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EXPANDING TREATMENT OPTIONS

Unraveling Complex Cellular Activity to Develop Targeted Therapies

Corporate Presentation

December 2022

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. New risks and uncertainties may emerge from time to time, and it is not possible to predict all risks and uncertainties. Any forward-looking statements in this presentation are based on management’s current expectations and beliefs and are subject to a number of risks, uncertainties and important factors that may cause actual events or results to differ materially from those expressed or implied by any forward-looking statements contained in this presentation, including, without limitation, risks relating to: (i) unforeseen delays in clinical trial enrollment or other activities that may affect the timing and success of our ongoing gedatolisib and CELsignia trials, (ii) the fact that preliminary data from a clinical study may not be predictive of the final results of such study or the results of other ongoing or future studies, (iii) unforeseen challenges in developing partnership opportunities with pharmaceutical companies, (iv) our ability to obtain and maintain FDA approval to commercialize gedatolisib, (v) our ability to raise additional capital for further product development and other activities, (vi) the development of products or services competitive with our products, including without limitation, other effective drug candidates, diagnostic tests and treatment options, and (vii) uncertainties and assumptions regarding the impact of the COVID-19 pandemic on our business, operations, clinical trials, supply chain, strategy, goals and anticipated timelines.

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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
  - **85% and 63% ORR** reported in 1st and 2nd line expansion arms in Phase 1b trial
  - **42.3** months mPFS in 1L patients; **12.9** months mPFS in 2L patients dosed with Phase 3 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. 37%)

Multiple Potential Indications

- Phase 3 trial for 2L+ patients with HR+/HER2- advanced breast cancer is currently enrolling
  - Addresses **100K+ annual patient population** globally
- Broad range of indications are possible given PI3K/mTOR's role in multiple tumor types

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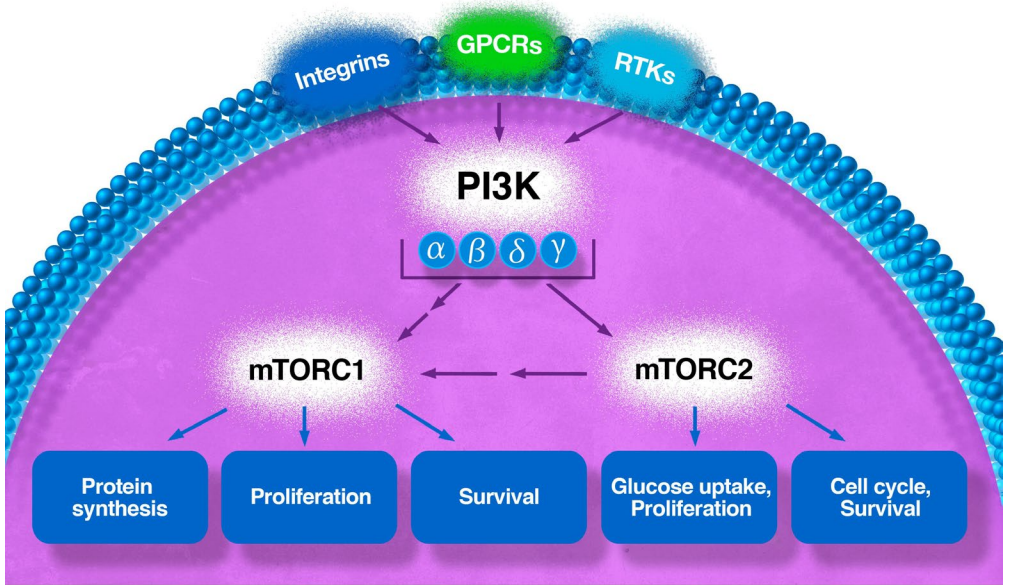
Abbreviations: SOC = standard-of-care; ORR = objective response rate; mPFS = median progression free survival; 1L = 1st line; 2L = 2nd line; TEAE = treatment emergent adverse events

PI3K/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%

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(1) Ciskova 2012; (2) Mills 2016

Difficult to Safely and Efficaciously Inhibit the PI3K/mTOR Pathway

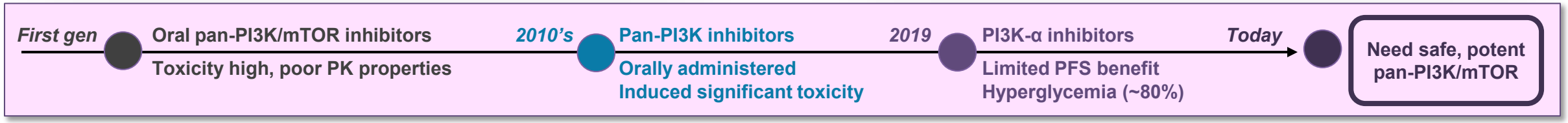
Maximum efficacy requires inhibition of all Class 1 PI3K isoforms and mTORC1 and mTORC2

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



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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
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

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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

Gedatolisib for Advanced Breast Cancer (ABC)

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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 *(PIK3CA WT)*	1.9[2,3]	6%[3]
Everolimus (mTOR) + Exemestane[4] *(PIK3CA WT)*	Unknown	Unknown
Alpelisib (PI3K-α) + Fulvestrant[5] *(PIK3CA MT)*	5.6 - 7.3	17% - 22%

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.[6]

(1) ORR is for patients with measurable disease; (2) Bardia 2021, EMERALD trial; (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. Abbreviations: ORR = objective response rate; PFS = progression free survival; WT = wild type; MT = mutant; NR = not reported.

