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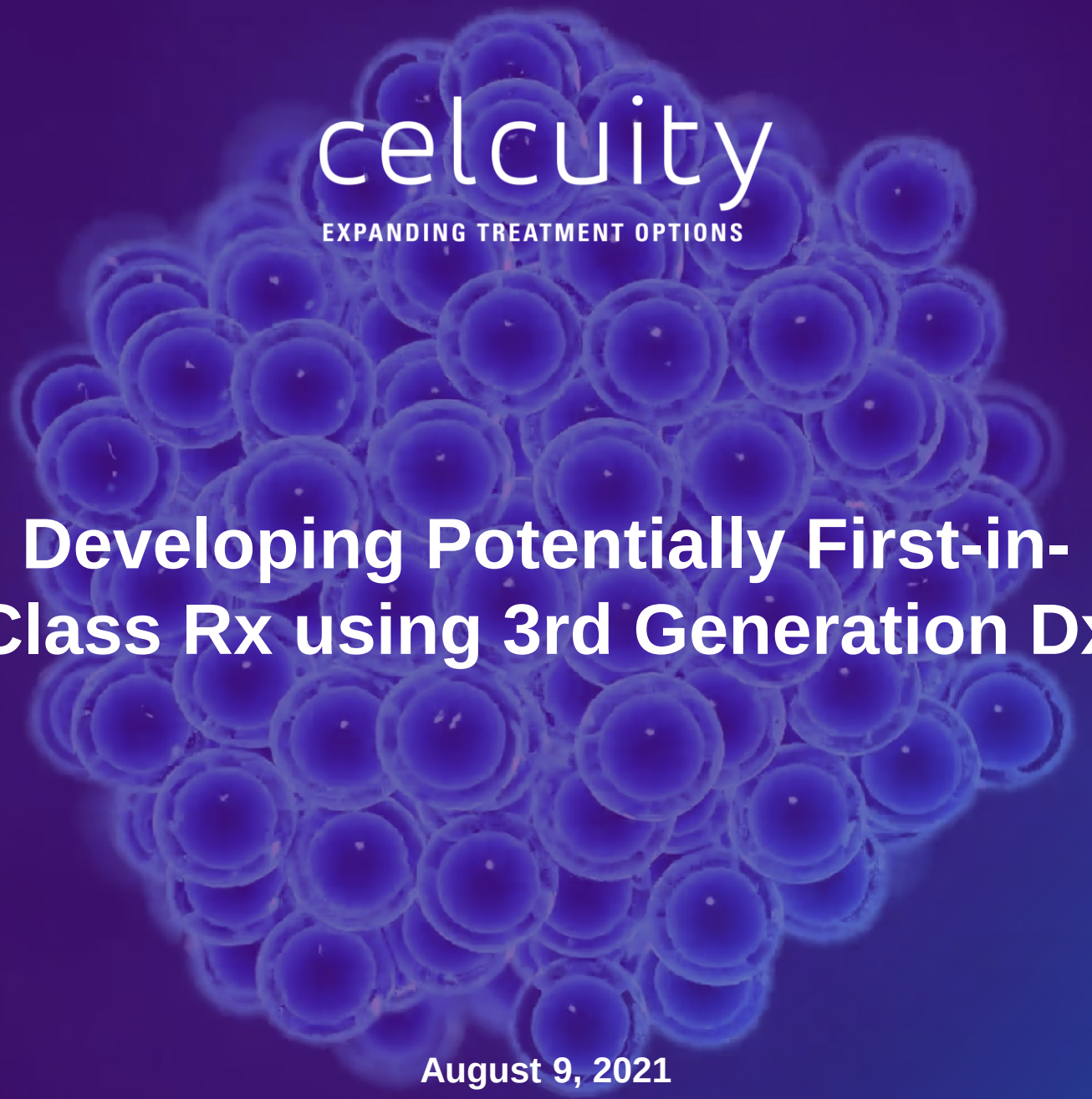

Developing Potentially First-in-Class Rx using 3rd Generation Dx

August 9, 2021

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, and FACT-5 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

Molecular tests can't detect the complex oncogenic activity driving many cancers

Our platform creates a **"movie" of signaling activity** in live patient tumor cells.

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

Leveraging our platform to **develop potentially first-in-class drugs**

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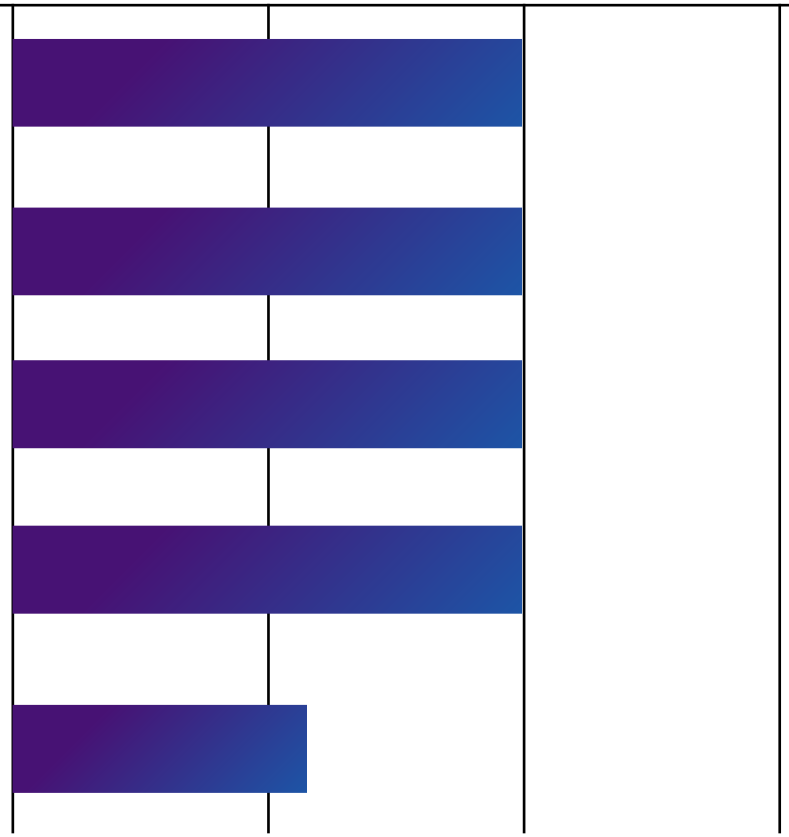
Current CELsignia Enabled Drug Programs for Breast Cancer

Supporting development of six targeted therapies to treat up to 30% of BC patients

