Olema ONCOLOGY

Improving the lives of women with breast cancer

August 2022

Forward-Looking Statements

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This presentation discusses product candidates that are under clinical study and which have not yet been approved for marketing by the U.S. Food and Drug Administration. No representation is made as to the safety or effectiveness of these product candidates for the use for which such product candidates are being studied.

Olema Oncology Overview

Developing Next-Generation Therapies in Women's Oncology

- Lead candidate: **OP-1250** - a Complete Estrogen Receptor Antagonist (CERAN) in development for the treatment of ER+/HER2- metastatic breast cancer
- Phase 2 clinical development ongoing with attractive emerging profile
  Preparing for initiation of pivotal Phase 3 monotherapy trial in mid-2023
- Internally-discovered, wholly-owned IP with no royalty burden
  Received Fast Track designation from U.S. FDA in July 2022
- Research pipeline of additional women's cancer programs complementary to OP-1250
- Strong balance sheet with $240.7M* cash position;
  Sufficient capital to fund clinical and development operations into 2H 2024

Nasdaq OLMA

*Cash, cash equivalents and marketable securities as of June 30, 2022

OP-1250: Best-in-Class Potential for ER+ / HER2- Breast Cancer

Complete Antagonism of ER	CERAN that completely blocks transcriptional activity in both wild-type and mutant ER
Attractive PK Profile	Highly favorable pharmacokinetics with oral bioavailability and steady-state plasma levels with minimal peak-to-trough variability
Robust Tumor Shrinkage	Robust anti-tumor activity in both wild-type and mutant ER in preclinical models and durable benefit in clinical settings
Favorable Tolerability	Favorable tolerability profile in heavily pretreated patients
CNS Penetration	Robust activity in nonclinical brain metastases studies
Combinability	Emerging combination data show tolerability profile consistent with expected profile of palbociclib plus endocrine therapy, with no DDI*

Data referenced above as of July 1, 2022. *In completed dose escalation cohorts from ongoing Phase 1b combination study with palbociclib.

OP-1250 Clinical Status as of July 2022

Rapidly Advancing Clinical Development of OP-1250 to Pivotal Studies

High investigator enthusiasm and robust enrollment continues across program; Granted FDA Fast Track Designation

Phase 1b Monotherapy Expansion

- As of July 1, 2022, 50 patients have been treated in Phase 1b (N=25 each for 60 and 120 mg dose cohorts)
- Favorable tolerability; most adverse events (AEs) were Grade 1 or 2, with most common treatment-related AEs were nausea, vomiting, fatigue and headache
- Encouraging anti-tumor activity in initial set of expansion patients evaluated
  - 4 partial responses in 31 efficacy-evaluable patients*
- Phase 2 enrollment ongoing at Recommended Phase 2 Dose of 120 mg OP-1250 once-daily

Phase 1b Combination with Palbociclib

- Dose escalation progressing
  - 30, 60 and 90 mg cohorts have completed DLT evaluation period; 120 mg cohort ongoing
- Combinability demonstrated in initial completed cohorts:
  - ✓ No dose limiting toxicities
  - ✓ Tolerability profile consistent with that of palbociclib plus endocrine therapy
  - ✓ No induced metabolism of palbociclib

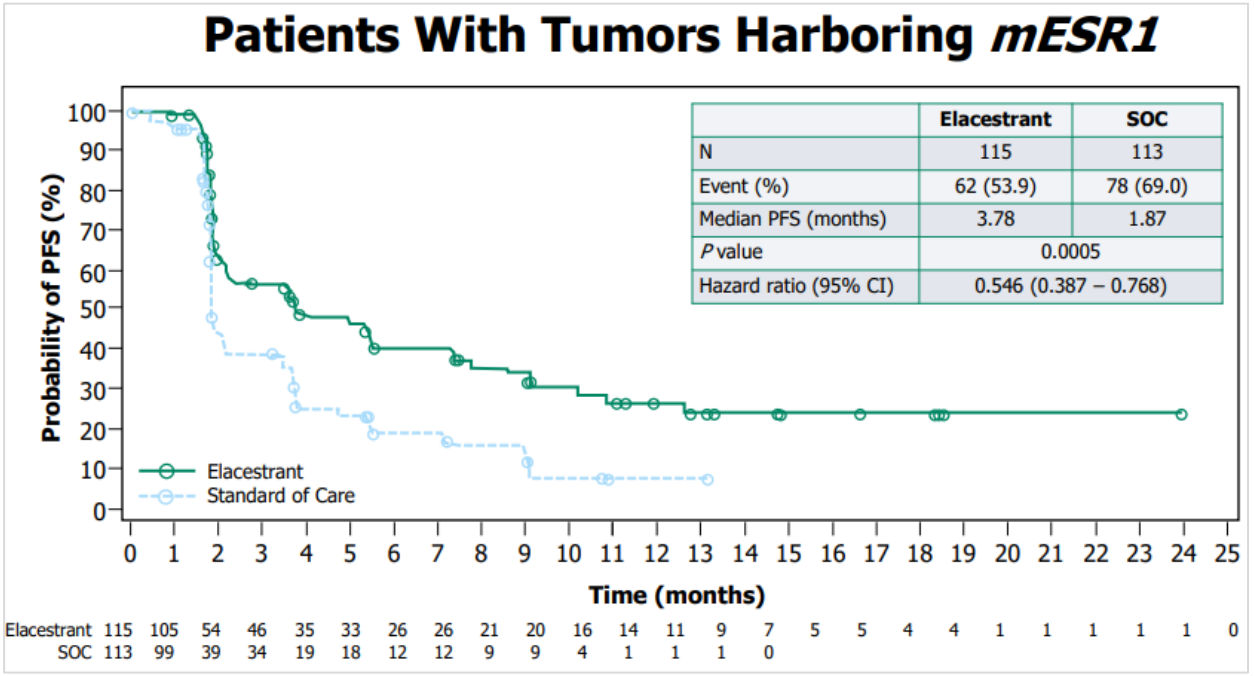
Upcoming Milestones

- Q3 2022: Initiate Phase 1b combo study with ribociclib and alpelisib
- Q4 2022: Present monotherapy and initial combination data
- Mid-2023: Initiate pivotal monotherapy study

Data and anticipated milestones stated above are as of July 1, 2022; *1 confirmed partial response at 60 mg and 3 unconfirmed partial responses, pending confirmation at a subsequent scan, at 120 mg

EMERALD Study Validates Opportunity in 2L+ Setting for OP-1250

- Hazard ratio of 0.55 in *ESR1*$_{mut}$ patients is clinically meaningful and validates the opportunity to beat SoC in the 2L+ setting
- ESR1 mutations are the most common resistance mechanism to SoC in the 1st line setting leading to progression in up to 50% of patients
- ESR1 mutations typically occur within the ligand binding domain resulting in a constitutively active ER in the absence of estrogen ligand binding
- Elecestrant, a SERM/SERD, acts as a partial antagonist on *ESR1*$_{mut}$ receptors but can have partial agonist activity on WT receptors
- OP-1250 has the potential to improve upon the EMERALD result given its activity on both mutant ESR1 and wild-type receptors



	Elacestrant	SOC
N	115	113
Event (%)	62 (53.9)	78 (69.0)
Median PFS (months)	3.78	1.87
P value	0.0005	
Hazard ratio (95% CI)	0.546 (0.387 – 0.768)	

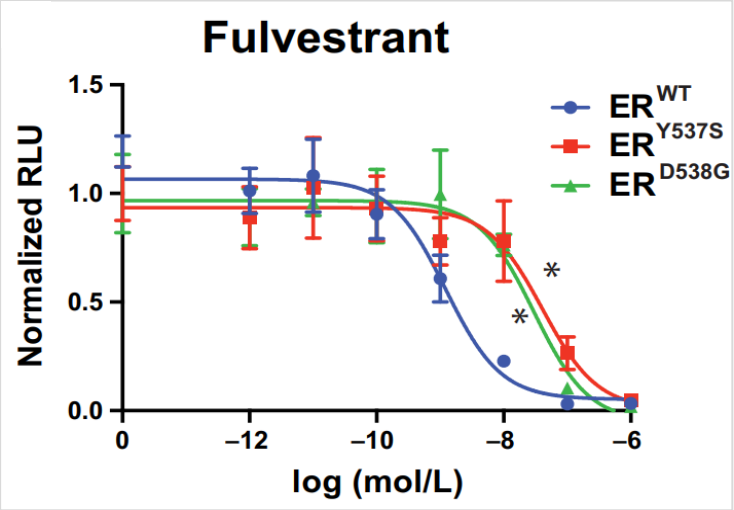
OP-1250, a Complete Estrogen Receptor Antagonist (CERAN), completely turns off both AF1 and AF2 transcriptional domains in <u>both</u> mutant ESR1 and wild-type receptors

Reference: Bidard et al. Journal of Clinical Oncology. May 18, 2022.