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Clinical Development Plan

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

- For patients with HR+/HER2- ABC who progressed on CDK4/6 therapy
- All-comer design (*PIK3CA*+/-) incorporates separate primary endpoints for mutated and non-mutated *PIK3CA* patients
- Breakthrough Therapy Designation for this indication was granted by the FDA in July 2022

Significant potential indications based on POC and nonclinical study data

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

Review of Preliminary Phase 1b Data

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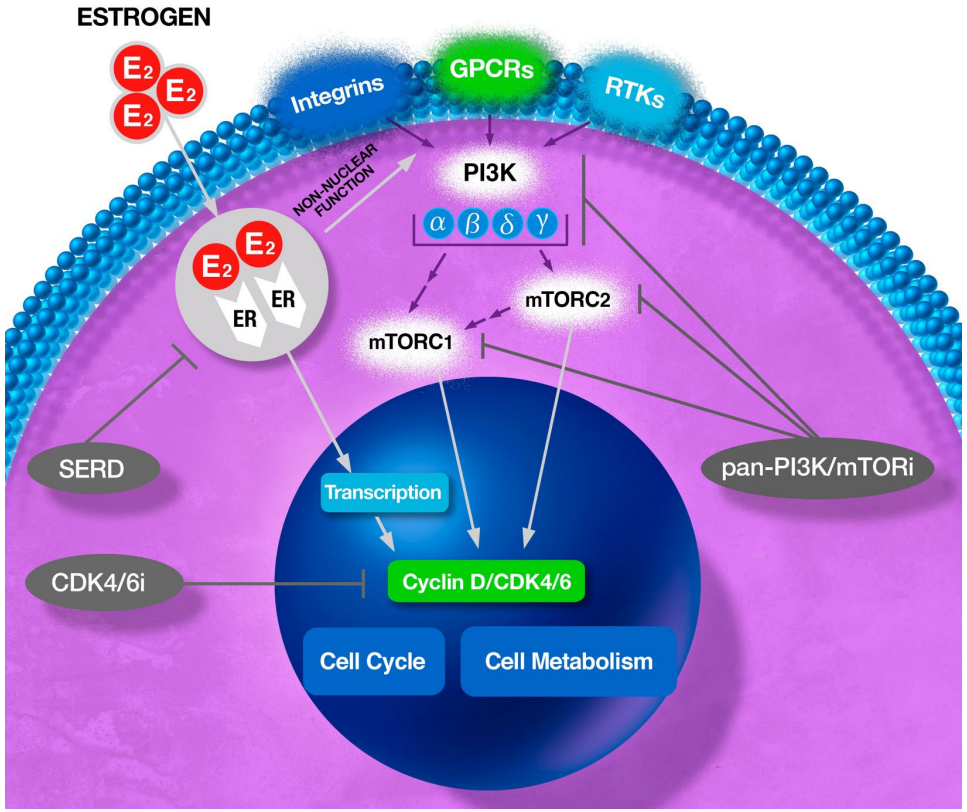
PI3K/mTOR, ER, and CDK4/6 are Interdependent Signaling Pathways

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



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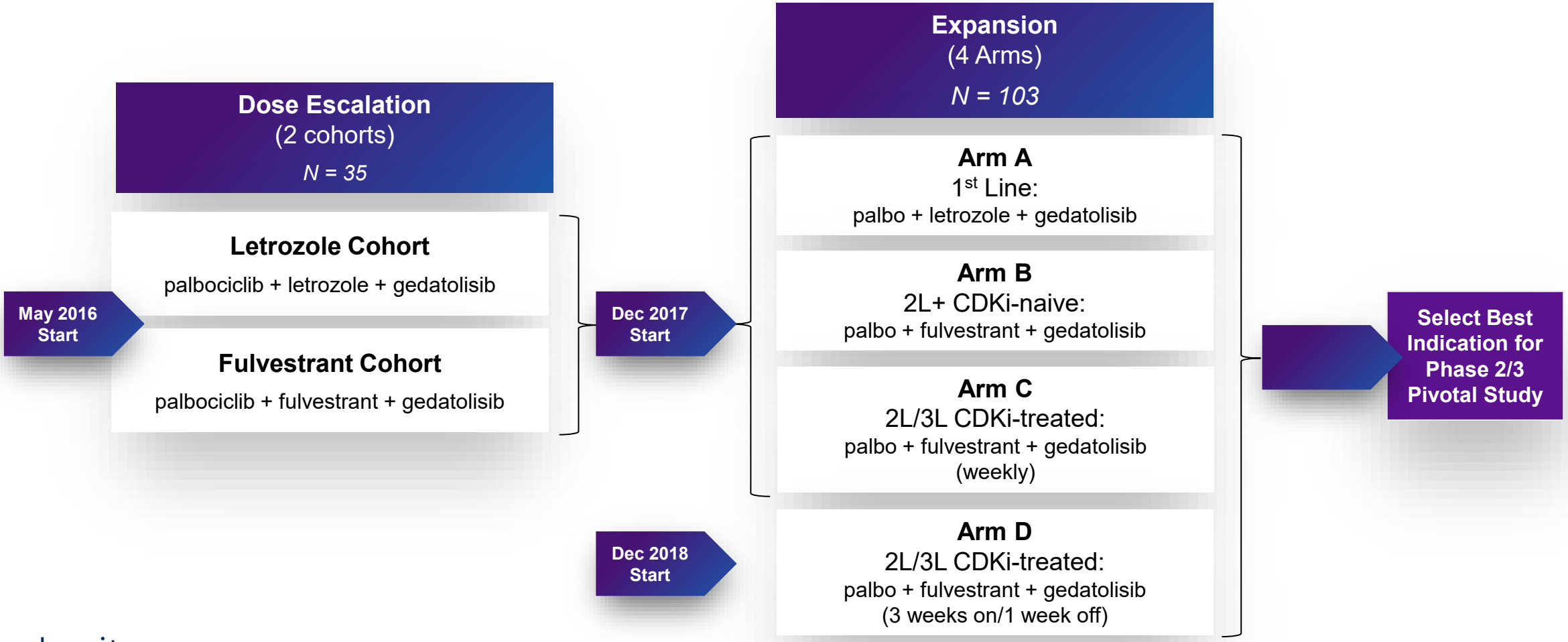
B2151009: Phase 1b Study (138 patients)

