



Developing Potentially First-in- Class Rx using 3rd Generation Dx

March 23, 2022

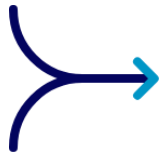
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Developing Potentially First-in-Class Rx using 3rd Generation Dx



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**

Gedatolisib is a Potential First-in-Class PI3K/mTOR Inhibitor

Phase 3 ready for 2L HR+ / HER2- metastatic breast cancer

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- MBC with gedatolisib + ET + CDK4/6i
 - Phase 1b trial (N=103) reported **62% ORR** in evaluable patients across four expansion arms
 - 31 months PFS in 1L arm and 12.9 months PFS in 2L arm with Phase 3 dosing schedule

Well-Tolerated

- Safety profile is well characterized - 492 patients treated with gedatolisib in eight clinical trials
- **Only 4% treatment discontinuation with Phase 3 dosing** - well-tolerated with manageable TEAE's
- Significantly lower Grade 3/4 hyperglycemia than approved oral PI3K-α inhibitor (7% vs. 39%)

Multiple Potential Indications

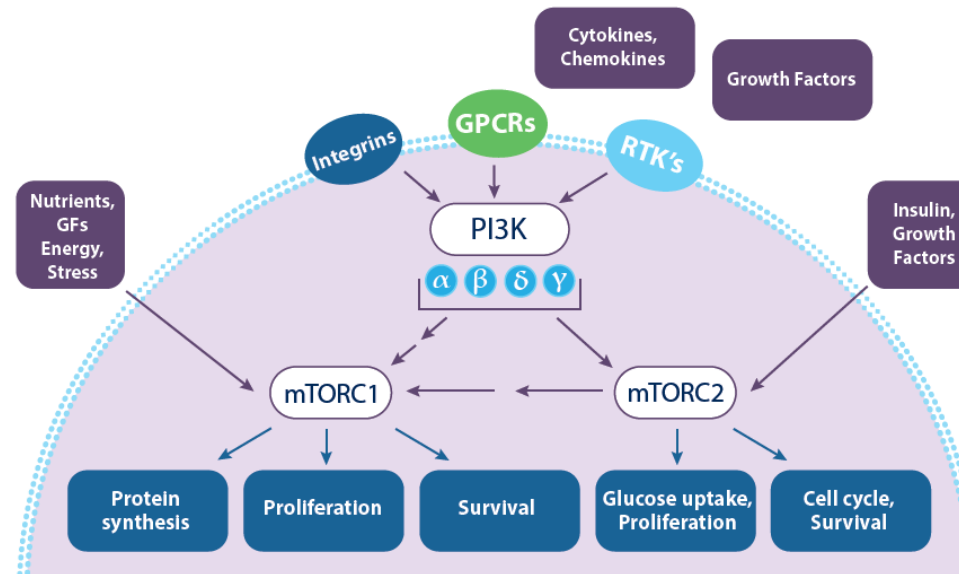
- Expect to initiate Phase 3 trial in 1H '22 for 2L+ patients with HR+ / HER2- metastatic breast cancer
 - Addresses **100K+ annual patient population** globally
- Broad range of indications are possible given PI3K/mTOR's role in multiple tumor types

PI3K/mTOR is One of Most Important and Complex Oncogenic Pathways

Key oncogenic driver and 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.
- Bypass resistance mechanism to CDK4/6, ER, AR, PARP inhibition



Tumor type	PIK3CA mutation	PTEN Loss or Mutated
ER+ BC ^{1,2}	~39% ¹	~46%
Endometrial ²	~37%	~82%
Cervix ²	~29%	~34%
HER2+ BC ^{1,2}	~25% ¹	~30%
Bladder ²	~22%	~35%
Colon ²	~17%	~51%
HNSCC ²	~14%	~36%
TNBC ^{1,2}	~13% ¹	~15%
Ovarian ²	~8%	~24%
Prostate ²	~6%	~66%

Difficult to Safely and Efficaciously Inhibit the PI3K/mTOR Pathway

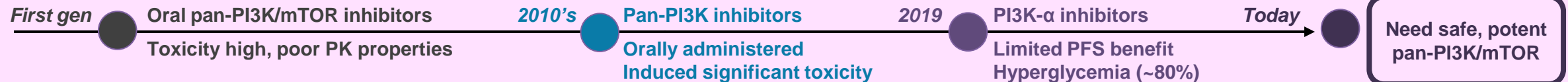
Maximum efficacy requires inhibition of all Class 1 PI3K isoforms and mTORC1 and mTORC2

Multiple pathway components must be targeted

- Feedforward and feedback loops between PI3K isoforms 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 administered pan-PI3K or pan-PI3K/mTOR inhibitors induced unacceptable toxicity



Gedatolisib Has a Highly Differentiated Mechanism of Action

Only 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 can occur with PI3K isoform or mTOR specific drugs
- Enhances potential synergy with other pathway inhibitors

Gedatolisib vs. Approved Solid Tumor PI3Ki or mTORi
IC₅₀ (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	-	-

Gedatolisib PK Properties vs. Other Approved PI3K Inhibitors

Differentiated chemical structure results in favorable PK profile and lower toxicity

	Gedatolisib¹	Alpelisib ²	Copanlisib ²	Duvelisib ²	Idelalisib ²	Umbralisib ²
Target(s)	Pan-PI3K mTOR	PI3K-α	Pan-PI3K	PI3K-δ	PI3K-δ	PI3K-δ CK1ε
Administration	IV	Oral	IV	Oral	Oral	Oral
Dosing in molar/month	0.88	19.03	0.37	3.22	20.22	32.3
Volume (distribution) L	30	114	871	29	23	312
AUC plasma ug.h/mL	47.1	33.2	1.6	7.9	10.6	141
Cmax ng/mL	8,594	2,480	463	1,500	1,861	7,300
Half-life (hours)	37	8-9	39	5	8	91
Hyperglycemia (G 3/4) ³	7%	39%	41%	-	-	-
Treatment related SAE's ³	15%	35%	26%	65%	68%	18%
Treatment related (TR) Discontinuations ³	4%	26%	16%	35%	17%	14%

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 > geda (molar/month)
 - Copanlisib 50x > retention liver vs plasma
- 75%-85% lower rate of TR discontinuations
- 4x-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 for Advanced Breast Cancer (ABC)

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

Finding more effective treatment for these patients is the biggest unmet need in breast cancer

2nd Line SOC HR+/HER2- Metastatic Breast Cancer (Post CDK4/6 inhibitor)

Treatment (Patient Group)	mPFS (months)	ORR ¹
Fulvestrant ² (PIK3CA WT)	1.9	NR
Everolimus (mTOR) + Exemestane ³ (PIK3CA WT)	Unknown	Unknown
Alpelisib (PI3K-α) ⁴ + Fulvestrant (PIK3CA MT)	7.3	21%

Treatment guidelines recommend use of sequential endocrine therapy before chemotherapy, in the absence of visceral crisis or until all endocrine therapy options have been exhausted.⁵

Clinical Development Plan Pending FDA Input

Phase 3 study for patients with HR+/HER2-ABC who progressed on CDK4/6 therapy

- Expect to initiate a pivotal Phase 3 clinical trial for gedatolisib with palbociclib + fulvestrant in 1H 2022
- All-comer design (PIK3CA+/-) that will incorporate separate primary endpoints for mutated and non-mutated PIK3CA patients
- Trial design finalized after receiving FDA input

