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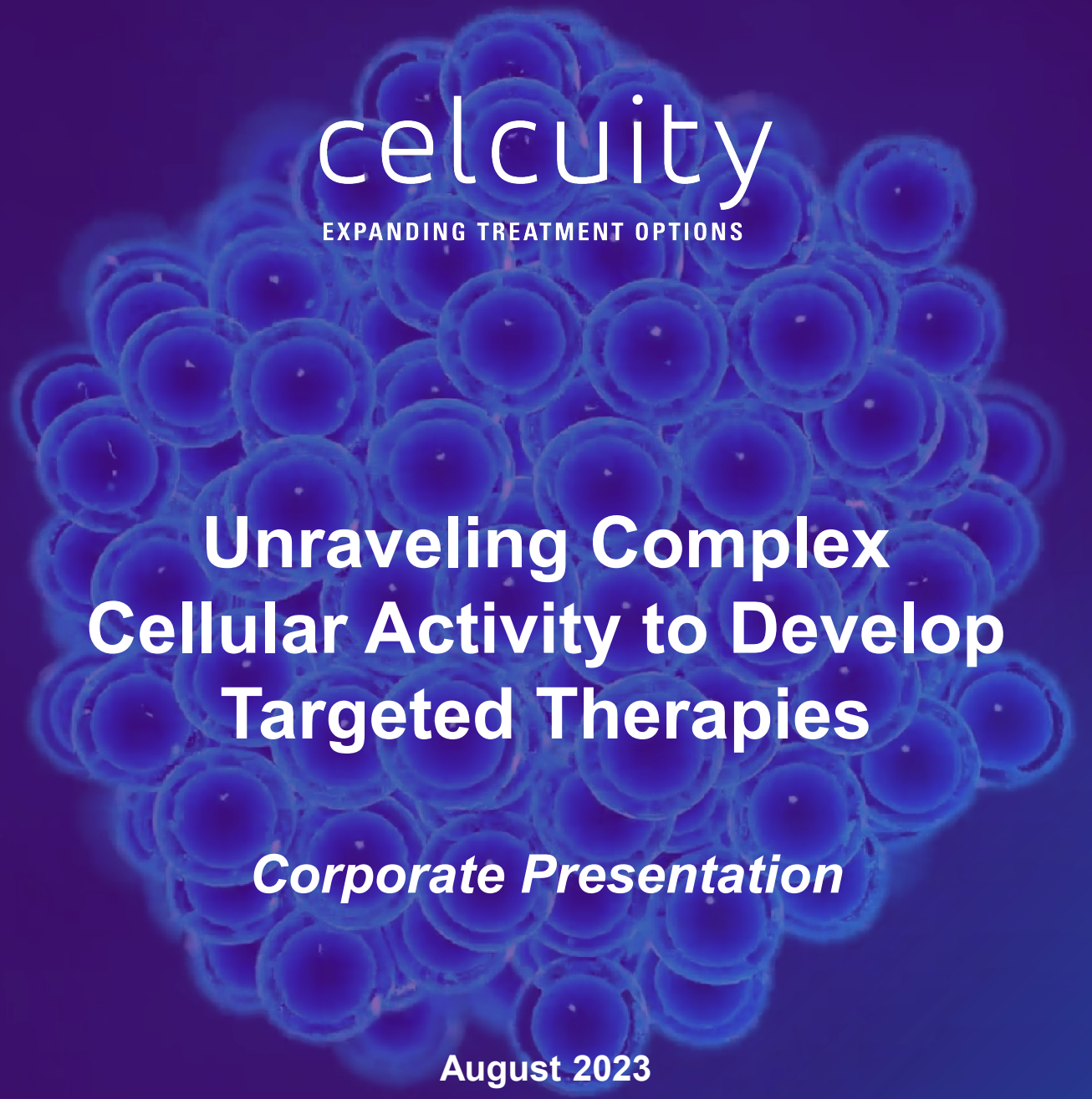
EXPANDING TREATMENT OPTIONS



Unraveling Complex Cellular Activity to Develop Targeted Therapies

Corporate Presentation

August 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 scope, protocol, and costs of our clinical development program and upcoming clinical trials for gedatolisib, including but not limited to our VIKTORIA-1 Phase 3 clinical trial and our Phase 1b/2 CELC-G-201 clinical trial, the expected results of VIKTORIA-1 and CELC-G-201, including but not limited to the anticipated efficacy of gedatolisib in combination with fulvestrant and with or without palbociclib, the anticipated efficacy of gedatolisib in combination with darolutamide, 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, “may,” “will,” “look forward to,” “believe,” “expects,” “anticipates,” “potential,” “intends,” “goal,” “estimates,” “predicts,” “intend,” “plans,” “would,” “should,” “could,” or “continue” and similar expressions or words, identify forward-looking statements.

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Because forward-looking statements are inherently subject to risks and uncertainties, some of which cannot be predicted or quantified and some of which are beyond our control, you should not rely on these forward-looking statements as predictions of future events. In our reports and filings with the SEC, including our Annual Report on Form 10-K for the year ended December 31, 2022, we present more information about the risks and uncertainties applicable to our business. New risks and uncertainties may emerge from time to time, and it is not possible to predict all risks and uncertainties. Except as required by applicable law, we do not plan to publicly update or revise any forward-looking statements contained herein, whether as a result of any new information, future events, changed circumstances or otherwise.

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Unraveling Complex Cellular Activity to Develop Potential First-in-Class Targeted Therapies

Our CELsignia platform creates a **“movie” of signaling activity** in live patient tumor cells.

Detects oncogenic pathway activity that molecular tests cannot identify

Enables discovery of new cancer drivers and **expands the market for targeted therapies**.

Leveraging our platform to develop gedatolisib, a potentially **first-in-class pan-PI3K/mTOR inhibitor**

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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

- **First** small molecule inhibitor of the PI3K/mTOR pathway administered intravenously
- Inhibits all isoforms of PI3K and mTOR at **low or sub-nanomolar** concentrations

Compelling Efficacy

- Compelling efficacy relative to 1st & 2nd line SOC with HR+/HER2- ABC with gedatolisib + ET + CDK4/6i
  - **79% ORR** and **48.6 months mPFS** in 1st line patients[1]
  - **63% ORR** and **12.9 months mPFS in** 2nd line patients[2]

Well-Tolerated

- Nominal Grade 3 and no Grade 4 TEAE's as a single agent
- Only 4% treatment discontinuation with Phase 3 dosing in combination with SERD + CDK4/6

Multiple Potential Indications

- **Breast Cancer**: Phase 3 trial for 2L patients with HR+/HER2- advanced BC is currently enrolling
- **Prostate Cancer**: Phase 1b/2 trial for 2L patients with mCRPC is expected to begin enrollment in Q1 '24
- **180,000 patients** potentially eligible globally with these two patient groups

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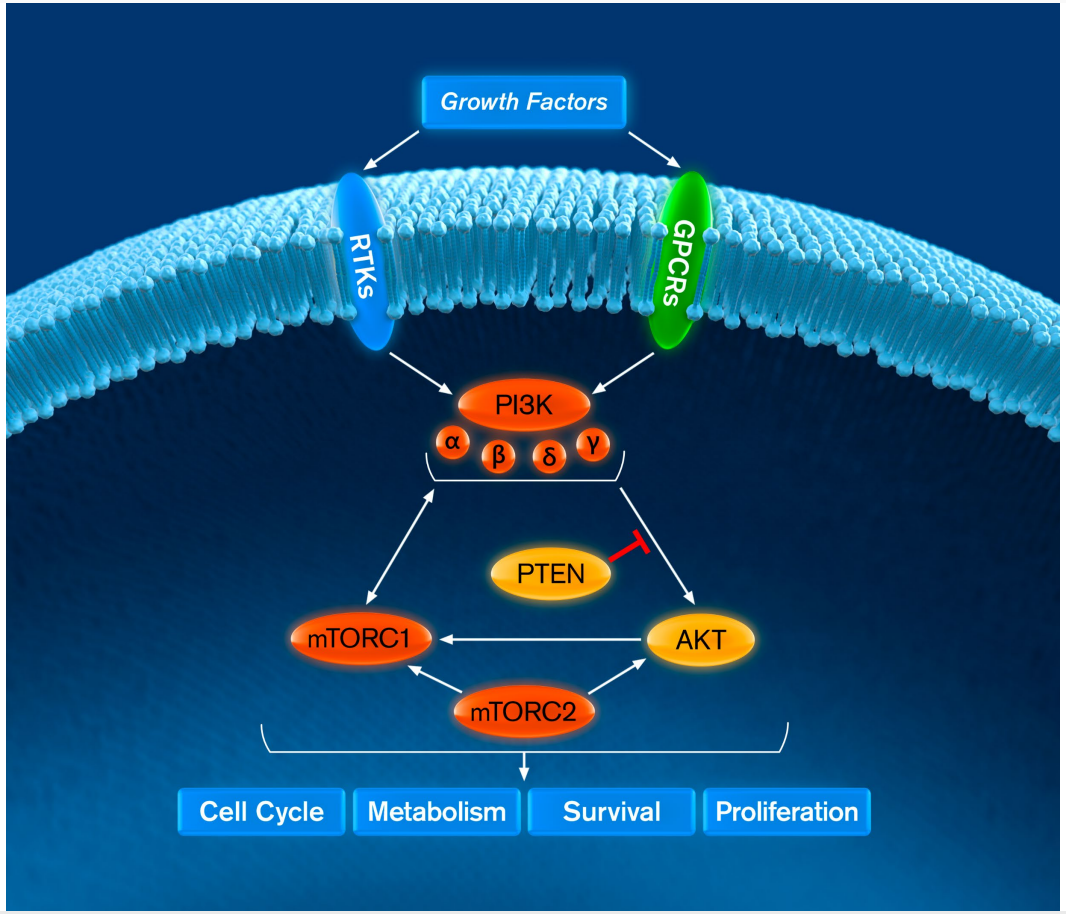
1) Combined data from treatment-naïve patients enrolled in Escalation Arm A and Expansion Arm A of the B2151009 Phase 1b clinical trial; 2) Data from Expansion Arm D of the B2151009 clinical trial. Abbreviations: SOC, standard-of-care; ORR, objective response rate; mPFS, median progression free survival; 1L, 1st line; 2L, 2nd line; TEAE, treatment emergent adverse events

PI3K/AKT/mTOR is One of Most Important & Complex Oncogenic Pathways

Key resistance mechanism for multiple oncogenic pathways

PI3K/mTOR regulates cell growth and metabolism

- Linked to multiple cell control decisions
- Can play a key role in driving cancer proliferation.
- Cross regulates ER, AR, CDK4/6 pathways
- >50% of breast, prostate, and endometrial tumors have PI3K pathway alterations



Tumor type	PI3K Pathway Alterations[1,2]
Endometrial	71%
Breast Cancer	59%
Prostate	56%
Colon	54%
Liver	54%
Cervix	53%
Kidney	46%
Bladder	44%
HNSCC	42%
NSCLC	29%
Ovarian	27%

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(1) Millis, Jama Oncol 2016; (2) PI3K pathway alterations include PIK3CA, PTEN, AKT mutations, and PTEN loss