	Indication	Treatment Approach (Pathways)	Trial	Drug Sponsor
Early Breast Cancer	**1L**	**Herceptin + Perjeta** + chemo *(HER2)*	FACT-1	Genentech
	1L	**Nerlynx** + chemo *(pan-HER)*	FACT-2	Puma
Metastatic Breast Cancer	**2L/3L**	**Xalkori + Vizimpro** *(c-Met + pan-HER)*	FACT-3	Pfizer
	2L/3L	Nerlynx + Fulvestrant *(pan-HER + ER)*	FACT-4	Puma
	2L/3L	**Tabrecta** + Nerlynx *(c-Met + pan-HER)*	FACT-5	NOVARTIS



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Integrating CELsignia with Gedatolisib, a Potentially First-in-Class PI3K/mTOR

CELsignia CDx creates more value for pharmaceutical companies than for Celcuity	Interest in gedatolisib was prompted while developing our CELsignia PI3K test	Unique set of circumstances drove the timing	Concluded this was a unique opportunity to leverage CELsignia
Gedatolisib may allow us to capture some of this value	Our proprietary assessment found gedatolisib may be superior to other PI3K inhibitors	Our research on PI3K signaling coincided with the availability of gedatolisib	Potentially first-in-class drugs with clinical data are rare and valuable

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An integrated CDx and Rx strategy maximizes the impact targeted therapies can have on patient outcomes

Leverage CELsignia to develop first-in-class drugs

Use CELsignia to maximize probability of getting approvals

Optimize regimens using our proprietary insights into pathway interactions

Discover synergistic drug combinations using CELsignia

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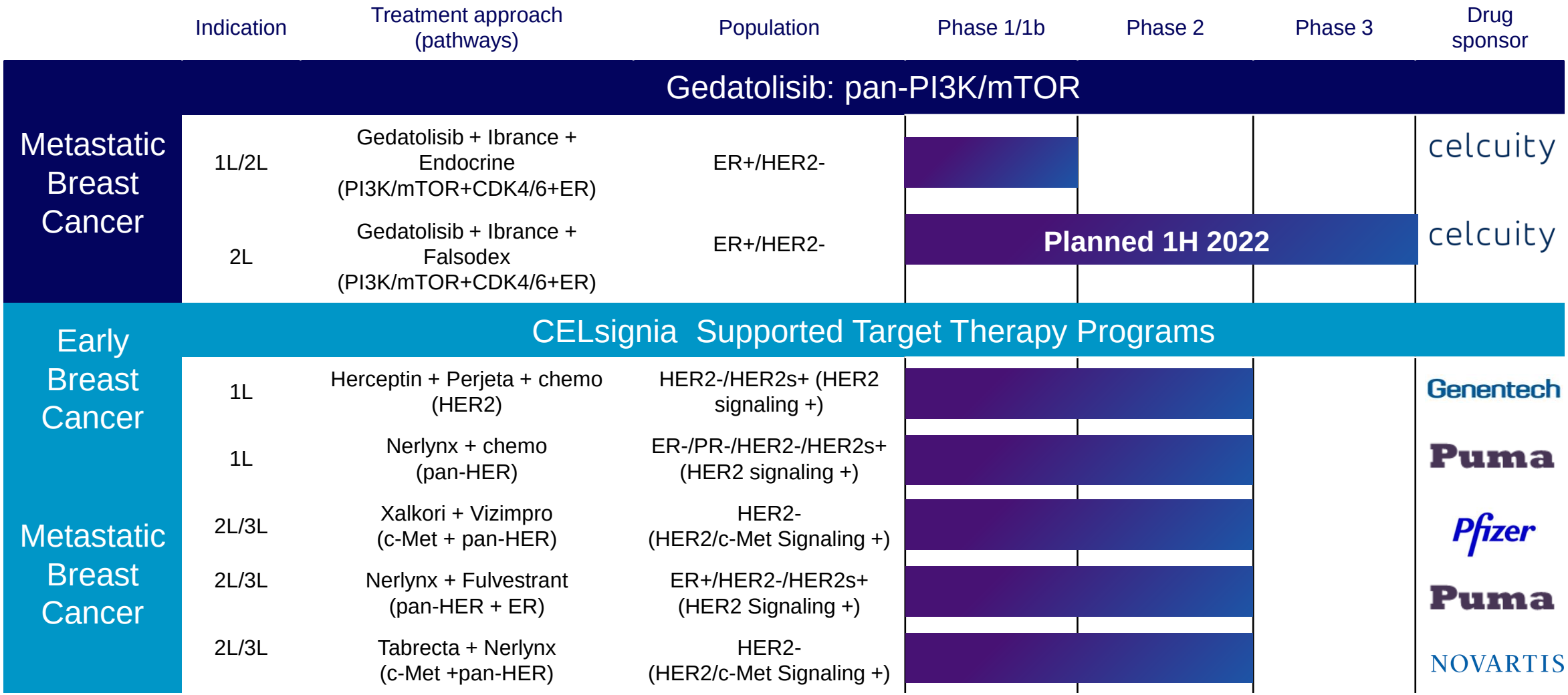
An Integrated Pipeline of Programs to Advance New Therapies for Breast Cancer



Gedatolisib

A PI3K/mTOR inhibitor

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Gedatolisib: Potential First-in-Class PI3K/mTOR Inhibitor

Compelling Phase 1b efficacy and safety data

Mechanism of Action

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

Key Clinical Data

- 457 patients with solid tumors have received gedatolisib in eight clinical trials sponsored by Pfizer
- Clinical development program focused on patients with ER+ / HER2- metastatic breast cancer (mBC)
- Expansion portion of Phase 1b trial treating HR+ / HER2- mBC with gedatolisib + ET + CDK4/6 inhibitor
  - 60% objective response rate: 53/88 evaluable patients with objective response
  - 75% clinical benefit rate: 66/88 evaluable patients with confirmed PR or stable disease > 24 weeks
- Primary TEAE's are manageable - 10% treatment discontinuation
- Significantly lower Grade 3/4 hyperglycemia than oral PI3K-α inhibitors (7% vs. 39%)