Additional potential indications based on POC and nonclinical study data

- Treating hormonally driven cancers has strong biological rationale
 - **Prostate cancer**
 - Nonclinical and clinical studies demonstrate linkage between androgen and PI3K/mTOR pathways
 - **Recurrent endometrial cancer**
 - **Ovarian cancer**
 - Favorable data from POC study
 - ORR = 80%

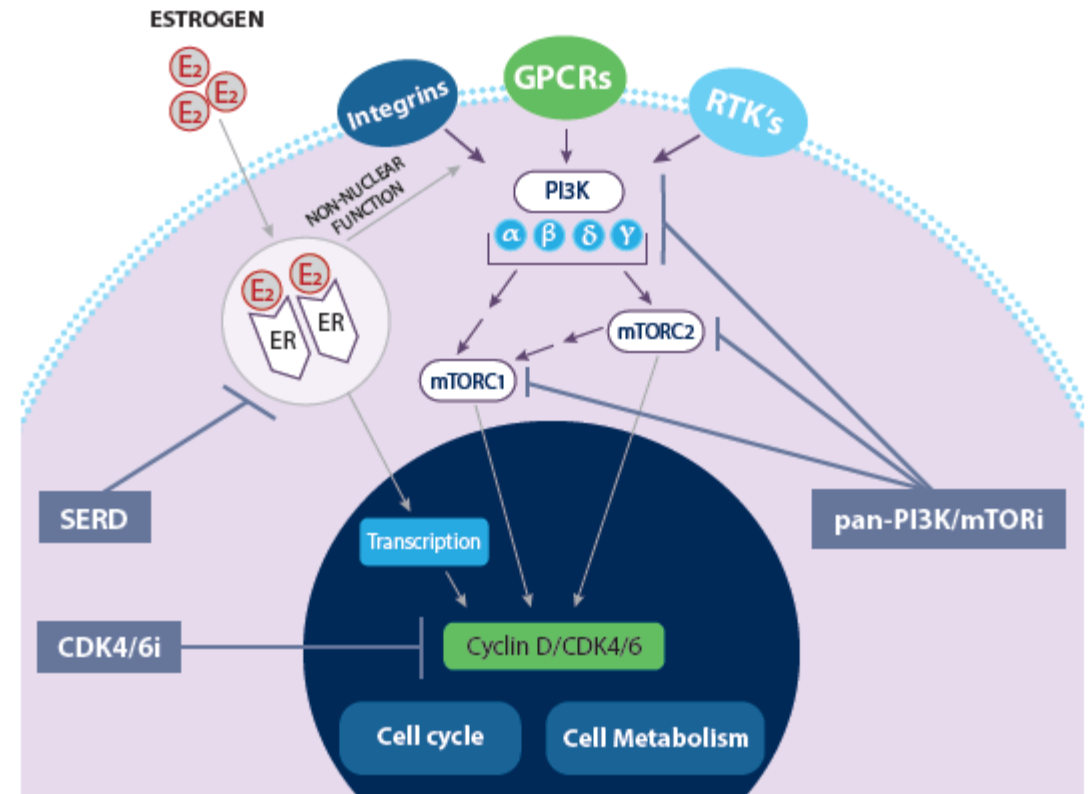
Review of Preliminary Phase 1b Data

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

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

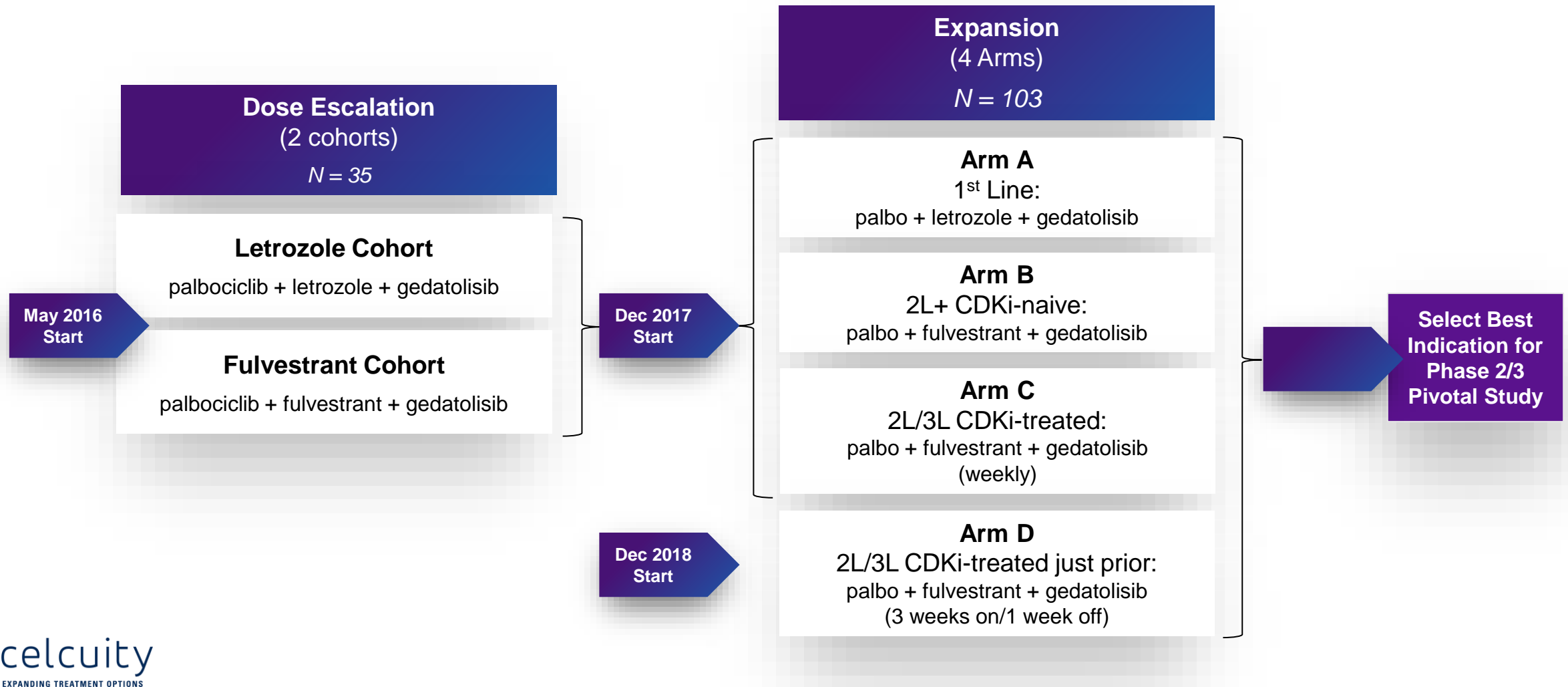
Treatment Strategy

- 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



B2151009: Phase 1b Study (138 patients)

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



63% ORR and 12.9 months PFS in Arm D with Phase 3 Dosing Schedule

ORR and PFS was superior to SOC in each arm for their respective lines of therapy

