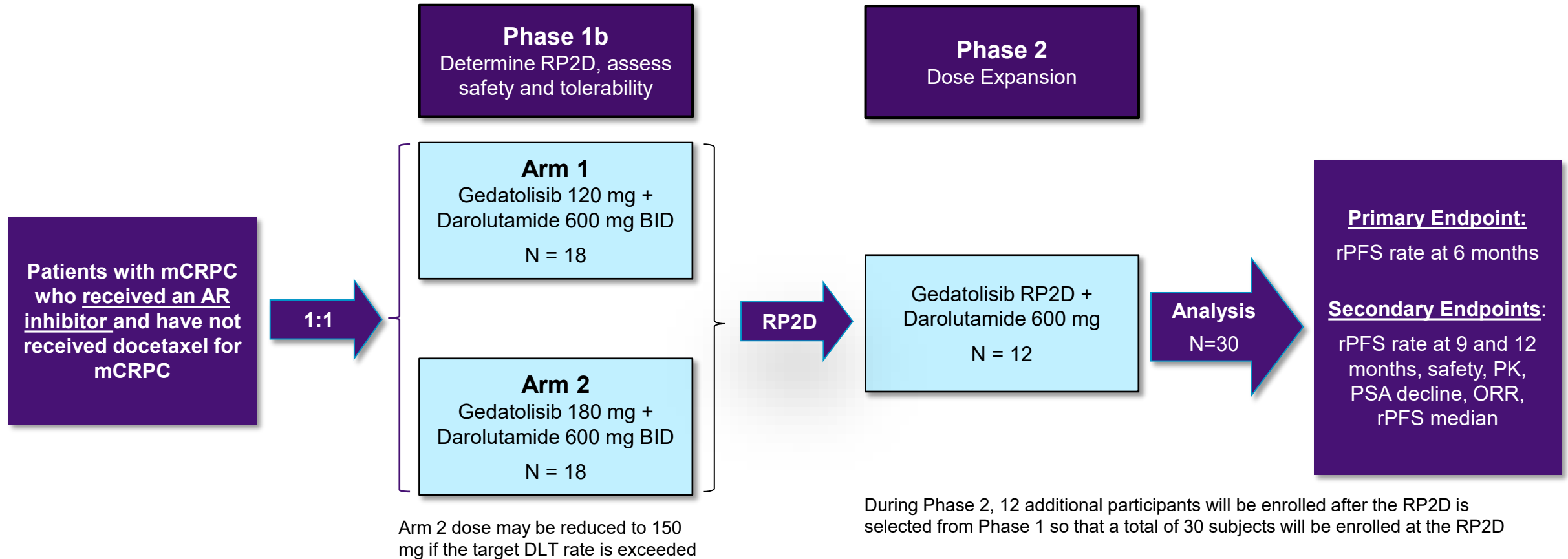
PI3K/mTOR + AR Inhibition *Treatment Rationale*

