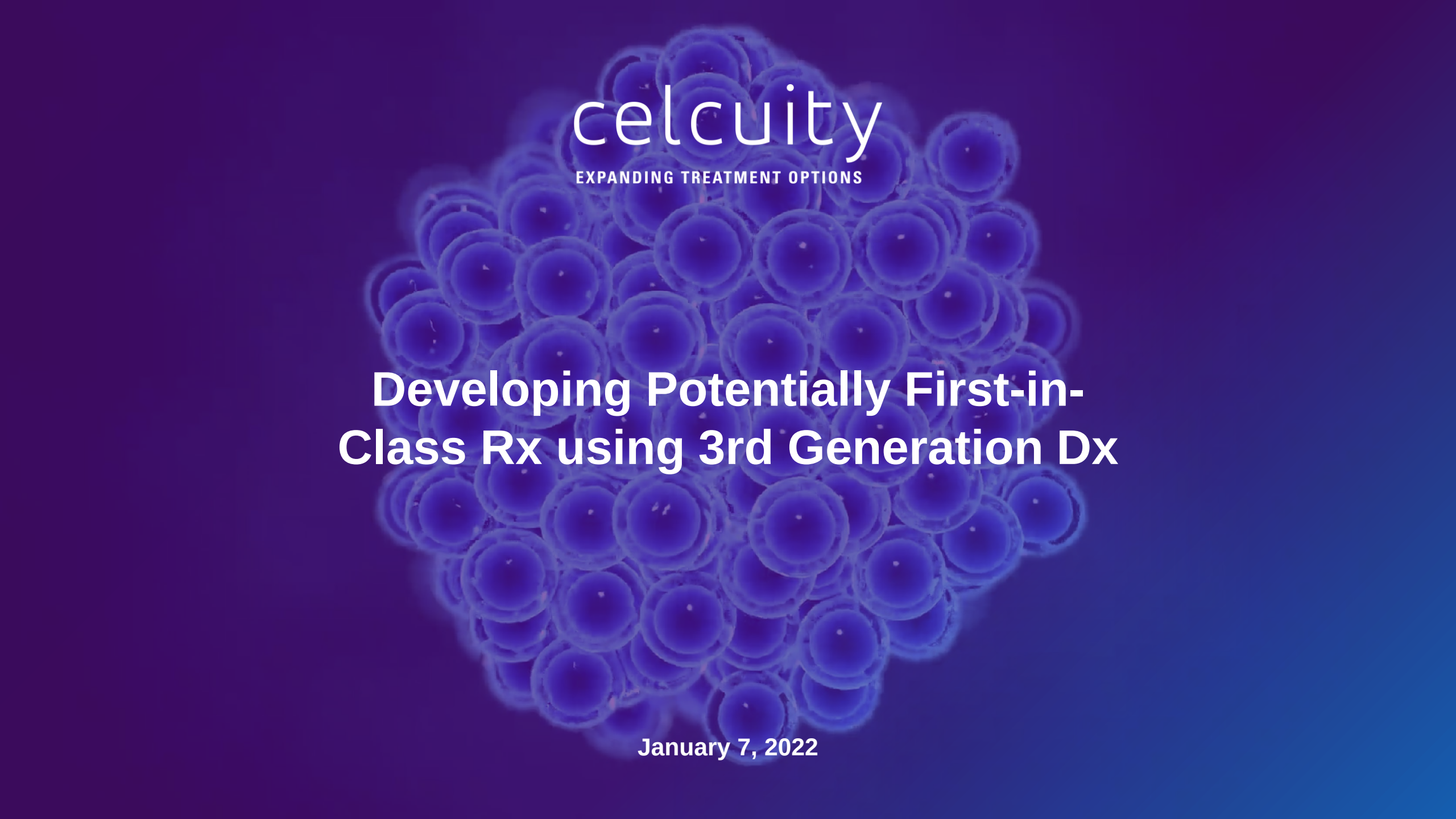

Developing Potentially First-in-Class Rx using 3rd Generation Dx

January 7, 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, the preliminary data of the B2151009 Phase 1b clinical trial, including its preliminary primary efficacy, safety and tolerability data, and other future conditions. 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) the success and timing of our ongoing FACT-1, FACT-2, FACT-3, FACT-4, FACT-5 and FACT-6 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) the success and timing of our product development activities and initiating clinical trials, (iv) expected partnership opportunities with pharmaceutical companies, (v) our ability to obtain and maintain regulatory approval of any of our product candidates, (vi) our plans to research, discover and develop additional product candidates, (vii) our ability to enter into collaborations for the development of new product candidates, (viii) our ability to meet any specific milestones set forth herein, and (ix) uncertainties and assumptions regarding the impact of the COVID-19 pandemic on Celcuity’s business, operations, clinical trials, supply chain, strategy, goals and anticipated timelines.

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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**

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

Novel Mechanism

- Potent 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

- In Phase 1b trial (N=103) treating HR+ / HER2- mBC with gedatolisib + ET + CDK4/6 reported:
  - 62% objective response rate (59/95) in evaluable patients
  - All four arms met their primary endpoint objective

Well-Tolerated

- Safety profile is well characterized - 492 patients treated with gedatolisib in eight clinical trials
- Well-tolerated with manageable TEAE's - 10% treatment discontinuation in mBC trial
- Significantly lower Grade 3/4 hyperglycemia than approved oral PI3K-α inhibitor (7% vs. 39%)

Significant Opportunities

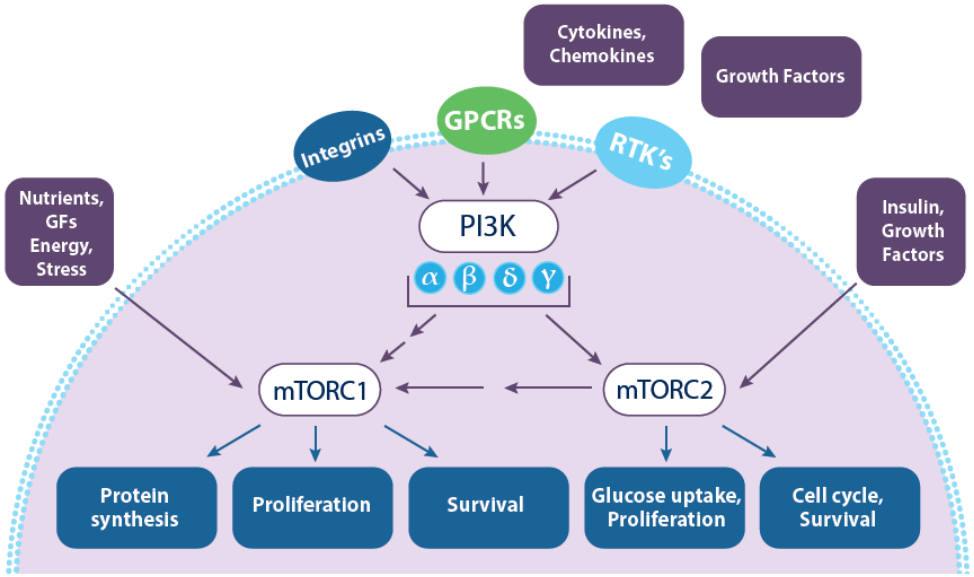
- Expect to initiate Phase 3 trial in 1H '22 for 2L+ patients with HR+ / HER2- metastatic breast cancer
  - Addresses 100 – 150K annual patient population globally
- Broad range of indications are possible given PI3K/mTOR's role in multiple tumor types