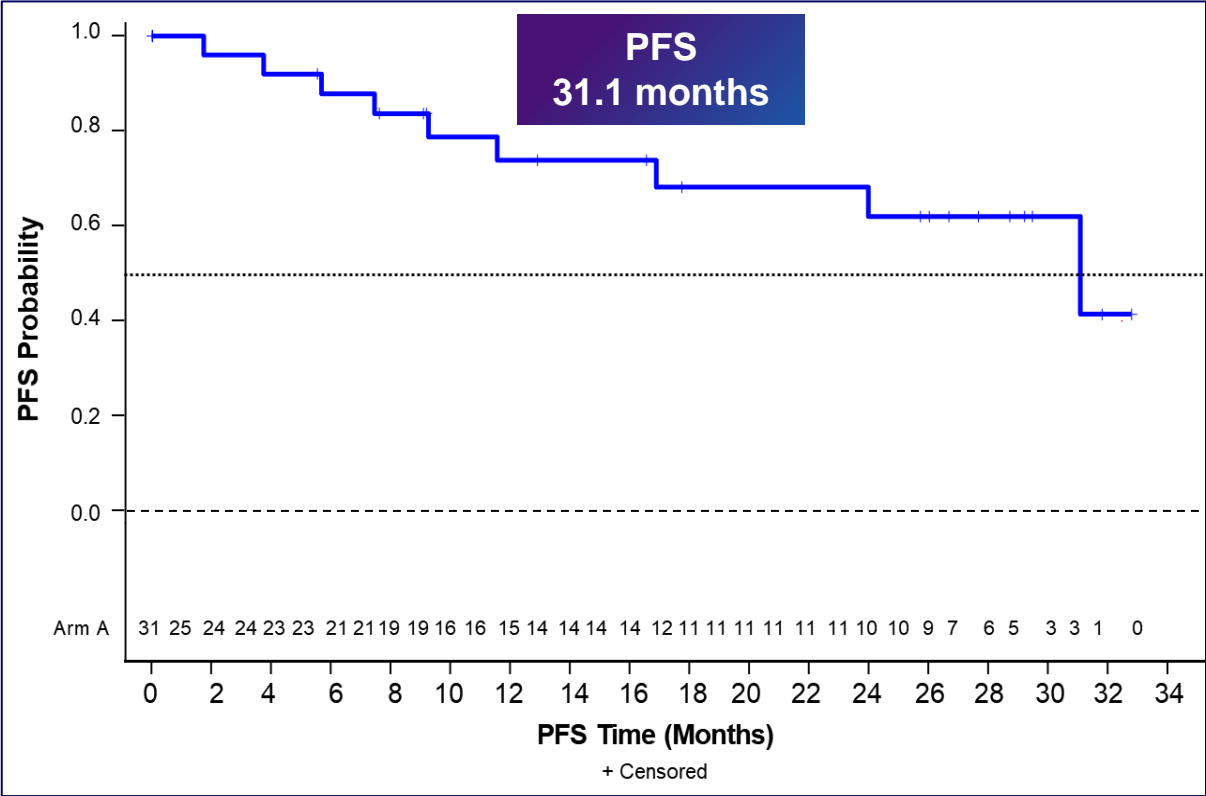
B2151009 Efficacy Summary (N=103)				
Patients	1L: CDKi-Naïve	2L+: CDKi-naïve	2L/3L: CDKi-pretreated	2L/3L: CDKi-pretreated
Arm	A (N=31)	B (N=13)	C (N=32)	D (N=27)
Study Treatment	G+ P + L (weekly)	G + P + F (weekly)	G + P + F (weekly)	G + P + F (3 week on/1 week off)
ORR ¹ (95% CI)	85% (66%-96%)	77% ² (46%-95%)	32% ^{2,3} (16%-52%)	63% ^{2,3} (42%-81%)
CBR ⁴ (95% CI)	96% (81%~100%)	100% (75%-100%)	79% (59%-92%)	96% (81%~100%)
Median PFS (mos) (95% CI)	31.1 (16.9, NR)	11.9 (3.7, NR)	5.1 (3.4, 7.5)	12.9 (7.4, 16.7)

Abbreviations: G = gedatolisib; P = palbociclib; L = letrozole; F = fulvestrant; CBR = clinical benefit rate; NR = not reached

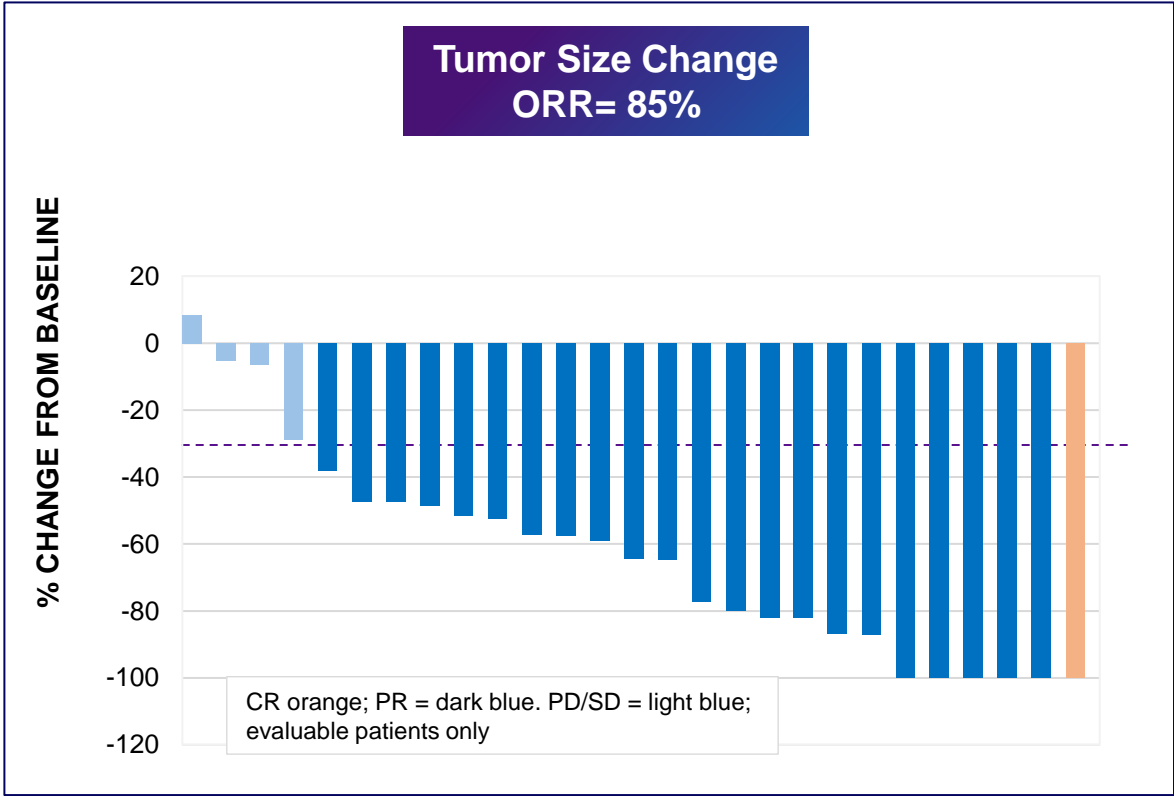
(1) ORR represents PR, except in Arm A, which had 1 CR. Responses by Physician Assessment per RECIST 1.1; (2) Includes 2 unconfirmed PR; (3) ORR was superior in Arm D relative to Arm C in patients regardless of the number of prior therapies for ABC. In Arm C and Arm D, ORR for patients receiving 1 prior line of therapy was 33% and 56% respectively and for ≥2 prior lines of therapy it was 32% and 78%; (4) CBR is clinical benefit rate. Source: Layman 2021 SABCS. Data presented for gedatolisib is from a preliminary data analysis as of a cutoff date of May 10, 2021, representing a database snapshot, and may change based on ongoing routine data monitoring