- 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



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

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



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

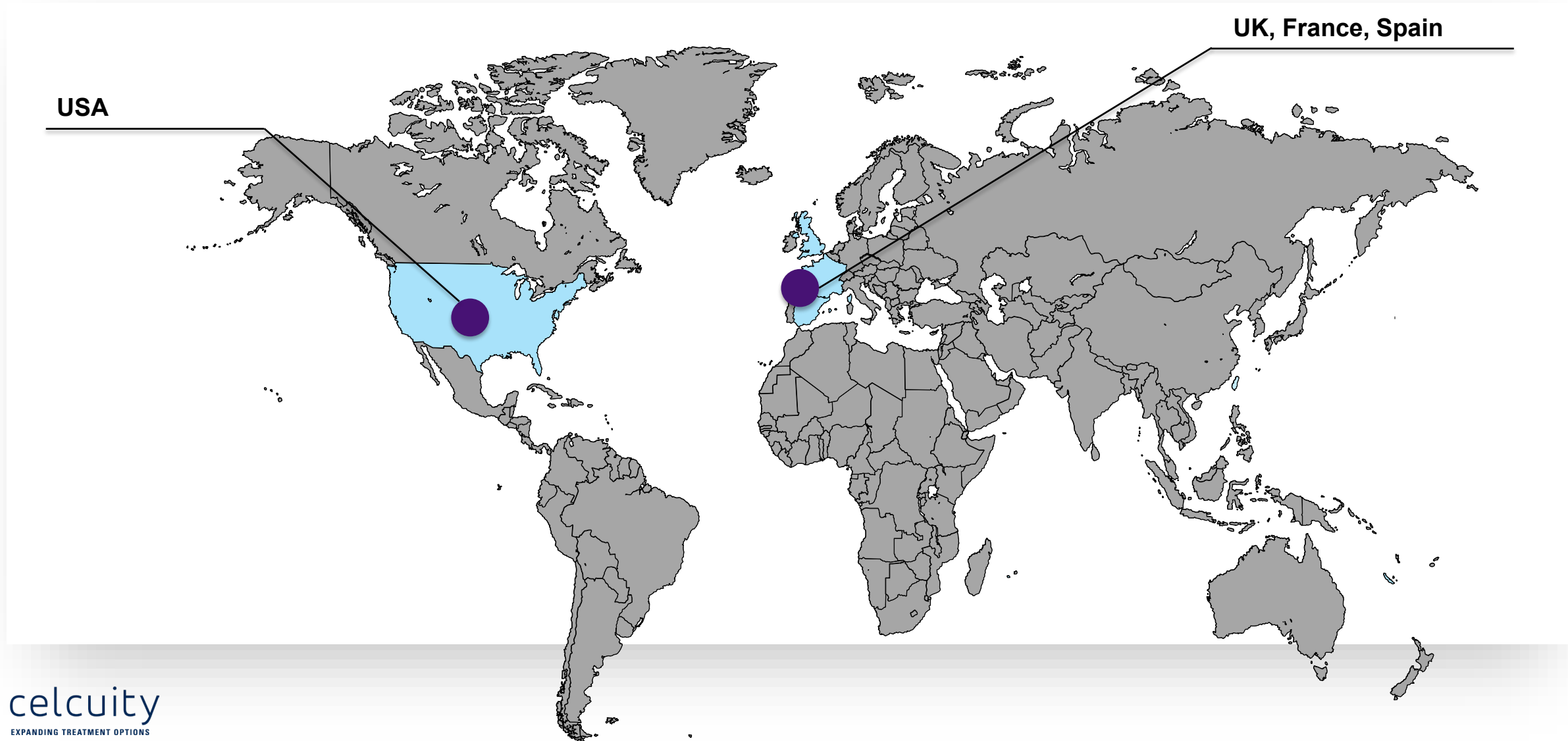
Darolutamide is More Potent and Better Tolerated than SOC 1L AR Inhibitors