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



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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)								
	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)	NR[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% [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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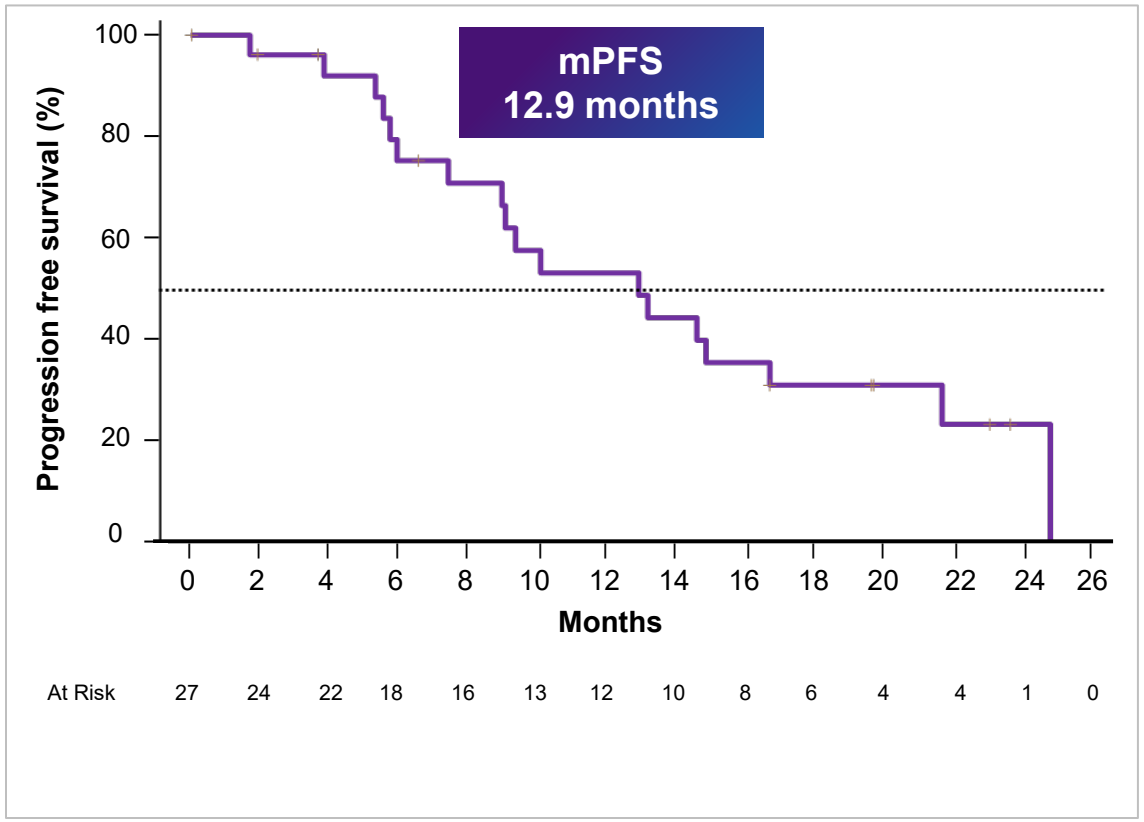
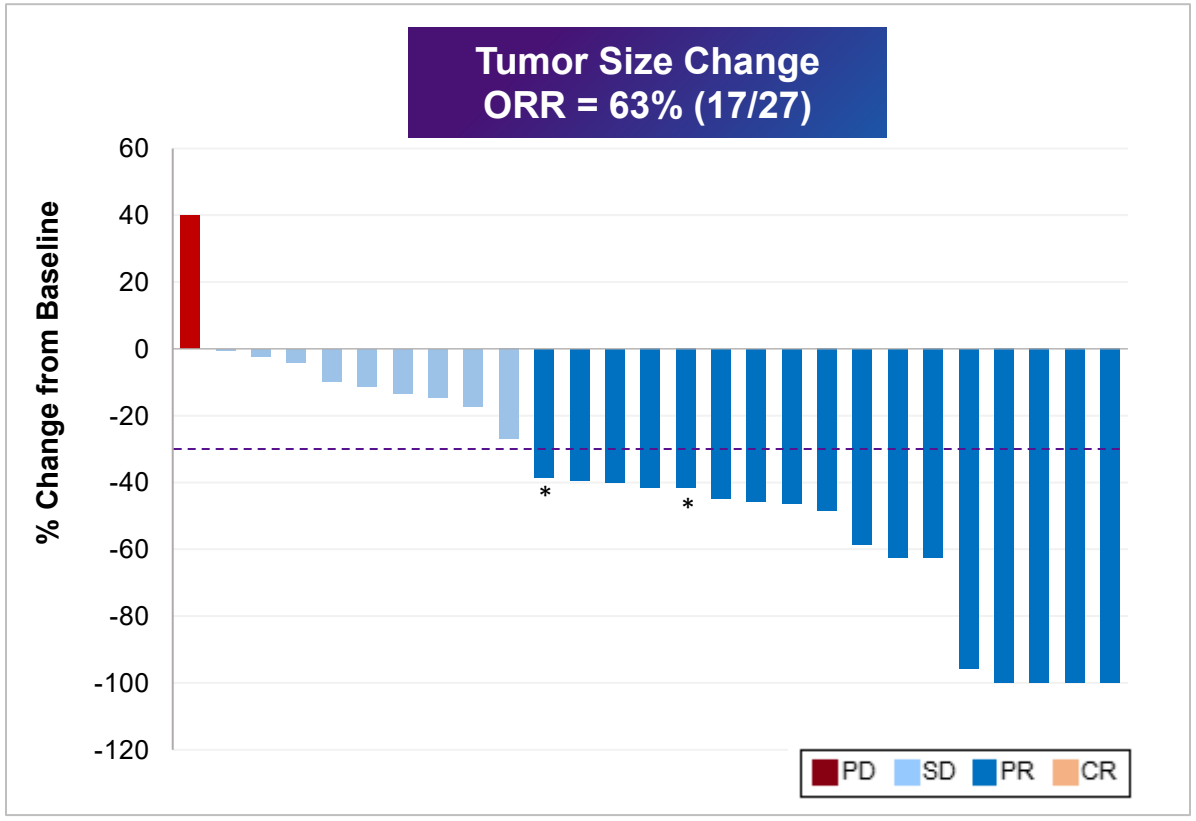
Source: Wesolowski 2022 SABCS. Footnotes: (1) Response evaluable analysis set per RECIST v1.1 including uPR; (2) full analysis set; (3) Baseline *PIK3CA* mutation status missing for one patient; (4) Median follow-up = 33.1 months. Abbreviations: 1L= first line, 2L= second line; mos = months; MT= *PIK3CA* mutation; NR = Not reached; ORR = objective response rate; mPFS = median progression free survival; SOC = standard of care; WT = wild type

