Safe harbor and forward-looking statements

This presentation contains forward-looking statements within the meaning of The Private Securities Litigation Reform Act of 1995 that involve substantial risks and uncertainties, including statements regarding vepdegestrant's potential to be a best-in-class treatment in estrogen receptor ("ER")-1 ("ESR1") mutant ER positive ("ER+"), human epidermal growth factor receptor-2 negative ("HER2-") advanced breast cancer, and being a best-in-class treatment without tradeoffs between efficacy, tolerability and patient reported outcomes; positive results from the VERITAC-2 Phase 3 clinical trial supporting potential regulatory filings; vepdegestrant's potential to address high unmet need for patients with tumors harboring ESR1 mutations; Arvinas, Inc.'s ("Arvinas') and Pfizer, Inc.'s plans to submit a New Drug Application to the U.S. Food and Drug Administration and the timing thereof; and the anticipated milestones, including submission of regulatory filings, presentation of data, and initiation of preclinical studies and/or clinical trials, the timings of such anticipated milestones, and the potential impacts of such anticipated milestones related to programs in Arvinas' pipeline, including vepdegestrant, ARV-102, ARV-393 and ARV-806. The words “anticipate,” “believe,” “estimate,” “expect,” “intend,” “may,” “might,” “plan,” “predict,” “project,” “target,” “potential,” “goal,” “will,” “would,” “could,” “should,” “continue,” and similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words.

We may not actually achieve the plans, intentions or expectations disclosed in our forward-looking statements, and you should not place undue reliance on our forward-looking statements. Actual results or events could differ materially from the plans, intentions and expectations disclosed in the forward-looking statements we make as a result of various risks and uncertainties, including but not limited to: whether we and Pfizer will be able to successfully conduct clinical development for vepdegestrant and receive results from our clinical trials of vepdegestrant on expected timelines, or at all; whether we will be able to successfully conduct and complete development for our other product candidates, including whether we initiate and complete clinical trials for our product candidates and receive results from our clinical trials on our expected timelines, or at all; whether we and Pfizer, as appropriate, will be able to obtain marketing approval for and commercialize vepdegestrant and other product candidates on current timelines or at all; whether Novartis will be able to successfully conduct and complete clinical development, obtain marketing approval for and commercialize ARV-766; whether we receive results from our preclinical trials on our expected timelines, or at all; our ability to protect our intellectual property portfolio; whether our cash and cash equivalent resources will be sufficient to fund our foreseeable and unforeseeable operating expenses and capital expenditure requirements; and other important factors, any of which could cause our actual results to differ from those contained in the forward-looking statements, discussed in the “Risk Factors” section of our quarterly and annual reports on file with the U.S. Securities and Exchange Commission. The forward-looking statements contained in this presentation reflect our current views as of the date of this presentation with respect to future events, and we assume no obligation to update any forward-looking statements, except as required by applicable law. These forward-looking statements should not be relied upon as representing our views as of any date subsequent to the date of this presentation.

The Arvinas name and logo are our trademarks. The trademarks, trade names and service marks appearing in this presentation are the property of their respective owners. We have omitted the ® and ™ designations, as applicable, for the trademarks named in this presentation. Throughout this presentation we may refer to "vepdegestrant" as "vepdeg".

This presentation may also contain estimates and other statistical data made by independent parties and by us relating to market size and other data about our industry. This data involves a number of assumptions and limitations, and you are cautioned not to give undue weight to such data and estimates. In addition, projections, assumptions and estimates of our future performance and the future performance of the markets in which we operate are necessarily subject to a high degree of uncertainty and risk. This presentation is intended for the investor community only. It is not intended to promote the products referenced herein or otherwise influence healthcare prescribing decisions. Cross-trial comparisons are not based on head-to-head studies and no direct comparisons can be made.

ARVINAS

IGNITING A

TRANSFORMATIVE CHANGE

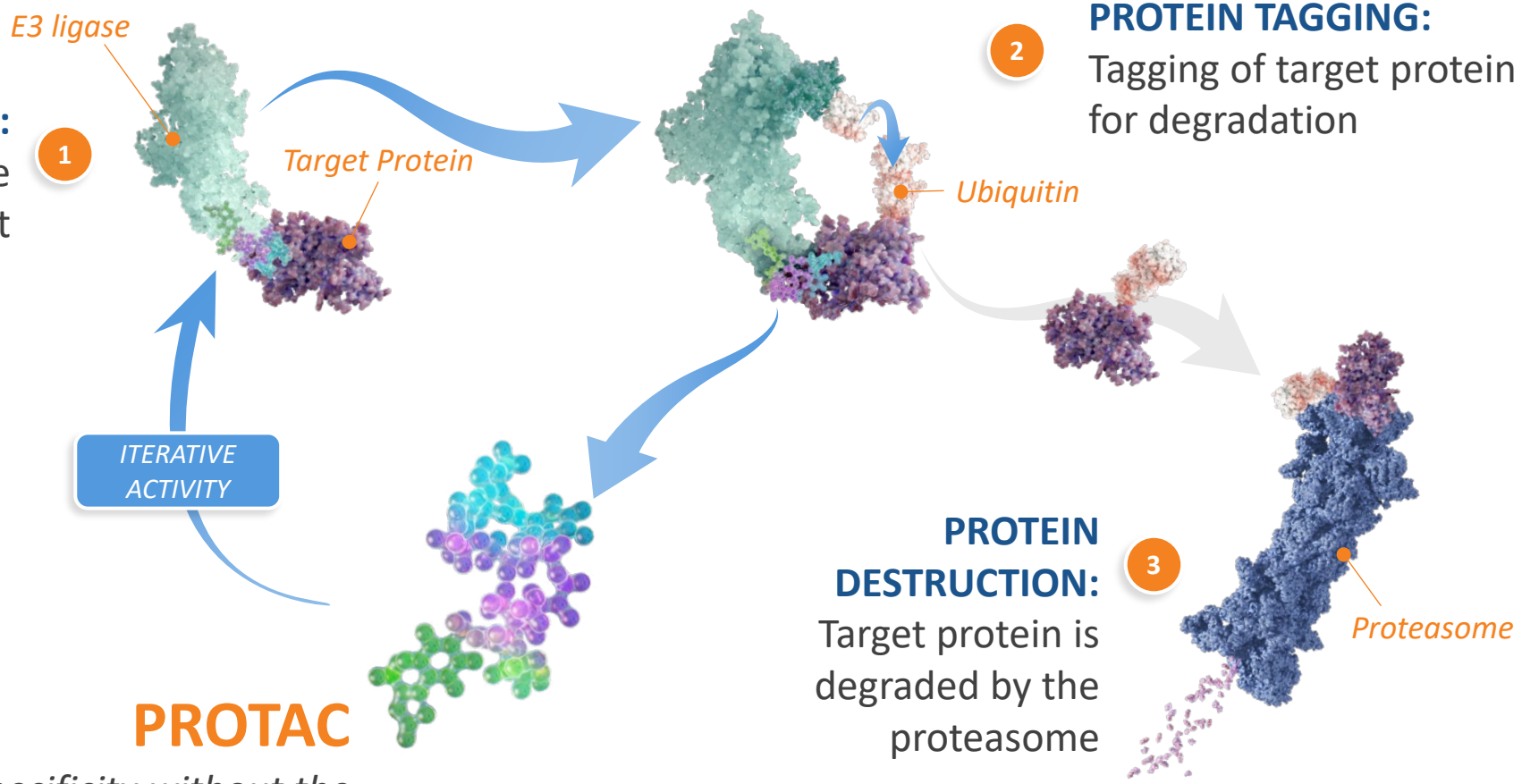
in the fight for patients with cancer and neurodegenerative diseases