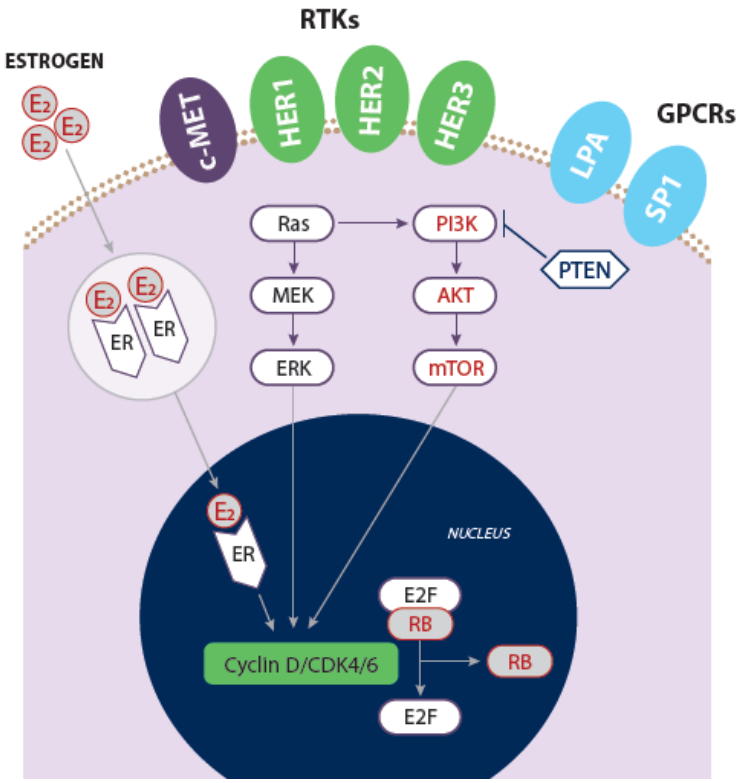
Market

- Initial clinical development program focused on breast cancer
- Breast cancer ~ $5 billion market potential

PI3K/mTOR is One of the Most Important Oncogenic Pathways

PI3K/mTOR regulates cell growth and metabolism

- Linked to multiple key pathways
- When overactivated, plays a key role in cancer progression
- Majority of patients with many solid tumors types have either a PI3KCA mutant or PTEN alteration[2]



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%
Prostate	~6%	~66%

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Source: (1) Ciskova 2012; (2) Mills 2016

Targeting PI3K and mTOR Efficaciously and Safely is Challenging

Compensatory pathways enable resistance to PI3K/mTOR inhibition

- Reflects the inherent adaptability and complexity of the PI3K/mTOR pathway
- Numerous feedforward and feedback loops and crosstalk with other pathways creates obstacles to effective inhibition

Therapeutic window for oral PI3K or mTOR inhibitors is narrow

- Difficult to achieve optimal pathway inhibition without inducing undue toxicities in patients
- Development of many promising pan-PI3K or pan-PI3K/mTOR inhibitors was halted due to toxicity challenges

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) Sedrani 2009; 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 Antagonists

PK properties responsible for favorable toxicity profile

	Gedatolisib[1]	Alpelisib[2]	Copanlisib[2]	Duvelisib[2]	Idelalisib[2]
Target(s)	Pan-PI3K mTOR	PI3K-α	Pan-PI3K	PI3K-δ	PI3K-δ
Organic class	Morpholino	Pyrrolidine	Quinazoline	*Isoquinoline*	*Isoquinoline*
Administration	IV	Oral	IV	Oral	Oral
Dosing in molar/month	0.88	***19.03***	0.37	***3.22***	***20.22***
Volume (distribution) L	30	114	***871***	29	23
AUC plasma	47.1 ug.h/mL	33.2 ug.h/mL	***1.6 ug.h/mL***	7.9 ug.h/mL	10.6 ug.h/mL
Cmax	8,594 ng/mL	2,480 ng/mL	463 ng/mL	1,500 ng/mL	1,861ng/mL
Half-life (hours)	37	8-9	39	5	8
Grade 3-4 hyperglycemia[3]	7%	***39%***	***41%***	-	-

Comments

- **Hyperglycemia is induced by PI3K-α inhibition and increased when drug has high affinity for the liver**
  - PI3K-α regulates glucose release
  - Liver is the primary site of glycolic 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 to process
  - Copanlisib – PK profile
    - 25x higher binding affinity for liver than plasma than gedatolisib
- **Other gedatolisib PK advantages**
  - 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 not well understood**
  - Likely due to amount of drug administered
    - 3.7x-23x more molar/month administered
  - Organic class may also be more toxic

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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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ER+/HER2- Metastatic Breast Cancer (mBC) Patient and Treatment Overview

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%
Letrozole[4]	9.4	32%
Fulvestrant[5]	6.5	14%

Prior CDKi

No prior CDKi

Second Line

Treatment (Patient Group)	mPFS (months)	ORR[1]
Everolimus (mTOR) + Exemestane[6]	**4.2[7]**	**NA**
Fulvestrant[8]	**3.7[7]**	**NA**
Alpelisib (PI3K-α)[9] + Fulvestrant *(PIK3CA+)*	**7.3**	**21%**
Everolimus + Exemestane[10]	7.8	13%
Alpelisib[11] + Fulvestrant *(PIK3CA+)*	11.0	36%

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Sources: (1) ORR is for patients with measurable disease; (2) PALOMA-2 trial; (3) PALOMA-3 trial; (4) PO25 trial; (5) CONFIRM trial; (6) Rozenblit 2019. Real world data (N=149) for CDKi-treated patients receiving everolimus + exemestane using electronic health records from Flatiron; (7) Duration of treatment; (8) Luhn 2018. Real-world data (N=147) for CDKi-treated patients using electronic health records from Flatiron; (9) BYLieve trial; (10) BOLERO-2 trial; (11) SOLAR-1 trial

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

- Combining gedatolisib with endocrine therapies in hormonally driven cancers has strong biological rationale
  - **Prostate cancer**
    - Nonclinical studies demonstrate linkage between androgen and PI3K/mTOR
  - **Recurrent endometrial cancer**
  - **HER2+ metastatic breast cancer**
    - Favorable data from gedatolisib + trastuzumab biosimilar POC study
    - ORR = 56%

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Review of Preliminary Phase 1b Data