Gedatolisib + Palbociclib + Letrozole in 1st Line HR+/HER2- ABC (N=31)¹

Data compares favorably to published data for palbociclib + letrozole therapy from PALOMA-2²



PALOMA-2 mPFS = 24.8 months

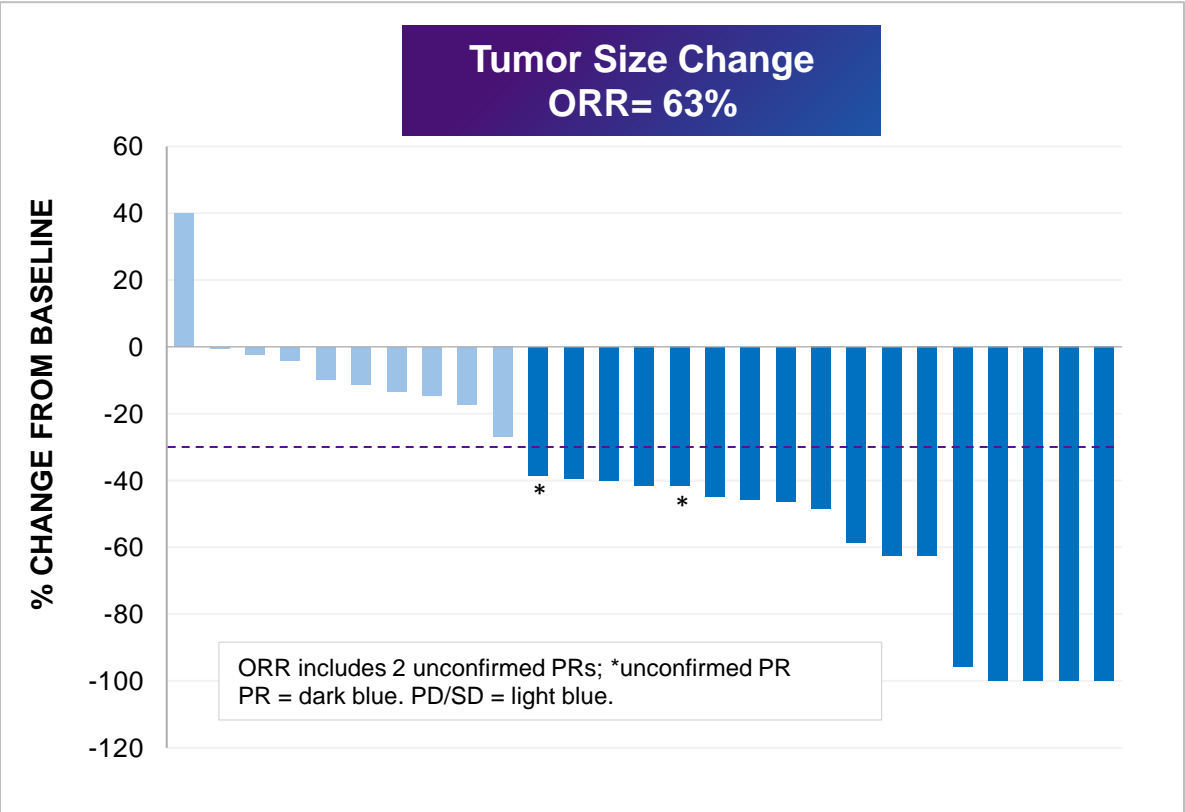
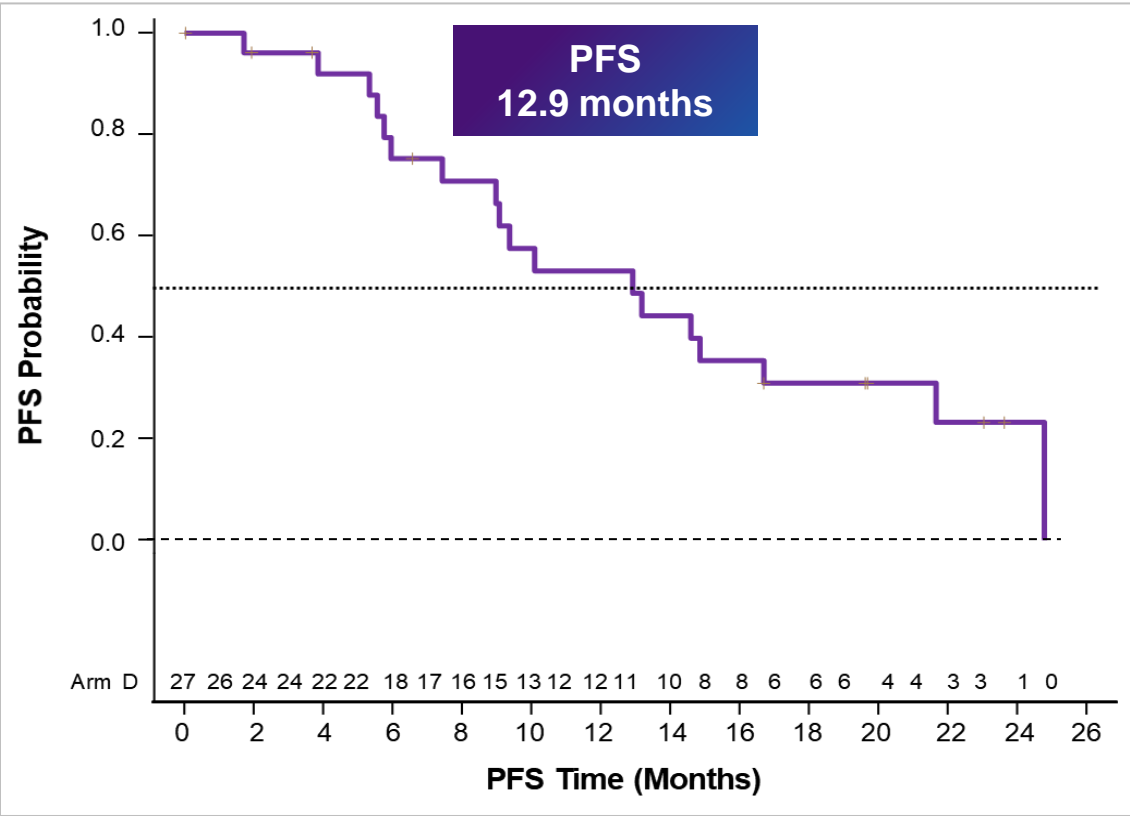


PALOMA-2 ORR = 55%

(1) Layman 2021 SABCS; Arm A data from B2151009 study. (2) Finn 2016 NEJM. Note: (1) ORR reported is for patients with measurable disease. (2) No head-to-head trials have been conducted; data collected from different trials, in different patient populations and may not be comparable. (3) Data presented is from a preliminary data analysis as of a cutoff date of May 10, 2021, representing a database snapshot, and may change based on ongoing routine data monitoring