Difficult to Safely and Efficaciously Inhibit PI3K/AKT/mTOR

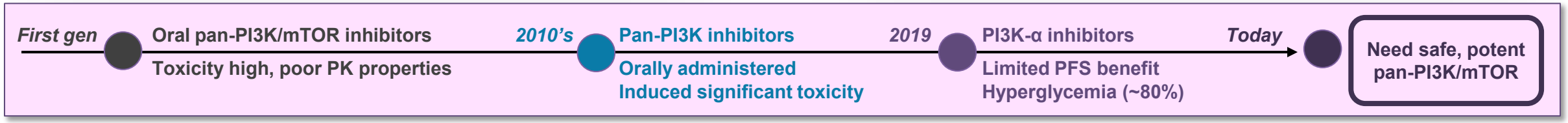
Maximum efficacy requires inhibition of all Class 1 PI3K isoforms and mTORC1 and mTORC2

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 or mTOR inhibitors is narrow

- Difficult to achieve optimal pathway inhibition without inducing undue toxicities in patients
- Orally administrated pan-PI3K or pan-PI3K/mTOR inhibitors induced unacceptable toxicity



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Gedatolisib Has a Highly Differentiated Mechanism of Action

Only equipotent pan-PI3K/mTOR inhibitor known to be under active development

Gedatolisib differentially targets one of the most important and complex oncogenic pathways

- First pan-PI3K/mTOR inhibitor with low nanomolar potency that is well tolerated with manageable toxicities
- Pan-PI3K/mTOR inhibition limits cross-activation that occurs with PI3K isoform, AKT or mTOR specific drugs
- MOA creates potential to induce anti-tumor activity independent of PIK3CA status

Gedatolisib vs. Approved Solid Tumor PI3Ki or mTORi
IC_{50} (nM)[1]

Target	Gedatolisib[2]	Alpelisib[3]	Everolimus[4]
PI3K-α (MT)	0.6	~4.0	-
PI3K-α (WT)	0.4	4.6	-
PI3K-β	6.0	1,156	-
PI3K-γ	5.4	250	-
PI3K-δ	6.0	290	-
mTORC1	1.6	-	~2.0
mTORC2	1.6	-	-

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(1) IC50 derived from cell-free biochemical dose response analysis; (2) Venkatesan 2010 for PI3K and mTORC1 IC50 values; (3) Fritsch 2014; (4) Schuler 1997; everolimus is an mTOR inhibitor that binds with high affinity to the FK506 binding protein-12 (FKBP-12), thereby forming a drug complex that inhibits the activation of mTOR

Gedatolisib PK Properties vs. Other Approved PI3K Inhibitors

Differentiated chemical structure results in favorable PK profile and lower toxicity

	Gedatolisib[1]	Alpelisib[2]	Copanlisib[2]	Duvelisib[2]	Idelalisib[2]
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)	30	114	*871*	29	23
AUC plasma (ug.h/mL)	47.1	33.2	*1.6*	7.9	10.6
Cmax (ug/mL)	8.6	2.5	0.5	1.5	1.9
Half-life (hours)	37	8-9	39	5	8
Hyperglycemia (G 3/4) [3]	7%	*37%*	*41%*	-	-
Treatment related SAE's[3]	7%	*35%*	*26%*	65-73%	50-77%
Treatment related (TR) Discontinuations[3]	4%	*26%*	*16%*	35%	17-53%

Gedatolisib vs. PI3K-α and pan-PI3K drugs

- 80% lower rate of Grade 3/4 hyperglycemia
  - Due to gedatolisib's lower liver exposure
  - Alpelisib dosage 22x > gedatolisib
  - Copanlisib 50x > retention liver vs plasma
- 75%-85% lower rate of TR discontinuations
- 3.5x-20x higher C_{max}
- 4x-30x more efficient distribution in plasma
- 1.5x-30x higher AUC plasma

Gedatolisib vs. PI3K-δ drugs

- 73%-97% lower dosage (molar/month)
- Minimal GI, liver, and infection-related AE's
  - Gedatolisib has lower GI exposure

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(1) Shapiro 2015; B2151009 Arm D; internal Celcuity studies; (2) US Package Insert; (3) 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; AUC, area under the curve

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 planned to begin Q1 2024

- Extensive literature describes androgen and estrogen 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

Gedatolisib for Advanced Breast Cancer (ABC)

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HR+/HER2- Advanced Breast Cancer (ABC)

Background

Significant Population with High Clinical Unmet Need [1,2]

- 58,000 women are diagnosed annually with HR+/HER2- ABC in the US.
- The five-year survival rate is only 30%.
- 43,700 women die of ABC annually in the US.

Endocrine Therapy (ET) with a CDK4/6 Inhibitor is 1L SOC

- The treatment landscape for 1st line ABC changed in 2015 when the first CDK4/6 inhibitors were approved.
- The typical median progression free survival period for ET + CDK4/6 is 25-28 months
- Efficacy of 2nd line therapies in post-CDK setting declined

PI3K-AKT-mTOR Pathway Dysregulation is a Disease Driver of HR+ ABC

- Clinical and nonclinical data confirm that ER, CDK4/6, and PI3K pathways cross-regulate each other
- PIK3CA and mTORC1 inhibitors are approved, confirming pathway's role
- ~60% of tumors have either PIK3CA mutations or PTEN alterations[3]

(1) Sheffield, K. M. et al. (2022); (2) American Cancer Society; (4) Millis Jama Oncol 2016;.

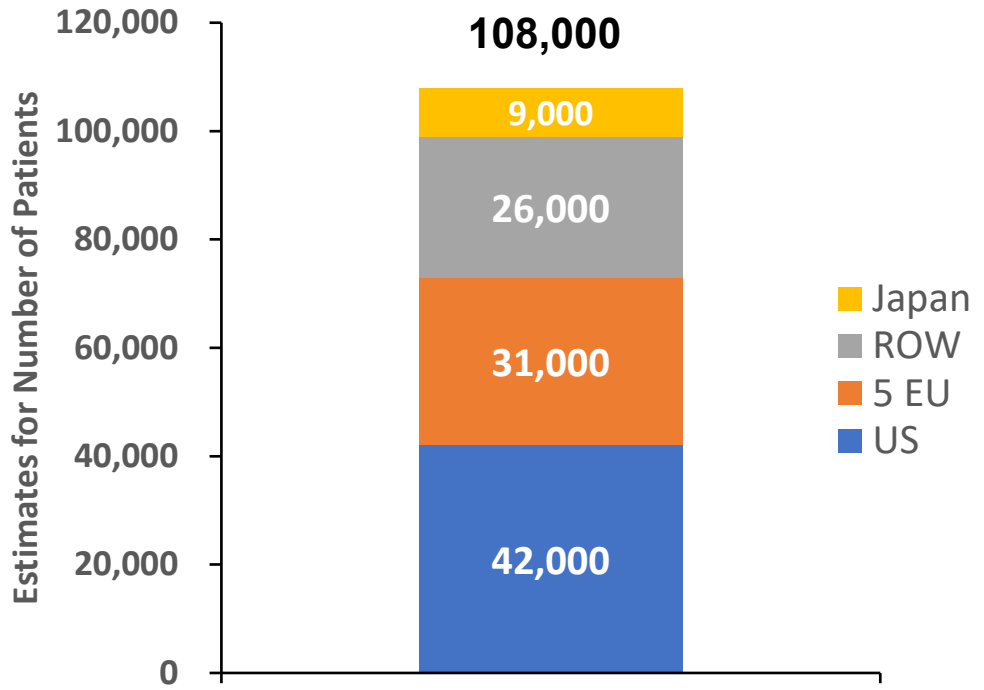
Limited Benefit for 2nd Line HR+/HER2- ABC Patients Post-CDK4/6 Treatment

Guidelines recommend sequential endocrine therapy until all endocrine therapy options have been exhausted

Current 2nd Line Standard-of-Care (Post CDK4/6 Treatment)

Treatment	Patient Sub-Group	mPFS (months)	ORR[1]
Fulvestrant [2, 3]	*ESR1* WT *PIK3CA* WT	1.9	6%
Elacestrant [2]	*ESR1* MT	3.8	7%
Everolimus + Exemestane [4]	*PIK3CA WT*	Unknown	
Alpelisib + Fulvestrant [5, 6]	*PIK3CA MT*	5.6 - 7.3	17% - 22%

Patient Population (Received CDK4/6 Therapy)



(1) ORR is for patients with measurable disease; (2) Bidard 2022, EMERALD trial; Elascestrant NDA/BLA Multi-Disciplinary Review and Evaluation (3) Lindeman 2021, VERONICA trial; (4) No prospective clinical trials have been conducted for this regimen in this patient population; (5) Rugo 2021, BYLieve trial; (6) B Moy 2021, JO Brett 2021; GJ Lindeman 2021. (7) Pfizer, Eli Lilly and Novartis 2021 annual reports; Datamonitor Healthcare; ROW calculated using 84% EU scale up factor. Abbreviations: ORR = objective response rate; mPFS, progression free survival; WT, wild type; MT, mutation

Review of Phase 1b Data

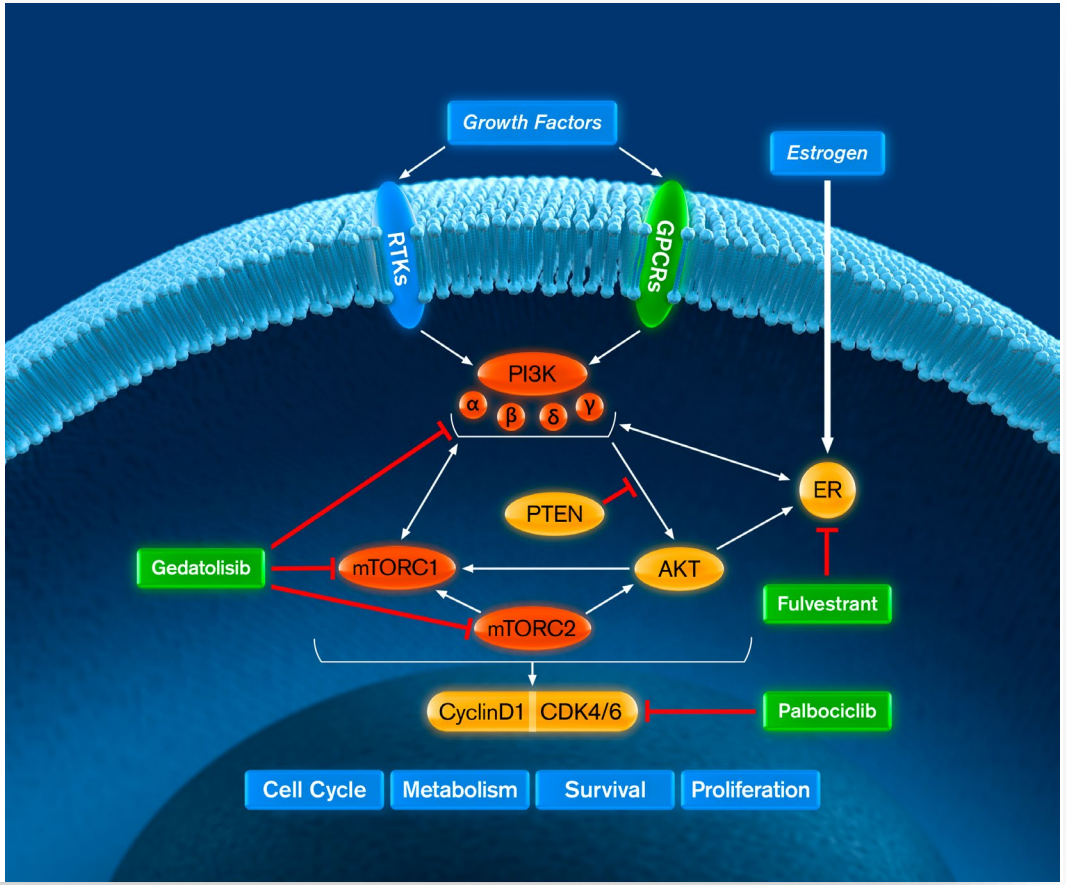
Gedatolisib + Palbociclib + Fulvestrant/Letrozole