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

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



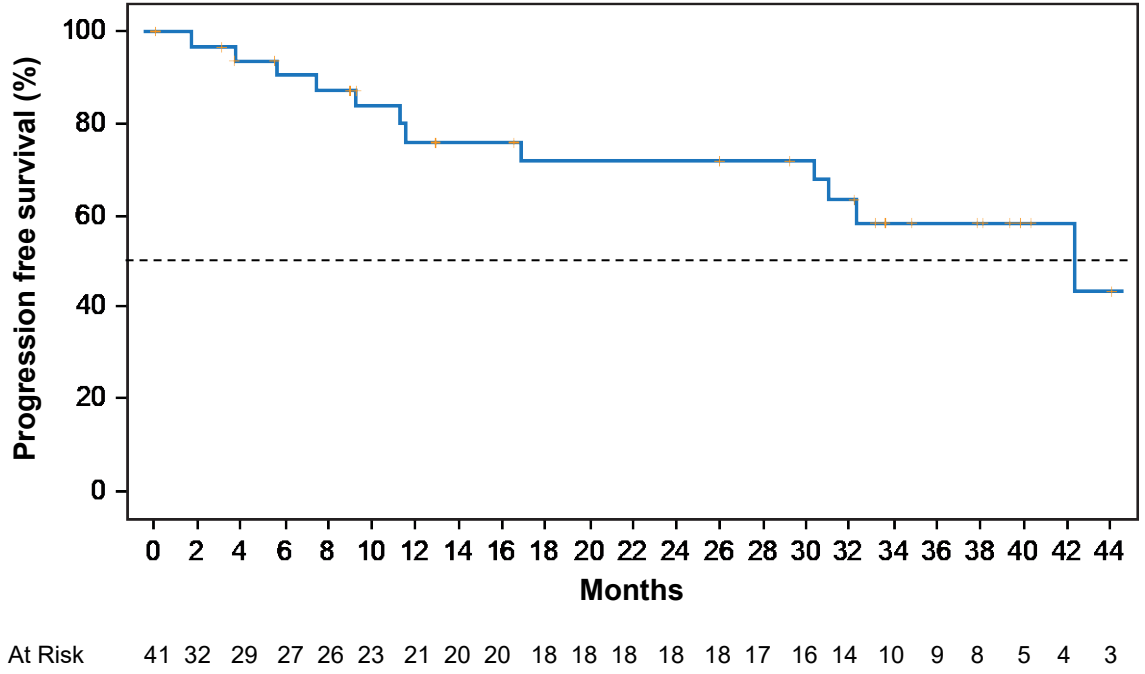


Source: Layman 2021 SABCS, Wesolowski 2022 SABCS - Arm D data from B2151009 study. ORR includes 2 unconfirmed PRs; *unconfirmed PR. Data presented is from a data analysis cutoff as of June 29, 2022

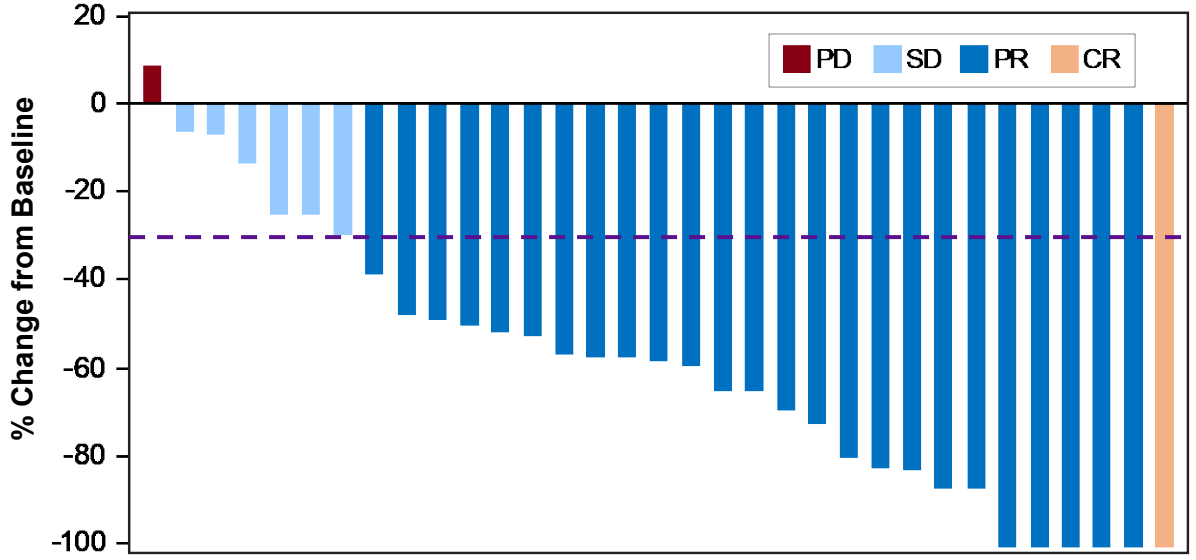
Gedatolisib + Palbociclib + Letrozole in 1st Line HR+/HER2- ABC (N=41)[1]

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

mPFS
42.3 Months



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



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(1) Wesolowski 2022 SABCS; 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.

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)	**42.3** (30.4, 45.8)	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

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

Gedatolisib Combo Offers Potential for Superior Efficacy Compared to SOC Options

2nd Line ER+/HER2- ABC (post-CDKi)	
Drug Regimen	**Efficacy**
Gedatolisib + Palbociclib + Fulvestrant[1] *(PIK3CA WT and MT patients)*	**mPFS 12.9 months** **ORR 63%**
Alpelisib + fulvestrant[2] *(PIK3CA MT patients only)*	**mPFS 5.6-7.3 months** **ORR 21%**
Fulvestrant[3] *(PIK3CA WT patients only)*	**mPFS 1.9** **ORR 6%**
Everolimus + Exemestane[4] *(PIK3CA WT patients only)*	**Unknown**

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(1) Wesolowski 2022 SABCS. (2) Rugo 2020 SABCS, BYLieve trial; Rugo 2021 SABCS, BYLieve trial (3) Bardia 2021, EMERALD trial mPFS only; Lindeman 2021, VERONICA trial mPFS and ORR; (4) No prospective clinical trials have been conducted for this regimen in this patient population. Note: No head-to-head trials have been conducted; data collected from different trials, in different patient populations and may not be comparable.

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

Gedatolisib Combo Offers Potential for Superior Efficacy Compared to SOC Options

1st Line ER+/HER2- ABC	
Drug Regimen	**Efficacy**
Gedatolisib + Palbociclib + Letrozole[1]	**mPFS 42.3 months** **ORR 79%**
Palbociclib + Letrozole[2]	**mPFS 24.5 months** **ORR 55%**
Letrozole[2]	**mPFS 14.5 months** **ORR 44%**

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Sources: (1) Wesolowski 2022 SABCS. (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.