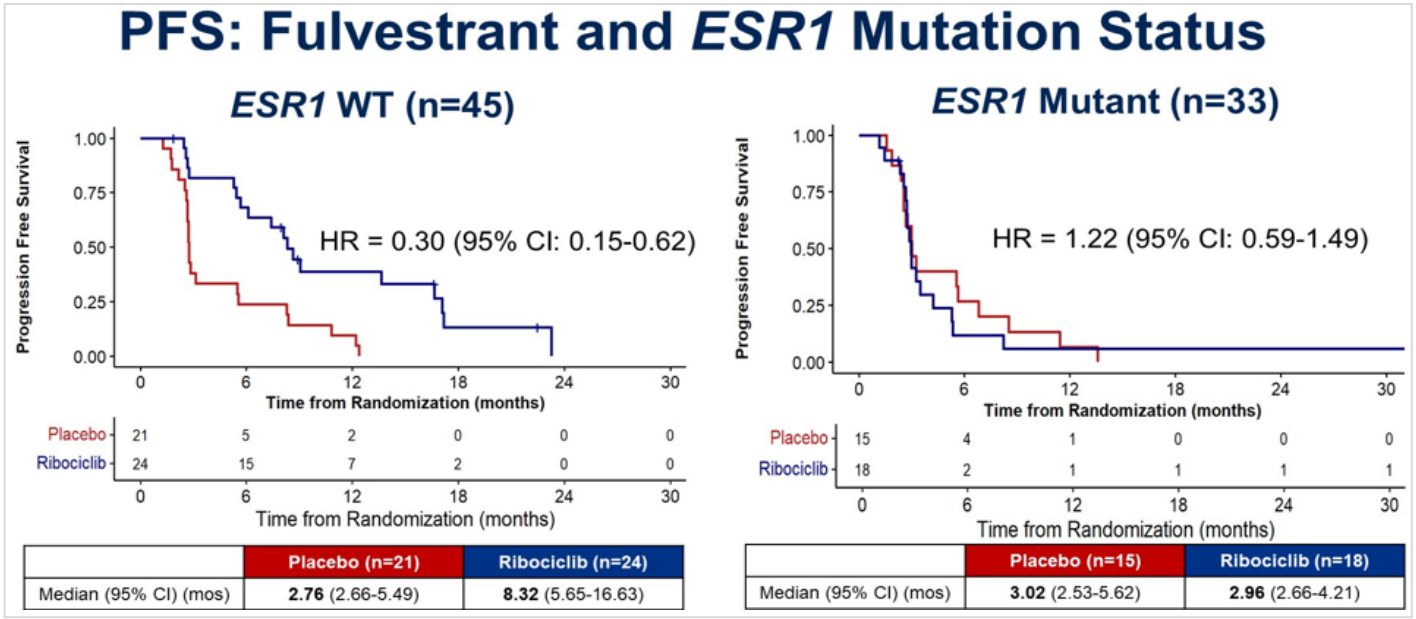
MAINTAIN Study Validates Opportunity in Both 1L and 2L+ Settings for OP-1250

Fulvestrant's pharmacology limited by:

- Poor bioavailability
- Reduced potency in *ESR1*$_{mut}$ receptors compared to WT due to mutation in ligand binding domain



- Fulvestrant's efficacy benefit limited to wild-type ESR1 receptors; suboptimal drug exposure results in an inability to shut-off mutant ESR1 receptor
- MAINTAIN results consistent with PARSIFAL; unable to shut-off the most common resistance mechanism to 1st line treatment



	Placebo (n=21)	Ribociclib (n=24)
Median (95% CI) (mos)	**2.76** (2.66-5.49)	**8.32** (5.65-16.63)

	Placebo (n=15)	Ribociclib (n=18)
Median (95% CI) (mos)	**3.02** (2.53-5.62)	**2.96** (2.66-4.21)

OP-1250 has potential to improve upon fulvestrant due to favorable pharmacokinetics, ~10-fold higher exposure levels enables stronger binding affinity in *ESR1*$_{mut}$ receptors

References: Andreano K et al. Molecular Cancer Therapeutics. May 7, 2020. Kalinsky et al. Proceedings: MAINTAIN trial presentation. ASCO Annual Meeting; June 3-7, 2022.

OP-1250: Designed to Shut Down ER-Driven Cell Growth and Proliferation in ER+/HER2- Breast Cancer

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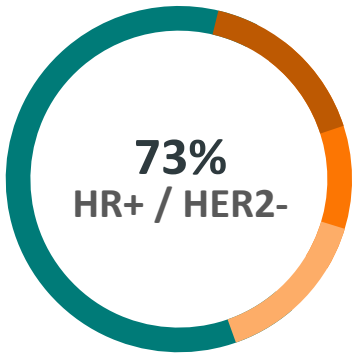
ER+ Breast Cancer — A Significant Unmet Need

Breast Cancer is the Most Common Diagnosed Cancer Worldwide

Majority of All Breast Cancers

express Estrogen Receptor (ER+)



73%
HR+ / HER2-

12%
HR- / HER2-

4%
HR- / HER2+

11%
HR+ / HER2+

Estimated $20B Market

for endocrine therapies and targeted agents for ER+ breast cancer

In 2022, approximately

288K

Women in the U.S. will be diagnosed with breast cancer

43,250

Women in the U.S. will succumb to metastatic breast cancer

New endocrine agents needed to suppress or overcome resistance, and delay toxic chemotherapy

References: World Health Organization; American Cancer Society. Facts and Figures 2022; SEER database

Segments of Therapy in ER+/HER2- Breast Cancer

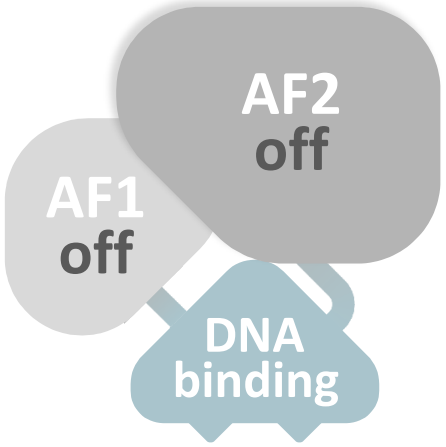
	HR+ / HER2-[1]				HR+ / HER2+[2]
LINE OF THERAPY	2L/3L+	1L	High-Risk Adjuvant	Early Breast Cancer	HER2+ w/ CNS Mets
PATIENTS	~150K	~115K	~75K	~285K+	~10K
DURATION OF THERAPY[3]	~6 MONTHS	~24 MONTHS	Up to 5 YEARS	Up to 5 YEARS	~12 MONTHS
MARKET POTENTIAL[4]	~$3-5B	$5-10B+	~$3-5B	$10B+	~$500M

[1]2025 incidence projection estimates. Olema internal data, Informa ER+ HER2- BC Prevalence Based Market Forecast. 26% of adjuvant eligible patients assumed to have Ki-67 ≥ 20%. Early breast cancer incidence includes high risk adjuvant segment. [2]2025 incidence projection estimates. Olema internal data, Informa HER2+ BC Prevalence Based Market Forecast. Forecast based on 3L+ HR+ HER2+ metastatic breast cancer projections. [3]Olema internal data. [4]2025 opportunity estimates for total endocrine therapy market (US and EU5). Olema internal data.

Understanding the Estrogen Receptor (ER)

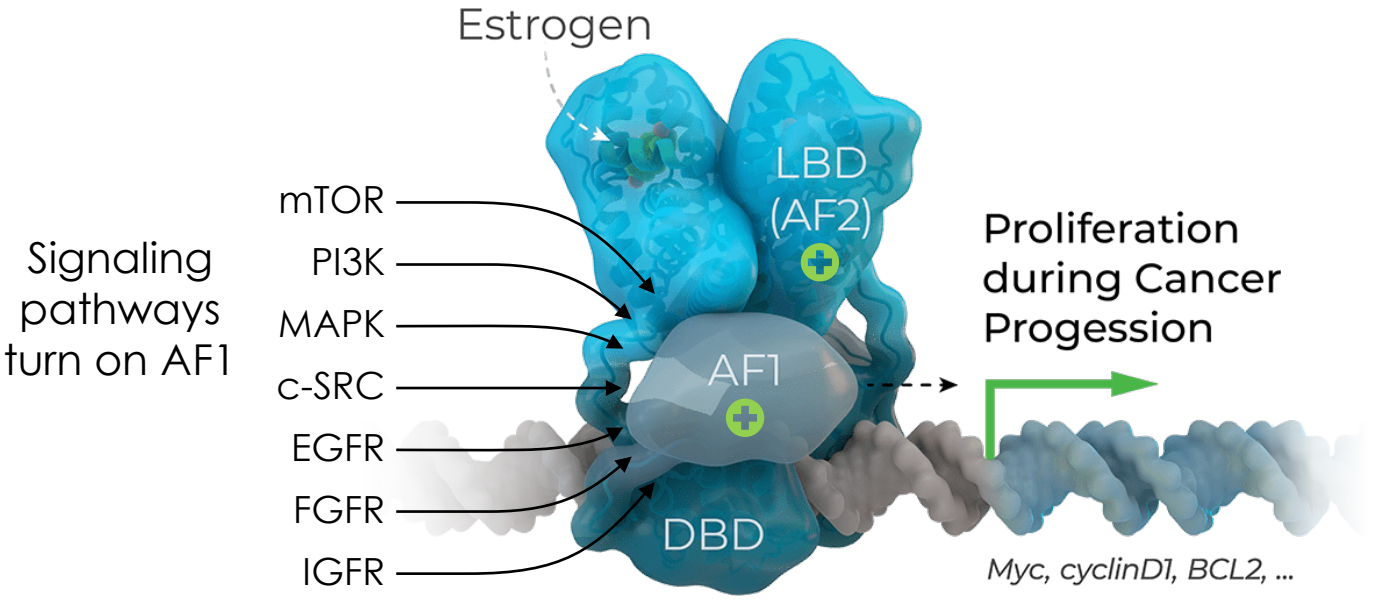
The Estrogen Receptor is a Tripartite Protein with Two Distinct Transcriptional Activation Domains (AF1 and AF2)

Unbound, both activation domains (AF1 and AF2) remain off



Estrogen receptor, unbound, remains in the off position.

When Estrogen binds and activates AF2, a conformational change exposes AF1



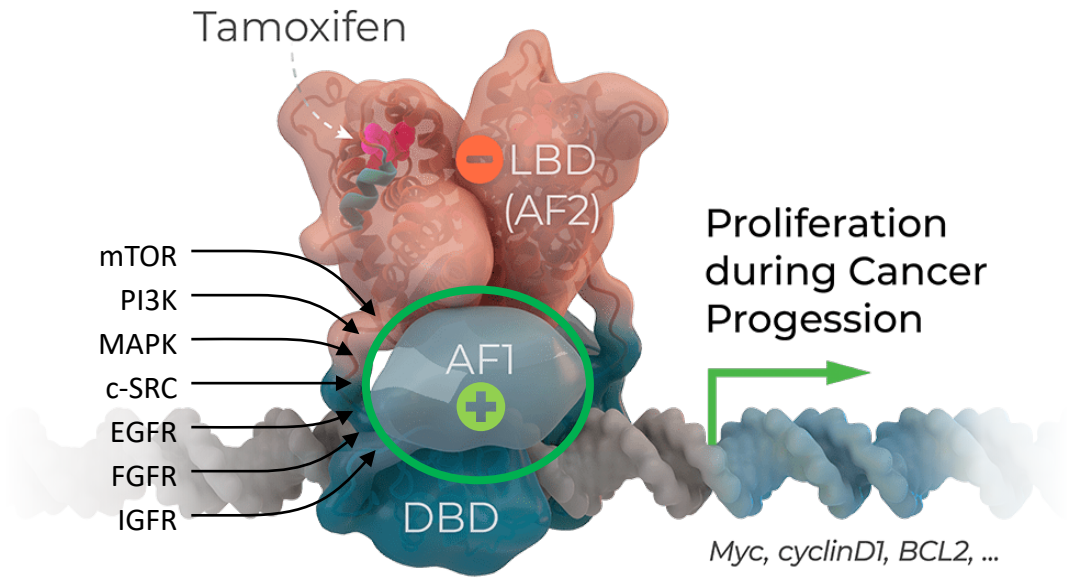
When Estrogen binds the ER, both AF1 and AF2 can drive transcription and cancer cell proliferation.