PI3K/mTOR, ER, and CDK4/6 are Interdependent Signaling Pathways

PI3K/mTOR is a key resistance mechanism to estrogen and CDK4/6 therapies

PI3K/mTOR + ER + CDK4/6 Inhibition *Treatment Rationale*

- Simultaneously blocking interdependent ER, PI3K, mTOR & CDK signaling pathways in ER+ breast cancer addresses ER and CDKi resistance mechanisms
- Inhibiting all PI3K isoforms and mTORC1/2 prevents resistance mechanisms that occur when only PI3K-α or mTOR are inhibited
- Leads to improved response rates and duration of response



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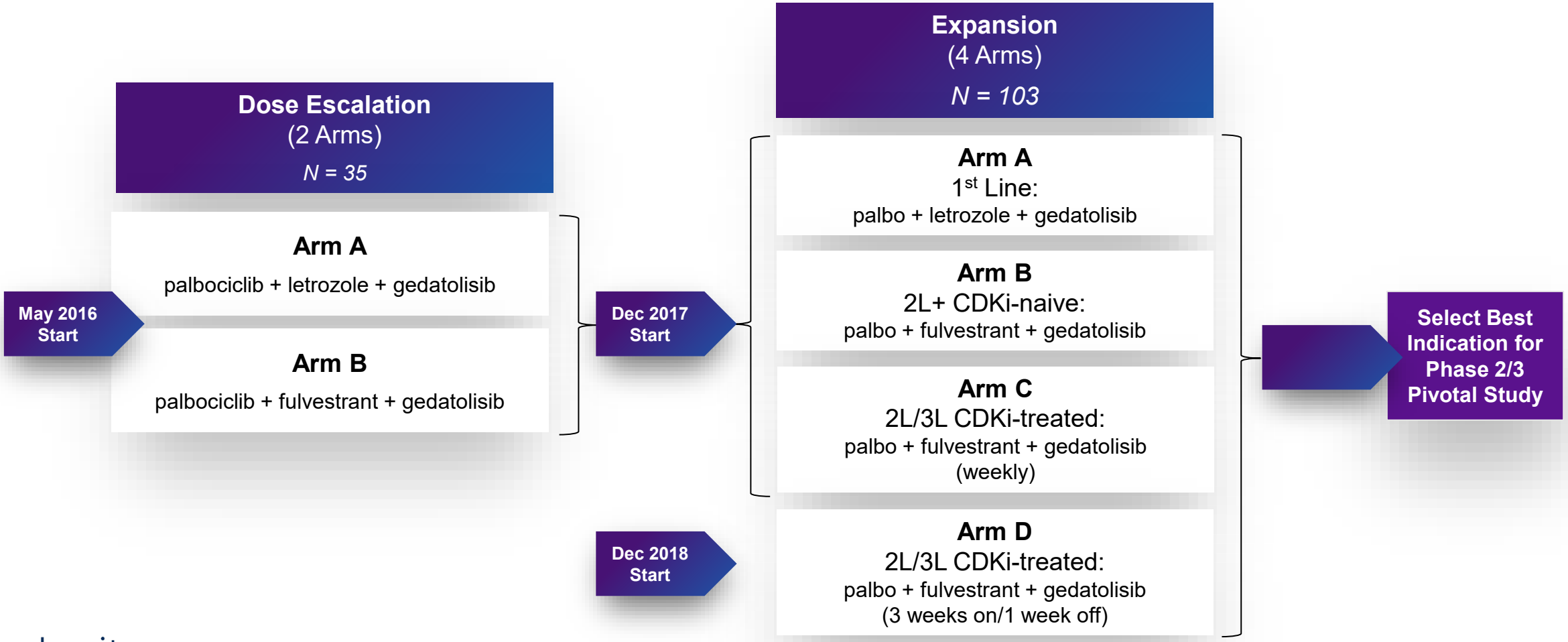
B2151009: Phase 1b Study (138 patients)

Dose escalation and safety/efficacy expansion (early signals of clinical activity)



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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 CDKi-naive		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)	48.6 (11.6, 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% [2,3]	16% [2,3]	69%	31%	75%[2]	25%[2]	**56% [2,3]**	**41% [2,3]**
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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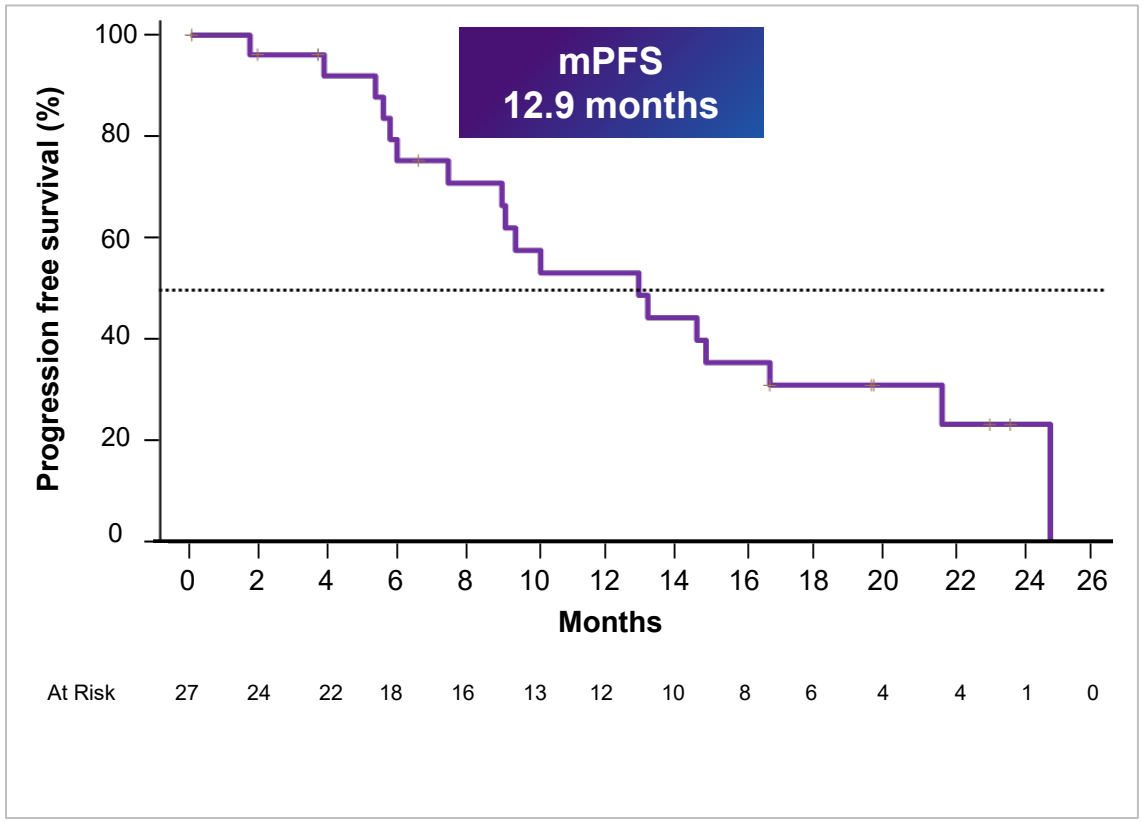
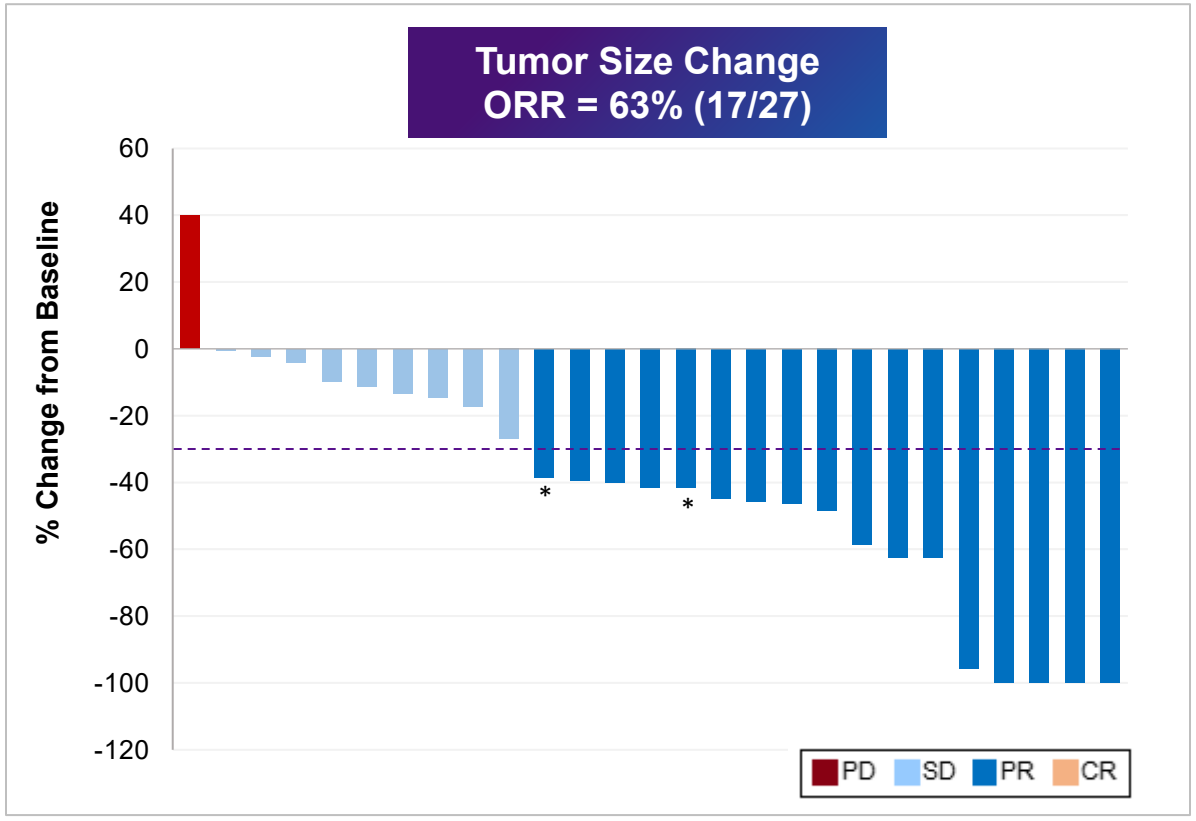
Source: Wesolowski 2022 SABCS; Rugo 2023 ESMO-Breast. Footnotes: (1) Response evaluable analysis set per RECIST v1.1 including uPR; (2) full analysis set; (3) Baseline *PIK3CA* mutation status missing for one patient. Abbreviations: 1L, first line, 2L, second line; mos, months; MT, *PIK3CA* mutation; 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