PROTAC degraders harness the body's natural machinery to degrade, not simply inhibit, disease-causing proteins

ARVINAS

COMPLEX FORMATION:
Recognizing disease causing protein of interest

1



PROTEIN TAGGING:
Tagging of target protein for degradation

PROTEIN DESTRUCTION:
Target protein is degraded by the proteasome

PROTAC

High specificity without the requirement for strong binding

Tumors develop common resistance mutations that do not utilize the ER pathway for growth

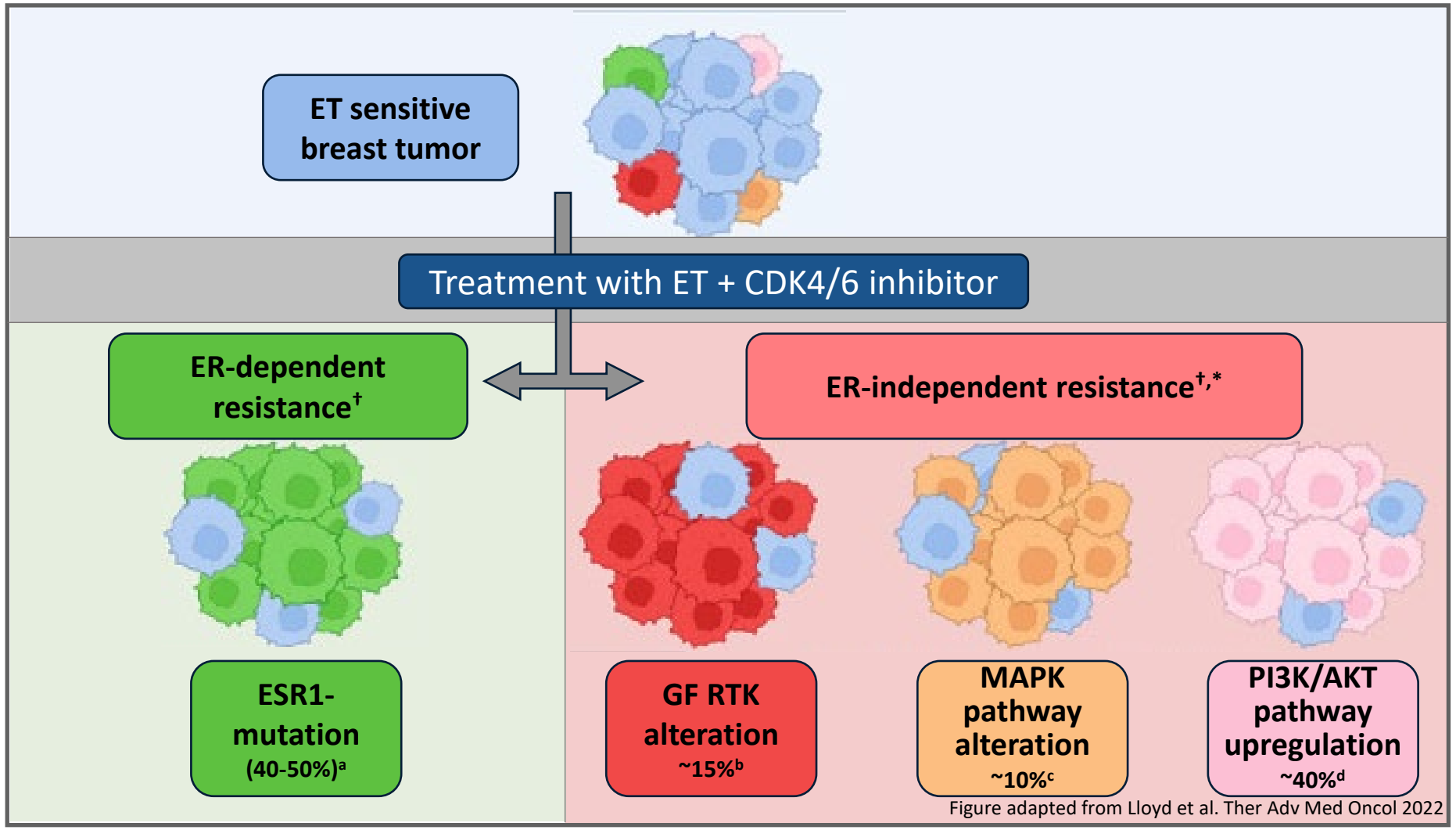


Figure adapted from Lloyd et al. Ther Adv Med Oncol 2022

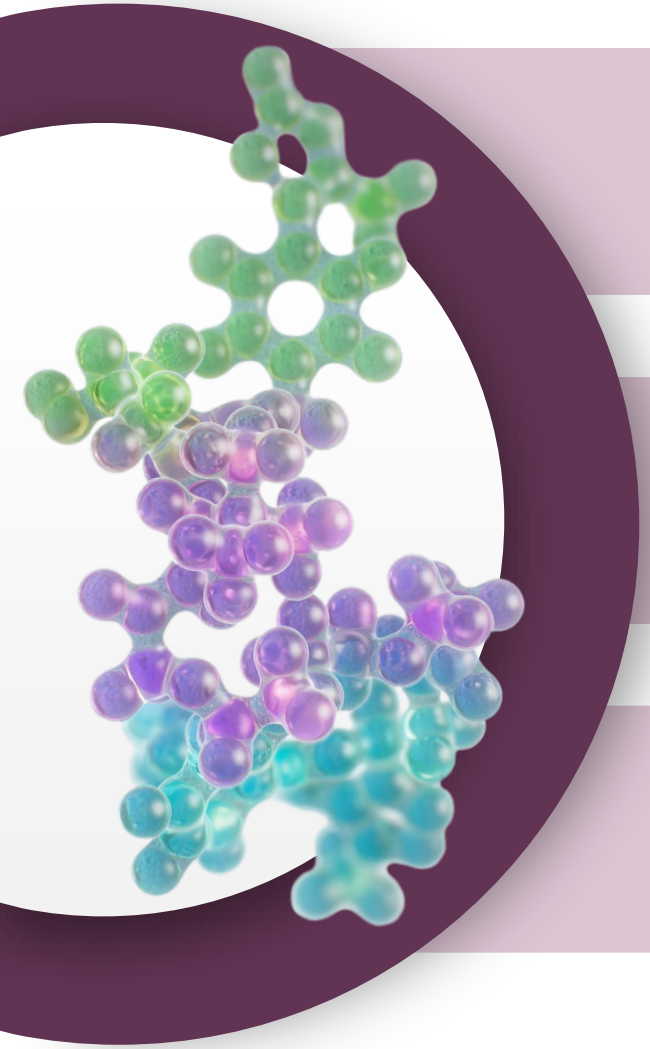
ET, estrogen therapy; CDK, cyclin-dependent kinase; ER, estrogen receptor; GF RTK; growth factor receptor tyrosine kinase; MAPK, mitogen-activated protein kinase; PI3K/AKT; phosphatidylinositol 3-kinase/protein kinase B

† Percentages are estimates of the overall patient population in 2L HR+/HER2- metastatic breast cancer after prior treatment with ET + CDK4/6i

*Includes ERBB2, FGFR1/2, EGFR, RAS, NF1, PIK3CA, AKT1, PTEN

a. Bidard et al. JCO 2022, Kalinsky et al. JCO 2025; Tolaney et al. JCO 2023. b. Includes ERBB2, FGFR1/2, EGFR: Wander et al. Cancer Discov 2020, Goetz et al. CCR 2024, Kingston et al. Nature Comms 2021, Chaudhary et al. npj Breast Cancer 2024. c. Includes RAS, NF1: Razavi et al. Cancer Cell 2018, Wander et al. Cancer Discov 2020, Goetz et al. CCR 2024, Kingston et al. Nature Comms 2021. d. Includes PIK3CA, AKT1, PTEN: Kalinsky et al. JCO 2025; Turner et al. NEJM 2023.

Vepdegestrant has potential to be a best-in-class treatment in ESR1 mutant ER+/HER2- advanced breast cancer