As of January 11, 2021 data cut-off

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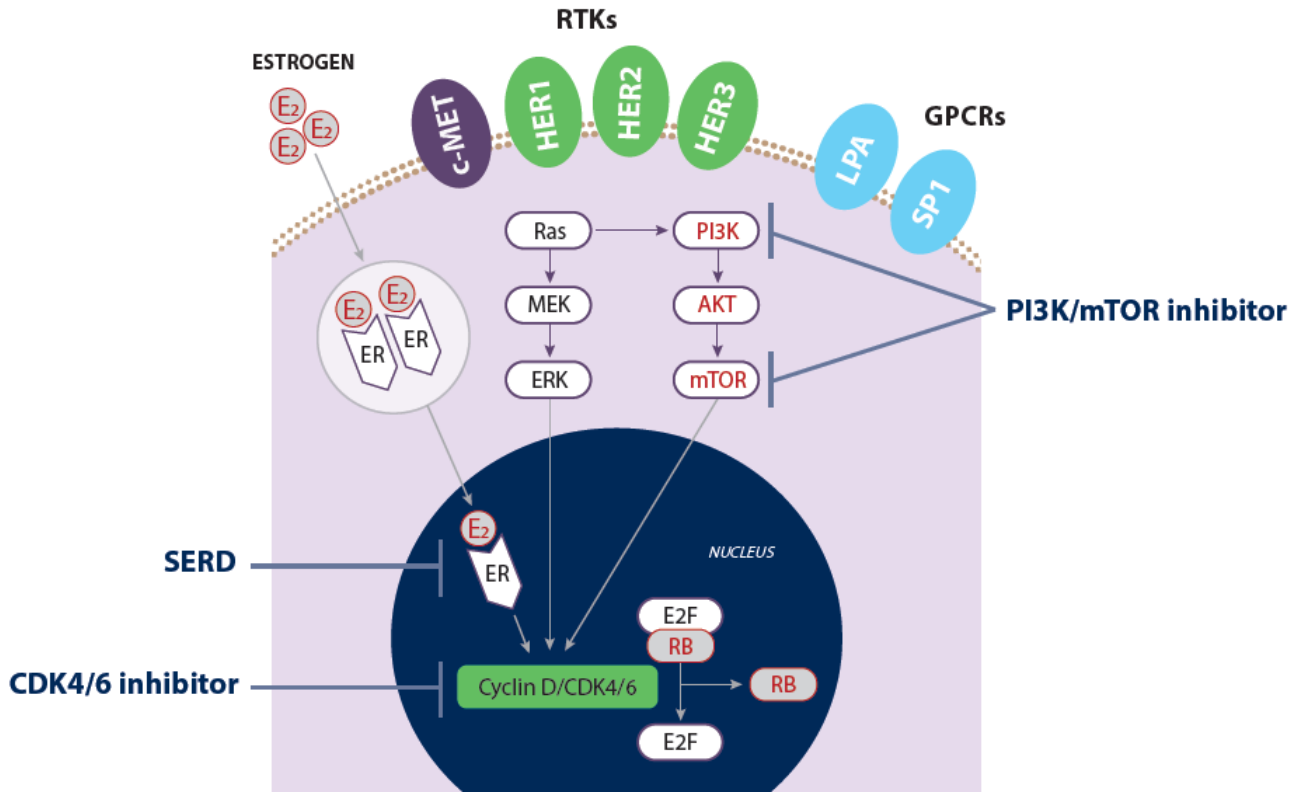
PI3K/mTOR, ER, and CDK4/6 are Interdependent Signaling Pathways

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

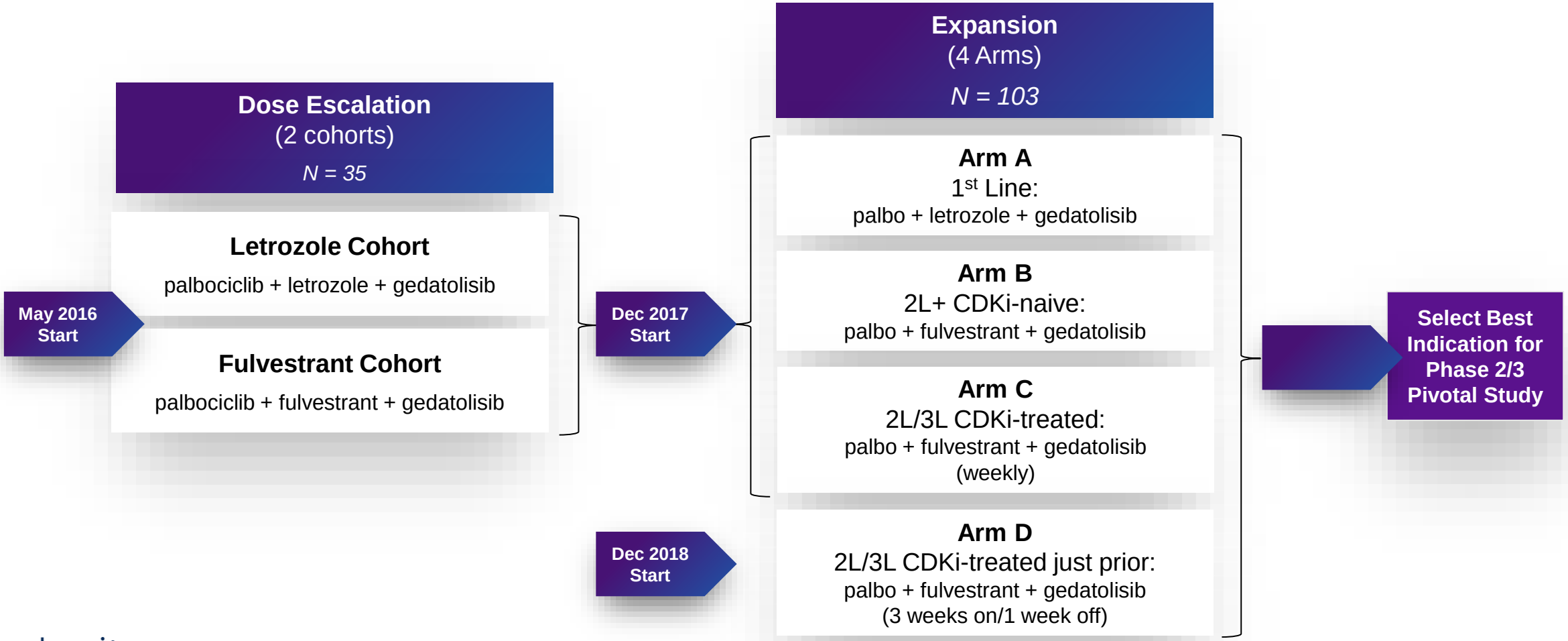
Treatment Strategy

- 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



B2151009: Phase 1b Study (138 patients)