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

Data from Phase 1b study with Phase 3 regimen (Arm D) compares favorably to published data with current SOC (N=27)



Adding Gedatolisib to Palbociclib + ET Resulted in Higher ORR (1.5-2.5x)

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	Arm A	PALOMA-3	Arm D
Evaluable Patients	N=338	N=27	N=267	N=27
Study Treatment	Palbociclib + Letrozole	Gedatolisib + Palbociclib + Letrozole	Palbociclib + Fulvestrant	Gedatolisib + Palbociclib + Fulvestrant
ORR (evaluable patients) (95% CI)	55% (50%-61%)	85% (66%-96%)	25% (20%-30%)	63% ⁵ (42%-81%)
Median PFS (months) (95% CI)	24.8 (22.1, NR)	31.1 (16.9, NR)	9.5 (9.2, 11.0)	12.9 (7.4, 16.7)

- Arm A ORR **1.55 times** higher than PALOMA-2 (85% vs. 55%)
- Arm D ORR **2.52 times** higher than PALOMA-3 (63% vs. 25%)

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

Gedatolisib Combo Offers Potential for Superior Efficacy Compared to SOC Options

2nd Line ER+/HER2- MBC (post-CDKi)

Drug Regimen	Efficacy
Gedatolisib + Palbociclib + Fulvestrant¹ (<i>PIK3CA</i> WT <u>and</u> MT patients)	PFS 12.9 months ORR 63%
Alpelisib + fulvestrant² (<i>PIK3CA</i> MT patients only)	PFS 7.3 months ORR 21%
Fulvestrant³ (<i>PIK3CA</i> WT patients only)	PFS 1.9 ORR NR
Everolimus + Exemestane⁴ (<i>PIK3CA</i> WT patients only)	Unknown

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)		
	DIPT <180 Days	DIPT <365 Days
Arm	D	D
# 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

Data presented for gedatolisib is from a preliminary data analysis as of a cutoff date of May 10, 2021, representing a database snapshot, and may change based on ongoing routine data monitoring

Arm D: High ORR Irrespective of Number of Prior Lines of Therapy

Number of Prior Lines of Therapy for Advanced Disease		
	≥ 2 Prior Lines	1 Prior Line
# of Evaluable Patients	9	18
# of Partial Responses	7	10
Objective Response Rate	78%	56%

Source: Layman 2021 SABCS

Data presented for gedatolisib is from a preliminary data analysis as of a cutoff date of May 10, 2021, representing a database snapshot, and may change based on ongoing routine data monitoring

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 (154 mg weekly IV)

Adverse Event	All Arms (n=42)		
	TEAE's > 20%		
	All Grades	Grade 3	Grade 4
Adverse Event	%	%	%
Stomatitis	55	7	-
Nausea	41	2	-
Hyperglycemia	26	2	-
Vomiting	24	2	-
Asthenia	21	2	-
Appetite decrease	21	-	-
Fatigue	21	-	-

Phase 1b Trial – Arm D: G + P + F

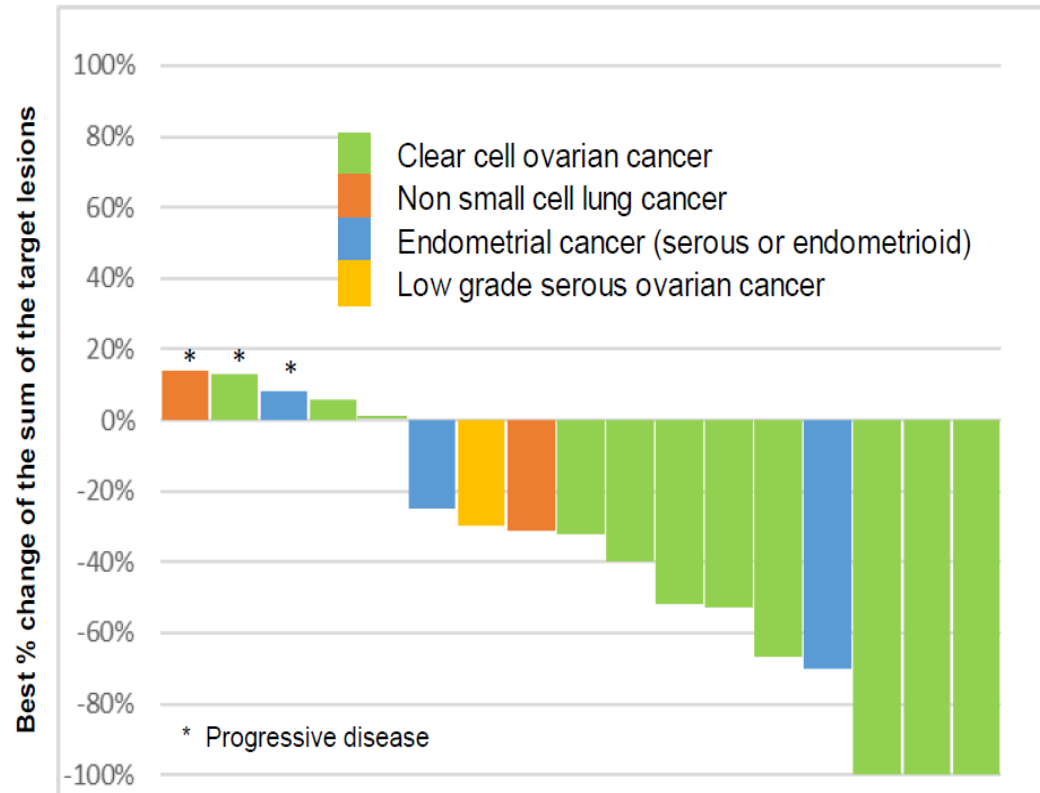
- Only <4% discontinued drug due to AE
 - Alpelisib – 26% discontinued
- 33% on treatment for >15 mos
- Few hyperglycemia-related adverse events (22% all Grades, 7% Grade 3/4)
 - Alpelisib (79% all, 39% Grade 3/4)
- Most TEAE's were Grade 1 or 2
- Stomatitis was treated at manifestation, not prophylactically
 - Prophylactic treatment reduces G2 incidence by 90%; G3 by 100%¹
 - Phase 3 study will include prophylaxis
- Neutropenia, leukopenia, and anemia AEs related to palbociclib

Phase 1b Trial – Arm D: G + P + F (180 mg IV, 3 weeks, one week off)

Adverse Event	Arm D (n=27)		
	TEAE's > 30%		
	All Grades	Grade 3	Grade 4
Adverse Event	%	%	%
Neutropenia	85	59	11
Stomatitis	85	22	-
Nausea	74	-	-
Fatigue	67	7	-
Dysgeusia	52	-	-
Leukopenia	41	19	4
Diarrhea	41	4	-
Vomiting	37	4	-
Constipation	37	4	-
Hyperglycemia	22	7	-