Novel treatment options are **needed for the ~20k patients**[a] with ESR1-mutated ER+/HER2- advanced or metastatic breast cancer in the 2L setting

In VERITAC-2, vepdegestrant demonstrated 5-month median PFS, with **robust 2.9-month improvement over fulvestrant** in patients with tumors harboring ESR1 mutations

Vepdegestrant's **novel mechanism of action** as a PROTAC ER degrader differentiates it from other ER targeting therapies in the 2L+ ESR1 mutant setting

2L, second-line; ESR1, estrogen receptor 1 gene; ER, estrogen receptor; ER+/HER2-, estrogen receptor-positive, human epidermal growth factor receptor 2-negative; PFS, progression free survival

a. Plan A ONCOSight™ Patient Populations, based on data from National Cancer Institute, SEER, 2023.

Phase 3 VERITAC-2 Results

VERITAC-2: Global Phase 3 Clinical Trial of Vepdegestrant

Key Eligibility Criteria

- Age ≥18 years old
- ER+/HER2- advanced or metastatic breast cancer
- Prior therapy:
  - 1 line of CDK4/6i + ET
  - ≤1 additional ET
  - Most recent ET for ≥6 months
  - No prior SERD (eg, fulvestrant, elacestrant)
  - No prior chemotherapy for advanced or metastatic disease
- Radiological progression during or after the last line of therapy

Randomization (1:1)

28-day Treatment Cycles

Vepdegestrant (n=313)
200 mg orally (once daily)

Fulvestrant (n=311)
500 mg IM
(days 1 and 15 of cycle 1; day 1 of subsequent cycles)

Stratification Factors:
- *ESR1* mutation[a] (yes vs no)
- Visceral disease (yes vs no)

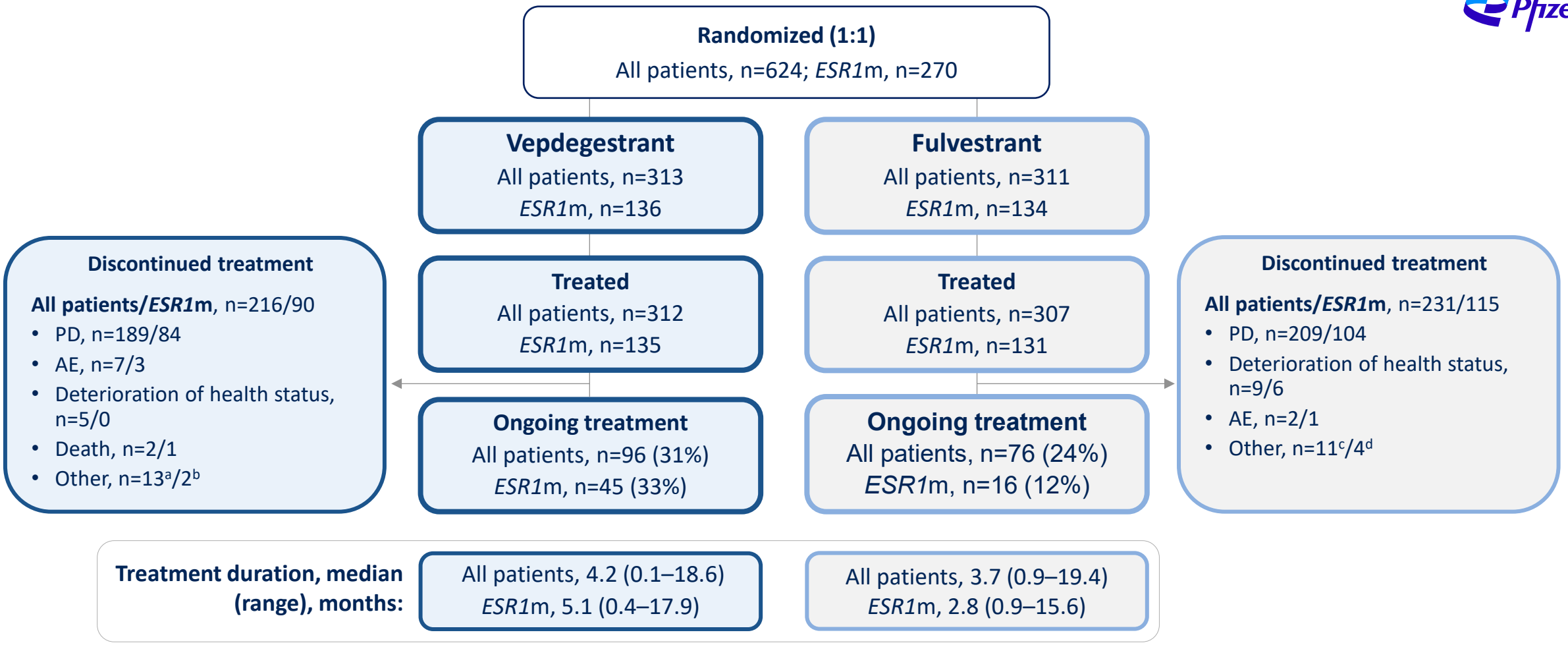
Primary Endpoints:
- PFS by BICR in
  - *ESR1*m population
  - All patients