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



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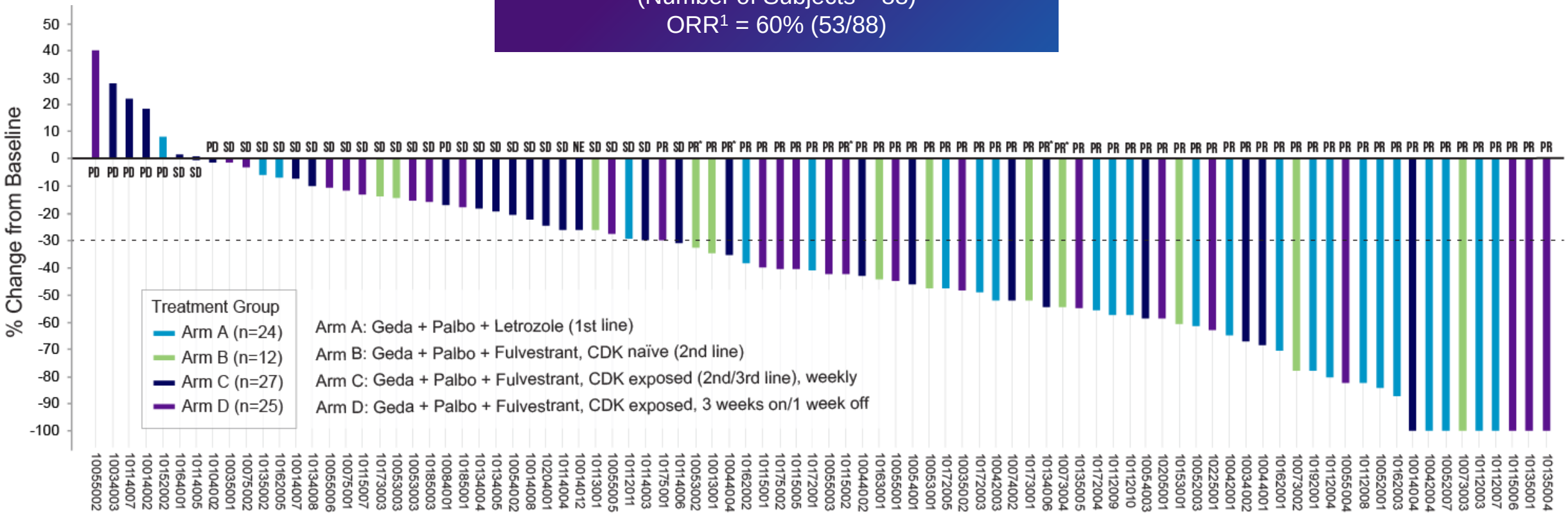
60% of Patients Achieved an Objective Response

Minimal correlation between PIK3CA status (mutant or WT) and response

Tumor Size Change Data
(Number of Subjects – 88)
ORR[1] = 60% (53/88)



Source: (1) ORR includes 5 Unconfirmed PR; Note: PR* is an unconfirmed PR
Note: Data presented is from a preliminary data analysis as of a cutoff date of January 11, 2021, representing a database snapshot, and may change based on ongoing routine data monitoring.

Palbociclib + Endocrine Therapy[1] + / - Gedatolisib

Patients	1L CDKi-naïve		1L+ CDKi-naïve	2L CDKi-naïve	2L/3L Prior CDKi
Evaluable Patients	N=338[2]	**N=24[3]**	N=267[4]	**N=12[5]**	**N=25[7]**
Study Treatment	Palbociclib + Letrozole	**Gedatolisib + Palbociclib + Letrozole**	Palbociclib + Fulvestrant	**Gedatolisib + Palbociclib + Fulvestrant**	**Gedatolisib + Palbociclib + Fulvestrant**
ORR (evaluable patients) (95% CI)	55% (50%-61%)	**83% (65%-94%)**	25% (20%-30%)	**75%[6] (47%-91%)**	**60%[8] (39%-74%)**
Median PFS (months) (95% CI)	24.8 (22.1, NR)	**Not Yet Reached >28 months**	9.5 (9.2, 11.0)	**11.9 (3.7, NR)**	**13.2 (9.0, 16.7)**

- Primary objective of B2151009 expansion portion (Arms A, B, C, D) was to determine if addition of gedatolisib to palbociclib + endocrine therapy produced a superior OR compared to historical control data
- Each fully enrolled arm met its endpoint target (Arm B did not meet enrollment target but ORR threshold was met)
- CDKi pre-treated patients receiving G + P + F in Arm D had higher ORR than 1L CDKi naïve patients receiving P + F alone
  - 60% vs. 25%

Sources: (1) Endocrine therapy – letrozole or fulvestrant; (2) PALOMA-2; (3) B2151009 – Arm A; (4) PALOMA-3; (5) B2151009 – Arm B; (6) Includes two unconfirmed partial responses; (7) B2151009 – Arm D; (8) Includes one unconfirmed response

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 January 11, 2021, representing a database snapshot, and may change based on ongoing routine data monitoring.

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

CDKi Status	CDKi-naïve			Prior CDKi	
Evaluable Patients	N=126[1]	N=485[2]	N=136[1]	N=100[3]	**N=25[4]**
Study Treatment	Alpelisib + Fulvestrant	Exemestane + Everolimus	Fulvestrant	Alpelisib + Fulvestrant	**Gedatolisib + Palbociclib + Fulvestrant (G+P+F)**
PIK3CA Status	M	M / WT	M / WT	M	**M / WT[5]**
Line of Therapy	2L	2L	2L	2L/3L	**2L/3L**
ORR (95% CI)	36% (27%-45%)	13% (10%-16%)	16% (10%-24%)	21% (14%-30%)	**60%[6,7] (39%-74%)**

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Sources: (1) SOLAR-1; (2) BOLERO-2; (3) BYLieve; (4) B2151009 trial - Arm D (5) 9 of 25 patients (36%) were PIK3CA+; (6) 6 of 9 PIK3CA+ patients (67%) had an OR; 9 of 16 PIK3CA- patients (56%) had an OR; (7) Includes 1 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 January 11, 2021, representing a database snapshot, and may change based on ongoing routine data monitoring.

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

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

Indication	Drug Regimen	Efficacy
1st/2nd Line ER+/HER2- Metastatic		
May have received prior mBC ET, no prior CDKi (quick progression patients)	Gedatolisib + Palbo + Fulvestrant[1]	PFS 11.9 months, ORR 75% 2L/3L, Phase 1b, N=13 (B2151009)
	Palbo + Fulvestrant[2]	PFS 9.5 months, ORR 25%, HR 0.46 1L/2L, Phase 3, N=521 (PALOMA-3)
2nd/3rd Line ER+/HER2- Metastatic (post CDKi)		
Progressed on CDKi + ET (AI or SERD)	Gedatolisib + Palbo + Fulvestrant[3]	PFS 13.2 months, ORR 60% 2L/3L, Phase 1b, N=27 (B2151009)
	Alpelisib + fulvestrant[4] *(PI3K-α + SERD for PIK3CA+)*	PFS 7.3 months, ORR 21% Phase 2, N=121 (BYLieve)
	Fulvestrant[5] (SERD)	PFS 3.7 mos 2L/3L, N=147
	Everolimus + Exemestane[6] (mTORi + AI)	PFS 4.2 mos 2L/3L, N=149