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

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

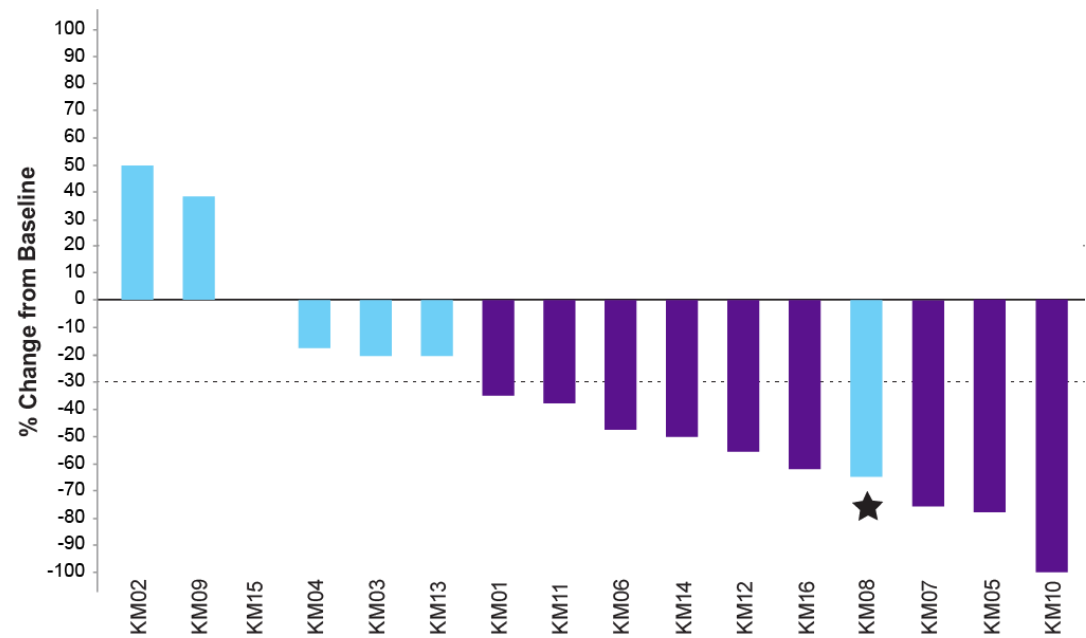


- Ovarian Cancer (N=11)
 - ORR: 82%
 - Clear cell ovarian cancer (CCOC) (N = 10)
 - ORR: 80% - 5/10 PR, 3/10 CR
 - Low grade serous ovarian (N=1)
 - 1/1 PR
- Other solid tumors (N= 6)
 - ORR = 33%
- Median PFS = 6.35 months (95% CI 4.6-11.11)
- Median duration of response = 7.6 months (95% CI 1.9-13.4)

- 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

56% ORR for Patients Receiving Gedatolisib + Trastuzumab Biosimilar

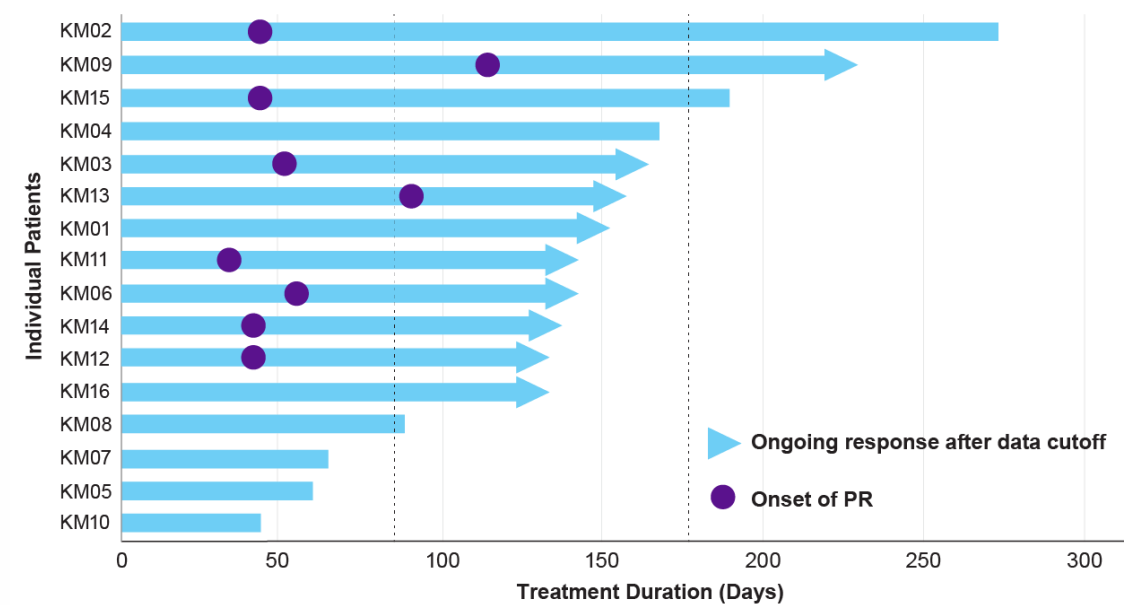
Best Response



*Patient whose target lesion decreased by 63% but a new leptomeningeal seeding occurred.

- 9 of 16 (56%) showed partial response (PR)
- 4 of 16 (25%) had stable disease (SD)

Duration of Response



Swimmer plot of the treatment duration

- At the time of the analysis, 9 patients had a continuing response.