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





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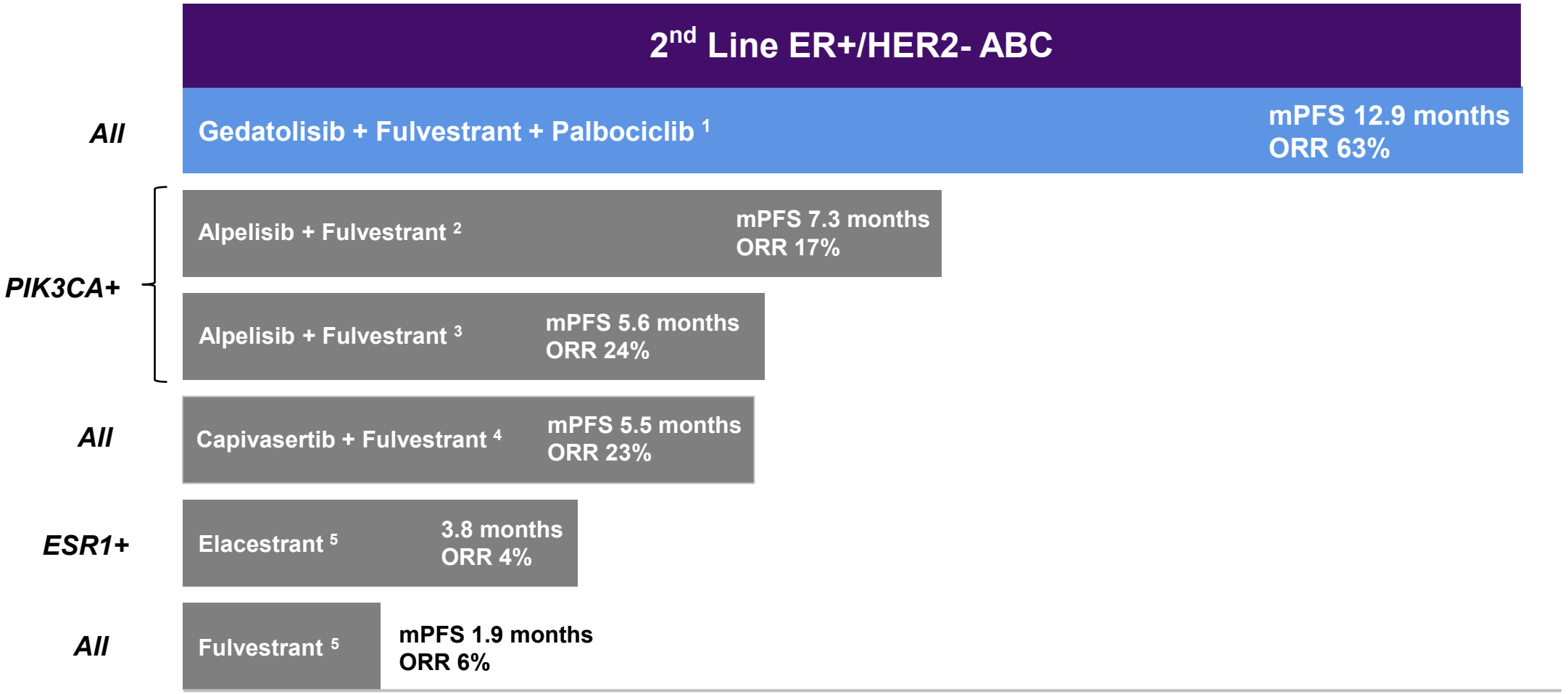
Source: Layman 2021 SABCS, Wesolowski 2022 SABCS - Arm D data from B2151009 study. ORR includes 2 unconfirmed PRs; *unconfirmed PR. Data presented is from a data analysis cutoff as of June 29, 2022

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

Gedatolisib Combo Offers Potential for Superior Efficacy Compared to Altneratives



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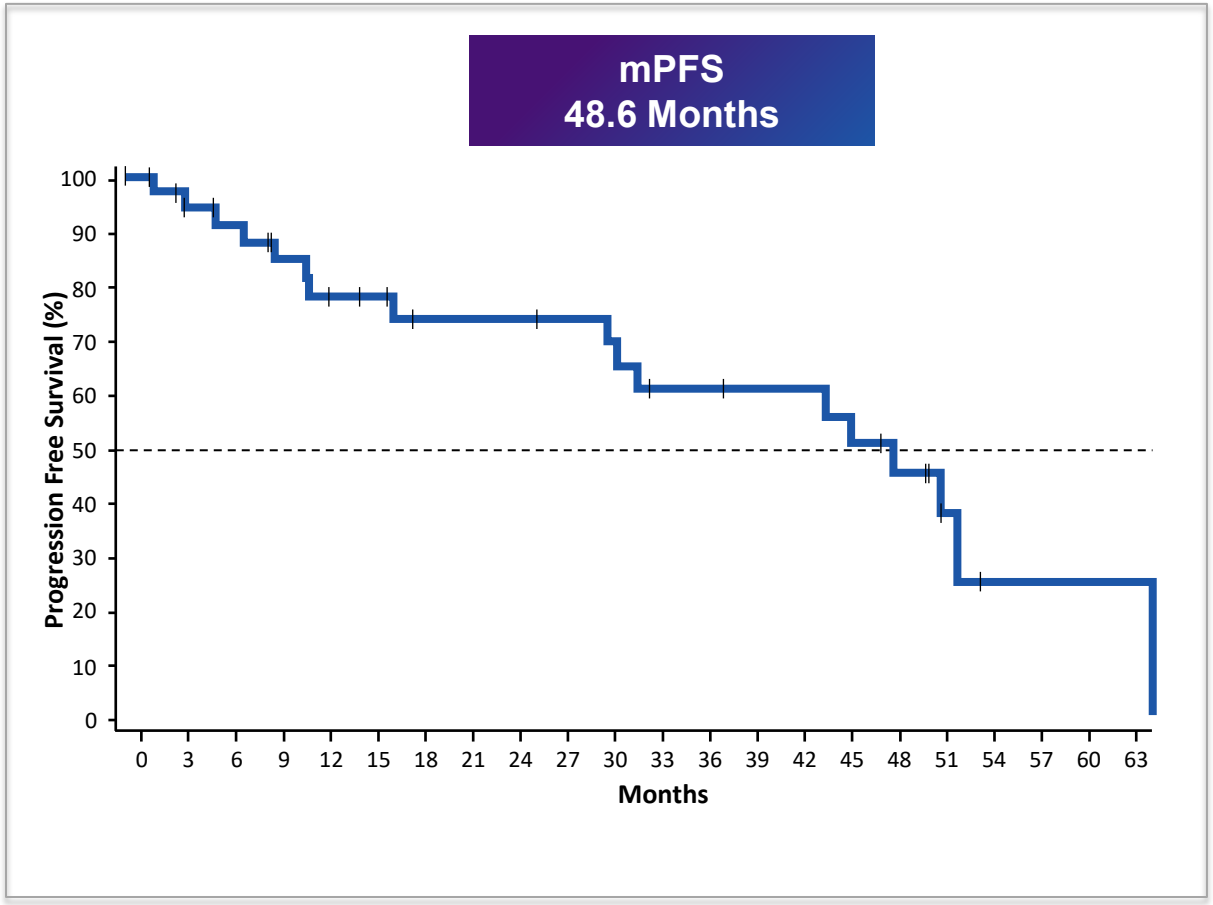
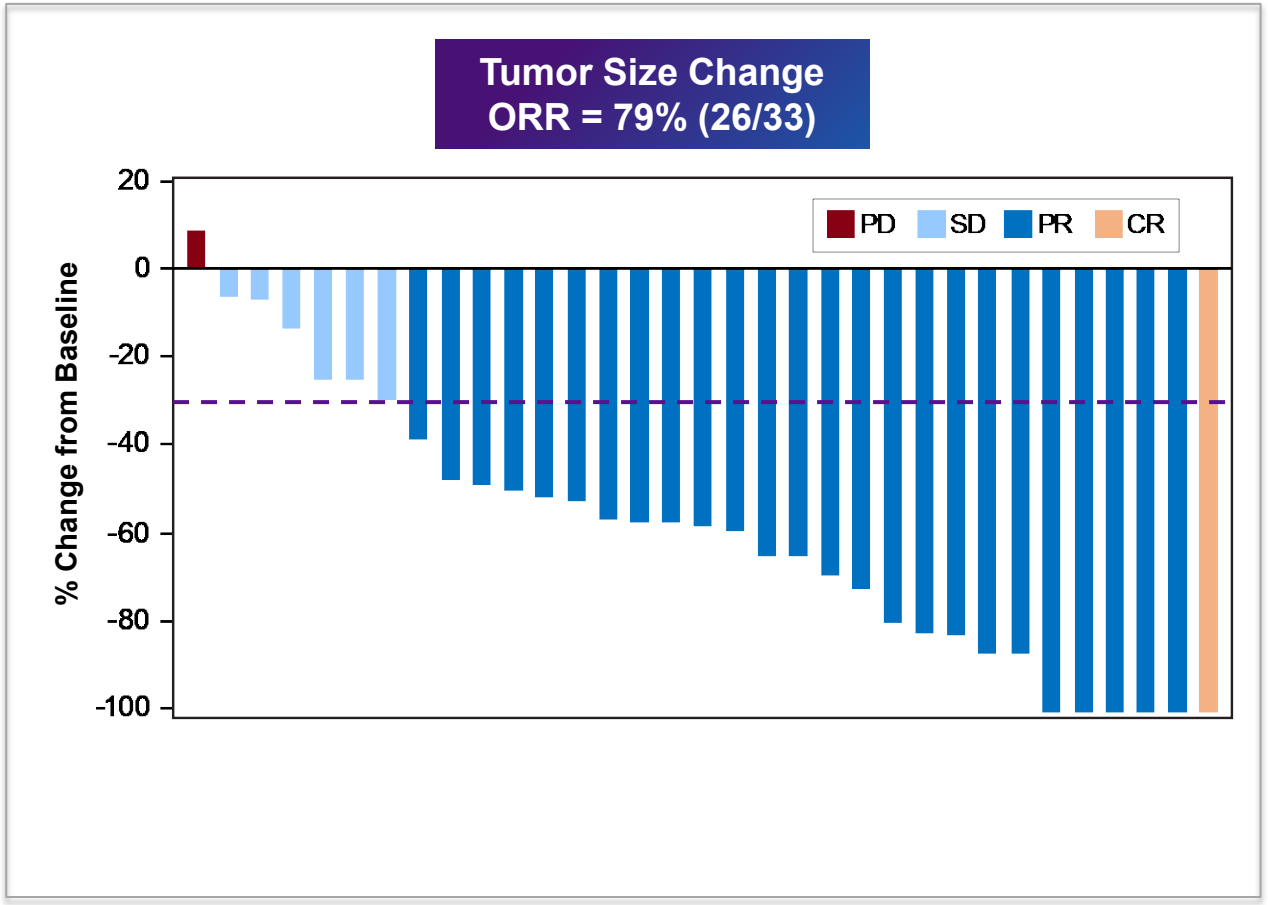
(1) Wesolowski SABCS 2022, Arm D; (2) Rugo, Lancet Onco, 2021; (3) Rugo, SABCS, 2021;(4) Oliveira, ESMO Breast, 2023, CDK4/6 prior treated patients (5) Bidard, JCO, 2022 and FDA
Note: All drugs listed are FDA approved, except for capivasertib

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]