Sources: (1) B2151009 – Arm B; (2) PALOMA-3 trial; (3) B2151009 – Arm D; (4) BYLieve; (5) Luhn 2018 SABCS. Real-world data for patients with prior-CDK4/6 treatment receiving fulvestrant using electronic health records from Flatiron; (6) Rozenblit 2019 SABCS. Real world data for patients with prior CDK4/6 treatment receiving everolimus + exemestane using electronic health records from Flatiron

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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
- Nearly 20% of patients were on treatment for >24 months
- Most TEAE's were Grade 1 or 2
- <10% discontinued the drug due to AE
- Stomatitis (mouth sores) was originally treated at manifestation
  - Steroid mouth rinse reduced severity
- Few hyperglycemia-related adverse events (22% all Grades, 7% Grade 3/4)
  - Significant contrast to PI3K-α drugs
- Neutropenia, leukopenia, and anemia AEs are 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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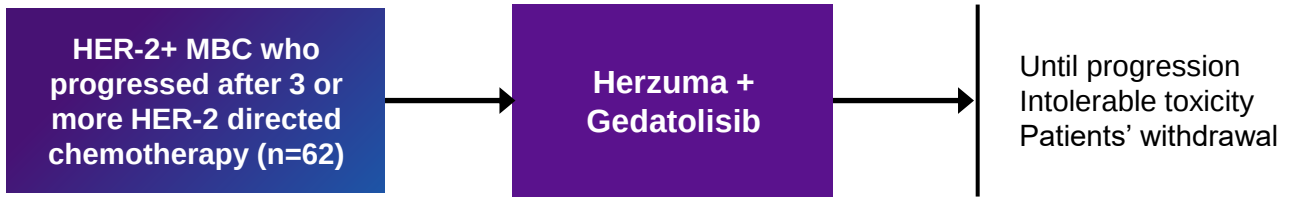
Note: Data presented for the B2151009 trial is from a preliminary data analysis as of a cutoff date of January 11, 2021, representing a database snapshot, and may change based on ongoing routine data monitoring.

Gedatolisib Opportunity in HER2+/PIK3CA+ mBC Patients

Patients received 3 or more lines of prior HER2 therapies

- Aberrant PI3K/mTOR is a known resistance mechanism of HER2 therapies
- A phase 2 pilot study[1] evaluated safety and efficacy of Herzuma® (trastuzumab biosimilar) plus gedatolisib in patients with HER2+ / PIK3CA+ mBC who progressed after 3 or more lines of prior HER2 therapy



- **Primary endpoints:** ORR
- **Secondary endpoints:** PFS, OS, Safety
- **Exploratory endpoints:** Biomarker, QoL

Clinical characteristics of participants

Clinical characteristics	N=16
Age (median, years)	54.5
Menopausal status	
Pre-menopausal	2 (12.5%)
Menopausal	14 (87.5%)
Metastatic site: target or non-target lesion	
Breast	2 (12.5%)
Lymph node	7 (43.8%)
Bone	5 (83.3%)
Lung	12 (75.0%)
Liver	6 (37.5%)
Etc.	3 (18.8%)

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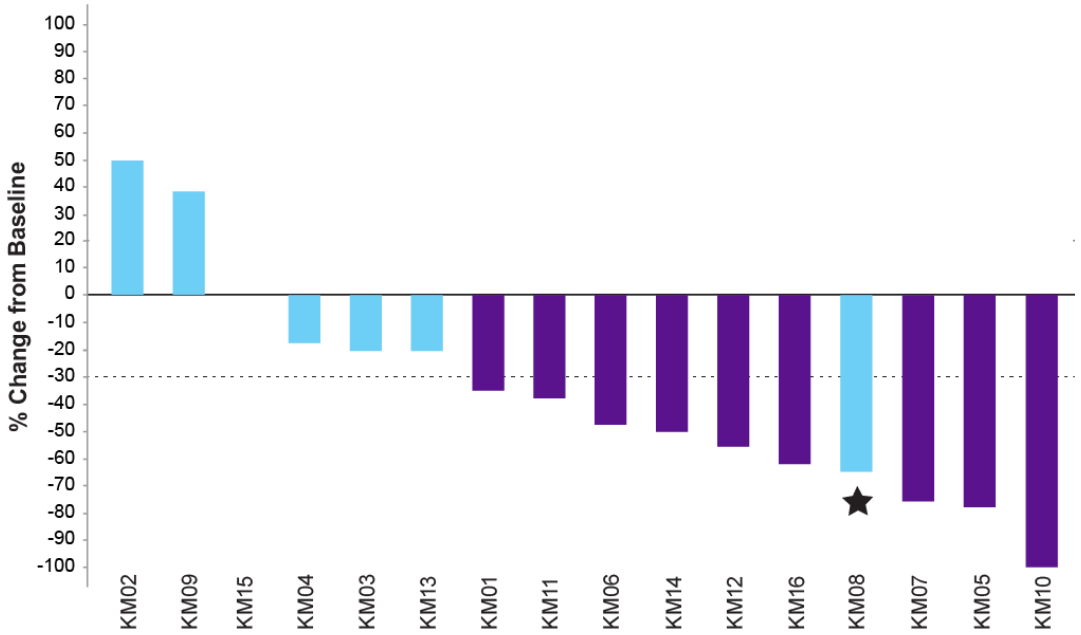
Source: (1) Kim 2020 SABCS