References: Shang and Brown, Science, 29 Mar 2002: Vol. 295, Issue 5564, pp. 2465-2468; Webb, Nguyen, and Kushner, JBC, Vol. 278, 2003, pp. 26912-26920

OP-1250 is a Complete Estrogen Receptor Antagonist (CERAN)

SERM / SERDs Only Block AF2 Activity but Allow AF1 Activation

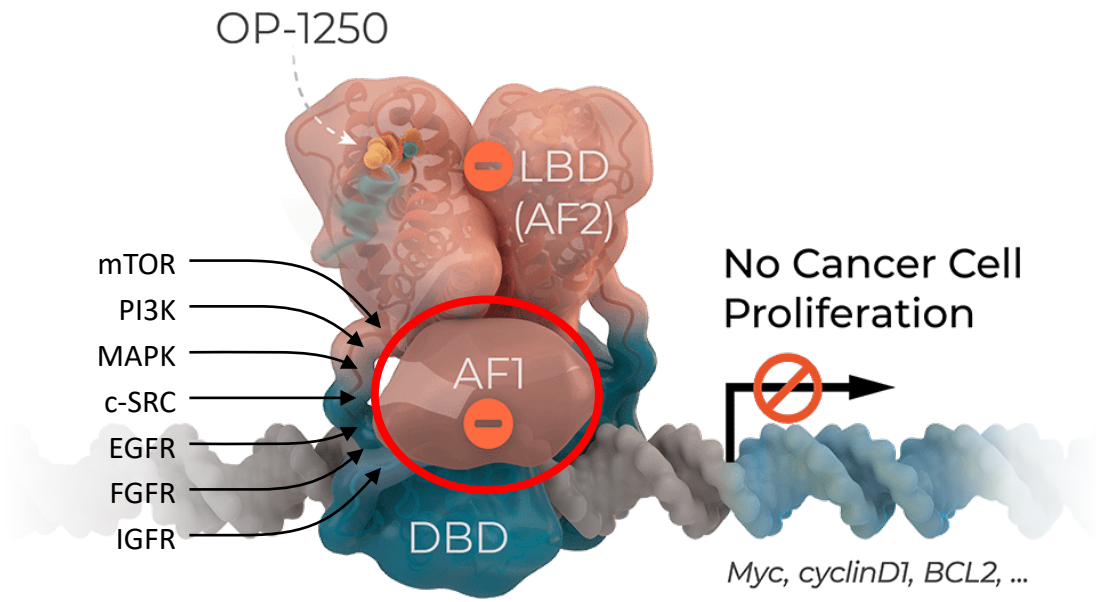
Tamoxifen, a partial antagonist, turns off AF2 but not AF1



Partial antagonists have a short duration of response for treatment of metastatic breast cancer

CERANs Completely Block Both AF1 and AF2 Activity

Complete antagonists turn off AF2 and recruit N-CoR to inactivate AF1



CERANs block AF1 activity, even in the presence of signaling, preventing cell proliferation

References:
Shang and Brown, Science, 29 Mar 2002: Vol. 295, Issue 5564, pp. 2465-2468
Webb, Nguyen, and Kushner, JBC, Vol. 278, 28 Feb 2003, pp. 6912–6920

OP-1250 Non-Clinical Data Summary

OP-1250 Non-Clinical Data Consistent with Emerging Clinical Profile

Complete Antagonism of ER

Activity in both wild-type and mutant ESR1 models by turning-off both AF1 and AF2 transcriptional activation domains

Strong Degradation of ERα

Across all tested ER+ cell lines

Attractive PK Profile

Orally bioavailable and attractive steady-state plasma levels

Robust Tumor Shrinkage

Superiority vs. fulvestrant in head-to-head nonclinical studies

Combination Therapy

Demonstrated additive effects in nonclinical models with other targeted agents

CNS Penetration

Robust activity in nonclinical brain metastases studies

OP-1250: Interim Phase 1 Clinical Data

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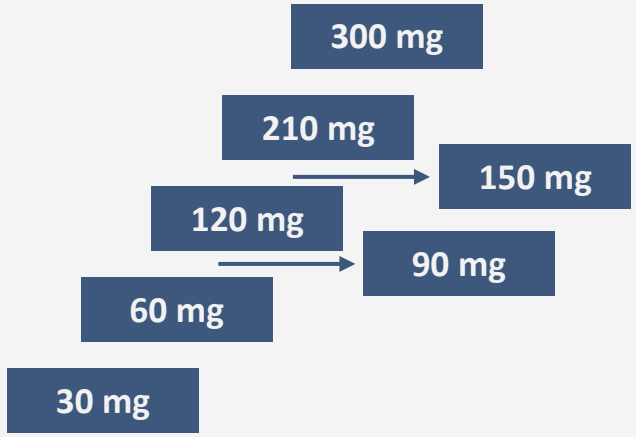
OP-1250 Study 001: First-in-Human Phase 1/2 Clinical Study Design

OP-1250 oral, once-daily dosing

We are here today

Phase 1a Dose Escalation



- Measurable and non-measurable disease

Phase 1b Dose Expansion

60 mg (n=15)

120 mg (n=15)

- Measurable disease only

RP2D (120 mg)

Phase 2 Cohorts

Several patients on study

Patients with measurable disease	(N=50)
Patients with non-measurable disease	(N=15)
Patients with CNS metastasis	(N=15)

Primary objectives: Pharmacokinetics, safety and tolerability, identify RP2D
Secondary objectives: ORR (CR + PR); CBR (CR + PR + SD ≥24 weeks)

Objectives: Antitumor activity in measurable disease cohort; safety and tolerability at RP2D

Phase 1 Key Inclusion Criteria:

- ER+/HER2- advanced breast cancer
- ≥1 prior endocrine therapy for advanced breast cancer
- ≤2 prior chemotherapy regimens for locally advanced or metastatic disease

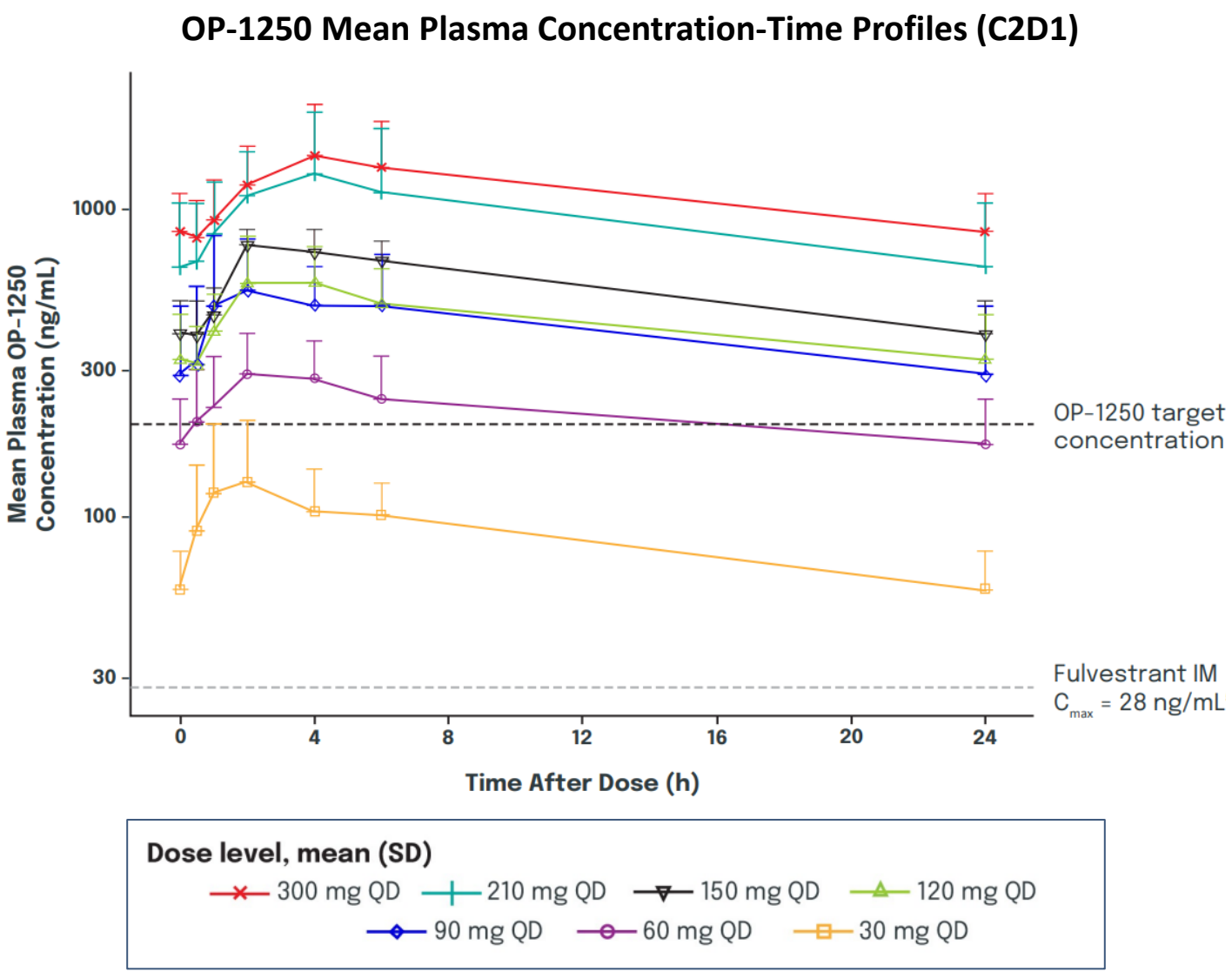
CNS, central nervous system; CBR, clinical benefit rate; ORR, overall response rate; CR, complete response; PR, partial response; SD, stable disease; RP2D, recommended phase 2 dose