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

Inhibition of all Class 1 PI3K isoforms and mTOR1 and mTORC2 required for maximum efficacy

PI3K/mTOR regulates cell growth and metabolism

- Linked to multiple cell control decisions
- Can play a key role in driving cancer proliferation.
- 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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Source: (1) Ciskova 2012; (2) Mills 2016

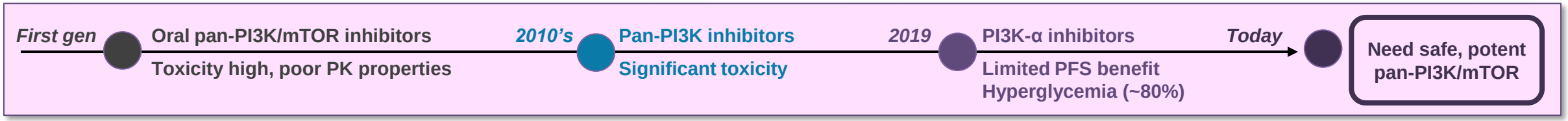
Difficult to Safely and Efficaciously Inhibit the PI3K/mTOR Pathway

Maximum efficacy requires equipotent pan-PI3K/mTOR inhibition, high bioavailability

- 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 is potent against all PI3K isoforms and mTORC1/2

Superior MOA minimizes potential for activation of resistance mechanisms

- **PIQRAY** (Novartis) - PI3K-α inhibitor for 2L therapy in ER+/PIK3CA+ mBC patients
  - PI3K-α inhibition can activate other PI3K isoforms and mTORC2
  - Doesn't address oncogenic signaling associated with other PI3K isoforms
- **AFINITOR** (Novartis) - mTOR inhibitor for 2L therapy in ER+/HER2- mBC patients
  - mTORC1 inhibition can activate PI3K signaling by relieving feedback regulatory mechanisms

IC_{50} (nM)
(cell-free biochemical dose response analysis)

Inhibitor	PI3K-α (m)	PI3K-α (WT)	PI3K-β	PI3K-γ	PI3K-δ	mTORC1	mTORC2
Gedatolisib[1]	0.6	0.4	6.0	5.4	6.0	1.6	1.6
PIQRAY (alpelisib)[2]	~4.0	4.6	1156	250	290	-	-
AFINITOR (everolimus)[3]	-	-	-	-	-	~2.0	-

No other pan-PI3K/mTOR inhibitor known to be under active development

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Sources: 1) Venkatesan 2010 for PI3K and mTORC1 IC50 values; 2) Fritsch 2014; 3) 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 vs. Other PI3K Inhibitors

PK properties are responsible for differentiated toxicity and efficacy profile

	Gedatolisib[1]	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
Organic class	Morpholino	Pyrrolidine	Quinazoline	*Isoquinoline*	*Isoquinoline*	*Pyrazolo-pyrimidine*
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
Grade 3-4 hyperglycemia[3]	7%	***39%***	***41%***	-	-	-

Comments

- **Hyperglycemia can be induced by PI3K-α inhibition and increased if drug has high affinity for the liver**
  - PI3K-α regulates glucose release and storage
  - Liver is the primary site of glucose regulation
- **6x higher hyperglycemia induced by alpelisib and copanlisib is due to higher liver exposure in each**
  - Alpelisib – daily oral administration
    - 22x more molar/month dosed than gedatolisib
    - Oral admin requires liver exposure
  - Copanlisib – PK profile
    - 25x higher retention by liver than plasma vs. gedatolisib
- **Other gedatolisib PK advantages vs. Alp and Cop**
  - 4x-20x higher C_{max} and superior AUC plasma
  - Distributed in blood/plasma 4x-30x more efficiently than alpelisib and copanlisib
- **Higher toxicity of PI3K-δ drugs**
  - Likely due to amount and route of administration
    - 3.7x-35x more molar/month administered
  - Significant GI, liver, and infection-related AE's

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Sources: 1) Venkatesan 2010; internal Celcuity studies; 2) FDA label; 3) No head-to-head trials have been conducted; data collected from different trials, in different patient populations and may not be comparable.

Gedatolisib for Breast Cancer

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HR+/HER2- Metastatic Breast Cancer (mBC) Treatment SOC

High unmet medical need for better options for 2L patients who have received a CDK4/6 inhibitor

First Line

Treatment (Patient Group)	mPFS (months)	ORR[1]
CDK4/6i + letrozole[2] *(TFI > 12 months)*	24.8	55%
CDK4/6i + fulvestrant[3] *(TFI < 12 months)*	9.5	25%

Second Line

Treatment (Patient Group)	mPFS (months)	ORR[1]
Everolimus (mTOR) + Exemestane[4]	**4.2**	**17%**
Fulvestrant[5]	**3.7[6]**	**NR**
Alpelisib (PI3K-α)[7] + Fulvestrant *(PIK3CA+)*	**7.3**	**21%**

Sources: (1) ORR is for patients with measurable disease; (2) PALOMA-2 trial; (3) PALOMA-3 trial; (4) Rozenblit 2019, Dhakali 2020. (5) Luhn 2018; (5) Duration of treatment; (6) EMERALD trial (N=165); (7) BYLieve trial

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Clinical Development Plan Pending FDA Input

Phase 2/3 study for patients with ER+/HER2- mBC who progressed on CDK4/6 therapy

- Goal is to begin enrollment of Phase 2/3 clinical trial for gedatolisib with palbociclib + fulvestrant in first half of 2022
- All-comer design (PIK3CA+/-) that will incorporate a CELsignia PI3Ks+ sub-group
- Trial design will be finalized upon 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%

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Review of Preliminary Phase 1b Data