Secondary Endpoints:
- OS (key secondary)
- CBR and ORR by BICR
- AEs

Data cutoff date: Jan 31, 2025
Clinicaltrials.gov: NCT05654623

AE, adverse event; BICR, blinded independent central review; CBR, clinical benefit rate; CDK4/6i, cyclin-dependent kinase 4/6 inhibitor; ER, estrogen receptor; *ESR1,* estrogen receptor 1 gene; *ESR1*m, estrogen receptor gene 1 mutation; ET, endocrine therapy; HER2, human epidermal growth factor receptor 2; IM, intramuscularly; ORR, objective response rate; OS, overall survival; PFS, progression-free survival; SERD, selective estrogen receptor degrader
a. *ESR1*m status was assessed in ctDNA by Foundation Medicine, except in China, where Origmed testing was used.

VERITAC-2: Patient Disposition



Data cutoff date: January 31, 2025

AE, adverse event; *ESR1m*, estrogen receptor 1 gene mutation; PD, progressive disease.

a. Other reasons included withdrawal by patient (n=9), physician's decision (n=2), and protocol deviation (n=2). b. Withdrawal by patient (n=2). c. Withdrawal by patient (n=7), physician's decision (n=3), and other (n=1). d. Withdrawal by patient (n=3) and physician's decision (n=1).

Patient population in VERITAC-2 representative of the real-world second-line setting in the U.S.

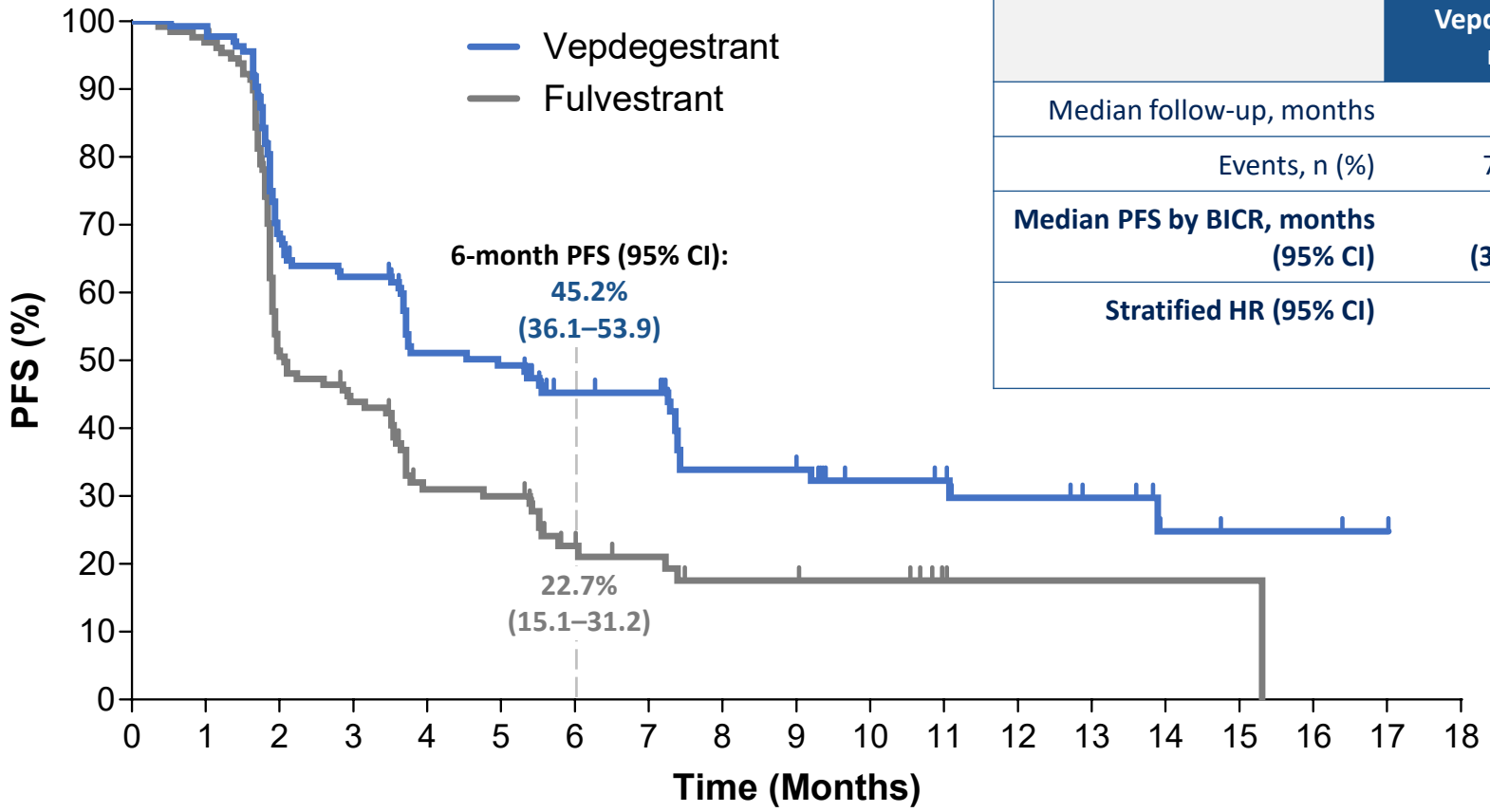
Characteristic	Patients With *ESR1*m		All Patients	
	Vepdegestrant (n=136)	Fulvestrant (n=134)	Vepdegestrant (n=313)	Fulvestrant (n=311)
Median age (range), y	60 (26–87)	60 (34–85)	60 (26–89)	60 (28–85)
Female, %	99	100	99	100
Postmenopausal, %	79	79	78	78
Race, %				
White	43	51	47	46
Black or African American	3	4	2	2
Asian	45	37	39	41
Unknown/NR	9	7	12	9
ECOG PS, %				
0	57	57	61	64
1	43	43	39	36
*ESR1*m, %[a]	100	100	43	43
Sites of disease, %				
Visceral disease	68	68	63	63
Liver metastasis	46	44	40	36
Bone-only disease	18	18	18	20