OP-1250 Phase 1a Study Population Received Extensive Prior Therapy

Patient Characteristics	N=41 (%*)
Median age (years)	63
ECOG performance status	
0	17 (41)
1	24 (59)
Measurable disease at baseline	31 (76)
Visceral disease (liver, lung, peritoneum, pleura, ascites)	25 (61)
Prior lines of therapy in advanced settings	Median=3 (Range 1-8)
Prior lines of endocrine therapy in advanced settings†	Median=2
1	12 (29)
2	13 (32)
3 or more	15 (37)
Types of prior therapies in advanced settings	
Chemotherapy	17 (42)
Aromatase inhibitor (AI)	31 (76)
Fulvestrant	28 (68)
CDK 4/6 inhibitor	39 (95)‡
***ESR1* mutations at baseline (ctDNA)**, n=39 evaluated	19 (49)§

*Sums may not total to 100% due to rounding †One patient had missing data ‡Nine patients received 2 prior CDK4/6i regimens
§ctDNA was not collected in 2 patients. ECOG, Eastern Cooperative Oncology Group; CDK4/6, cyclin-dependent kinases

Dose-Proportional PK with Attractive Steady-State Plasma Concentrations



- High oral bioavailability and steady-state plasma levels with minimal peak-to-trough variability
- Doses ≥60 mg QD exceed predicted efficacy thresholds
- Enables complete antagonism of the estrogen receptor without the need for higher daily doses
- Effective half life ($T_{1/2}$)=51-73 hours, supporting once-daily dosing

Favorable Tolerability Profile

TRAEs in ≥15% of Patients	30 mg (n=5)		60 mg (n=6)		90 mg (n=6)		120 mg (n=6)		150 mg (n=4)		210 mg (n=7)		300 mg (n=7)		Total N=41 (%)	
	All	G≥3	All	G≥3	All	G≥3	All	G≥3	All	G≥3	All	G≥3	All	G≥3	All	G≥3
Patients with ≥1 event	4	1	3	0	2	0	5	0	2	1	6	1	6	1	28 (68)	4 (10)
Nausea	1	0	2	0	1	0	5	0	1	0	4	0	6	1	20 (49)	1 (2)
Fatigue	2	0	3	0	0	0	1	0	1	1	3	0	4	0	14 (34)	1 (2)
Vomiting	0	0	1	0	1	0	2	0	0	0	1	0	4	0	9 (22)	0
Headache	0	0	1	0	0	0	1	0	1	0	0	0	4	0	7 (17)	0

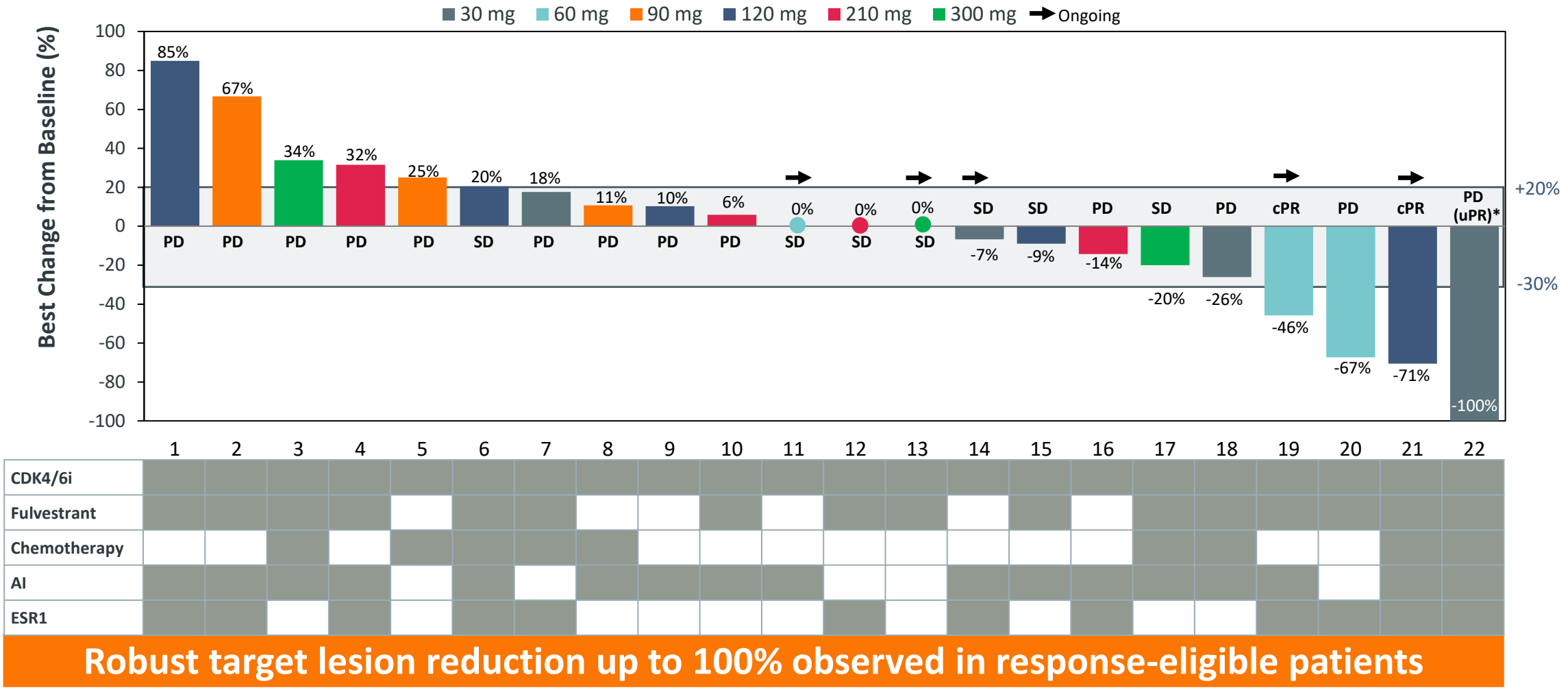
- Adverse events primarily grade 1 or 2 across all dose levels
- No dose limiting toxicities and maximum tolerated dose not reached
- No clinically significant bradycardia, ocular toxicity or diarrhea

Targeted RP2D range of 60 to 120 mg based on pharmacokinetics, favorable tolerability, and initial efficacy

TRAE, treatment-related adverse event as assessed by study investigator.

OP-1250 Demonstrated Meaningful Anti-Tumor Activity

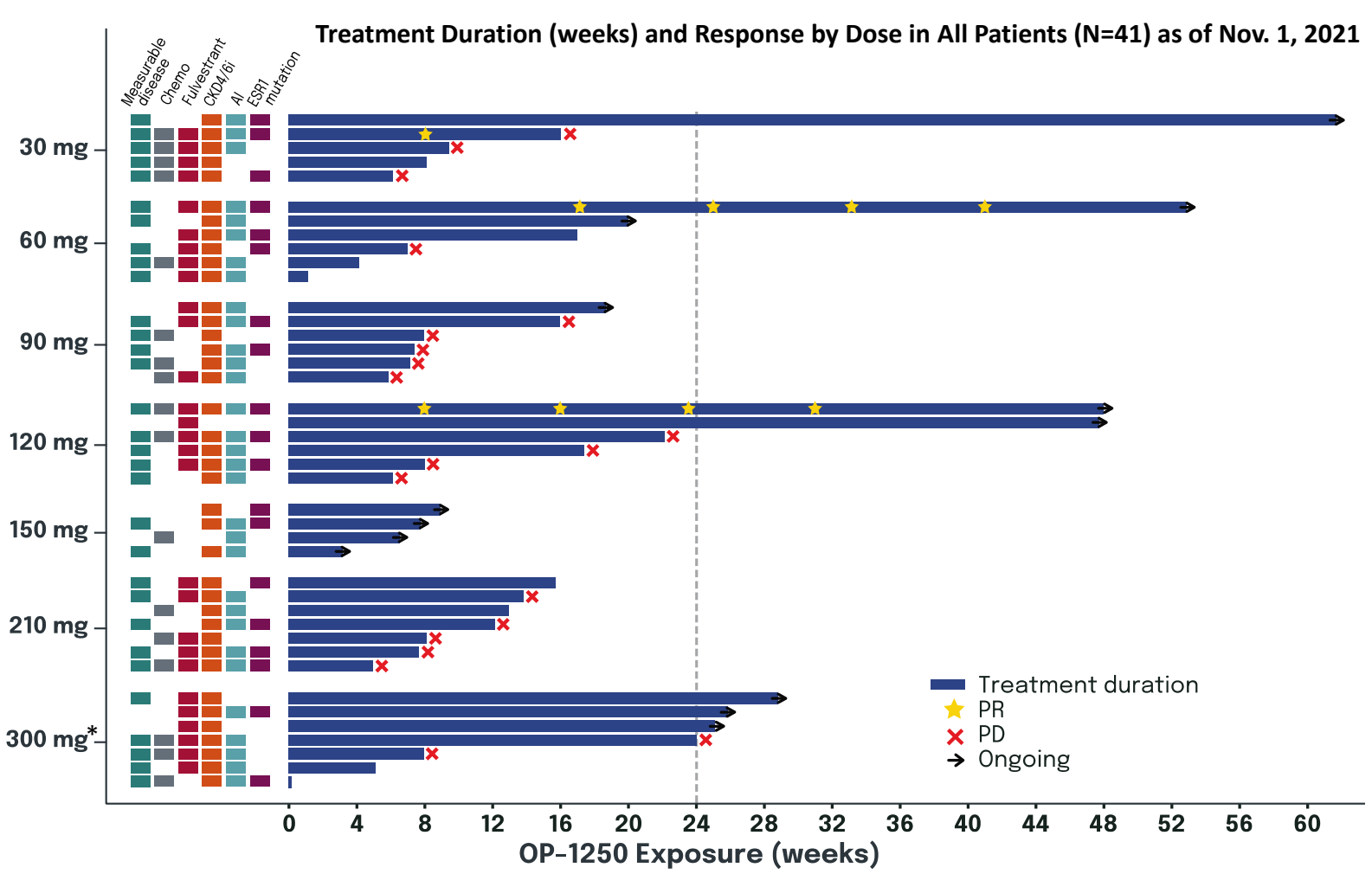
Best Response of Target Lesion in Patients with Measurable Disease (N=22)



Robust target lesion reduction up to 100% observed in response-eligible patients

*Patient's response unconfirmed due to progression with a new non-target lesion at follow-up visit.
Efficacy-evaluable patients include those with measurable disease at baseline and at least one post-baseline scan. Data cut-off: November 1, 2021.
CDK4/6i, cyclin-dependent kinases inhibitor; AI, aromatase inhibitor.