As of May 10, 2021 data cut-off

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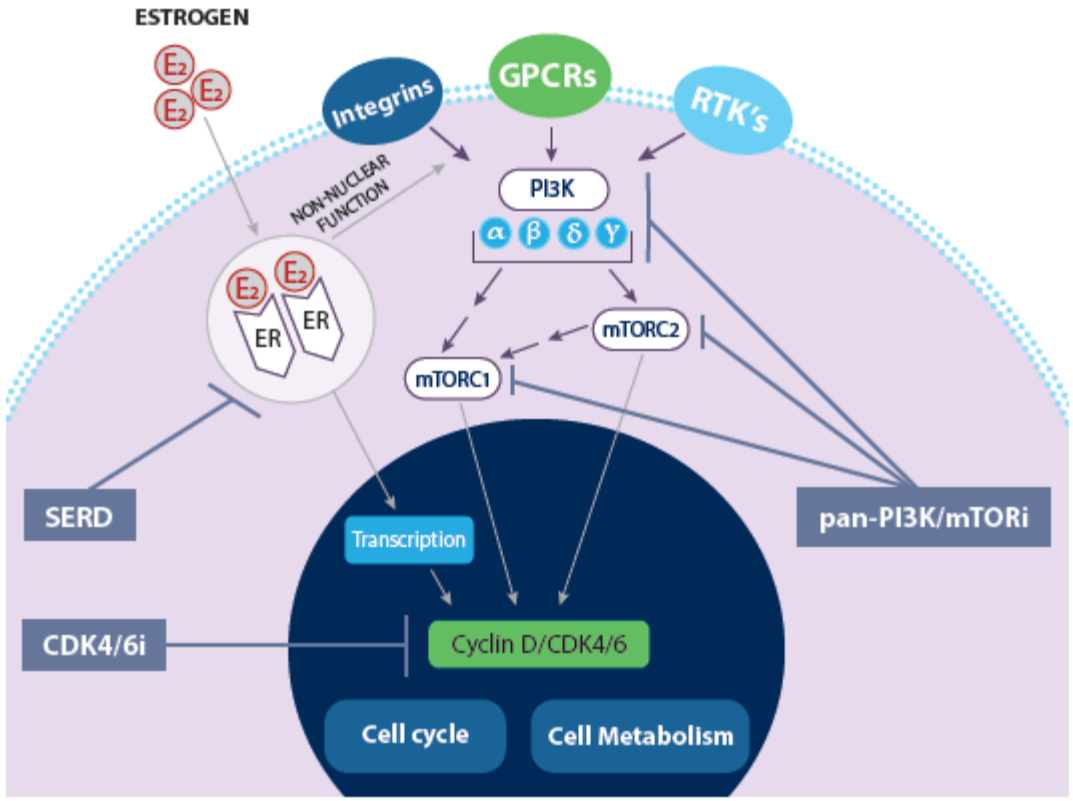
PI3K/mTOR, ER, and CDK4/6 are Interdependent Signaling Pathways

PI3K/mTOR is a key resistance mechanism to ER and CDKi treatment

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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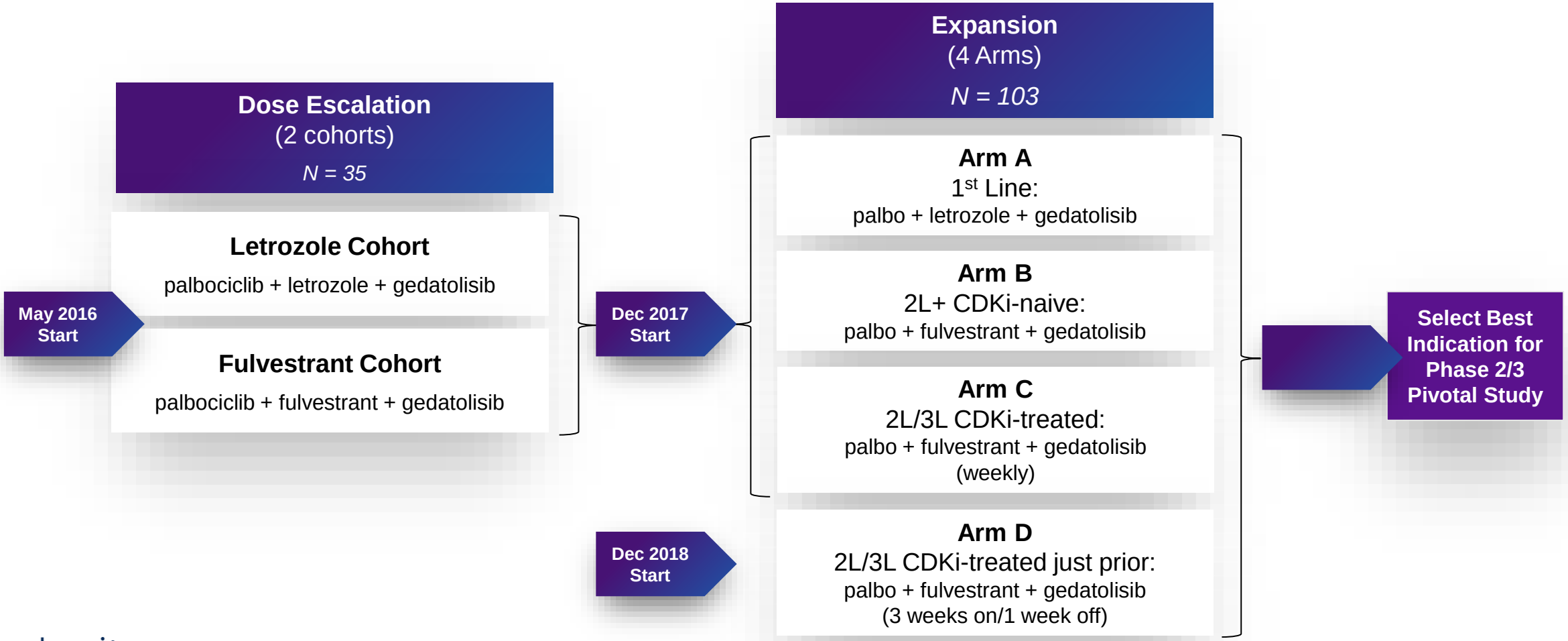
B2151009: Phase 1b Study (138 patients)

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



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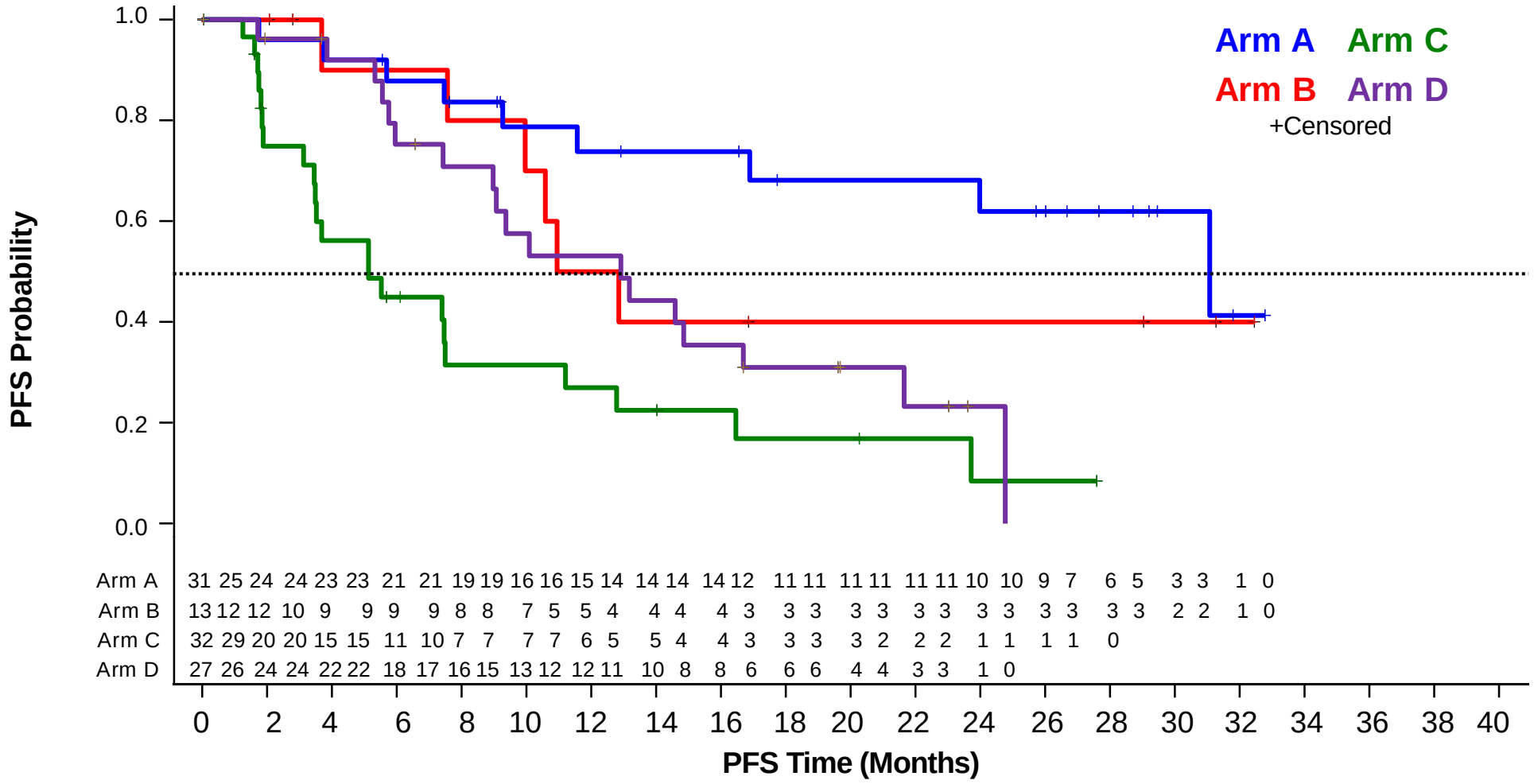
Efficacy Summary