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

~12 Sites Across US and Europe

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

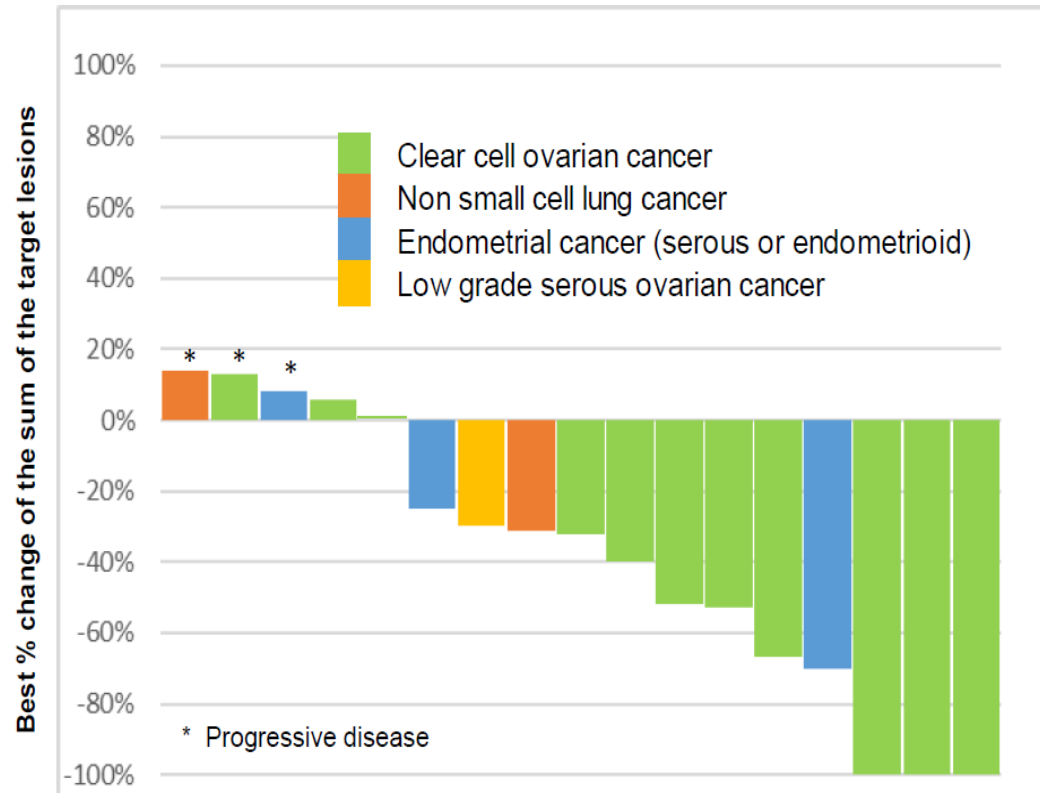




Additional Early Phase Clinical Data

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

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



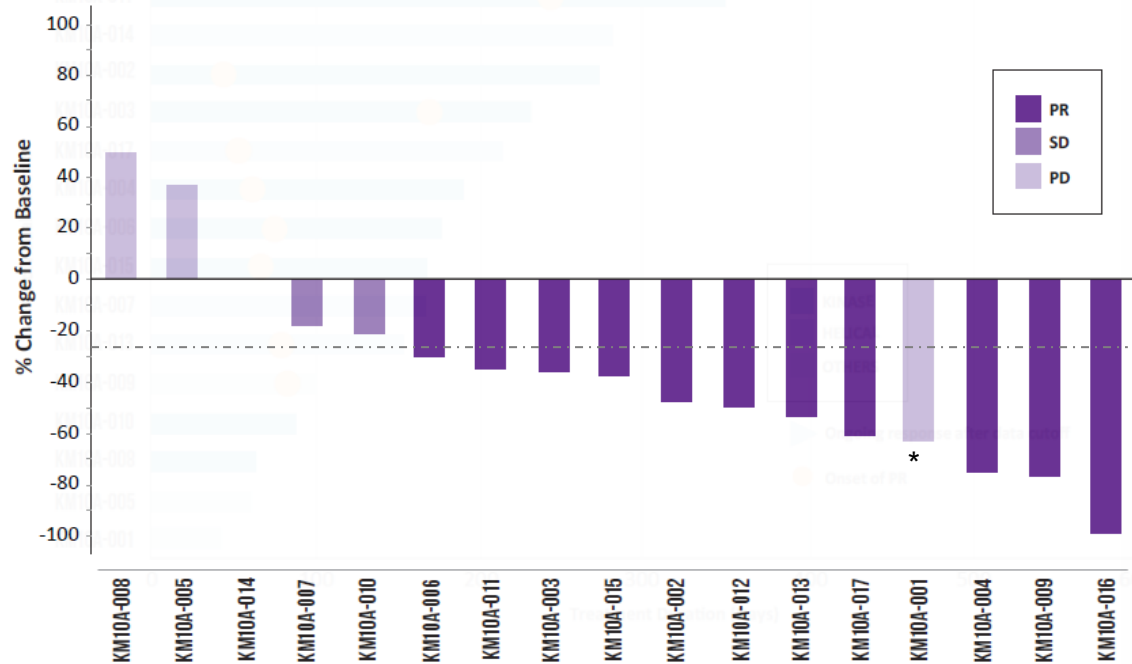
- Ovarian Cancer (N=11)
 - ORR: 82%
 - Clear cell ovarian cancer (CCOC) (N = 10)
 - ORR: 80% - 5/10 PR, 3/10 CR
 - Low grade serous ovarian (N=1)
 - 1/1 PR
- Other solid tumors (N= 6)
 - ORR = 33%
- Median PFS = 6.35 months (95% CI 4.6-11.11)
- Median duration of response = 7.6 months (95% CI 1.9-13.4)