Durable Clinical Benefit Observed in Heavily Pretreated Population



- Across dose levels:
  ORR: 8% (2/24) , CBR: 29% (7/24)
- RP2D range (60-120 mg):
  ORR: 17% (2/12), CBR: 46% (6/13)

- 2 confirmed partial responses (cPR) maintained ≥8 months

- 1 patient had 100% target lesion reduction on 30 mg OP-1250; response remained unconfirmed due to PD with new lesion identification at a follow-up visit

- Long duration of benefit, with 8 patients on therapy ≥6 months and 2 ≥1 year

- 32% of patients (13/41) still on treatment as of Nov. 1, 2021

*Four patients in the 300 mg cohort dose reduced, 3 to 120 mg and 1 to 60 mg with most occurring at the beginning of cycle 2. These patients were included in RP2D CBR calculation. CBR defined as SD persisting ≥24 weeks, or a best response of confirmed CR or PR. ORR includes patients with measurable disease only. AI, aromatase inhibitor; CDK4/6i, cyclin-dependent kinases inhibitor. Data cut-off: November 1, 2021.

OP-1250: Successful Phase 1a with Key Objectives Achieved

Highly Attractive Pharmacokinetics

- High oral bioavailability
- Dose proportional PK with exposures supporting once-daily dosing
  - Smooth profile with minimal peak-to-trough variability
- Effective half-life of 51-73 hours
- Doses ≥60 mg QD exceed predicted efficacy thresholds
  - Enables complete antagonism of ER without the need for higher daily doses

Favorable Tolerability

- Generally well tolerated
- Adverse events (AEs) were mostly Grade 1 or 2 at all dose levels
- No DLTs observed and MTD not reached
- No clinically significant bradycardia, ocular toxicity, or diarrhea. Low rates of neutropenia seen and identified as an AE of special interest
- RP2D range of 60 to 120 mg identified based on favorable PK, tolerability, and initial efficacy

Promising Anti-Tumor Efficacy

- Clear efficacy signals observed in heavily pretreated patients
- 3 partial responses observed in patients with ESR1 mutations*
  - 2 confirmed and 1 unconfirmed
  - Durable cPRs ≥8months
- RP2D range (60-120 mg):
  ORR: 17% (2/12), CBR: 46% (6/13)
- Robust target lesion reductions ≥30% observed in 4 response-eligible patients
- 13 of 41 (32%) patients remain on study as of data cut-off date

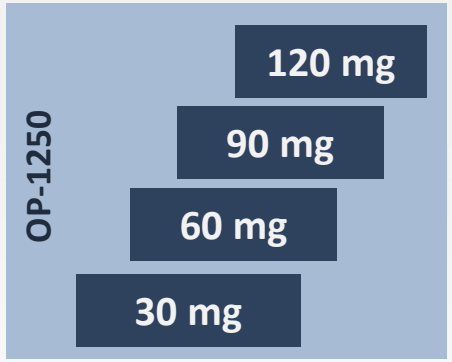
Potential best-in-class backbone endocrine therapy of choice for ER+ breast cancer

*Best overall response as of data cut-off date of November 1, 2021

Phase 1b Combination Study with Palbociclib: Study Design

Initiated January 2022

Dose Escalation



\+ Palbociclib 125 mg

- 3+3 design
- N=12-18 patients

Primary objectives: Pharmacokinetics, safety and tolerability, identify RP2D of OP-1250 for combination with palbociclib
Secondary objectives: ORR (CR + PR), CBR (CR + PR + SD ≥24 weeks)

Dose Expansion

RP2D OP-1250 + Palbociclib 125 mg

- N=12-18 patients

Objectives: Safety, tolerability and antitumor activity of OP-1250 at RP2D in combination with palbociclib

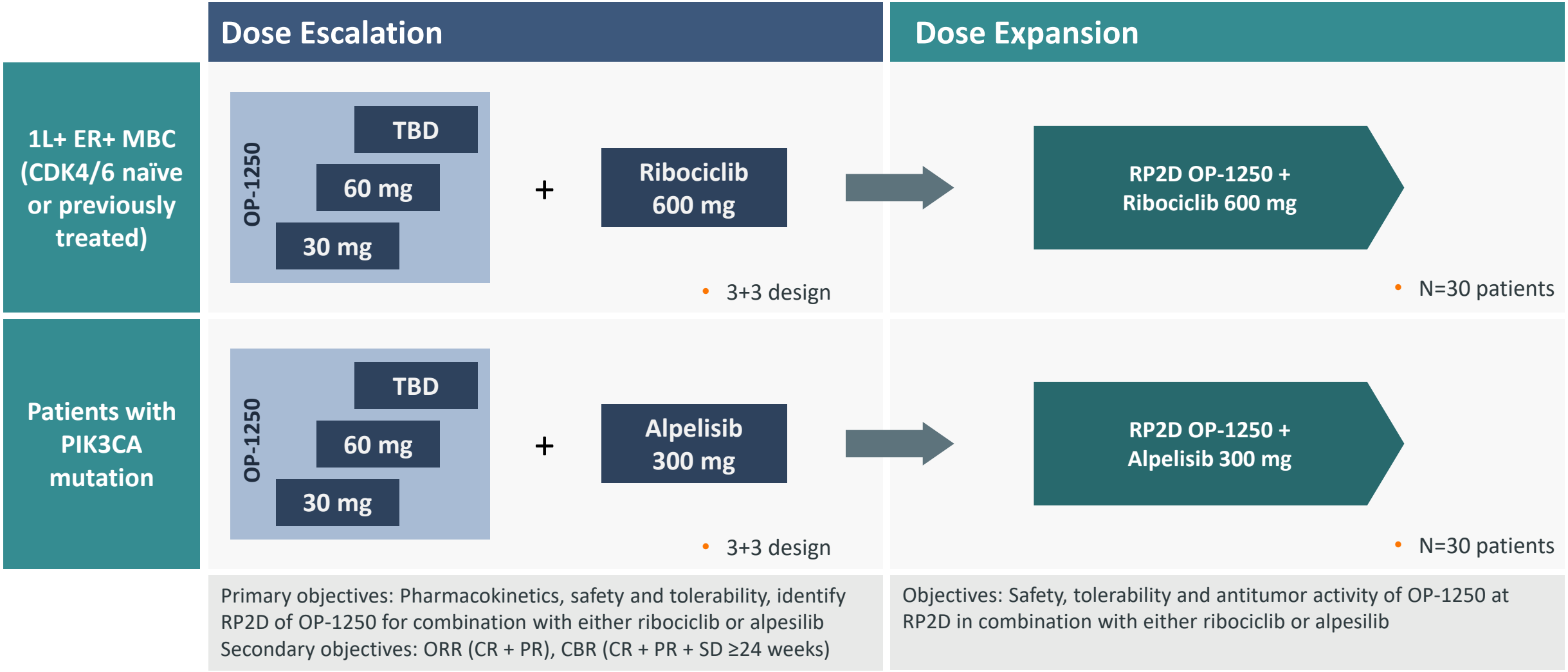
Key Inclusion Criteria:

- ER+/HER2- advanced breast cancer
- Evaluable disease (measurable and non-measurable)
- ≤ 1 prior hormonal regimen for locally advanced or metastatic disease
- Can be CDK4/6i naïve or pre-treated

CBR, clinical benefit rate; ORR, overall response rate; CR, complete response; PR, partial response; SD, stable disease; RP2D, recommended phase 2 dose

Phase 1b Combination Study with Ribociclib and Alpelisib: Study Design

Initiating in Q3 2022



Primary objectives: Pharmacokinetics, safety and tolerability, identify RP2D of OP-1250 for combination with either ribociclib or alpesilib
Secondary objectives: ORR (CR + PR), CBR (CR + PR + SD ≥24 weeks)

Objectives: Safety, tolerability and antitumor activity of OP-1250 at RP2D in combination with either ribociclib or alpesilib

Rapidly Advancing OP-1250 Clinical Development Toward Pivotal Phase 3 Studies

1H 2022

- ✓ Select RP2D
- ✓ Initiate Phase 2 cohorts
  - Measurable disease (N=50)
  - Non-measurable disease (N=15)
  - CNS metastasis (N=15)
- ✓ Initiated Phase 1b combination study with palbociclib