Each arm met its primary endpoint objective

B2151009 (Phase Ib) (N=103)				
Arm	A (N=31)	B (N=13)	C (N=32)	D (N=27)
Patients	1L: CDKi-Naïve	2L+: CDKi-naïve	2L/3L: CDKi-pretreated	2L/3L: CDKi-pretreated
Study Treatment	G+ P + L (weekly)	G + P + F (weekly)	G + P + F (weekly)	G + P + F **(3 week on/1 week off)**
# of Evaluable Patients	27	13	28	27
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)

(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

Progression Free Survival (PFS) Kaplan-Meier Curves

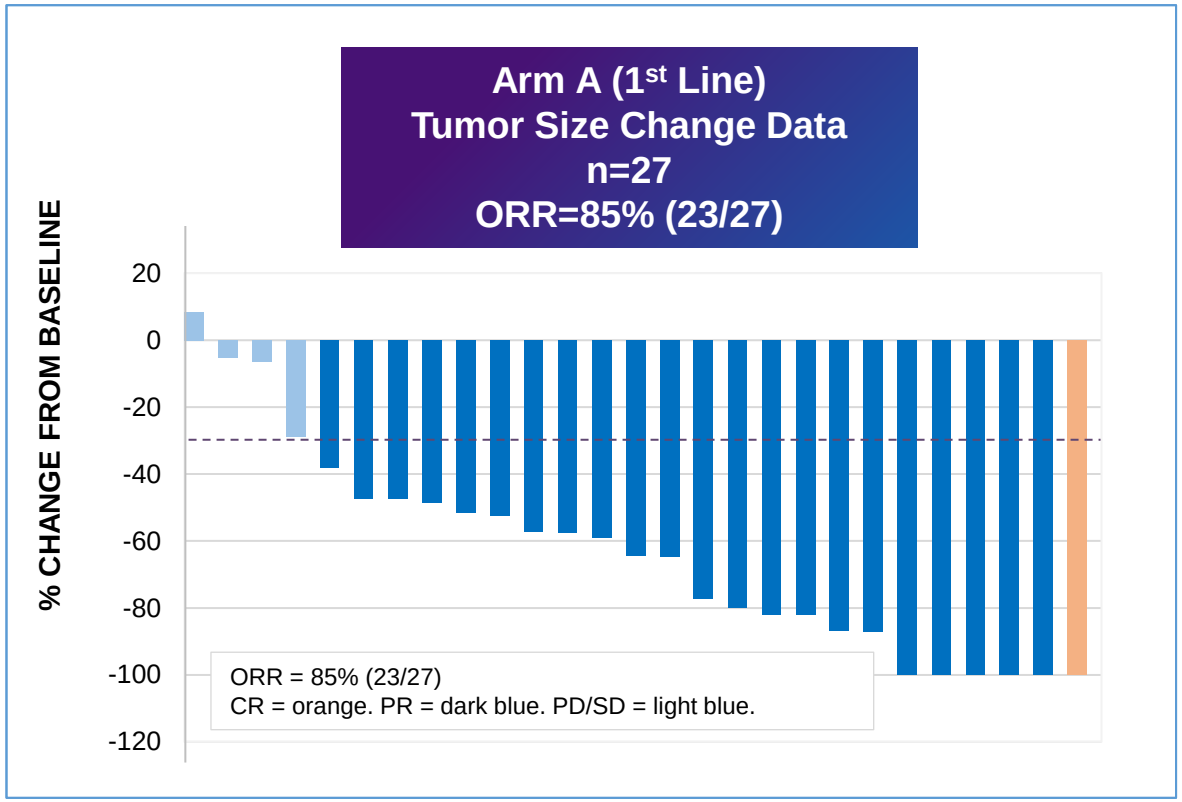


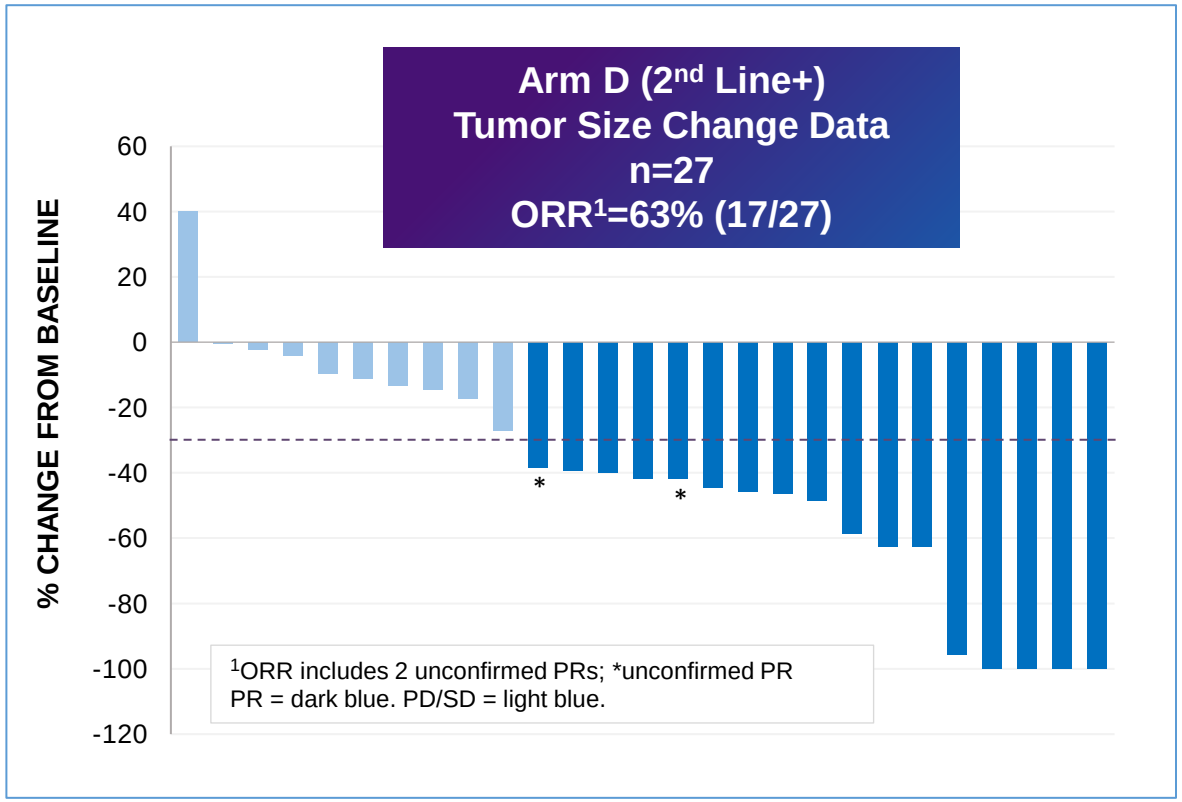
Source: Layman 2021 SABCS

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 A and D: Best Response - Tumor Size

85% ORR in 1st line and 63% ORR in 2nd line+ patients





Source: Layman 2021 SABCS. 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

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SOC Palbociclib + Endocrine Therapy + / - Gedatolisib

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

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Source Arm D: Layman 2021 SABCS