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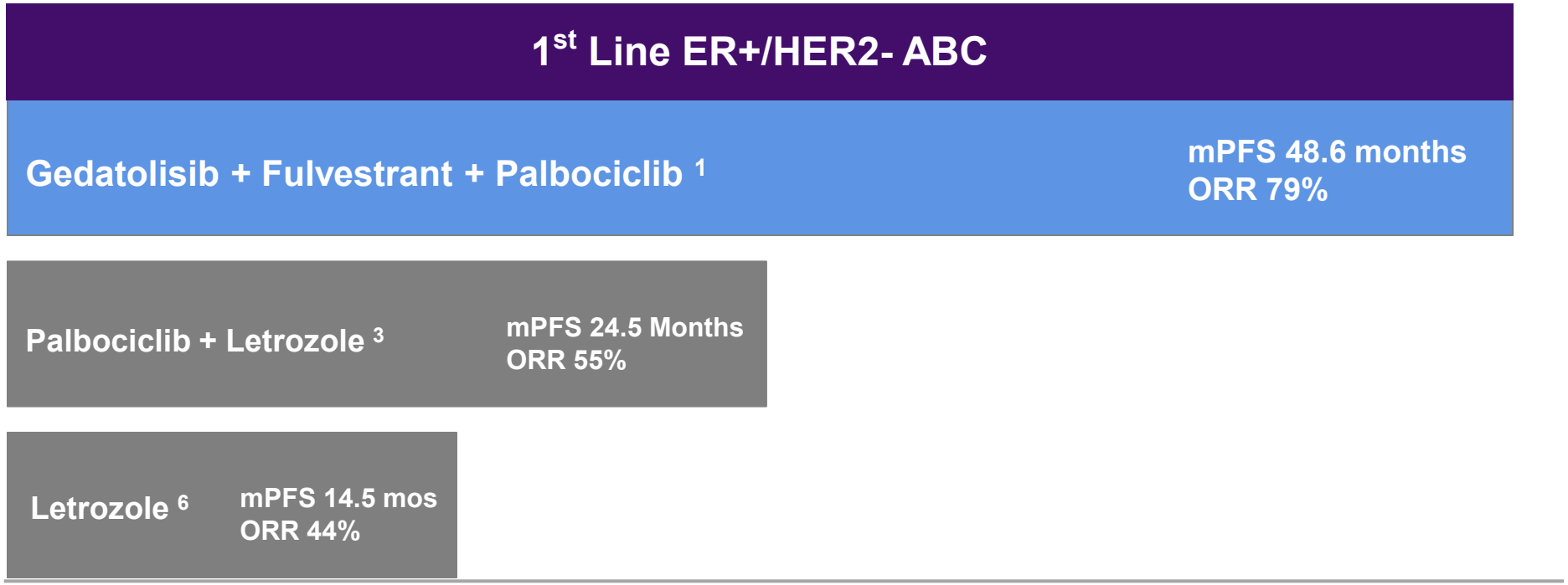
(1) Rugo 2023 ESMO-Breast; Escalation Arm A & Expansion Arm A data from B2151009 study. Median time from the last prior therapy was 1 month for Escalation Arm A vs 26 months for Expansion Arm A; (2) Finn 2016 NEJM – PALOMA-2; (3). Note: (a) ORR reported is for patients with measurable disease of a target 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 SOC



Sources: (1) Rugo 2023 ESMO-Breast. (2) 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.

Adding Gedatolisib to Palbociclib + ET Resulted in Higher ORR (1.4-2.5x)

Arm D vs. PALOMA-3 ORR and PFS results are particularly significant since PALOMA-3 patients were CDKi-naïve

Patients	1L CDKi-naïve		1L+ CDKi-naïve	2L/3L Prior CDKi
Study	PALOMA-2[1]	**Esc Arm + Exp Arm A[2]**	PALOMA-3[3]	**Arm D[2]**
N, (full, evaluable)	666, 338	**41, 33**	521, 267	**27, 27**
Study Treatment	Palbociclib + Letrozole	**Gedatolisib + Palbociclib + Letrozole**	Palbociclib + Fulvestrant	**Gedatolisib + Palbociclib + Fulvestrant**
ORR [a] (95% CI)	55% (50%-61%)	**79% (62%-89%)**	25% (20%-30%)	**63%[c]** (44%-78%)
Median PFS [b] (months) (95% CI)	24.8 (22.1, NR)	**48.6** (30.4, NR)	9.5 (9.2, 11.0)	**12.9** (7.4, 16.7)

- 1L ORR **1.43 times** higher than PALOMA-2 (79% vs. 55%)
- 2L/3L ORR **2.52 times** higher than PALOMA-3 (63% vs. 25%)
- Extended mPFS of gedatolisib regimen in 1st line setting suggests PI3K/mTOR is likely intrinsically, not just adaptively, involved as a disease driver

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(1) Finn 2016 NEJM; (2) Wesolowski 2021 SABCS; (3) Turner 2015 NEJM. Notes: (a) Response evaluable analysis set per RECIST v1.1 including uPR; (b) full analysis set. (c) Includes one unconfirmed partial response. No head-to-head trials have been conducted; data collected from different trials, in different patient populations and may not be comparable. Abbreviations: ET, endocrine therapy; NR, not reached

Arm D: Duration of Treatment in Patients' Refractory to Prior Therapy

Gedatolisib treatment duration significantly greater than patient's prior line of therapy

Duration of Immediate Prior Treatment (DIPT) Arm D		
	DIPT <180 Days	**DIPT <365 Days**
# Evaluable patients with DIPT <185 or 365 days (% of evaluable)	7 (27%)	11 (42%)
Median DIPT (days)	106	155
Median Duration of Study Treatment (DST, days)	270	276
Ratio of median DST vs. DIPT	**2.6**	**1.8**
Objective Response Rate to Study Treatment (95% CI)	**71%** (29%-96%)	**73%** (39%-94%)

Source: Layman 2021 SABCS

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Safety Summary: Treatment-Emergent Adverse Events

G + P + ET was well tolerated overall; < 4% discontinuation rate with Phase 3 dosing (Arm D)

Phase 1 Trial: Gedatolisib alone[1]

(154 mg weekly IV)

	All Arms (n=42)		
	TEAE's > 20%		
	Grade 1	Grade 2	Grade 3/4
Adverse Event	**%**	**%**	**%**
Stomatitis	46	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	-

Phase 1b Trial – Arm D: G + P + F[2]

- **Only <4% discontinued** drug due to AE
  - Alpelisib – 26% discontinued[3]
- **33% on treatment for >15 months**
- **Few hyperglycemia-related adverse events (26% all Grades, 7% Grade 3/4)**
  - Alpelisib (79% all, 39% Grade 3/4)[3]
- Most TEAE's were Grade 1 or 2
- Stomatitis was not treated prophylactically
  - **Prophylactic treatment may reduce G2 incidence by 90%; G3 by 100%[4]**
  - Phase 3 study will include prophylaxis
- Neutropenia and leukopenia, and anemia AEs related to palbociclib

Phase 1b Trial – *Arm D*: G + P + F[2]

(180 mg IV, 3 weeks, one week off)

	Arm D (n=27)		
	TEAE's > 30%		
	Grade 1	Grade 2	Grade3/4
Adverse Event	**%**	**%**	**%**
Stomatitis	11	56	22
Neutropenia	0	15	67
Nausea	44	30	-
Fatigue	22	37	7
Dysgeusia	44	7	-
Leukopenia	-	19	22
Diarrhea	37	-	4
Constipation	30	4	4
Vomiting	22	11	4
Anemia	4	15	15
Hyperglycemia	15	4	7

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(1) Shapiro 2015; (2) Wesolowski 2022 SABCS; (3) USPI Alpelisib; (4) Rugo 2017. Abbreviations: ET = endocrine therapy; G = gedatolisib; P = palbociclib; F = fulvestrant; TEAE = treatment emergent adverse events; AE = adverse event. Note: Data presented for the B2151009 trial is as of a cut-off date June 29, 2022.

Phase 3 Study Design
VIKTORIA-1

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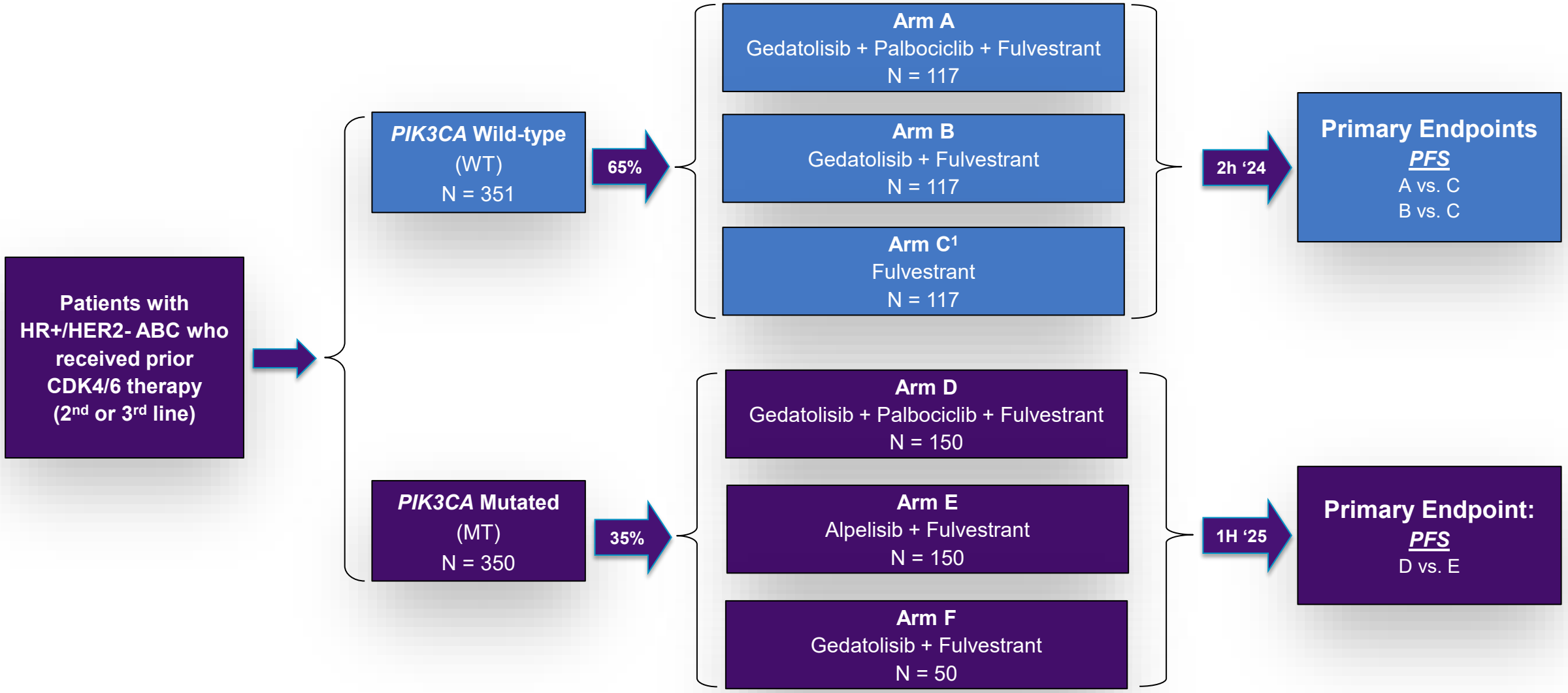
Pivotal Trial Design Considerations for 2nd Line HR+/HER2- ABC

- Standard-of-care 2nd line treatment differs based on *PIK3CA* status
  - *PIK3CA* wildtype (WT): Fulvestrant or everolimus + exemestane
  - *PIK3CA* mutated (MT): Alpelisib + fulvestrant
- 35% of patients have *PIK3CA* mutations in HR+/HER2- breast cancer
- Must formally test efficacy for each *PIK3CA* sub-group (WT and MT)
- PFS is the standard primary end point for randomized studies in 1st / 2nd line HR+/HER2- ABC
  - Pivotal studies for all current FDA approved therapies used PFS

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 Clinical Trial Results for VIKTORIA-1 Study Arms

Each trial evaluated patients who received prior treatment with a CDK4/6 therapy

	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%	4%	6%	24%	17%
PFS % at 12 months	53% (overall) WT 49% M 60%	10%	12%	22%	27%

Sources: (1) Wesolowski 2022 SABCS; (2) Includes 2 unconfirmed PR.(3) Bidard 2022 – EMERALD trial and FDA Multi-Disciplinary Review; (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.