Characteristic, %	Patients With *ESR1*m		All Patients	
	Vepdegestrant (n=136)	Fulvestrant (n=134)	Vepdegestrant (n=313)	Fulvestrant (n=311)
Measurable disease[b]	71	75	71	71
Prior lines of therapy in advanced/metastatic setting[c]				
1	82	80	82	76
2	18	20	18[d]	23[d]
Prior endocrine therapy	100	100	100	100[e]
Aromatase inhibitor	99	100	99	99
SERM	15	16	16	20
Prior CDK4/6 inhibitor	100	100	100	100
Palbociclib	50	54	46	52
Ribociclib	38	28	36	31
Abemaciclib	16	25	20	21
Other[f]	1	5	4	4

CDK4/6, cyclin-dependent kinase 4/6; ECOG PS, Eastern Cooperative Oncology Group performance status; *ESR1*m, estrogen receptor 1 gene mutation; NR, not reported; SERD, selective estrogen receptor degrader; SERM, selective estrogen receptor modulator

a. *ESR1*m status was assessed in pretreatment circulating tumor DNA; b. Measurable disease assessed by blinded independent central review using Response Evaluation Criteria for Solid Tumors v1.1; c. Disease progression during or within 12 months from the end of adjuvant therapy was counted as a line of therapy in the advanced/metastatic setting; d. one additional patient in the vepdegestrant group and 3 additional patients in the fulvestrant group received 3 prior lines of therapy; e. one patient received a prior SERD; f. other CDK4/6 inhibitors included birociclib, dalpiciclib, lerociclib.

Vepdeg met the primary endpoint with a ~3-month improvement in mPFS in patients with tumors harboring ESR1 mutations



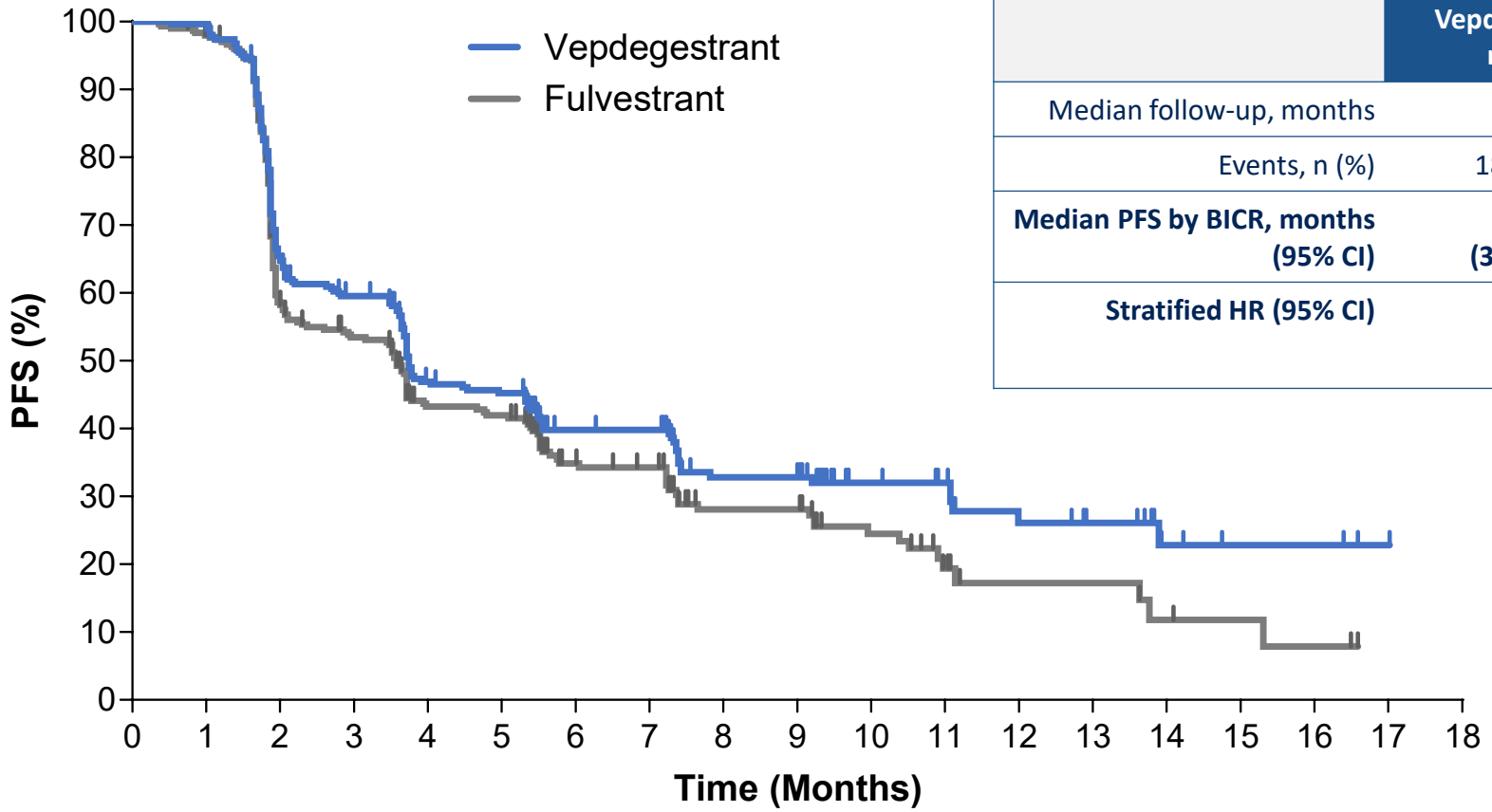
No. at risk

Vepdegestrant	136	134	87	78	55	53	38	37	22	22	15	14	10	8	4	3	3	2	0
Fulvestrant	134	125	62	52	30	29	15	12	8	8	7	2	1	1	1	1	0	0	0