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 May 10, 2021, representing a database snapshot, and may change based on ongoing routine data monitoring.

2L/3L Gedatolisib + Palbociclib + Fulvestrant vs. 2L SOC

	Prior CDKi			
Evaluable Patients	N=142[1]	N=165[3]	N=100[5]	**N=27[6]**
Study Treatment	Everolimus + ET	Fulvestrant	Alpelisib + Fulvestrant	**G + P + F**
PIK3CA Status	M / WT	M / WT	M	**M / WT[5]**
Line of Therapy	2L	2L	2L/3L	**2L/3L**
ORR (95% CI)	17%[2] (9%-31%)	16%[4] (10%-24%)	21% (14%-30%)	**63%[6,7]** (42%-81%)
PFS	4.2[2]	3.7[3]	7.3	12.9

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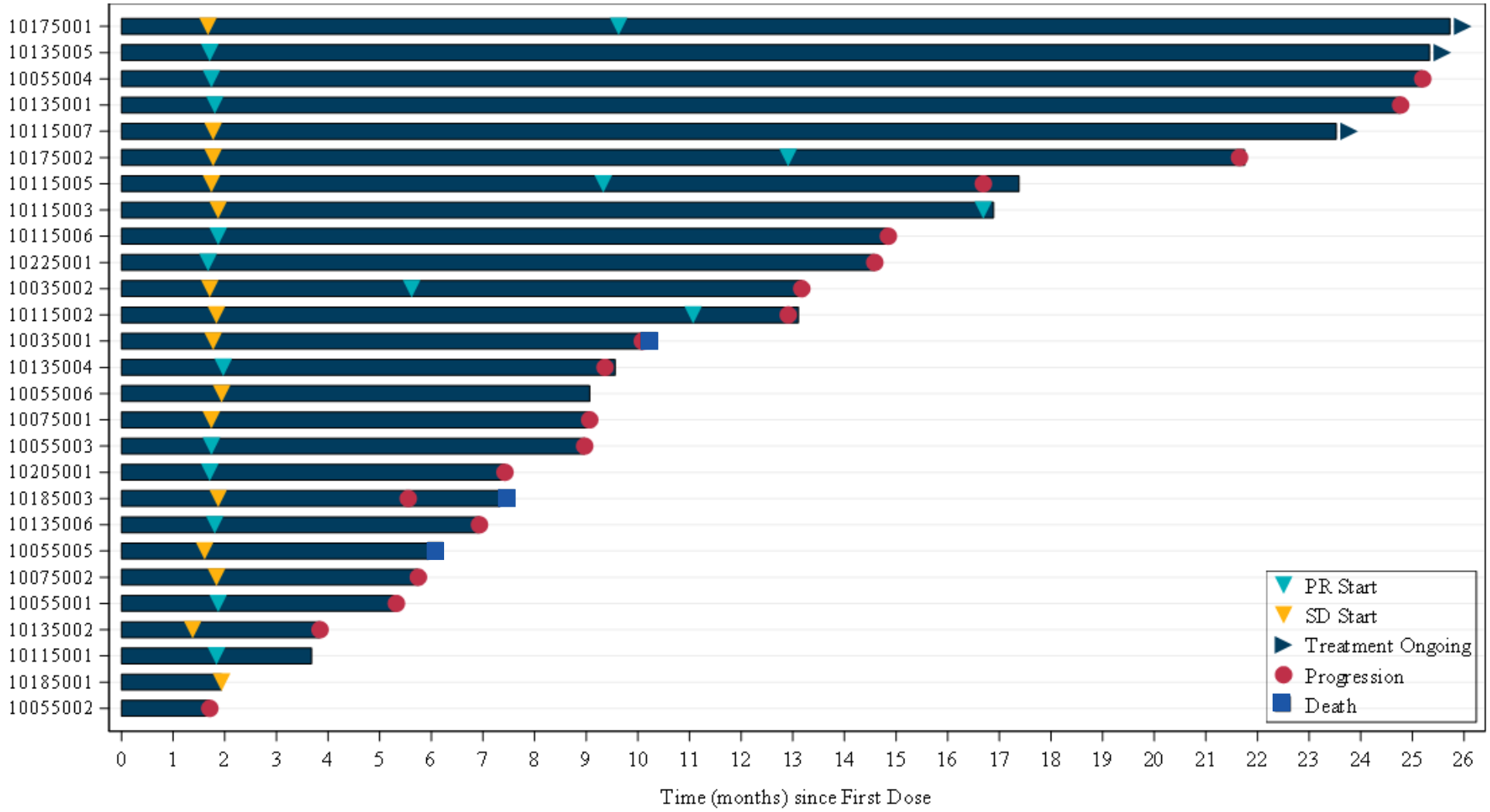
Sources: (1) Rozenblit 2019; (2) Dhakali 2020; (2) BOLERO-2 trial; (3) Luhn 2018 – duration of treatment; (4) SOLAR-1 trial; (5) BYLieve trial; (6) B2151009 trial - Arm D (5) 9 of 27 patients (33%) were PIK3CA+; (6) 6 of 9 PIK3CA+ patients (67%) had an OR; 11 of 18 PIK3CA- patients (61%) had an OR; (7) Includes 2 unconfirmed partial responses
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 May 10, 2021, representing a database snapshot, and may change based on ongoing routine data monitoring.