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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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

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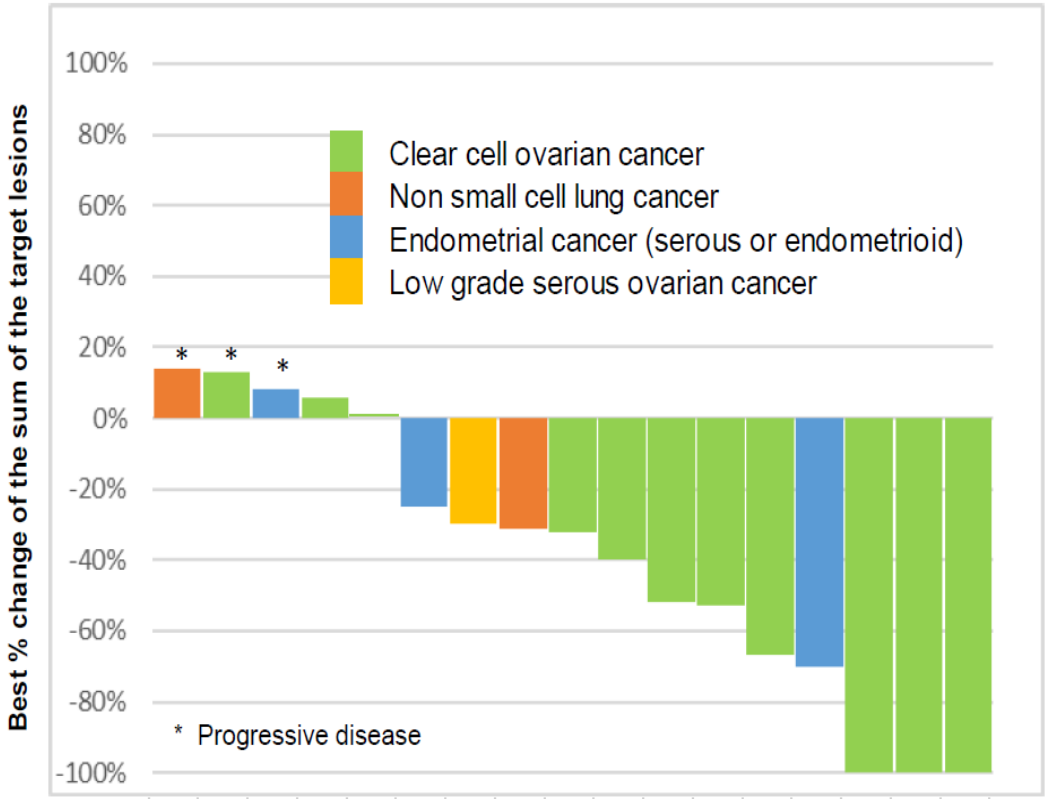
(1) Shapiro 2015; (2) Wesolowski 2022 SABCS; (3) USPI Alpelisib; (4) Rugo 2017. Abbreviations: ET = endocrine therapy; G = gedatolisib; P = palbociclib; F = fulvestrant; TEAE = treatment emergent adverse events; AE = adverse event. Note: Data presented for the B2151009 trial is as of a cut-off date June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

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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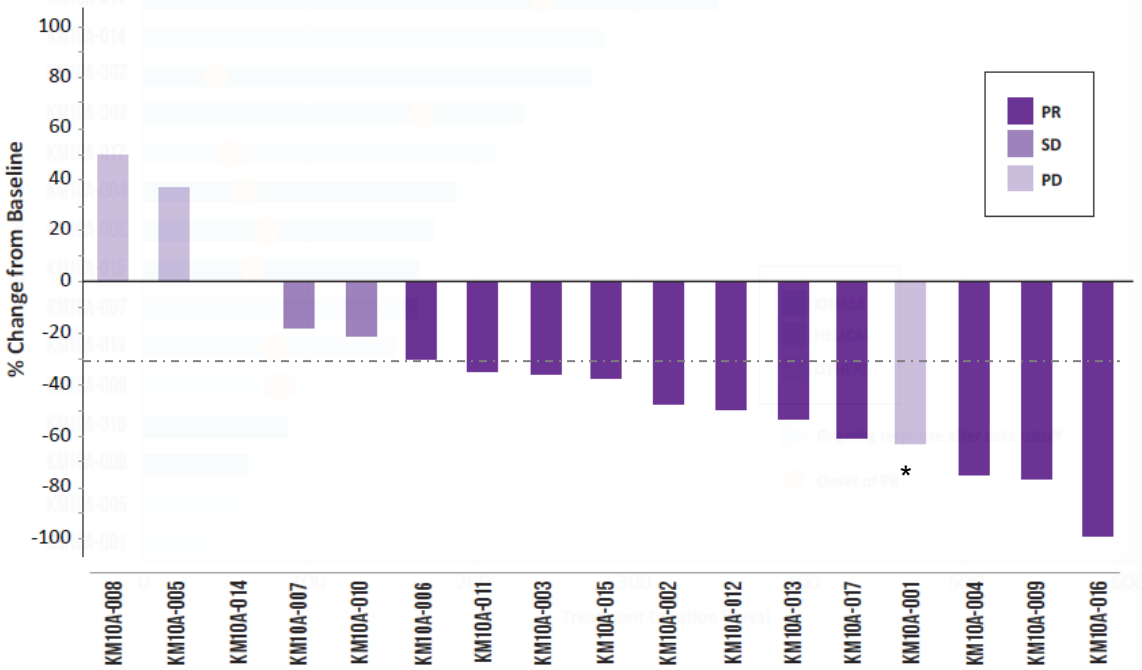
(1) Columbo 2021 CCR

59% ORR for Patients Receiving Gedatolisib + Trastuzumab Biosimilar[1]