2H 2022

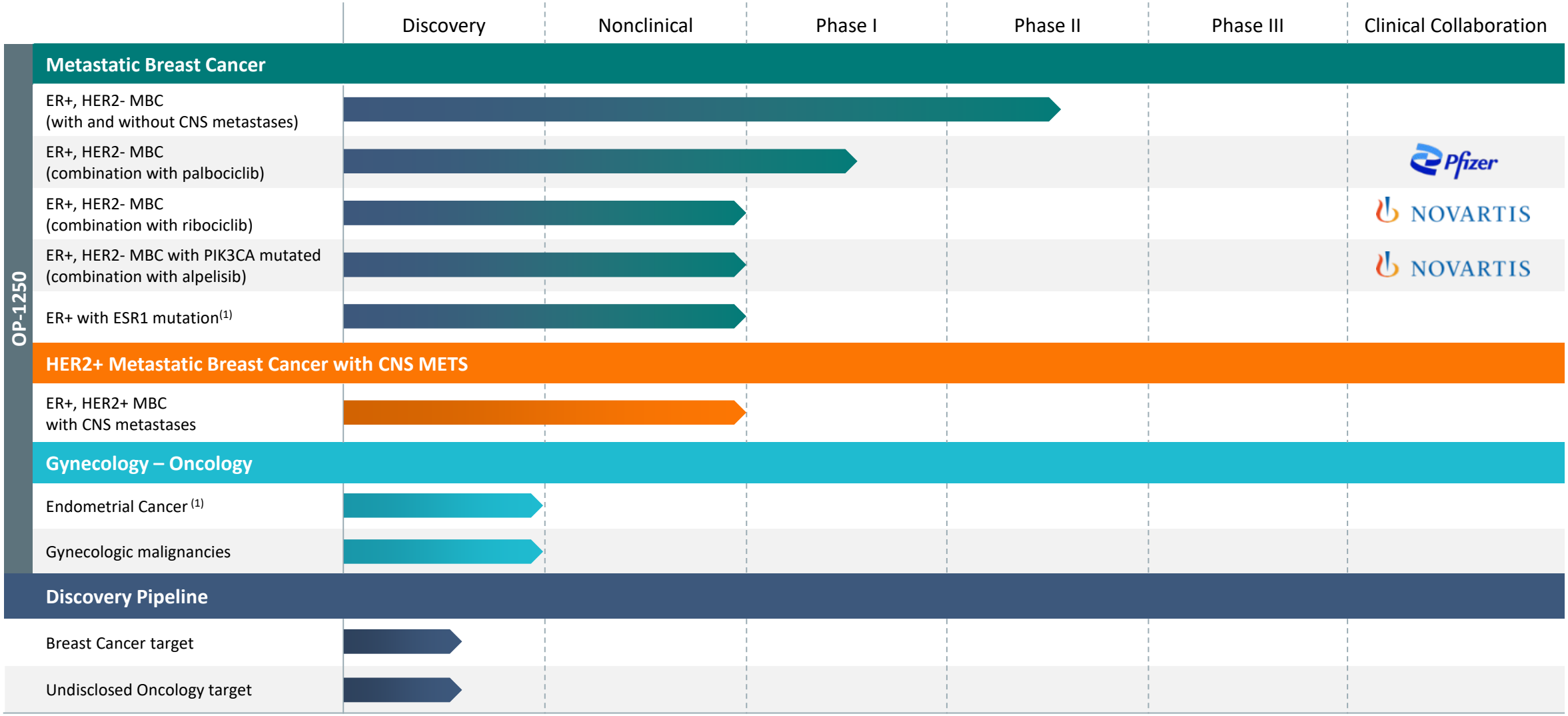
- Initiate Phase 1b study of OP-1250 in combination with each of ribociclib and alpelisib
- Present updated monotherapy and initial combination data in Q4 2022

2023

- Present additional monotherapy and combination data
- Initiate pivotal 2L+ monotherapy study mid-2023

Building evidence to support OP-1250's potential as a differentiated, best-in-class CERAN

Olema Oncology Pipeline



MBC = metastatic breast cancer; PI3Kα = phosphatidylinositol 3-kinase alpha; CDK4/6i = CDK4/6 inhibitor
(1) Patient population may be studied as additional cohort(s) of current Phase 1/2 clinical trial or may be studied in a separate clinical trial.

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Appendix

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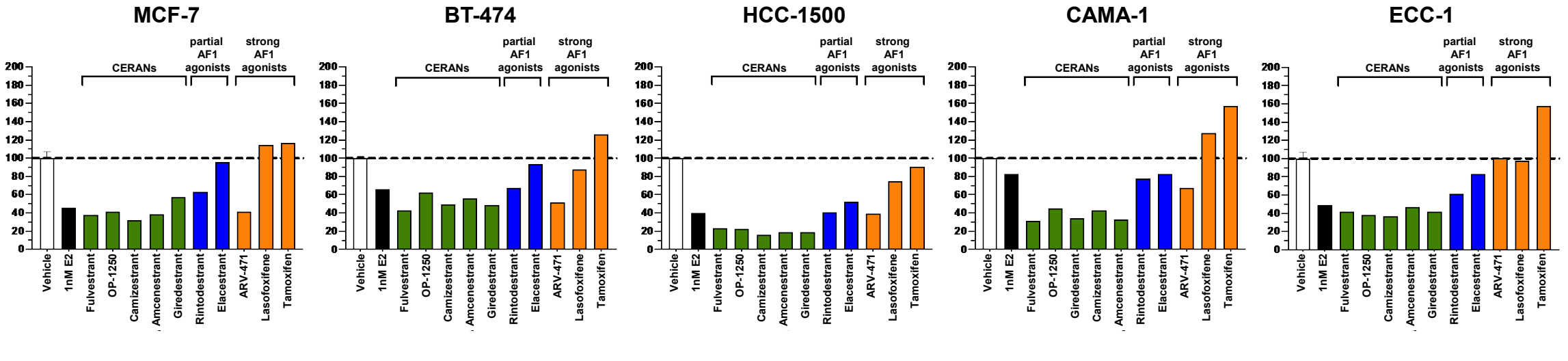
Both CERANs and SERM/SERDs are Strong Degraders of ERα



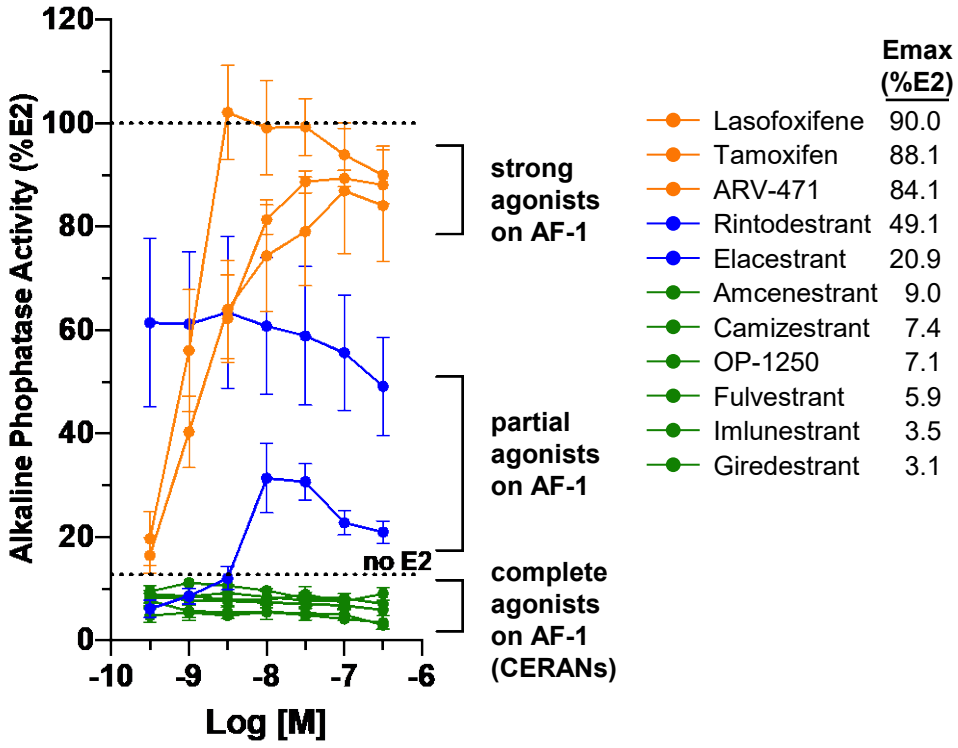
- OP-1250 and CERANs strongly degraded the estrogen receptor (ER) in five ER+ cell lines
- Partial and strong agonists demonstrated variable and inconsistent ER degradation
- Estradiol (E2), the prototypical agonist of ERα, degraded ERα in all five ER+ cell lines

In all cases, none of the compounds achieved full ER degradation;
Complete ER antagonism is independent from degradation and key to inactivating remaining ER receptor

Reference: Sun et al., Proceedings: 2021 JCA-AACR Precision Cancer Medicine International Conference; September 10-12, 2021

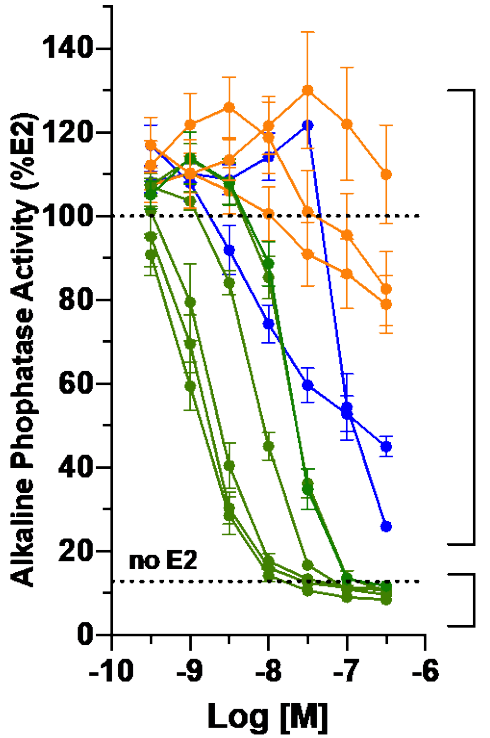
CERANs Inactivate Both AF1 & AF2 Activity While SERM/SERDs Only Inactivate AF2

Agonist Mode (No Estrogen)



	Emax (%E2)
Lasofoxifene	90.0
Tamoxifen	88.1
ARV-471	84.1
Rintodestrant	49.1
Elacestrant	20.9
Amcenestrant	9.0
Camizestrant	7.4
OP-1250	7.1
Fulvestrant	5.9
Imlunestrant	3.5
Giredestrant	3.1

Antagonist Mode (+ Estrogen)