Arm D: Time to First Response, Duration of Response & Treatment



Each bar (patient) starts from first dose to last dose, last assessment, or death, whichever occurs last

Source: Layman 2021 SABCS

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

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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

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Gedatolisib Combinations vs. SOC Benchmarks for ER+ / HER2- mBC

Biggest unmet need is in the 2nd line setting where gedatolisib combo has most differentiation

2nd/3rd Line ER+/HER2- Metastatic (post CDKi)	
Drug Regimen	**Efficacy**
Gedatolisib + Palbo + Fulvestrant[1]	**PFS 12.9 months, ORR 63%**
Alpelisib + fulvestrant[2] (PI3K-α + SERD for PIK3CA+)	**PFS 7.3 months, ORR 21%**
Fulvestrant[3] (SERD)	**PFS 3.7 months**
Everolimus + Exemestane[4] (mTOR + AI)	**PFS 4.2 months**

Sources: (1) B2151009 – Arm D; (2) BYLieve; (3) Luhn 2018 SABCS. Real-world data for patients with prior-CDK4/6 treatment receiving fulvestrant using electronic health records from Flatiron; (4) Rozenblit 2019 SABCS. Real world data for patients with prior CDK4/6 treatment receiving everolimus + exemestane using electronic health records from Flatiron
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 May 10, 2021, representing a database snapshot, and may change based on ongoing routine data monitoring.

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Safety Summary: Treatment-Emergent Adverse Events

Single Agent gedatolisib and gedatolisib + palbociclib + ET

Phase 1 Trial: Gedatolisib alone

(154 mg weekly IV)

	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: G + P + ET

- Combo has been well tolerated
- <10% discontinued the drug due to AE
- Nearly 20% of patients were on treatment for >24 months
- Most TEAE's were Grade 1 or 2
- Stomatitis was treated at manifestation, not prophylactically
  - Prophylactic treatment reduces incidence and severity 80%
- Few hyperglycemia-related adverse events (22% all, 7% Grade 3/4)
  - Alpelisib (79% all, 39% Grade 3/4)
- Neutropenia, leukopenia, and anemia AEs related to palbociclib

Phase 1b Trial: G + P + ET

(180 mg IV, once weekly or 3 weeks, one week off)

	All Arms (n=27)		
	TEAE's > 30%		
	All Grades	Grade 3	Grade 4
Adverse Event	%	%	%
Stomatitis	81	27	-
Neutropenia	80	53	14
Nausea	75	11	-
Fatigue	68	-	-
Dysgeusia	46	-	-
Vomiting	45	1	-
Anemia	40	12	-
Constipation	37	4	-
Diarrhea	34	4	-
Decreased appetite	32	4	-
Leukopenia	32	13	3

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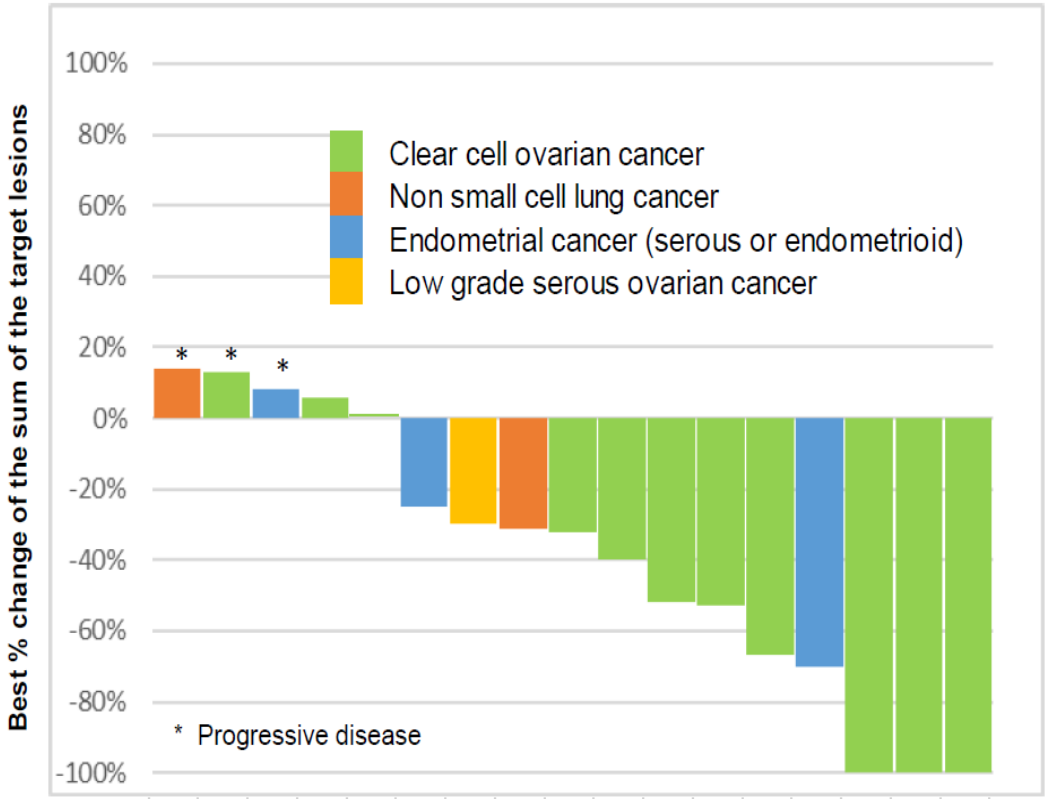
Note: Data presented for the B2151009 trial is from a preliminary data analysis as of a cutoff date of May 10, 2021, representing a database snapshot, and may change based on ongoing routine data monitoring.

Gedatolisib with Paclitaxel and Carboplatin in Patients with Solid Tumors

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



Study was an IST and the results were published in Clinical Cancer Research in July

- Seventeen patients were enrolled:
  - 10 clear cell ovarian, 4 endometrial, 2 NSCLC, 1 low grade ovarian
- The safety profile was favorable
- Clear cell ovarian cancer (CCOC)
  - ORR overall: 80% - 5/10 PR, 3/10 CR
  - ORR by platinum status: 6/7 in platinum naïve, 2/3 in prior platinum
- Low grade serous ovarian
  - 1/1 PR (prior platinum)
- NSCLC
  - 1/2 PR (prior platinum) and 1/2 PD
- Endometrial Cancer
  - 1/4 PR (no prior platinum), 2/4 SD, and 1/4 PD
- Prior platinum (all tumors)
  - 4/9 PR (45%)
- 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 sample size is very small, but the CCOC data is interesting. ORR for platinum therapy reported in platinum-naïve CCOC patients ranges from 25%-50%
- CCCO only accounts for 5-10% of ovarian cancers in US (~15% in Japan) so we must assess practicality of pursuing this indication.
- Will assess likelihood other ovarian sub-types may benefit from gedatolisib + platinum therapy