56% ORR for Patients Receiving Gedatolisib + Trastuzumab Biosimilar

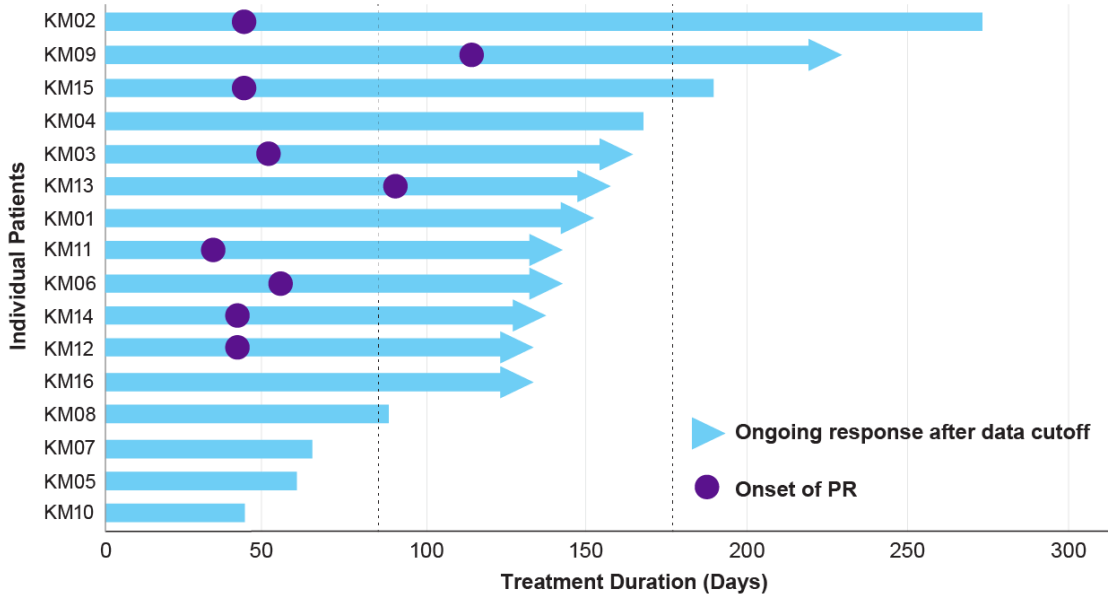
Best Response



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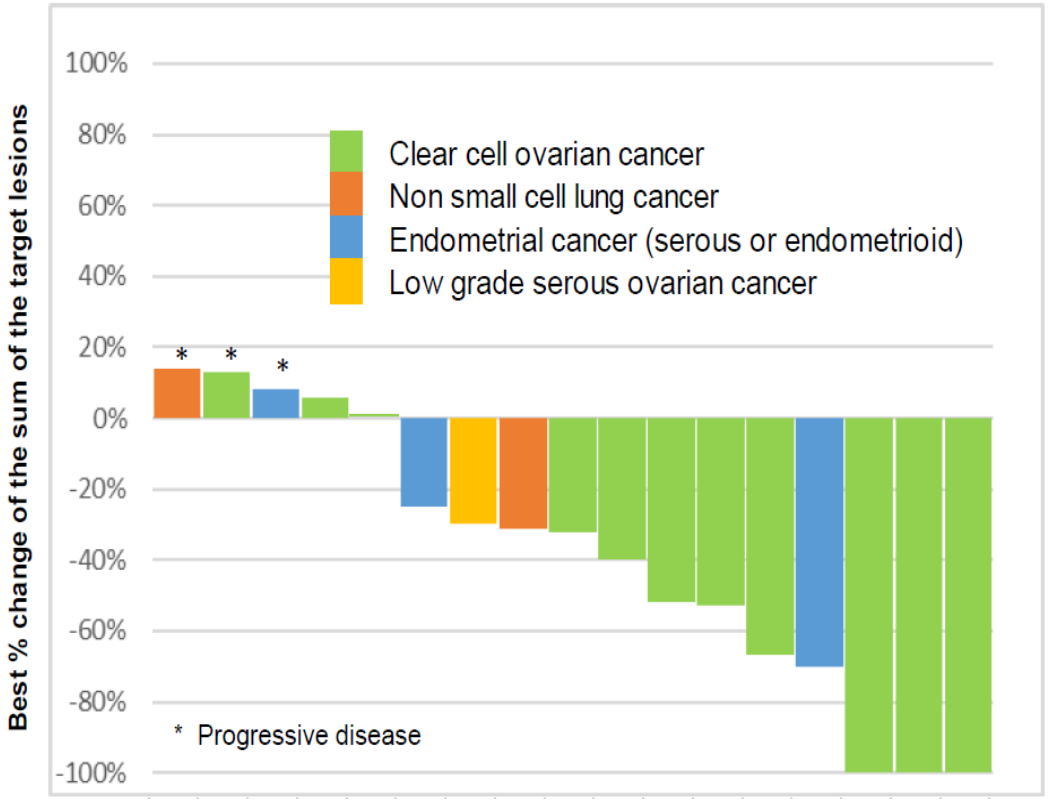
Note: Data presented is from an interim analysis of data as of a cutoff date of October 30, 2020, representing a database snapshot, and may change based on ongoing routine data monitoring and enrollment.

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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Experienced drug development team

CMO

Art DeCillis, MD

CMO for Eleven Biotherapeutics (now Sesen Bio)

VP Clinical Research and Medical Affairs at Exelixis.

Executive Director of Oncology Development at Novartis

Group Director at Bristol-Myers Squibb

- Involved in development of SPRYCEL®, AFINITOR®, FARYDAK®, and CABOMETYX®

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.

VP Clin Dev

Igor Gorbatchevsky, MD

VP Clinical Dev at MEI Pharma

- Responsible for zandelisib (PI3K-δ inhibitor)

VP Clinical Science at Iovance Biotherapeutics

Global Clinical Leader at Bayer Pharmaceuticals

- Responsible for ALIQOPA, a pan-PI3K inhibitor

Senior Medical Director at Daiichi-Sankyo

VP Clin Ops

Jill Krause

VP Clinical Operations Quality and VP Study Management and Clinical Affairs 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

Head CMC

Bernhard Lambert, PhD

Executive Director, Pharmaceutical R&D at Chimerix

- Served in various CMC roles at Gilead and Glaxo Wellcome

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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

Hung Khong M.D.

MOFFITT CANCER CENTER

Bora Lim M.D.

Baylor College of Medicine

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.

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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 In-licensed

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

- Phase 3 ready asset[1]
- 60% objective response rate
- Well tolerated safety profile with 10% gedatolisib discontinuation rate

Experienced Team

Experienced clinical team with successful track record of getting drug approvals

Financial Resources

Strong balance sheet

- 6/30/21 - $41.6 million cash on hand
- 7/1/21 – Received $52.8 million net proceeds from follow-on equity offering
- Approximately $94.4 million cash-on-hand after follow-on 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."