- 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

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

59% ORR and 83% clinical benefit rate

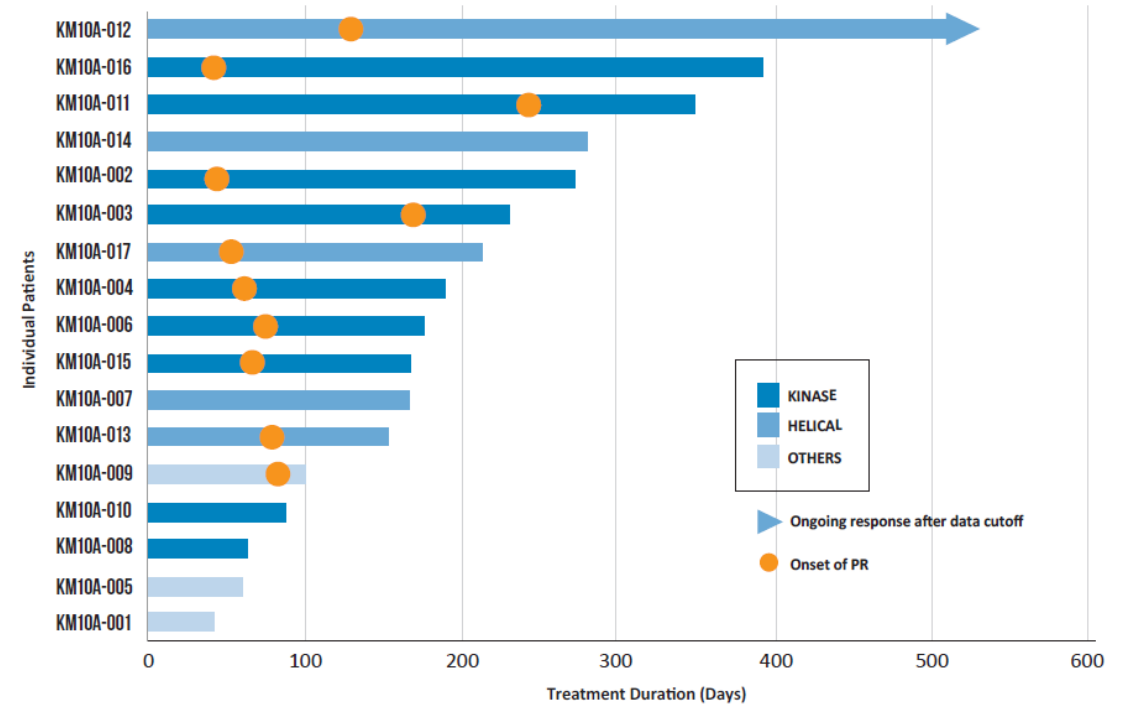
Best Response



* 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

Leading cancer KOLs are participating in our research

Clinical Advisory Board



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



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VP, Medical Affairs



Sunni Miller

VP, Regulatory Affairs

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 \$146M at end of Q2 '23 expected to fund operations through data readouts in ABC and mCRPC