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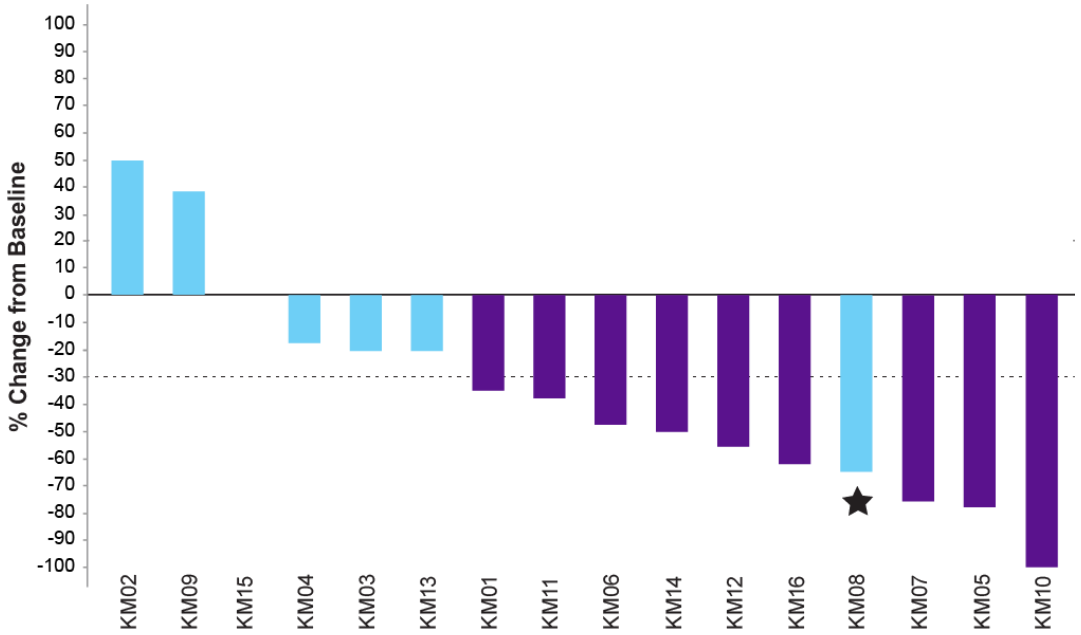
56% ORR for Patients Receiving Gedatolisib + Trastuzumab Biosimilar

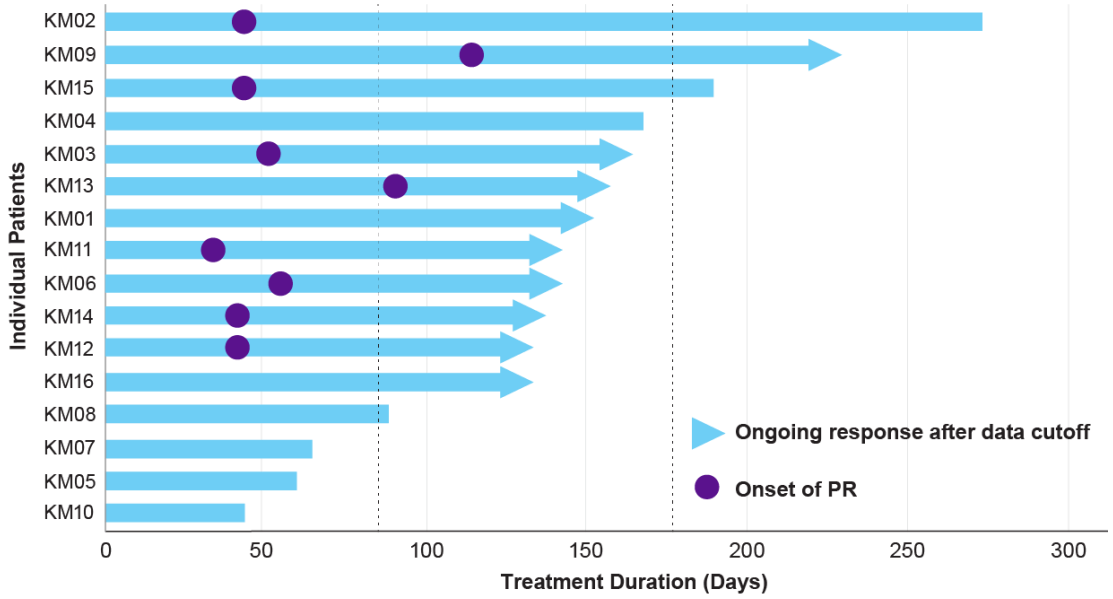
Best Response



*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.

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Note: Data presented is from an interim analysis of data as of a cutoff date of October 30, 2020, representing a database snapshot, and may change based on ongoing routine data monitoring and enrollment.

Experienced drug development team

SVP Clin Dev

Igor Gorbatchevsky, MD

VP Clin Dev at MEI Pharma

- Responsible for zandelisib (PI3K-δ inhibitor)

VP Clin Science at Iovance Biotherapeutics

Global Clin Leader at Bayer Pharmaceuticals

- Responsible for ALIQOPA, a pan-PI3K inhibitor

Senior Medical Director at Daiichi-Sankyo

VP Clin Ops

Jill Krause

VP Clin Ops Quality and VP Study Mgmt at Odonate

- Nine years of experience managing breast cancer clinical trials
- Over 10 years experience at Pfizer in various clinical operations roles.
- Led clinical operations teams at various CRO's

VP Pharma Dev

Bernhard Lambert, PhD

Executive Director, Pharmaceutical R&D at Chimerix

- Served in various CMC roles at Gilead and Glaxo Wellcome

VP Quality

David Bridge

Director of QA at Duke Clinical Research Institute

Senior Director, QA at Chimerix

Clinical QA Lead at EMD Pharmaceuticals

VP Program Mgmt.

Michael Snitkovsky

Senior Director, Program Leadership at Finch Ther

Senior Director of Operations, Red Oak Med

Associate Director, Head of Project Mgmt at Alnylam Pharmaceuticals

SVP R&D

John MacDonald, PhD

SVP R&D at MGI Pharma

- Senior executive responsible for all drug discovery, preclinical, and clinical teams at MGI Pharma
- Obtained FDA approvals for a number of oncology therapeutics while leading those teams. He began his career at Warner Lambert.

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Leading cancer KOLs are participating in our research

Clinical Advisory Board

Mark Pegram M.D. Ph.D.

Stanford University

Sara Hurvitz M.D.

Ucla

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

VANDERBILT UNIVERSITY

Adam Brufsky M.D., Ph.D.

UPMC
LIFE CHANGING MEDICINE

Filip Janku M.D., Ph.D.

THE UNIVERSITY OF TEXAS MD Anderson Cancer Center

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MOFFITT CANCER CENTER

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Mothaffar Rimawi M.D.

Baylor College of Medicine

Alberto Montero M.D.

University Hospitals
The Science of Health. The Art of Compassion.

Lee Schwartzberg M.D.

The West Clinic
Advancing Cancer Care. Together.

Scientific Advisory Board

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University of Minnesota

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Ron McGlennen M.D.

access genetics

Benita Katzenellenbogen Ph.D.

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 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 – Respirtech (medical devices)

Controller – SterilMed (medical devices)

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

CBO

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

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Summary – Strategic Overview

Proprietary CELsignia Technology

CELsignia can identify new indications for targeted oncology therapies

- Collaborations with numerous pharma partners to determine new indications for their compounds
- Applying CELsignia to our own compound leverages its potential

Gedatolisib

Preliminary results from phase 1b clinical trial show encouraging anti-tumor activity

- Phase 3 ready asset[1]
- 62% objective response rate
- Well tolerated safety profile with <10% gedatolisib discontinuation rate

Key Milestones

Laying groundwork for robust development plan

- Obtain FDA feedback Phase 2/3 study in early ‘22
- Activate Phase 2/3 study in 1H ‘22
- Lifecycle development update in 1H ‘22

Financial Resources

Strong balance sheet

- 9/30/21 - $90.4 million cash on hand
- 7/1/21 – Received $52.8 million net proceeds from follow-on equity offering

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Note: 1) Pending FDA feedback

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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."