	Vepdegestrant n=136	Fulvestrant n=134
Median follow-up, months	7.4	6.0
Events, n (%)	79 (58)	95 (71)
Median PFS by BICR, months (95% CI)	**5.0 (3.7–7.4)**	**2.1 (1.9–3.5)**
Stratified HR (95% CI)	**0.57 (0.42–0.77)**	
	2-sided *P*<0.001	

BICR, blinded independent central review; *ESR1m*, estrogen receptor 1 gene mutation; HR, hazard ratio; mPFS, median progression-free survival

Vepdeg did not meet the primary endpoint in the ITT population



	Vepdegestrant n=313	Fulvestrant n=311
Median follow-up, months	7.4	7.2
Events, n (%)	186 (59)	198 (64)
Median PFS by BICR, months (95% CI)	**3.7 (3.6–5.3)**	**3.6 (2.2–3.8)**
Stratified HR (95% CI)	**0.83 (0.68–1.02)**	
	2-sided *P*=0.07	

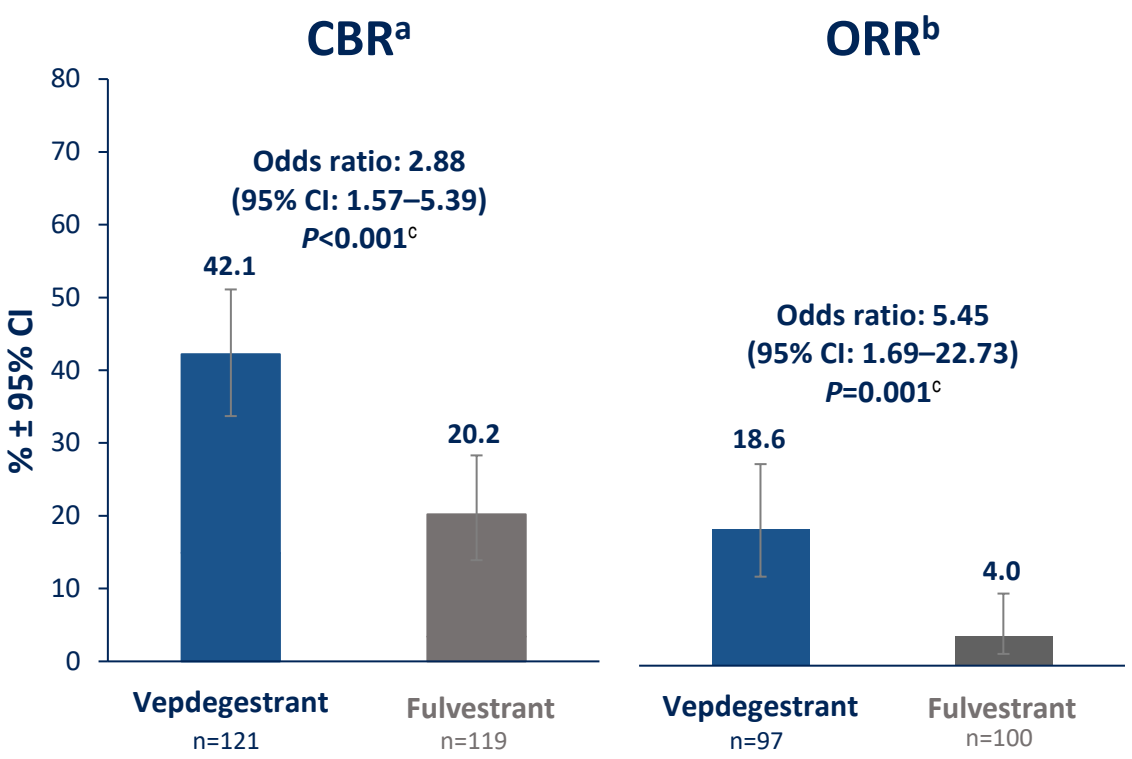
No. at risk

Vepdegestrant	313	306	189	168	113	108	74	72	46	46	28	24	15	12	6	4	4	2	0
Fulvestrant	311	292	162	143	101	98	58	54	36	36	23	12	7	7	4	3	2	0	0

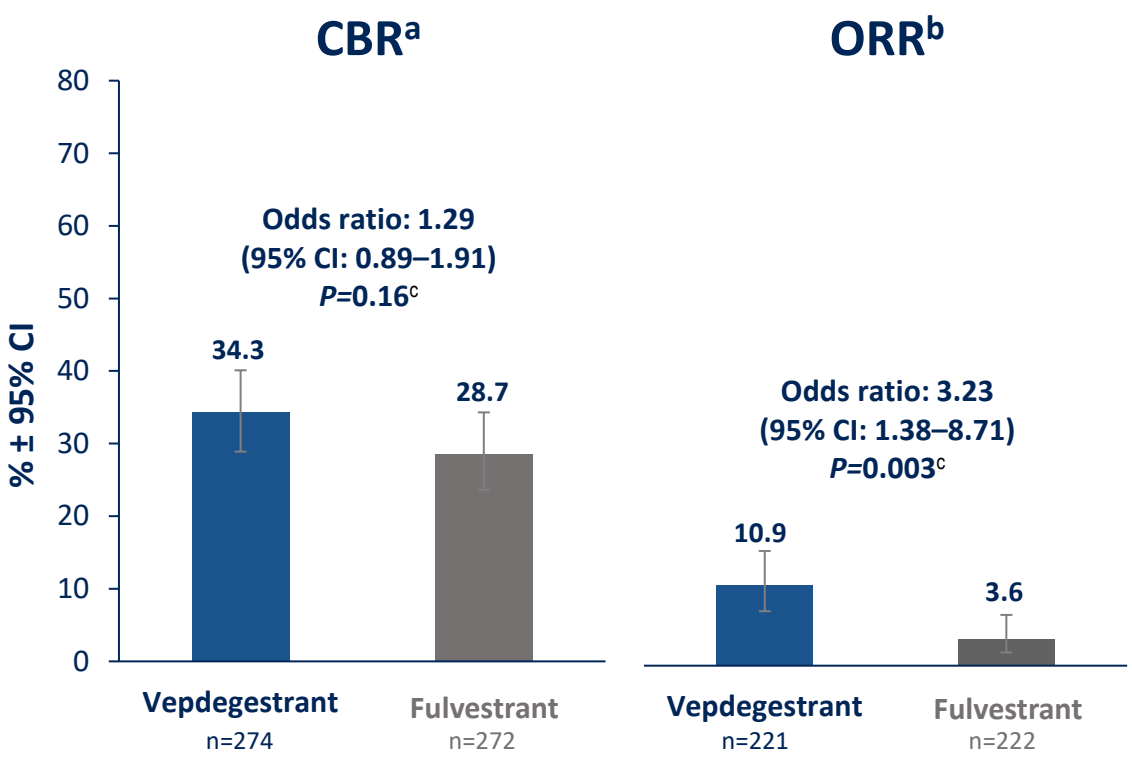
BICR, blinded independent central review; HR, hazard ratio; ITT, intent-to-treat; PFS, progression-free survival