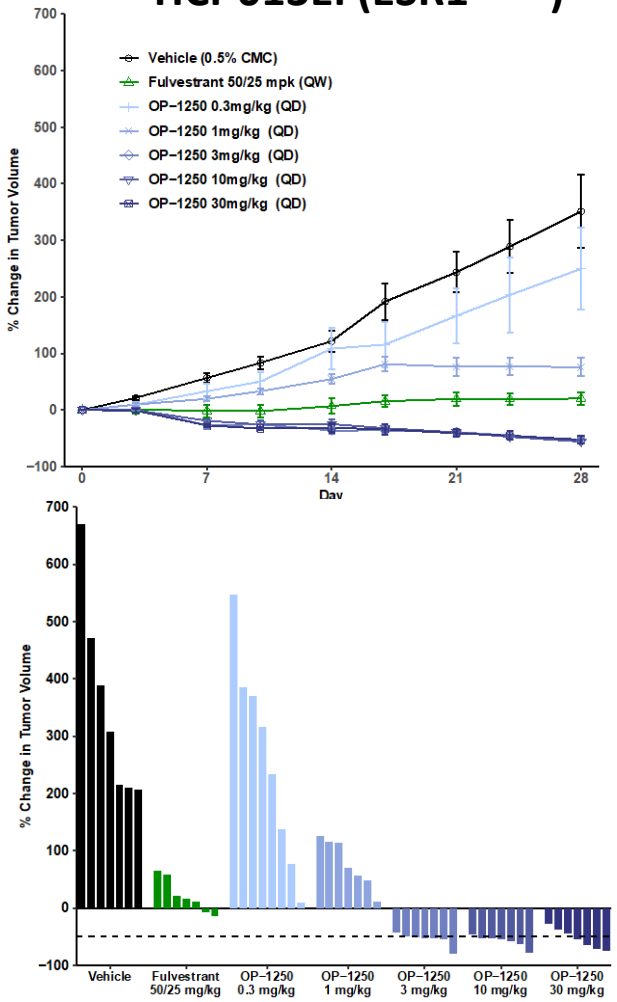
	pIC50	Emax (%E2)
Tamoxifen	NA	109.9
Lasofoxifene	NA	82.6
ARV-471	NA	78.9
Elacestrant	6.71	25.9
Rintodestrant	7.00	44.9
Imlunestrant	7.53	11.7
Giredestrant	8.71	10.7
Amcenestrant	7.54	10.5
Camizestrant	8.54	10.5
OP-1250	7.98	9.6
Fulvestrant	8.80	8.4

CERANs completely inactivate AF1 activity of the estrogen receptor, while partial and strong agonists (SERM/SERDs) do not fully inhibit AF-1 activity

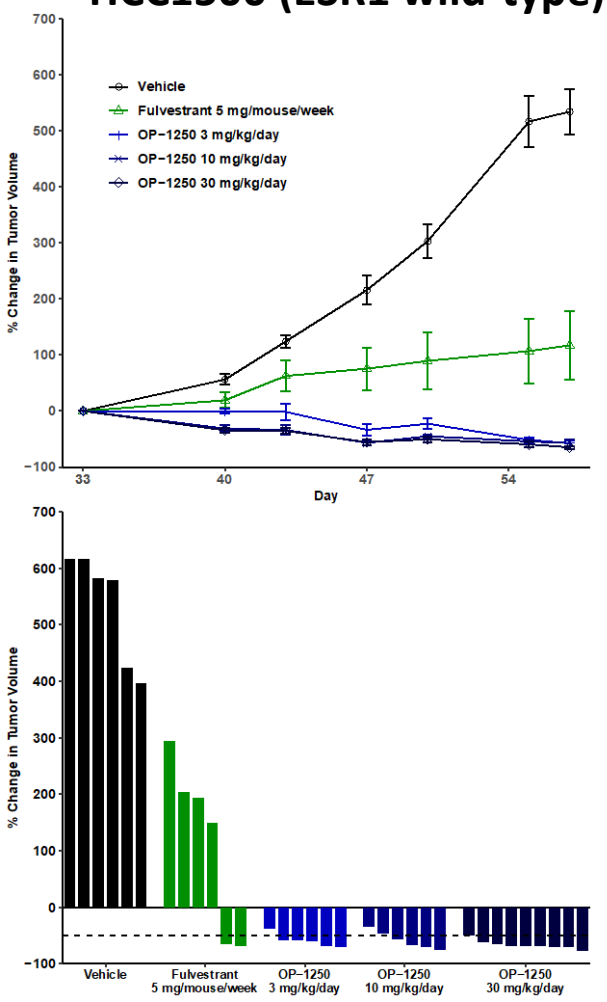
Reference: Sun et al., Proceedings: 2021 JCA-AACR Precision Cancer Medicine International Conference; September 10-12, 2021

OP-1250 Has Demonstrated Robust Tumor Shrinkage in Both ESR1 Wild-Type and Mutant Xenograft Models

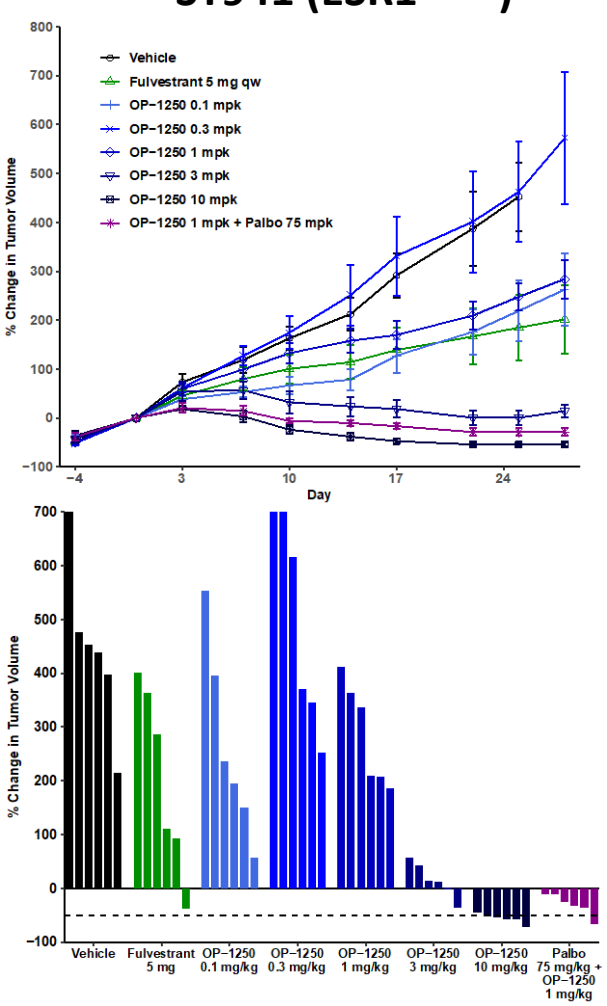
HCI-013EI (ESR1^{Y537S})



HCC1500 (ESR1 wild-type)



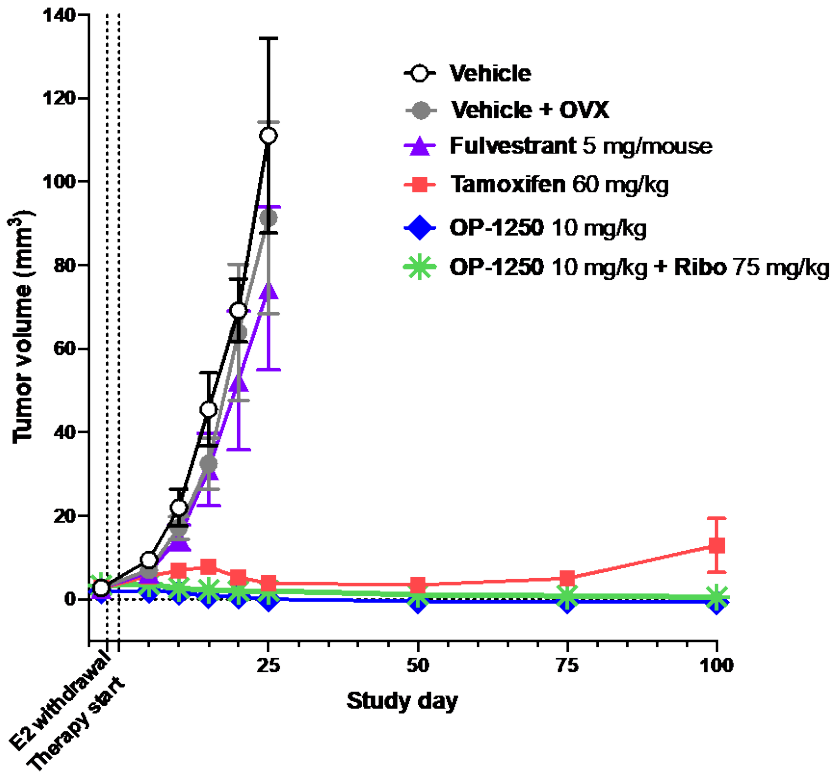
ST941 (ESR1^{Y537S})

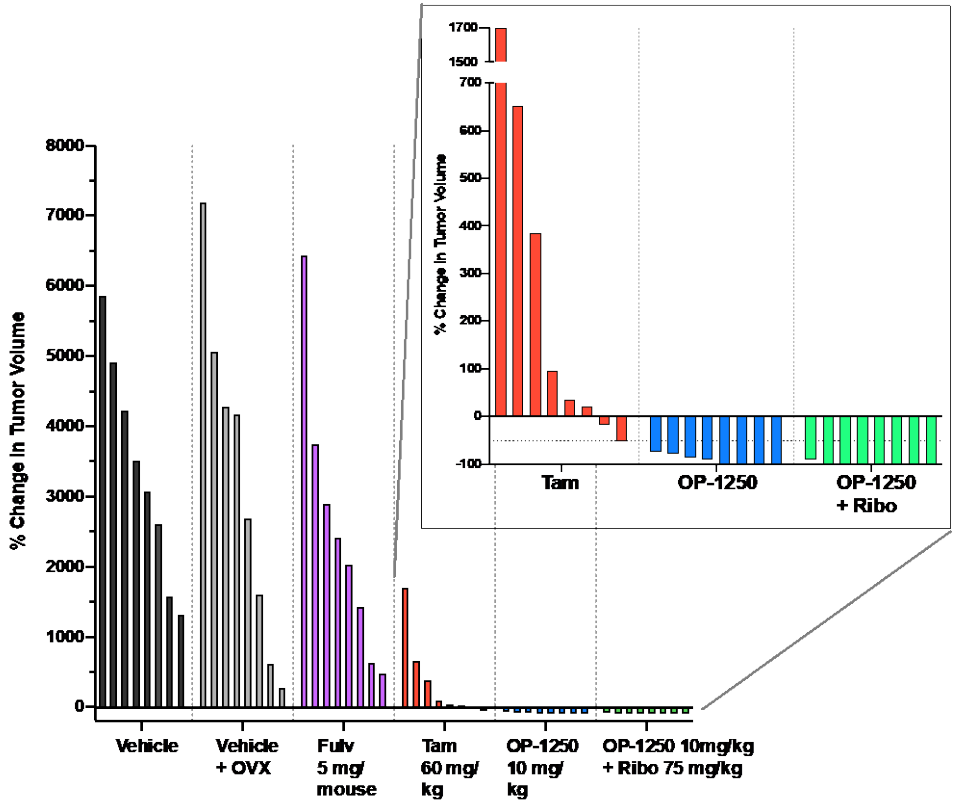


References:
Hodges-Gallagher et al., Abstract P5-05-02, Abstracts: 2019 San Antonio Breast Cancer Symposium; December 10-14, 2019; San Antonio, Texas
Hodges-Gallagher et al., Abstract 4376, Proceedings: AACR Annual Meeting 2020; April 27-28, 2020 and June 22-24, 2020; Philadelphia, PA