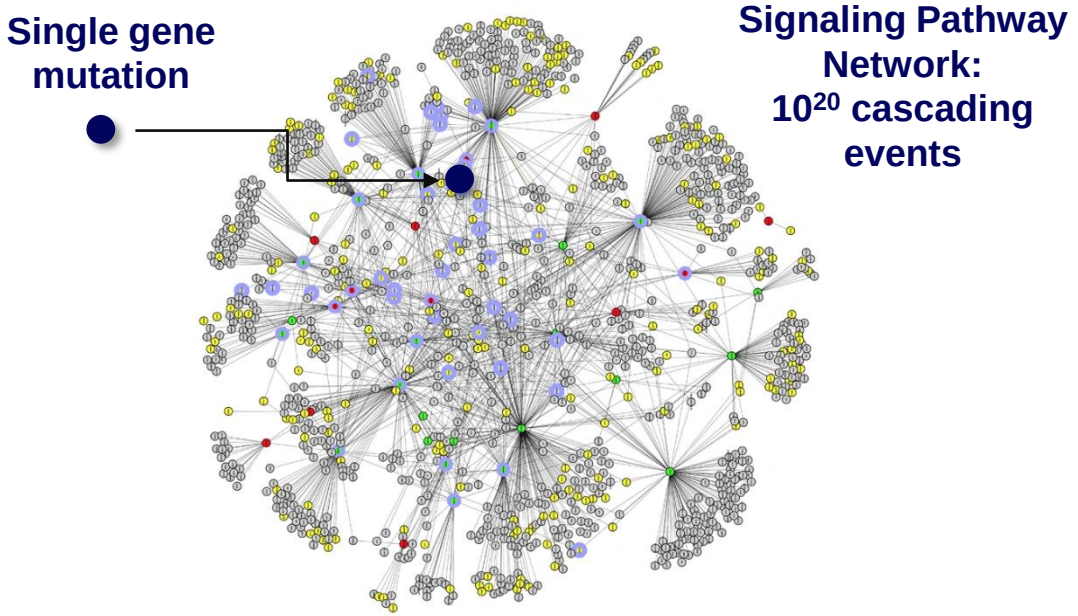
Neal Rosen, MD, PhD
Director, Center for Mechanism-Based Therapy
Memorial Sloan Kettering Cancer Institute

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



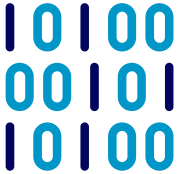
CELsignia – the first 3rd generation diagnostic

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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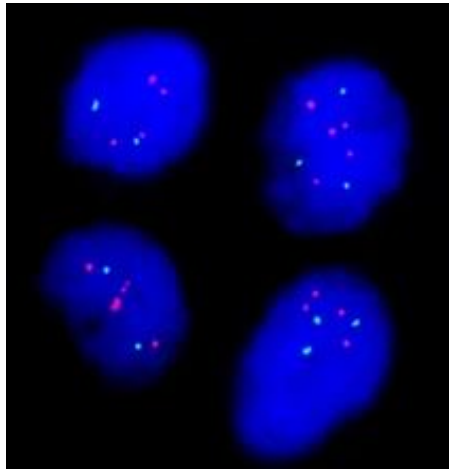
Source: 1) Internal Celcuity Studies

Current Molecular Diagnostics vs. CELsignia – HER2 Example

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

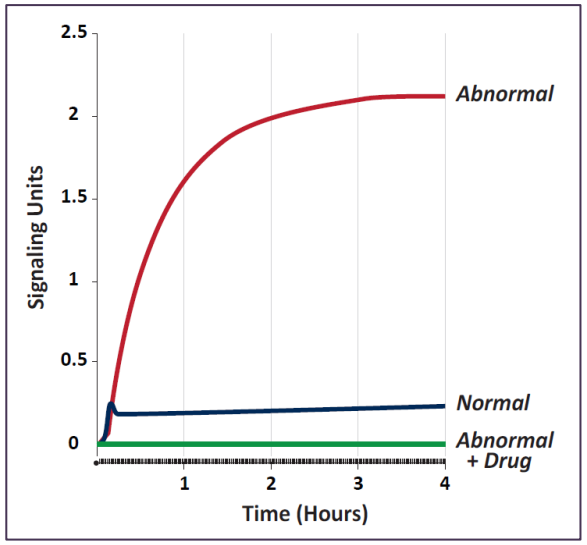
FISH HER2 Dx
(1 pathway gene)



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

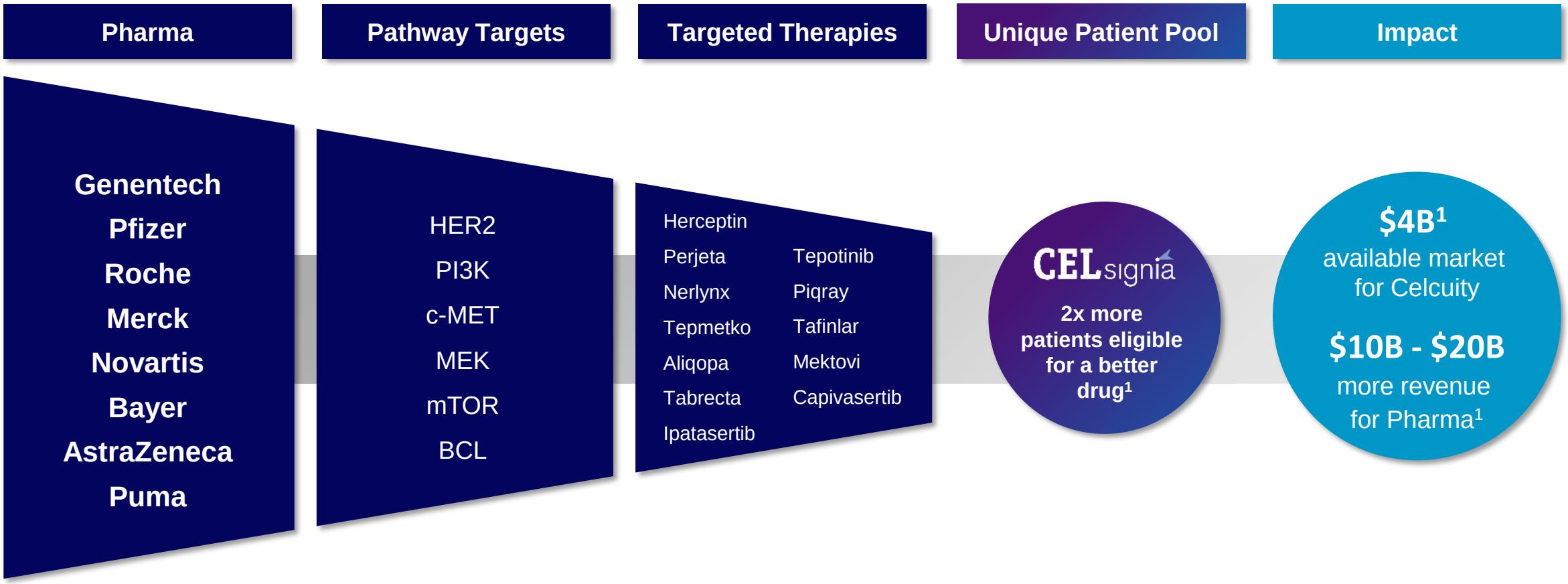
Drug resistance mechanisms characterized

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