Vepdeg showed statistically significant improvements in CBR and ORR in the ESR1 mutant population

Patients With *ESR1*m



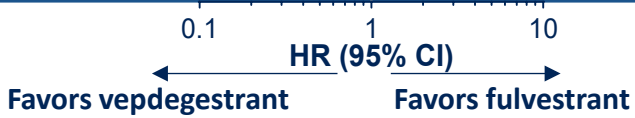
All Patients



BICR, blinded independent central review; CBR, clinical benefit rate; CR, complete response; *ESR1*m, estrogen receptor gene 1 mutation; ORR, objective response rate; PR, partial response; SD, stable disease
a. CBR was defined as the rate of confirmed CR or PR at any time, or SD, non-CR, or non-progressive disease for ≥24 weeks and was estimated in CBR-evaluable patients (those enrolled for ≥24 weeks prior to data cutoff or those with confirmed CR or PR). b. ORR was defined as the rate of confirmed CR or PR and was estimated in patients with measurable disease at baseline. c. Nominal p-value.

Consistent PFS benefit across pre-specified prognostic characteristics/ baseline disease demographics in the ESR1m population

Subgroup		Events/n Vepdegestrant	Events/n Fulvestrant	HR (95% CI)
All patients (stratified)		79/136	95/134	0.57 (0.42–0.77)
Age, years	<65	50/86	66/88	0.51 (0.35–0.74)
	≥65	29/50	29/46	0.75 (0.45–1.26)
Menopausal status	Pre/perimenopausal	14/28	20/28	0.48 (0.24–0.95)
	Postmenopausal	65/108	75/106	0.60 (0.43–0.85)
Geographic region	Asia	32/56	38/50	0.43 (0.26–0.70)
	Europe	25/41	39/56	0.65 (0.40–1.08)
	North America	13/20	10/16	0.73 (0.32–1.69)
	Other	9/19	8/12	0.71 (0.27–1.89)
ECOG PS	0	46/78	49/76	0.69 (0.46–1.04)
	1	33/58	46/58	0.46 (0.29–0.73)
Visceral disease	Yes	59/92	69/91	0.54 (0.38–0.77)
	No	20/44	26/43	0.65 (0.36–1.18)
Liver disease	Yes	45/63	49/59	0.50 (0.33–0.75)
	No	34/73	46/75	0.60 (0.38–0.94)
Bone-only disease	Yes	8/25	15/24	0.47 (0.20–1.23)
	No	71/111	80/110	0.58 (0.42–0.80)
Lines of prior therapy	1	65/112	76/107	0.54 (0.39–0.75)
	2	14/24	19/27	0.86 (0.43–1.72)



BICR, blinded independent central review; ECOG PS, Eastern Cooperative Oncology Group performance status; *ESR1*m, estrogen receptor 1 gene mutation; HR, hazard ratio; PFS, progression-free survival

Vepdeg was generally well-tolerated, with low rates of discontinuation and dose reductions; majority of TRAEs Gr 1/2

Overview

TEAEs, %	Vepdegestrant (n=312)	Fulvestrant (n=307)
Any grade	87	81
Grade ≥3	23	18
Serious	10	9
Leading to treatment discontinuation	3	1
Leading to dose reduction	2	NA
TRAEs, %		
Any grade	57	40
Grade ≥3	8	3

QT prolongation

- TEAEs: vepdegestrant,10%; fulvestrant, 1%
- A QT interval sub-study (n=88) confirmed a mild increase (11.1 ms) from baseline in mean QTcF, with upper 90% CI (13.7 ms) <20 ms,[f] **indicating no large QT-prolonging effect**

TEAEs in >10% of Patients in Either Group

TEAE, %	Vepdegestrant (n = 312)		Fulvestrant (n = 307)	
	Any Grade	Grade 3/4	Any Grade	Grade 3/4
Fatigue[a]	27	1	16	1
ALT increased[b]	14	1	10	1
AST increased[b]	14	1	10	3
Nausea	13	0	9	1
Anemia[b, c]	12	2	8	3
Neutropenia[d]	12	2[e]	5	1[e]
Back pain	11	1	7	<1
Arthralgia	11	1	11	0
Decreased appetite	11	<1	5	0

ALT, alanine aminotransferase; AST, aspartate aminotransferase; GI, gastrointestinal; QTcF, corrected QT interval using Fridericia's method; TEAE, treatment-emergent adverse event; TRAE, treatment-related adverse event
a. Includes fatigue and asthenia. b. No between-group differences were observed for ALT/AST increases or anemia based on laboratory values. c. Includes anemia, hemoglobin decreased, and iron deficiency anemia. d. Includes neutropenia and neutrophil count decreased. No events led to dose reductions or treatment discontinuation in either treatment group. There were no events of febrile neutropenia in the vepdegestrant group and 1 event of grade 2 febrile neutropenia in the fulvestrant group. e. One patient with grade 4 event. f. Based on a concentration-QTc population modeling analysis.

High unmet need remains for improved treatment options for patients in 2L+, ESR1 mutant setting

Current treatment landscape in 2L+, ESR1 mutant mBC

~20K patients

treated each year in the U.S.