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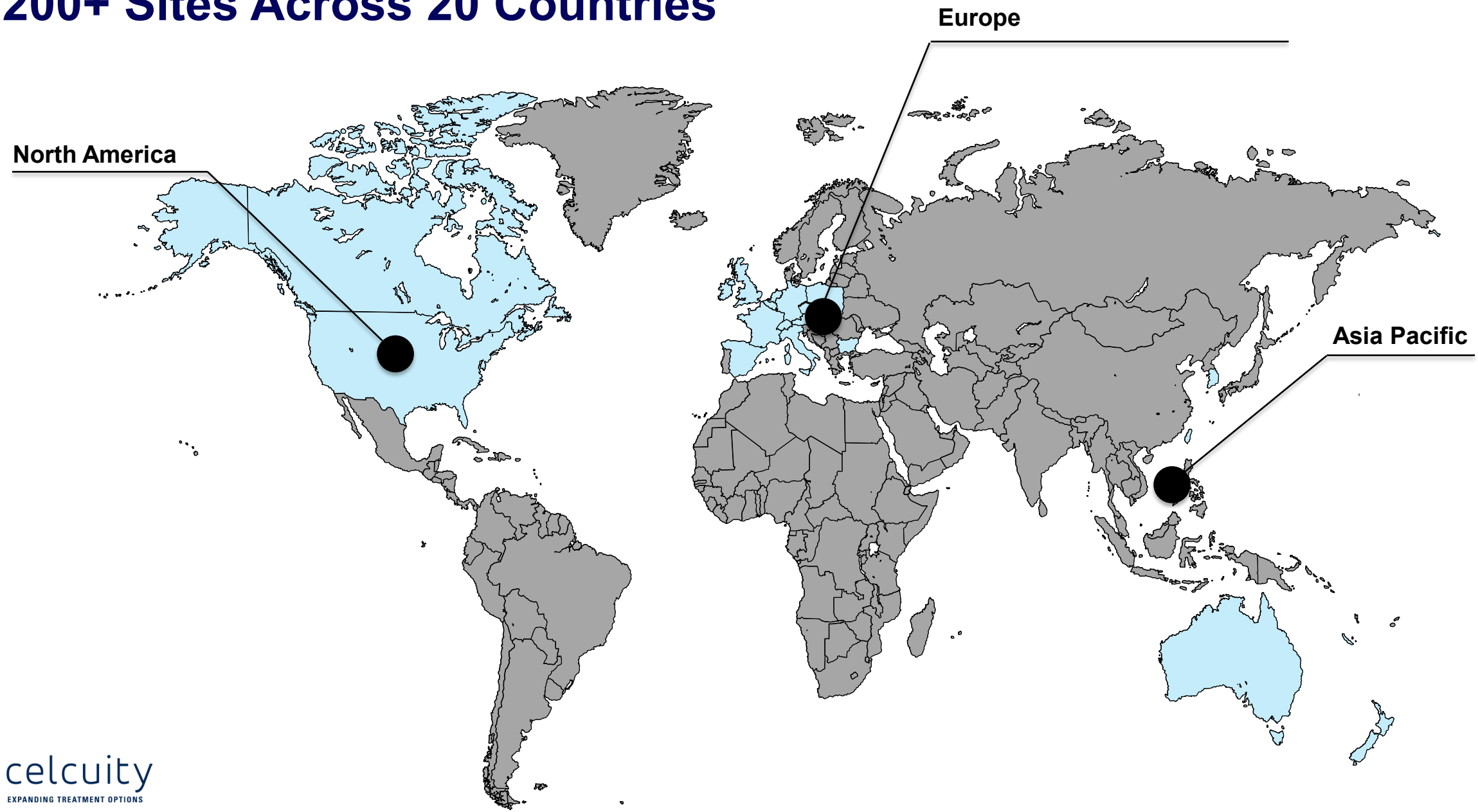
VIKTORIA-1 Pivotal Study Features

- Global open-label randomized study
- Key eligibility criteria:
  - Any *PIK3CA* status
  - Progressed on prior CDK4/6 treatment
  - Any menopausal status
  - ≤ 2 prior endocrine therapy
- 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)

Designed to support 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



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Gedatolisib for Prostate Cancer

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Metastatic Castration Resistant Prostate Cancer (mCRPC)

Background

Significant Population with High Clinical Unmet Need [1,2]

- 40,000 men diagnosed annually with mCRPC in the US, 125,000 globally [1]
- The five-year survival rate is only 32% [2]
- 34,000 men die of mCRPC annually in the US.

Aberrant Androgen Receptor Signaling is the Primary Driver

- The androgen receptor (AR) is the primary therapeutic target for mCRPC currently
- PARP and AR inhibitor combos are options for the ~15% of patients with an HRD.[3]

PI3K-AKT-mTOR Pathway Dysregulation Contributes to mCRPC Tumor Growth

- 56% - 70% of mCRPC tumors have PI3K/AKT/mTOR related pathway alterations.[4]
- Mutations dispersed across PTEN, PI3K, AKT, and mTOR sub-units
- mTOR plays a key role when pathway dysregulated and PTEN is functional

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(1) Scher, PLOS One, 2015; (2) National Cancer Institute, SEER Program 2023; (3) Heeke et al, JCO Precis Oncol 2018; (4) Millis Jama Oncol 2016; Carver et al, Cancer Cell 2011; El Sheikh et al, Neoplasia 2008. Abbreviations: HRD = homologous recombination repair deficiency

Limited Benefit for 2nd Line mCRPC Patients Post-AR Treatment

Guidelines recommend docetaxel or treatment with another AR therapy for patients lacking an HRD

First Line Treatment (HRP mCRPC)

Treatment	PFS (months)	OS (months)
Enzalutamide	19.5	35.3
Abiraterone	16.5	34.7

After ARi progression

Second Line Treatment (Prior ARi)

Treatment	mPFS (months)	OS (months)
Docetaxel [1]	6.7 – 8.5	20.3
Abiraterone [2]	5.6	NA
Enzalutamide [3]	5.5	NA

(1) Crabb J Clin Oncol 2021; (2) Attard J Clin Oncol 2018; (3) Sweeny Clin Cancer Res 2022; (4) US – SEER; Japan and EU - Datamonitor Healthcare; ROW calculated using 84% EU scale up factor. Abbreviations: AR = androgen receptor; NA = not available; HRD = homologous recombination deficient; HRP = homologous recombination proficient

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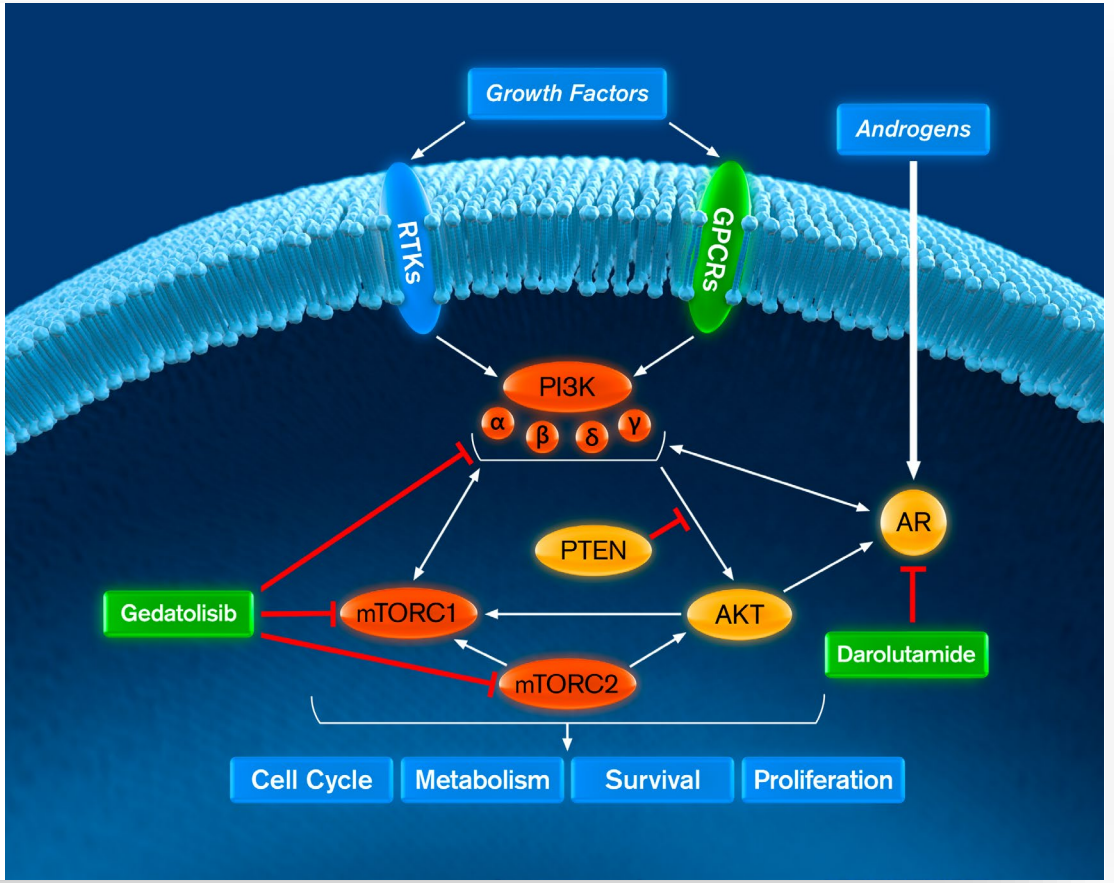
PI3K/AKT/mTOR (PAM) is a Key Resistance Mechanism to AR Inhibitors

The PAM pathway and AR signaling cross regulate each other by reciprocal feedback [1]

PI3K/mTOR + AR Inhibition
Treatment Rationale

- AR inhibition increases PAM pathway signaling [2]
  - For patients who progressed on an AR inhibitor, PI3K inhibition may resensitize them to an AR inhibitor
- PI3K inhibition increases AR protein levels and activation [3]
  - mTOR inhibition is particularly critical in patients when the tumor suppressor, PTEN, is functional
- Strong rationale to combine an AR inhibitor with a PAM inhibitor in patients who progressed on an AR inhibitor



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(1) Carver et al, Cancer Cell 2011; (2) Mulholland et al, Cancer Cell 2011; (3) Crumbaker et al, Cancers 2017: (4) Pungsinont at al, Int. J. Mol. Sci 2021
(5) Taylor et al, Cancer Cell 2010; Carver et al, Cancer Cell 2011; El Sheikh et al, Neoplasia 2008

In Vitro Activity of PI3K/AKT/mTOR (PAM) Inhibitors in Prostate Cancer Cell Lines

Gedatolisib is 2.5X+ more cytotoxic and 7X-50X more potent than other PAM inhibitors

Average Values for Six Prostate Cancer Cell Lines
(22RV1, MDA-PCa-2b, DU145, LNCaP, C4-2, PC3)

Potency
Low — High

Efficacy
Cytostatic — Cytotoxic

	GR50 (nM)				GR-Max (-1 to 1)			
	Gedatolisib *PI3K/mTOR*	**Alpelisib** *PI3K-α*	**Capivasertib** *AKT*	**Samotolisib** *PI3K/mTOR*	**Gedatolisib** *PI3K/mTOR*	**Alpelisib** *PI3K-α*	**Capivasertib** *AKT*	**Samotolisib** *PI3K/mTOR*
Average all	12	802	290	82	-0.43	0.7	0.02	-0.17
PTEN WT	13	504	383	119	-0.41	0.62	0.21	-0.05
PTEN altered	11	1000	197	45	-0.44	0.76	-0.16	-0.28

Gedatolisib vs. Other PAM Inhibitors

- **More cytotoxic** than other PAM inhibitors
  - Alpelisib and capivasertib not cytotoxic
  - Geda is 2.5x more cytotoxic than samotolisib
- **More potent** than other PAM inhibitors
  - **65x** more potent than alpelisib
  - **24x** more potent than capivasertib
  - **7x** more potent than samotolisib
- Same potency and efficacy regardless of PTEN status unlike other PAM inhibitors.