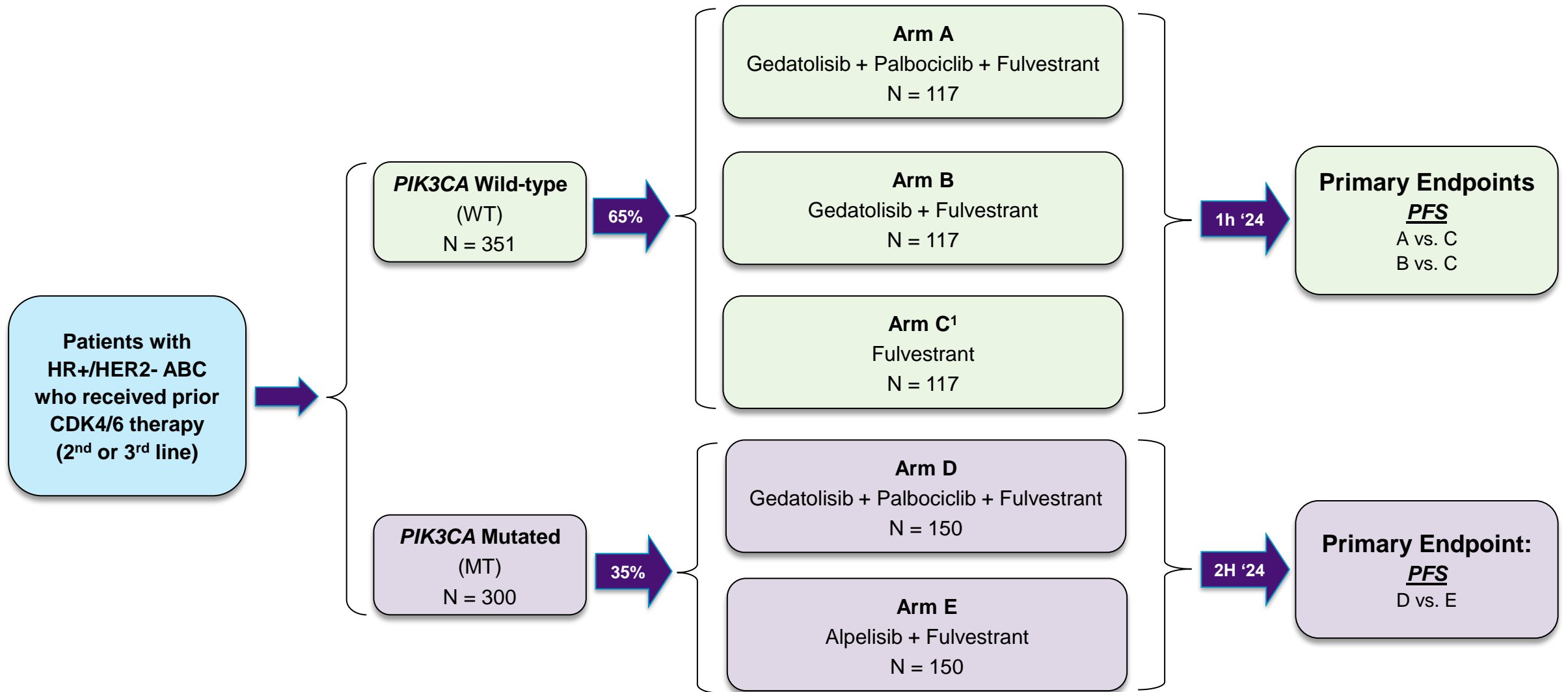
Phase 3 Study Design VIKTORIA-1

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



Relevant Clinical Trial Results for VIKTORIA-1 Study Arms

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

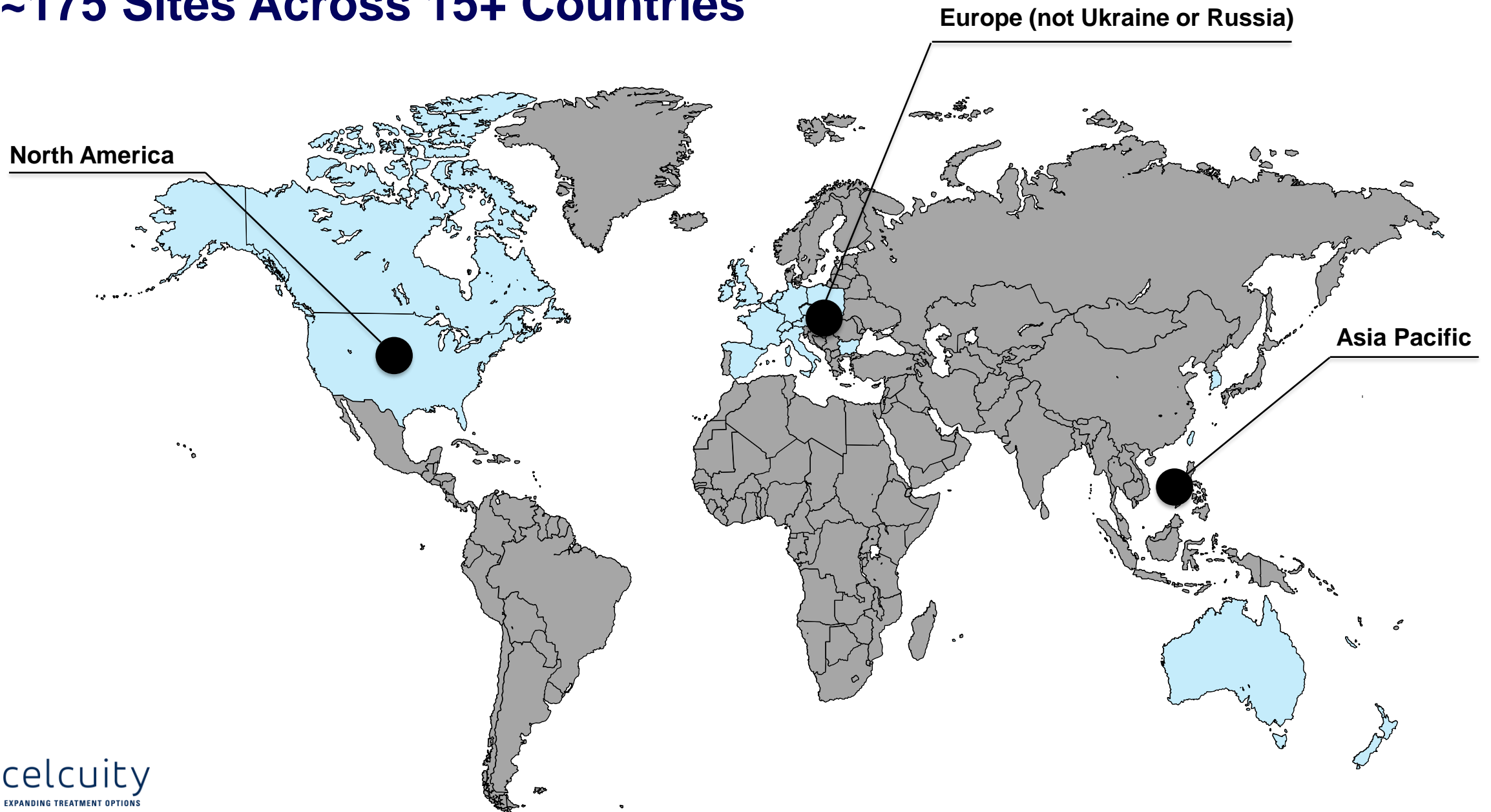
	Gedatolisib + Palbociclib + Fulvestrant N=27 ¹	Fulvestrant N=165 ⁴	Alpelisib + Fulvestrant N=121 ⁵
PIK3CA Status	WT / M (67% / 33%)	WT	M
Line of Therapy (% by line)	2L / 3L+ (67% / 33%)	2L / 3L+ (NR) ³	1L / 2L / 3L+ (12% / 70% / 19%)
mPFS (months)	12.9	1.9	7.3
ORR	63% (overall) ^{2,3} <u>WT</u> 59% <u>M</u> 78%	NR	21%
PFS % at 12 months	53.2% (overall) ³ <u>WT</u> 48.5% <u>M</u> 60.0%	10%	27%
Treatment Related Discontinuation	4%	1%	18%

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 and ≤ 1 prior chemotherapy
- 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 patients enables evaluation of two different regimens and shows contribution of gedatolisib
- Stratification by geography, prior chemotherapy (yes/no), prior treatment response (\leq or $>$ 6 months), presence of visceral 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

~175 Sites Across 15+ Countries



Experienced drug development team

CMO



Igor Gorbachevsky, MD



celcuity
EXPANDING TREATMENT OPTIONS

VP Clin Ops



Nadene Zack



VP Pharma Dev



Bernhard Lambert, PhD



VP Quality



David Bridge



VP Program Mgmt.