EFFICACY MEASURES

- HCP seeking additional benefits
  - Less than 2-month mPFS improvement in clinical trials with oral SERDs

TOLERABILITY

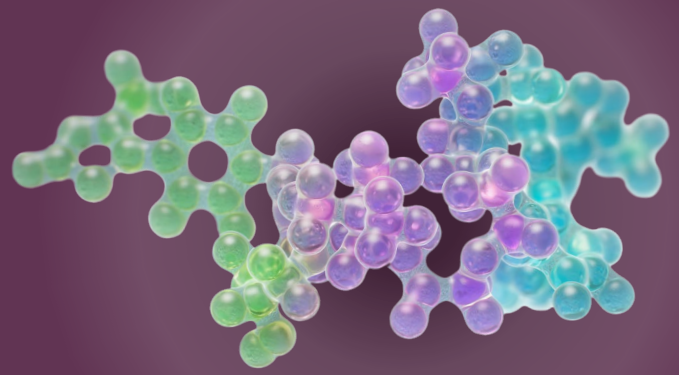
- GI-related events commonly reported with oral SERDs
  - AEs that negatively impact patients' daily lives

REAL-WORLD APPLICABILITY

- Patients enrolled in recent Phase 3 trials not representative of real-world 2L+ experience
  - Prior CDK4/6i + ET, pre/peri menopausal

2L+, second-line plus; AEs, adverse events; CDK, CDK, cyclin-dependent kinase; ESR1, estrogen receptor 1 gene; ET, estrogen therapy; GI, gastrointestinal; HCP, health care providers; mBC, metastatic breast cancer; mPFS, median progression free survival; SERDs, selective estrogen receptor degraders.

Vepdegestrant has the potential to be best-in-class treatment in the 2L+, ESR1 mutant setting



Vepdegestrant has the potential to be:

BEST-IN-CLASS TREATMENT

without tradeoffs between efficacy, tolerability, and patient reported outcomes

EFFICACY MEASURES

2.5x
Improvement in mPFS over SoC

~3-month
Improvement in mPFS
(5.0m vs. 2.1m)
vs. fulvestrant

TOLERABILITY

- Favorable safety/tolerability profile
- Low rates and severity of GI-related events

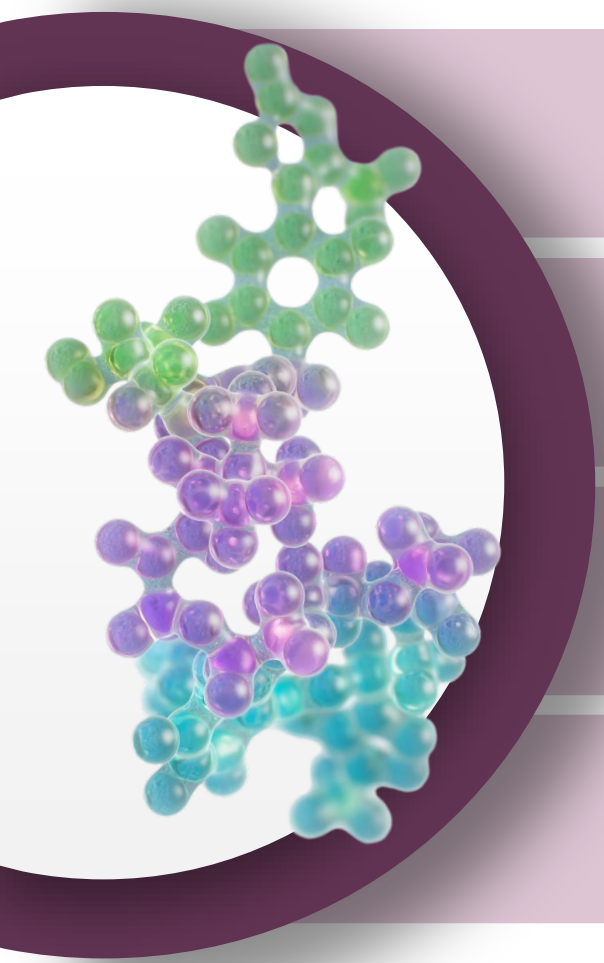
REAL-WORLD EXPERIENCE

- Patients enrolled in VERITAC-2 were representative of real-world 2nd line setting
  - 100% prior CDK4/6i + ET, pre/peri menopausal

2L+, second-line plus; AEs, adverse events; CDK, CDK, cyclin-dependent kinase; ESR1, estrogen receptor 1 gene; ET, estrogen therapy; GI, gastrointestinal; mBC, metastatic breast cancer; mPFS, median progression free survival; SERDs, selective estrogen receptor degraders; SoC, standard of care.

Positive results from VERITAC-2 Phase 3 clinical trial support potential regulatory filings[a]

Vepdegestrant is the first PROTAC degrader to demonstrate clinical benefit in a Phase 3 trial



- Potential to address high unmet need for patients with tumors harboring an ESR1 mutation in 2L+ setting
- Vepdeg showed a clinically meaningful median PFS benefit over fulvestrant, a current standard of care, in patients w/ESR1 mutations
- Vepdeg's safety and tolerability provide further evidence of a potential best-in-class profile
- **Arvinas and Pfizer are on track to submit New Drug Application to U.S. Food and Drug Administration in the coming weeks**

2L+, second-line plus; ESR1, estrogen receptor 1 gene; PFS, progression free survival
a. Data will be shared with global regulatory authorities to potentially support regulatory filing .

Impactful near-term milestones across Arvinas' clinical portfolio

Program	Anticipated Milestone	Timing	Potential Impact
Vepdeg with Pfizer	Submit NDA	2H 2025	Rapid progress towards potential commercialization in 2026
ARV-102 (LRRK2 PROTAC)	Data from Ph 1 SAD cohort in patients with PD	2H 2025	Potential to show biomarkers changes and degradation in PD patients (likely to have elevated LRRK2/ pathway impact)
	Initiate Ph 1 MAD cohort in patients with PD	2H 2025	Provide data supporting RP2D and explore impact of daily dosing in patients with Parkinson's disease
ARV-393 (BCL6 PROTAC)	Present preclinical combination data	June (EHA)	Demonstrate the potential of ARV-393 as a monotherapy in a rare and aggressive NHL, and in combination with SMIs in DLBCL
	Present initial Ph 1 data	2H 2025	First-in-human data supporting initiation of Phase 2 trial
ARV-806 (KRAS G12D PROTAC)	Initiate Ph 1 trial	2H 2025	Opportunity to show clinical impact of differentiated MOA versus clinical inhibitors/degraders

These agents are currently under investigation; their safety and effectiveness for these investigational uses have not been established.

BCL6, B cell lymphoma 6; EHA, European Hematology Association; DLBCL, diffuse large B-cell lymphoma; G12D, mutations in codon 12 on KRAS oncogene; KRAS, Kirsten rat sarcoma; LRRK2, leucine-rich repeat kinase 2; MAD, multiple ascending dose; MOA, mechanism of action; NDA, new drug application; NHL, non-Hodgkin's lymphoma; PD, Parkinson's disease; RP2D; recommended phase 2 dose; SAD, single ascending dose; SMI, small molecule inhibitor

ARVINAS

IGNITING A

TRANSFORMATIVE CHANGE

in the fight for patients with cancer and neurodegenerative diseases