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



“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



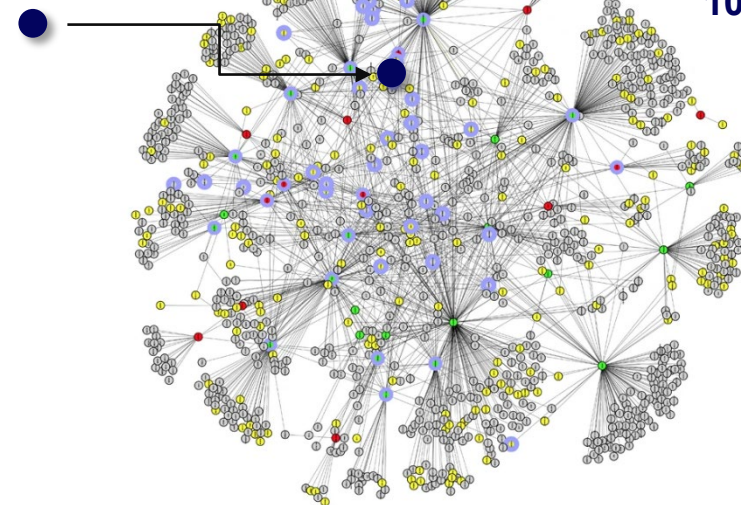
Memorial Sloan Kettering
Cancer Center

“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



Single gene mutation

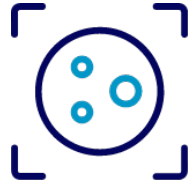


**Signaling Pathway
Network:
 10^{20} cascading
events**

CEL_{signia} – the first 3rd generation diagnostic

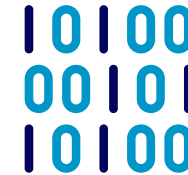
Measures dynamic cell signaling activity to identify cancer drivers genomic tests cannot detect

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 activity¹

Algorithmic Analysis

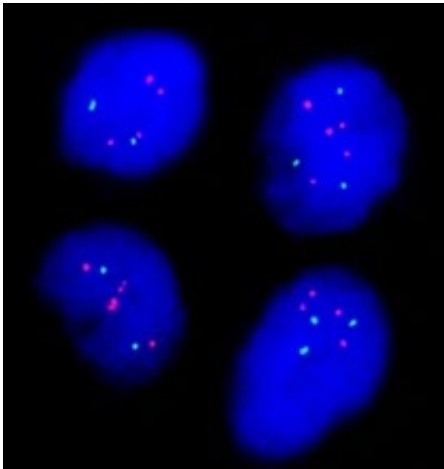


A **proprietary algorithm analyzes this “big data”** set to identify signaling activity 5 standard deviations from normal

Current Molecular Diagnostics vs. CELsignia – HER2 Example

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

FISH HER2 Dx
(1 pathway gene)



\$9 billion
anti-HER2 drug annual revenue¹

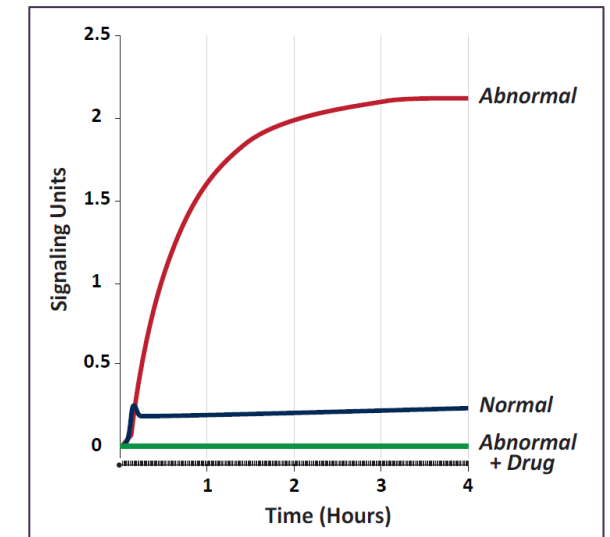
FISH+
15%

CELsignia+
15%-20%



CELsignia identifies new
patients for anti-HER2 drugs

CELsignia HER2 Activity
(4 hours of pathway signaling events)



\$Billions additional
anti-HER2 drug revenue potential

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