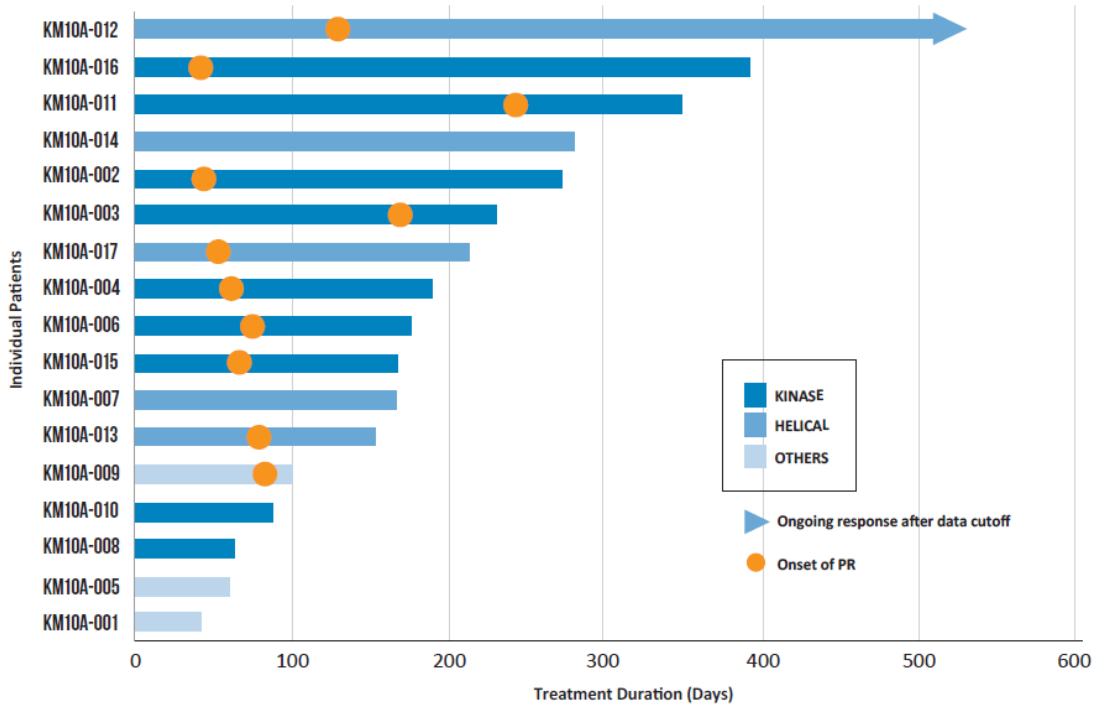
Best Response



* 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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(1) Kim 2021 SABCS. Note: Data presented is from an interim analysis of data as of a cutoff date of October 30, 2021, representing a database snapshot, and may change based on ongoing routine data monitoring and enrollment.

Phase 3 Study Design
VIKTORIA-1

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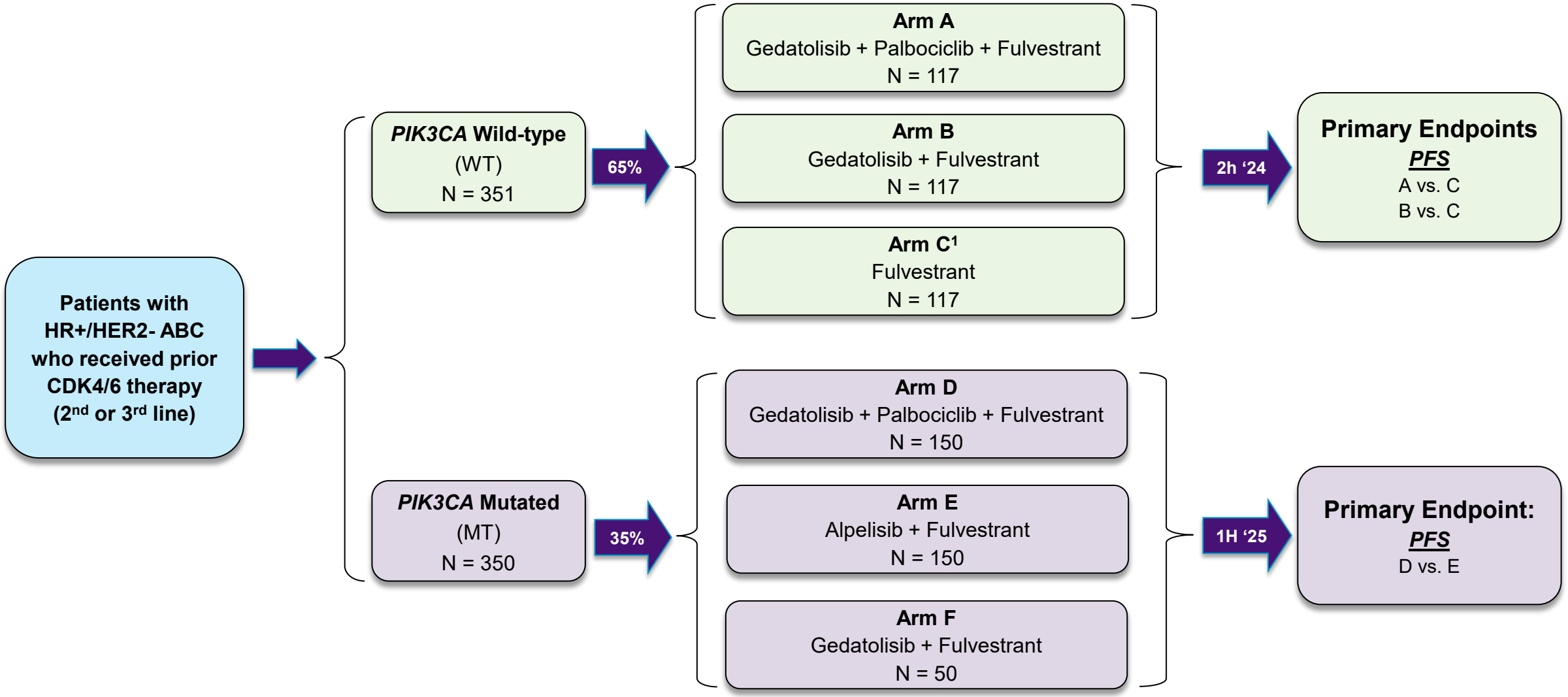
Pivotal Trial Design Considerations for 2nd Line HR+/HER2- ABC