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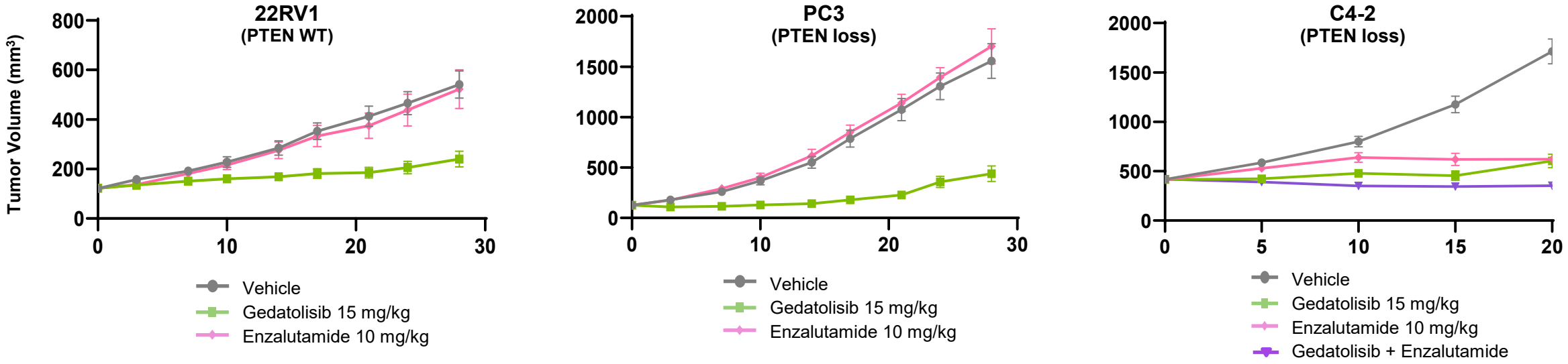
(1) Shapiro 2015; B2151009 Arm D; internal Celcuity studies; (2) US Package Insert; (3) No head-to-head trials have been conducted; data collected from different trials, in different patient populations and may not be comparable. Abbreviations: G, Grade; SAE, serious adverse event; AUC, area under the curve

In Vivo Activity of Gedatolisib in Prostate Cancer Xenograft Models

Gedatolisib induced 80%+ tumor growth inhibition (TGI) regardless of PTEN status and ARi sensitivity



- **Robust single-agent TGI in PC xenograft models regardless of sensitivity to enzalutamide (ARi) and PTEN status**
- **Gedatolisib + enzalutamide induced significantly greater TGI than enzalutamide alone in enzalutamide sensitive model**

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Benefit of Combining PAM and AR inhibitors Demonstrated Clinically

Favorable results obtained despite limitations of samotolisib (low potency) and ipatasertib (limited MOA)

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
					  ▪ 7X more potent overall; 100x for mTOR
					  ▪ 2.5X 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 ▪ Confirms mTOR is a resistance mechanism to AKT inhibition when PTEN is functional
		PTEN loss by NGS	209	**19.1 vs. 14.2 months** *(HR = 0.65; P = 0.02)*	

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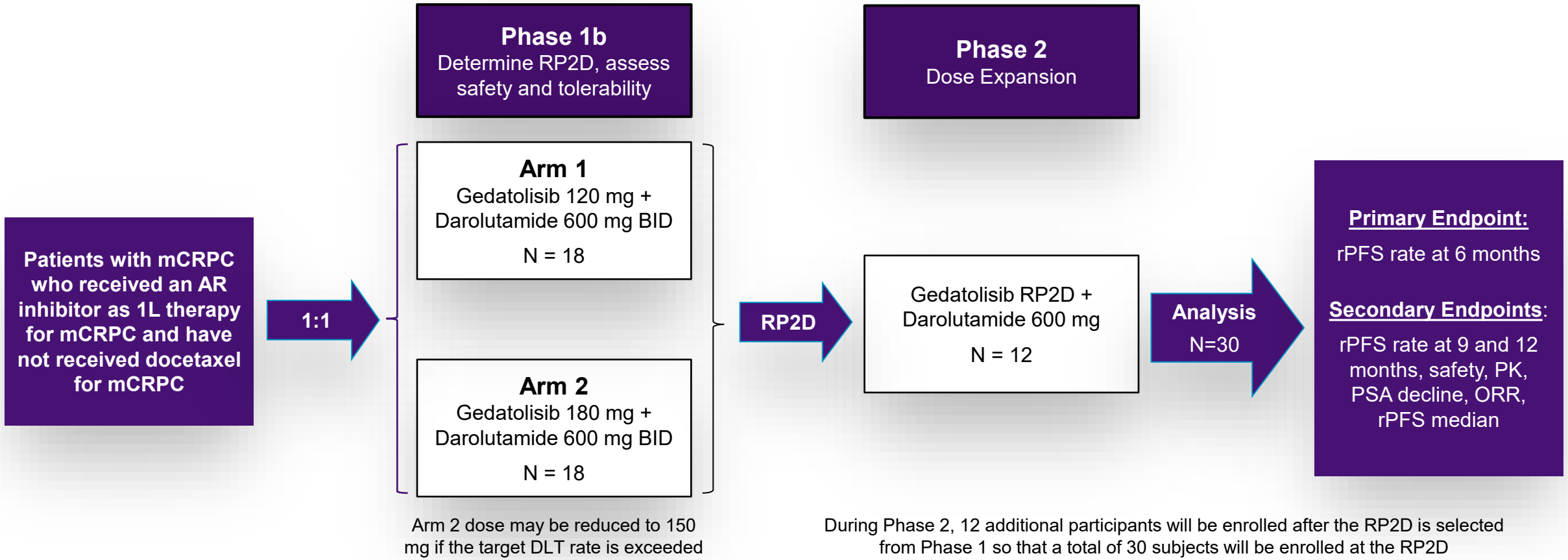
(1) Sweeny Clin Cancer Res 2022; (2) De Bono, Lancet, 2021

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

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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**	
IC50 [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: CRPC = castration resistant prostate cancer; nm = non-metastatic; HSPC = hormone sensitive prostate cancer

Additional Early Phase Clinical Data

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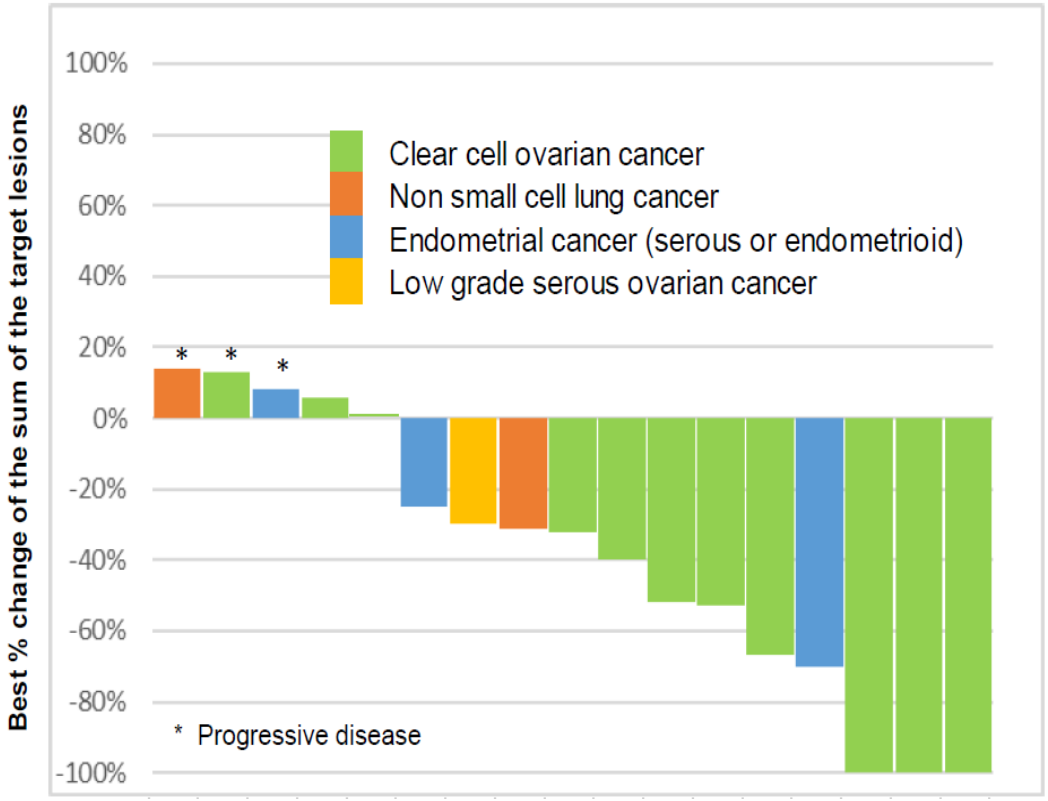
Gedatolisib + Paclitaxel + Carboplatin in Patients with Solid Tumors (N=17)[1]

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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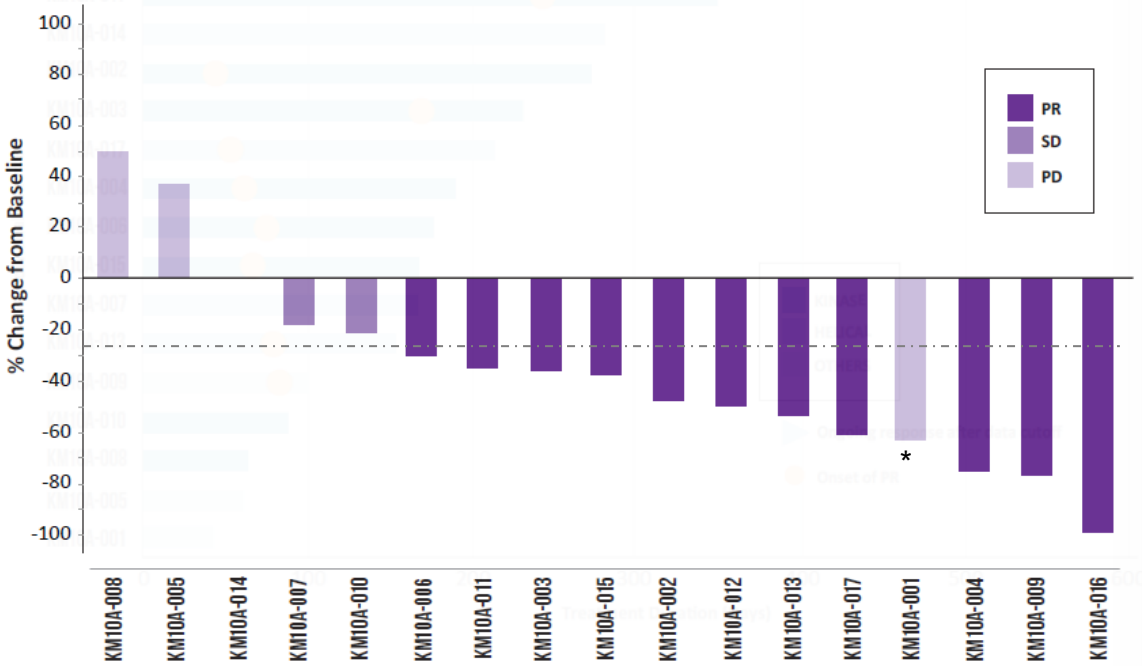
(1) Columbo 2021 CCR