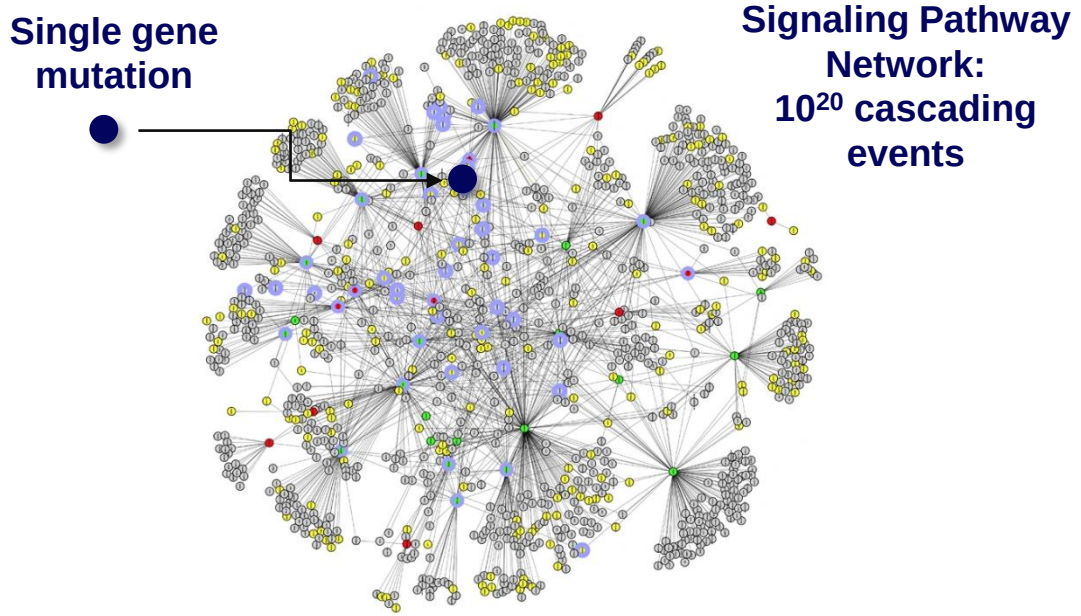
Neal Rosen, MD, PhD
Director, Center for Mechanism-Based Therapy
Memorial Sloan Kettering Cancer Institute

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



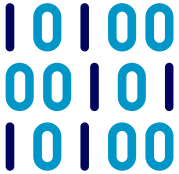
CELsignia – the first 3rd generation diagnostic

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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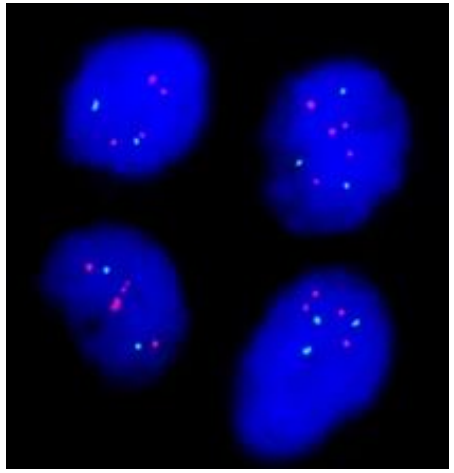
Source: 1) Internal Celcuity Studies

Current Molecular Diagnostics vs. CELsignia – HER2 Example

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

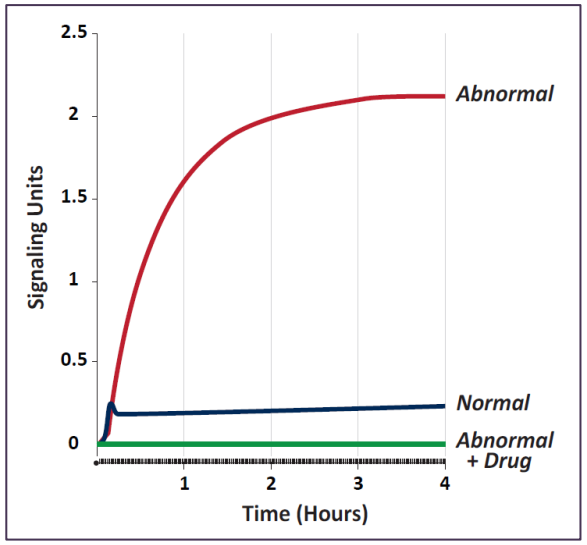
FISH HER2 Dx
(1 pathway gene)



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 cause 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

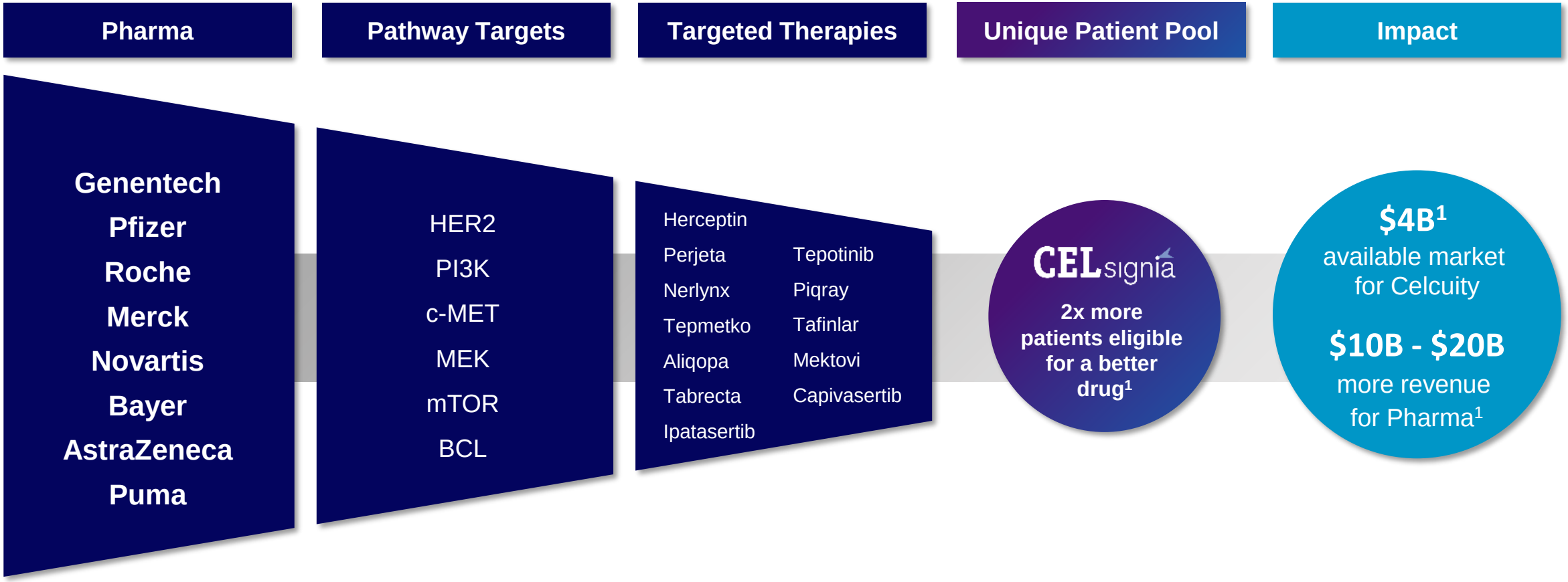
Drug resistance mechanisms characterized

- 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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