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

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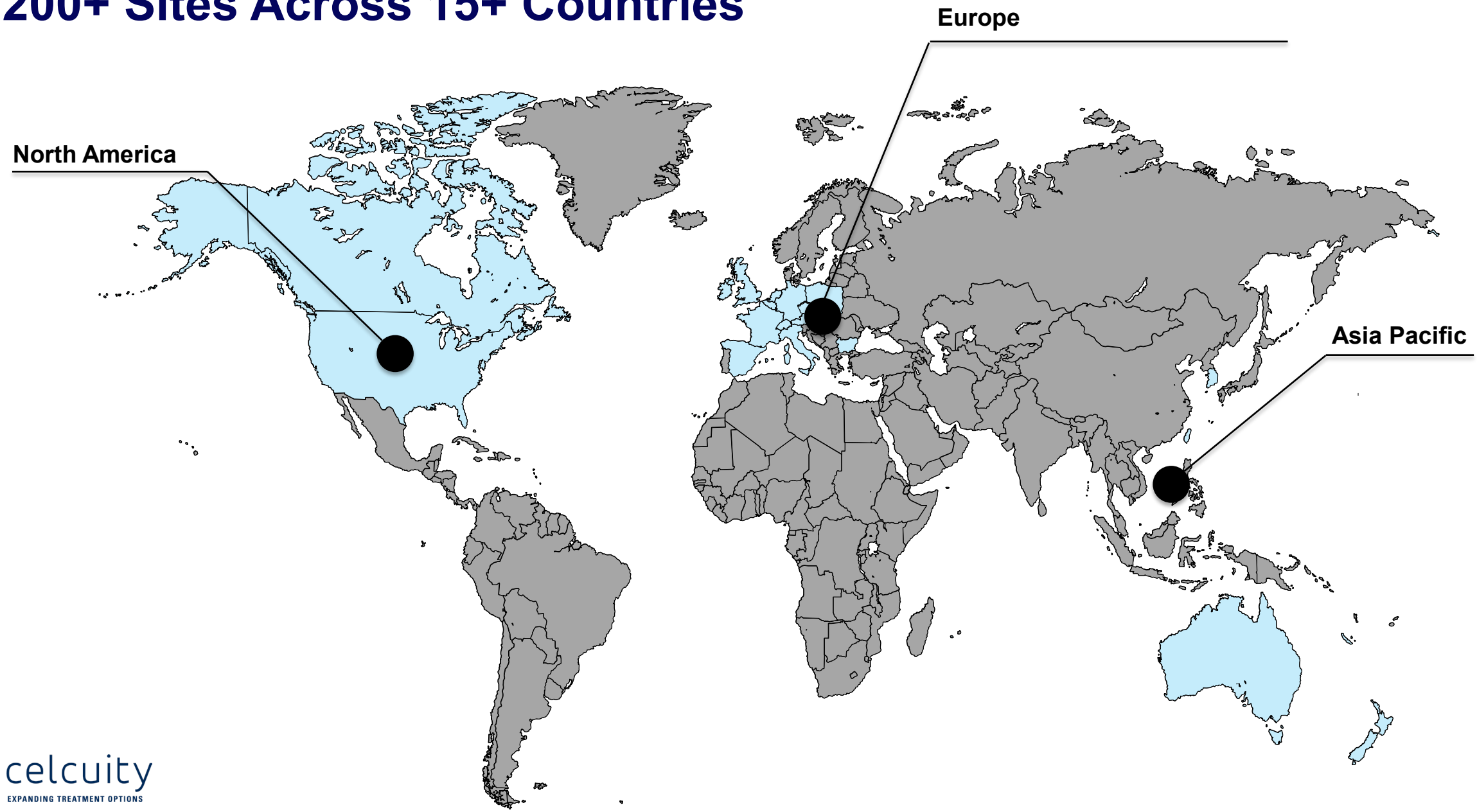
Sources: (1) Wesolowski 2022 SABCS; (2) Includes 2 unconfirmed PR.(3) Bidard 2022 – EMERALD trial; (4) 73% of patients had 1 prior line of endocrine therapy and 80% of patients had no prior chemotherapy in the advance setting; (5) Lindeman 2021, VERONICA trial; (6) Rugo 2021 SABCS (7) Rugo 2021 Lancet. Note: No head-to-head trials have been conducted; data collected from different trials, in different patient populations and may not be comparable. Data presented for gedatolisib is from a preliminary data analysis as of a cutoff date of June 29, 2022.

VIKTORIA-1 Pivotal Study Features

- Global open-label randomized study
- Key eligibility criteria:
  - Any *PIK3CA* status
  - 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 15+ Countries



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Experienced drug development team

CMO	VP Clin Ops	VP Pharma Dev	VP Quality	VP Program Mgmt.	VP Medical Affairs
					
Igor Gorbatchevsky, MD	**Nadene Zack**	**Bernhard Lambert, PhD**	**David Bridge**	**Fred Kershaw**	**Pratima Nayak, MD**
BAYER MEI pharma Daiichi-Sankyo J&J IOVANCE BIOTHERAPEUTICS	Pfizer darébio REGULUS	gsk CHIMERIX GILEAD Creating Possible	CHIMERIX EMD SERONO QUINTILES	MERCK J&J élan CYTOMX THERAPEUTICS	Takeda ATARA BIO rigel agios

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Leading cancer KOLs are participating in our research

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Ucla

Ben Ho Park M.D., Ph.D.

VANDERBILT UNIVERSITY

Adam Brufsky M.D., Ph.D.

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LIFE CHANGING MEDICINE

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UNIVERSITY OF ILLINOIS
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

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

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 – 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

- Arm D of Phase 1b (basis for Phase 3)
  - 63% ORR, 12.9 mos mPFS
  - High ORR and PFS rate at 12 mos for PIK3CA MT and PIK3CA WT
  - <4% discontinuation rate

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 enrolled first patient in Q4 ‘22
- Lifecycle development update in 1H ‘23

Financial Resources

Strong balance sheet

- $57.5 million cash on hand at end of Q3 2022
- Closed $100 million equity round in Q4 ‘22
- $20 million tranche of term loan available in Q4 ‘22

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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."