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

59% ORR and 83% clinical benefit rate

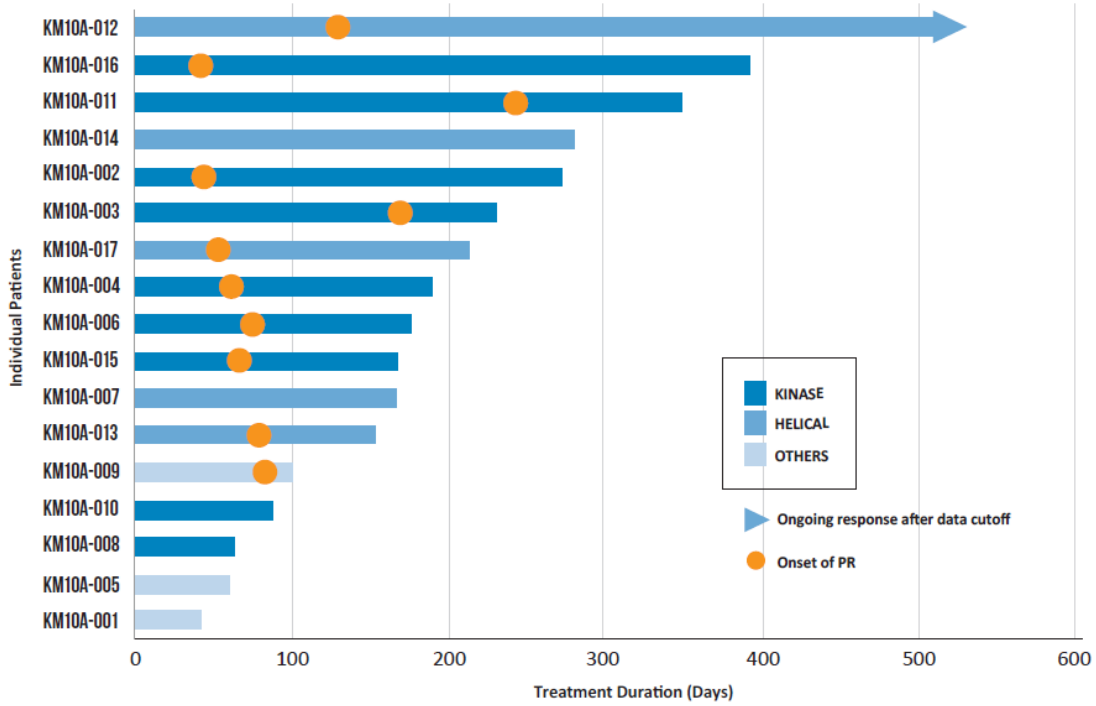
Best Response



* 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.

Experienced drug development team

CMO

Igor Gorbatchevsky, MD

BAYER
MEI pharma
Daiichi-Sankyo
J&J
IOVANCE BIOTHERAPEUTICS

VP Clin Ops

Nadene Zack

Pfizer
darébio
REGULUS

VP Pharma Dev

Bernhard Lampert, PhD

gsk
CHIMERIX
GILEAD Creating Possible

VP Quality

David Bridge

CHIMERIX
EMD SERONO
QUINTILES

VP Program Mgmt.

Fred Kerwood

MERCK
J&J
élan
CytomX THERAPEUTICS

VP Medical Affairs

Pratima Nayak, MD

Takeda
ATARA BIO
rigel
agios

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Leading cancer KOLs are participating in our research

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URBANA-CHAMPAIGN

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Celcuity Leadership Team

Co-Founder and CEO

Brian Sullivan

CEO, Founder - PUR Water Filters
- Sold to Proctor & Gamble in 1999 for $265 million

CEO - SterilMed, med devices
- Sold to Johnson & Johnson in 2011 for $330M

A.B. Harvard University, magna cum laude with distinction

9 U.S. patents received

Co-Founder and CSO

Lance Laing, PhD

Scientist at Scriptgen/Anadys (purchased by Novartis)

Director, Chemistry, Product Development – ACEA (purchased by Agilent)

PhD in biophysics and biochemistry - The Johns Hopkins University

Post-doc: Washington Univ. as NIH fellow

23 U.S. patents received

CFO

Vicky Hahne

CFO – SimonDelivers (on-line grocery)

Controller – Respirtech (medical devices)

Controller – SterilMed (medical devices)

15 years as controller and CFO at high-growth VC and PE backed companies

CMO

Igor Gorbatchevsky, MD

VP Clin Dev – MEI Pharma

VP Clin Science – Iovance

Global Clinical Leader – Bayer

Senior Med Dir – Daiichi-Sankyo

Senior Med Dir – Cell Therapeutics

NDA's
- Aliqopa (copanlisib)
- Raplixa (fibrocaps)
- Zevalin (ibritumumab tiuxetan
- Pixuvri (pixantrone)

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Gedatolisib – A Phase 3 Asset with Multiple Potential Indications

Phase 1b data in HR+/HER2- MBC reported better ORR and PFS than SOC in 1st and 2nd lines

Compelling Efficacy in Advanced Breast Cancer

Very promising results in 1L and 2L relative to SOC

- **2nd Line[1]**
  - 12.9 mos mPFS, 63% ORR
- **1st Line[2]**
  - 48.6 mos mPFS, 79% ORR

Multiple Potential Indications

Numerous tumor types involve PI3K/mTOR

- Compelling POC clinical data with PI3K therapies that have inferior MOA, higher toxicity
- Prostate, endometrial, cervical, and head & neck cancers involve PI3K/mTOR pathway

Key Milestones

Laying groundwork for robust development plan

- Phase 3 VIKTORIA-1 study first primary analysis expected in 2H ‘24
- Lifecycle development update in 2H ‘23

Financial Resources

Strong balance sheet

- $146.2 million cash and cash equivalents at the end of Q2 2023

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1) Data from Expansion Arm D of the B2151009 clinical trial; 2) Combined data from treatment-naïve patients enrolled in Escalation Arm A and Expansion Arm A of the B2151009 Phase 1b clinical trial.

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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."

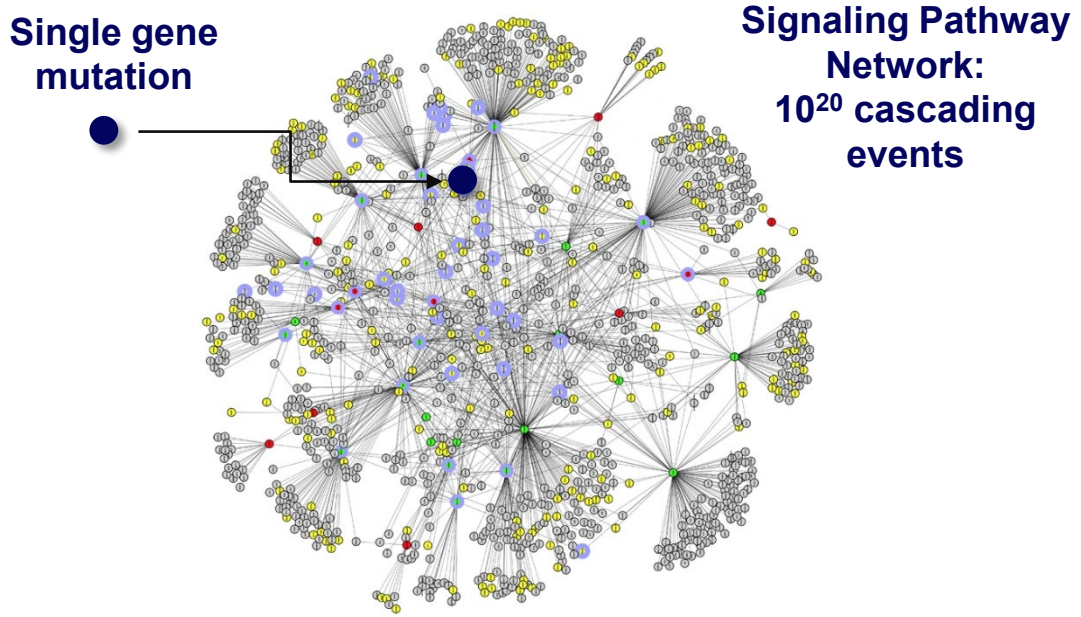
Neal Rosen, MD, PhD
Director, Center for Mechanism-Based Therapy
Memorial Sloan Kettering Cancer Institute

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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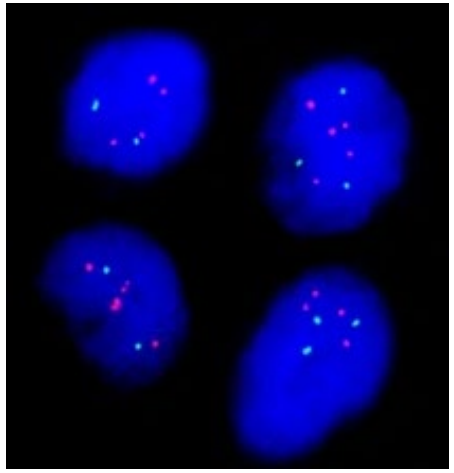
Source: 1) Internal Celcuity Studies

Current Molecular Diagnostics vs. CELsignia – HER2 Example

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

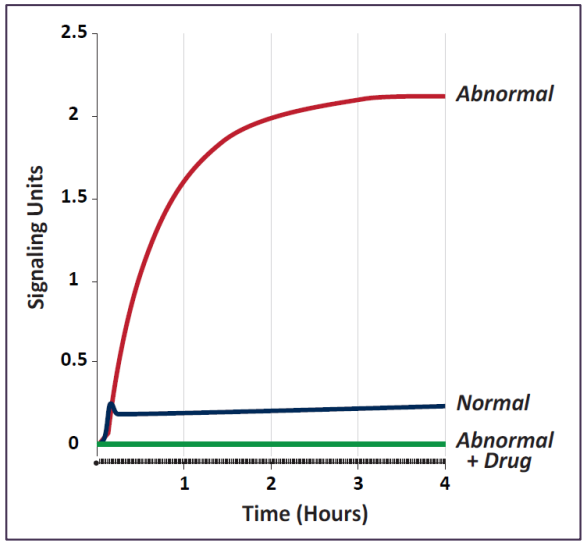
FISH HER2 Dx
(1 pathway gene)



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 a clinical stage biotechnology company that discovers previously undetectable cancer drivers and develops drugs to treat them.

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

celcuity
EXPANDING TREATMENT OPTIONS