OP-1250 Demonstrated Robust Activity in an Intracranial Breast Cancer Brain Metastases Xenograft Study

10 mg/kg OP-1250 is superior to tamoxifen, fulvestrant and ovariectomy in shrinking mutant ESR1-Y537S tumors in an intracranial model of ER+ breast cancer brain metastasis





Treatment	Endpoint	n
Vehicle PO, QD	PD SD PR CR	8 0 0 0
Vehicle + OVX PO, QD	**PD** **SD** **PR** **CR**	7 1 0 0
5 mg Fulvestrant SC, QW	**PD** **SD** **PR** **CR**	8 0 0 0
60 mg/kg Tamoxifen PO, QD	**PD** **SD** **PR** **CR**	6 1 1 0
10 mg/kg OP-1250 PO, QD	**PD** **SD** **PR** **CR**	0 0 4 4
10 mg/kg OP-1250 + 75 mg/kg Ribociclib PO, QD	**PD** **SD** **PR** **CR**	0 0 1 7

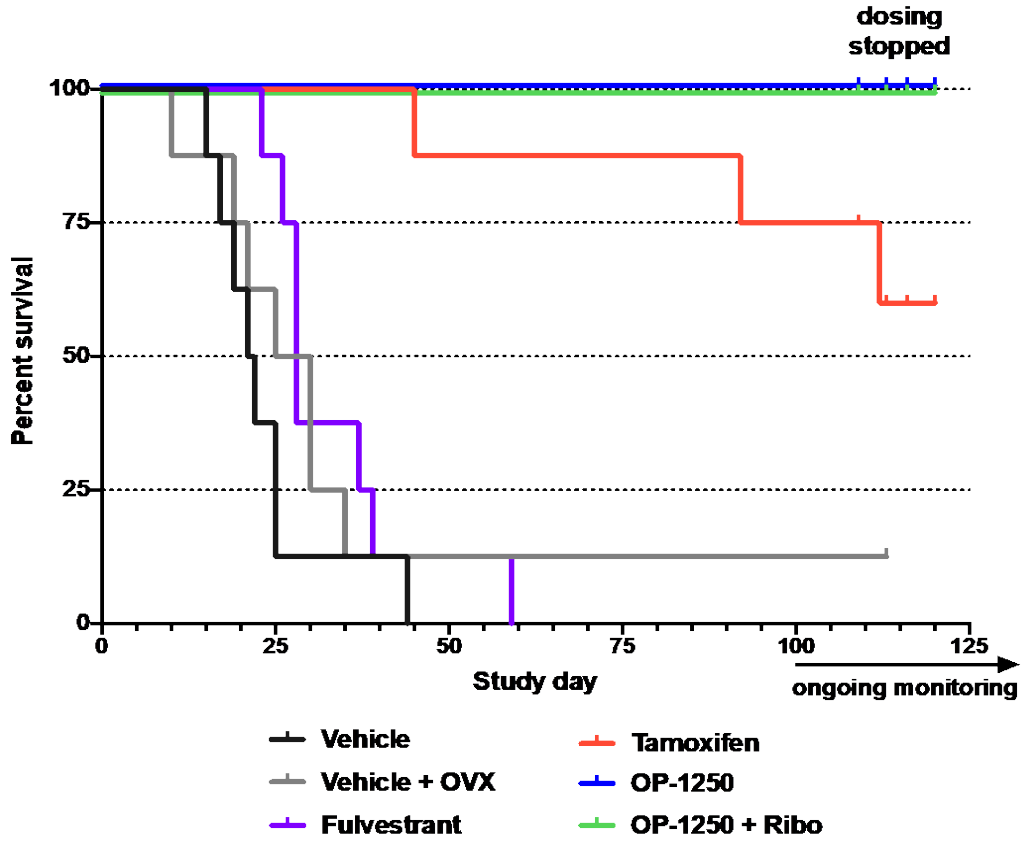
Endpoint criteria: PD (progressed disease) >20% increase tumor size; PR (partial response) >30% decrease in tumor size; CR (complete response): no tumor observed; SD (stable disease): does not meet above criteria.

After 100 days, tumors in mice treated with OP-1250 remain small or undetectable while tumors in mice treated with fulvestrant and tamoxifen have started to grow.

Reference: Hodges-Gallagher et al., Proceedings: AACR Annual Meeting 2021; April 9-14, 2021

AACR 2021: Intracranial Breast Cancer Brain Metastases Xenograft Study

OP-1250 vs. Fulvestrant and Tamoxifen: Prolonged Survival Impact



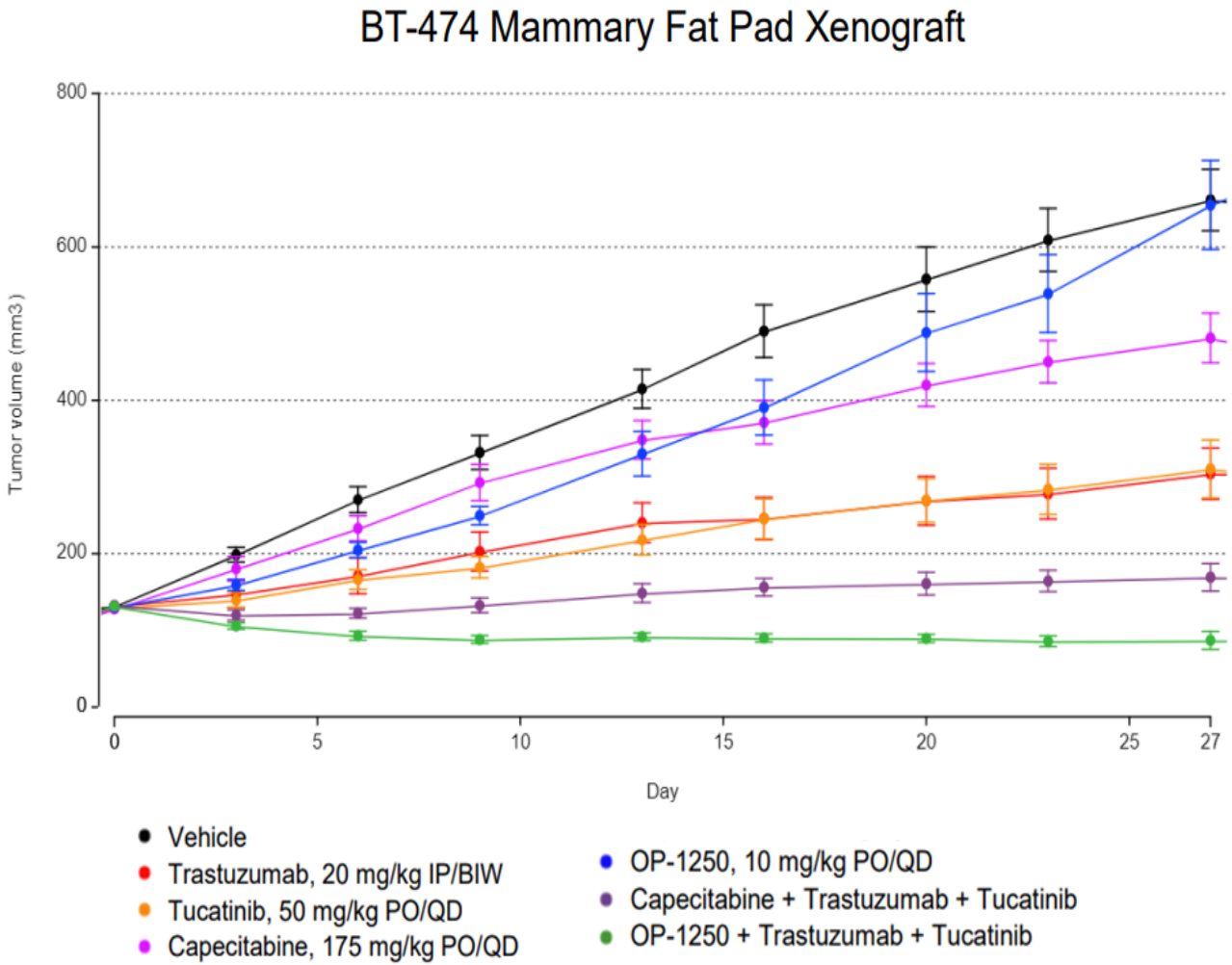
- Dosing stopped in OP-1250 10 mg/kg group at 100 days

- OP-1250 prevented death in all animals at day 120. Data suggest that OP-1250 may be an active treatment for patients with brain metastasis from ER+ breast cancer.

OP-1250 was superior in shrinking ER+ tumors compared to other endocrine therapies tested, including fulvestrant and tamoxifen.

Reference: Hodges-Gallagher et al., Proceedings: AACR Annual Meeting 2021; April 9-14, 2021

SABCS 2021: Addition of OP-1250 to Anti-HER2 Agents Improved Inhibition of Tumor Growth in Nonclinical Models of ER+/HER2+ Breast Cancer



- Approximately 25% of breast cancer tumors are HER2+, about half of which are also ER+
- HER2+ tumors have a high rate of brain metastasis
- HER2CLIMB regimen (tucatinib + trastuzumab + capecitabine) has shown efficacy for treatment of HER2+ brain metastases
- OP-1250 in combination with HER2 inhibitors, trastuzumab and tucatinib, inhibited ER+/HER2+ xenograft growth at least as well as capecitabine

Reference: Parisian, A. et al, Proceedings: 2021 SABCS; December 7-10, 2021


Thank you
Olema
ONCOLOGY