Michael Snitkovsky



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Pratima Nayak, MD



Leading cancer KOLs are participating in our research

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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

7 U.S. patents received

4 U.S. patents pending

Co-Founder and CSO



Lance Laing, PhD

Scientist at Scriptgen/Anadys (purchased by Novartis)

Director of Chemistry and Product Development for two instrument companies

PhD in biophysics and biochemistry - The Johns Hopkins University

Post-doc: Washington Univ. as NIH fellow

19 U.S. patents received

25 U.S. patents pending

CFO



Vicky Hahne

CFO – SimonDelivers (on-line grocery)

Controller – Respiritech (medical devices)

Controller – SterilMed (medical devices)

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

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Eric Lindquist

Global VP of BD at Natera (Signatera)

Global VP of CDx at Asuragen

CBO Cynvenio (CTC HER2, EGFR test)

Director of CDx at Ventana / Roche

Gedatolisib – A Phase 3 Ready 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 from Arm D of Phase 1b (basis for Phase 3)

- 63% ORR, 12.9 months mPFS
- High ORR and PFS rate at 12 mo for both – PIK3CA MT and PIK3CA WT
- <4% discontinuation rate

Multiple Potential Indications



Numerous other tumor types involve PI3K/mTOR signaling

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

Key Milestones



Laying groundwork for robust development plan

- Activate VIKTORIA-1 Phase 3 study in 1H '22
- Lifecycle development update in 1H '22
- CELsignia data readouts in 2023

Financial Resources



Strong balance sheet

- 12/31/21 - \$84.3 million cash on hand



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



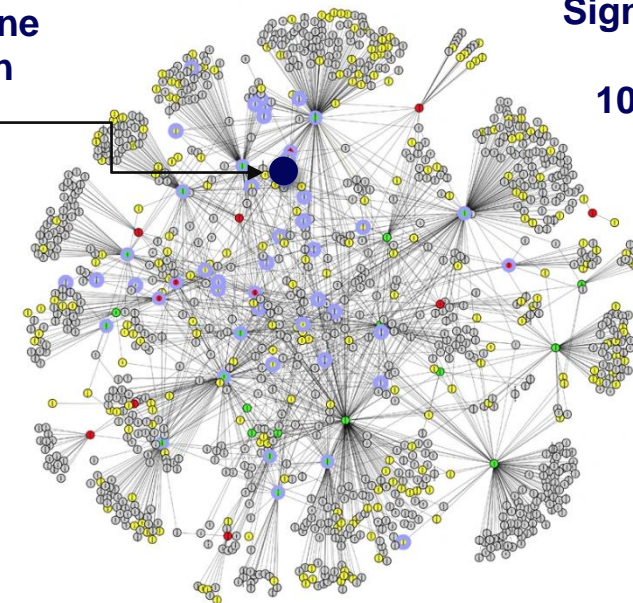
Memorial Sloan Kettering
Cancer Center

“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

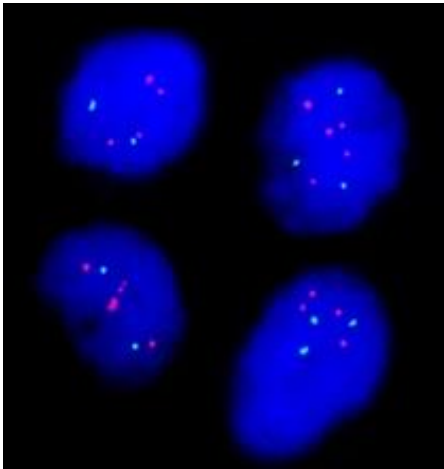


A **proprietary algorithm analyzes this “big data”** set to identify signaling activity 5 standard deviations from normal

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

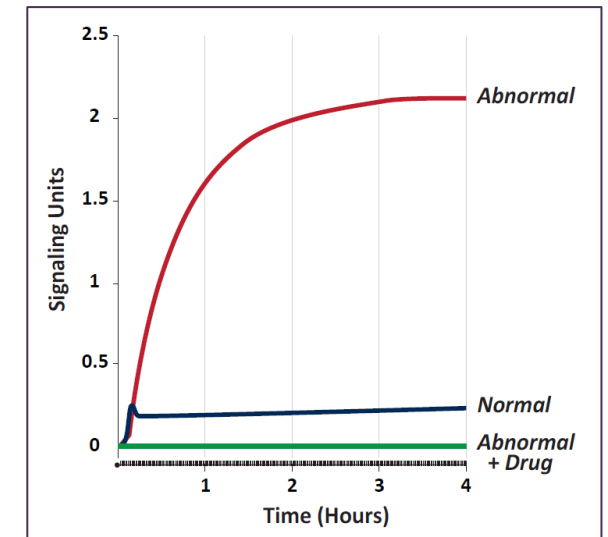
FISH+
15%

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

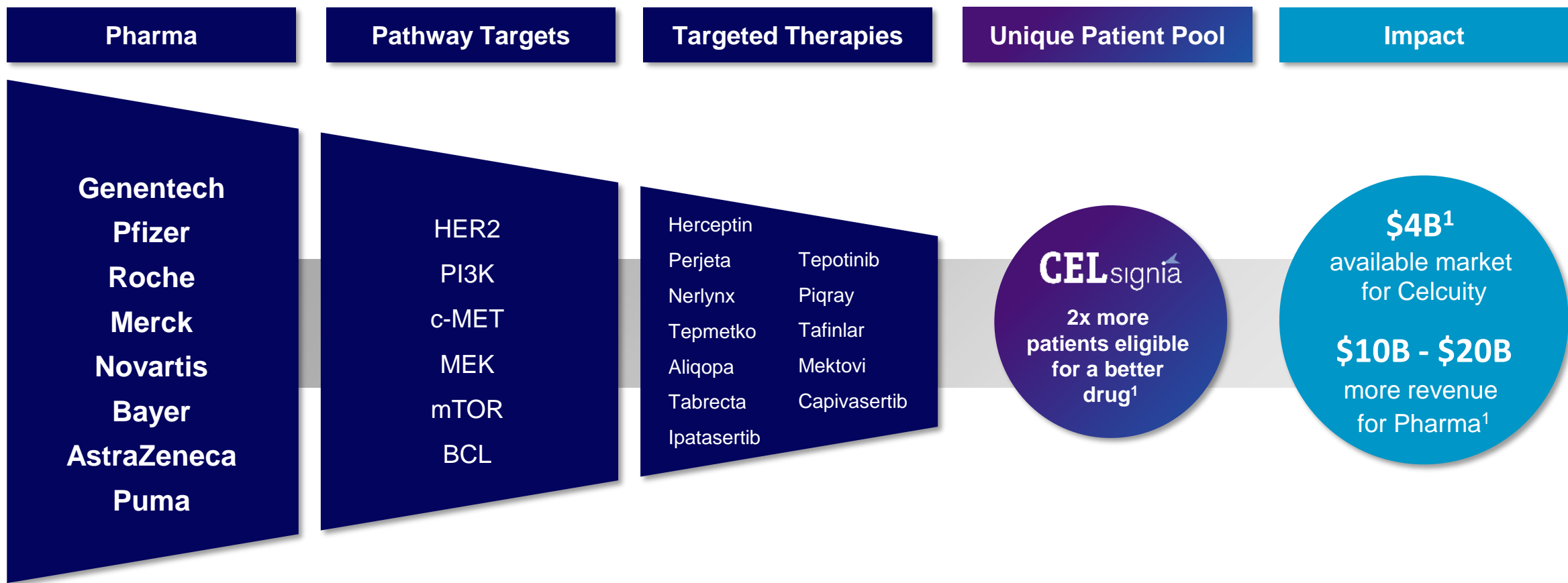
Drug resistance mechanisms characterized

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

Implications

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

CELsignia CDx identifies new patients for targeted therapies



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