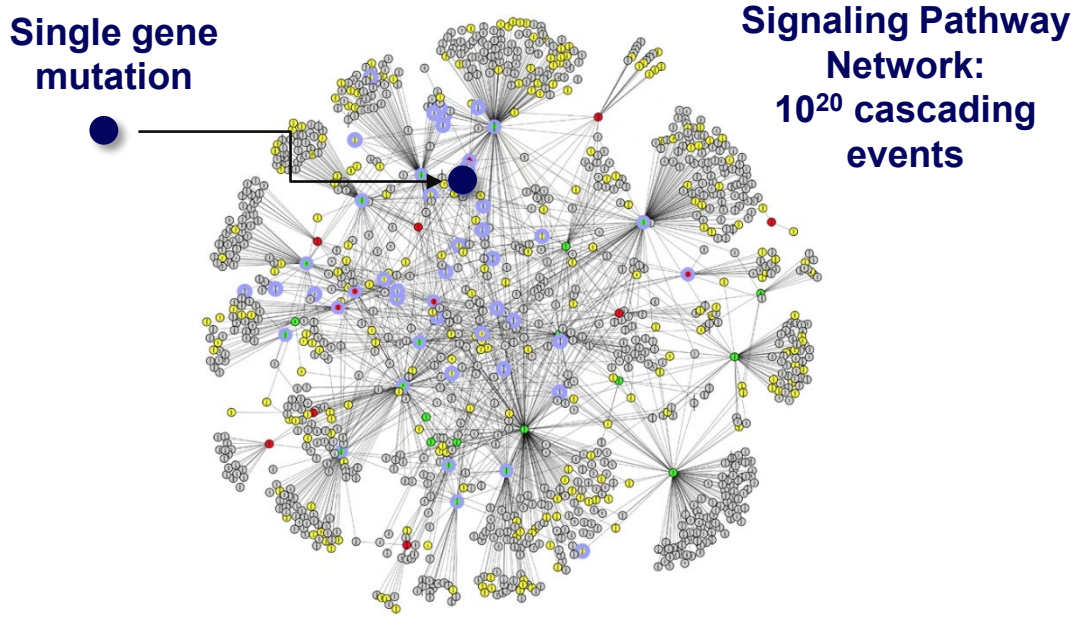
Neal Rosen, MD, PhD
Director, Center for Mechanism-Based Therapy
Memorial Sloan Kettering Cancer Institute

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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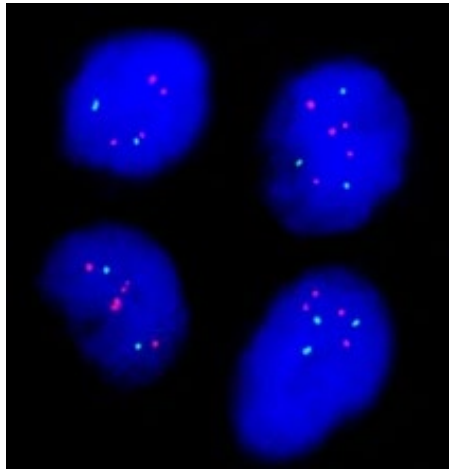
Source: 1) Internal Celcuity Studies

Current Molecular Diagnostics vs. CELsignia – HER2 Example

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

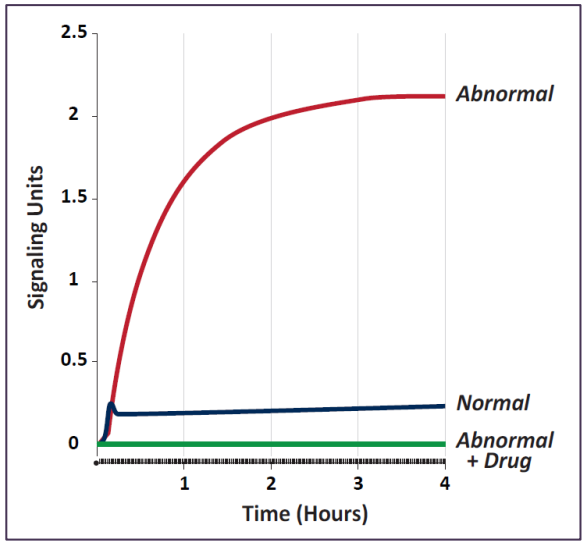
FISH HER2 Dx
(1 pathway gene)



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

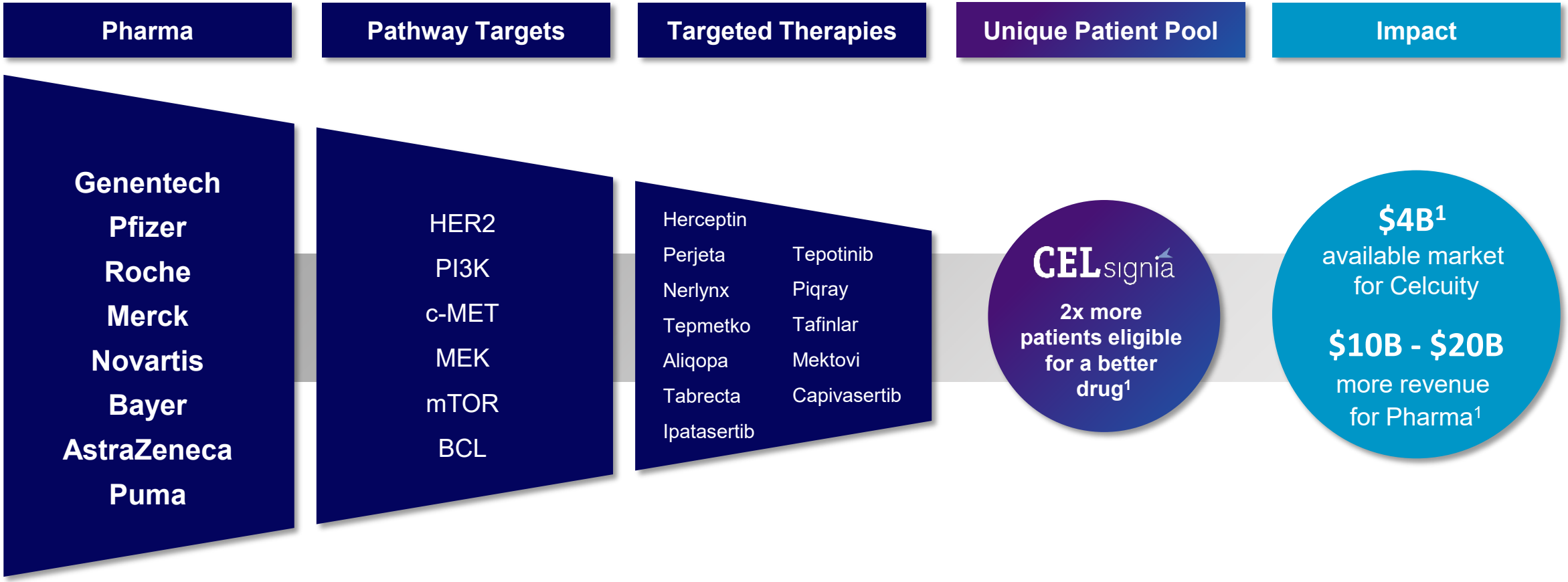
Drug resistance mechanisms characterized

- 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

CELsignia CDx identifies new patients for targeted therapies



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Source: 1) Internal Celcuity analysis

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

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