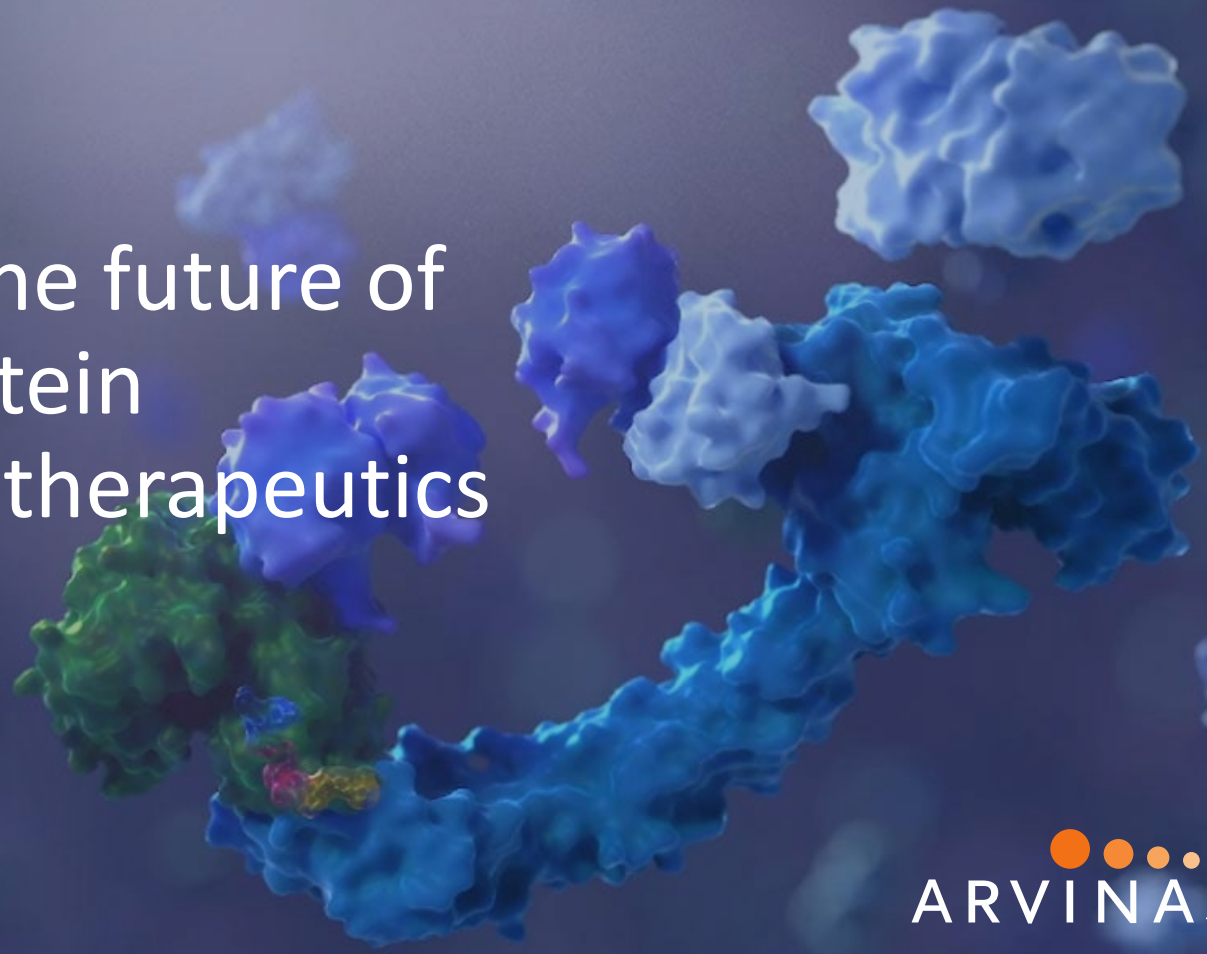
Pioneering the future of targeted protein degradation therapeutics



ARVINAS

August 2022

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 the receipt of upfront, milestone and other payments under the Pfizer collaboration, the potential benefits of our arrangements with our collaborative partnerships, statements regarding the potential advantages and therapeutic benefits of bavdegalutamide (ARV-110), ARV-471, ARV-766 and our other discovery programs, the development and regulatory status of our product candidates, such as statements with respect to our lead product candidates, bavdegalutamide (ARV-110), ARV-471 and ARV-766 and other candidates in our pipeline, and the timing of clinical trials and data from those trials and plans for registration for our product candidates, and our discovery programs that may lead to our development of additional product candidates, the potential utility of our technology, the potential commercialization of any of our product candidates and companion diagnostic partnering, and the sufficiency of our cash resources. All statements, other than statements of historical facts, contained in this presentation, including statements regarding our strategy, future operations, future financial position, future revenues, projected costs, prospects, plans and objectives of management, are forward-looking statements. The words “anticipate,” “believe,” “estimate,” “expect,” “intend,” “may,” “might,” “plan,” “predict,” “project,” “target,” “potential,” “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: each party’s performance of its obligations under the Pfizer collaboration, whether we and Pfizer will be able to successfully conduct and complete clinical development for ARV-471, whether we will be able to successfully conduct and complete development for bavdegalutamide and 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 our cash 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 the Company’s quarterly and annual reports on file with the 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.

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This presentation also contains 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.

Arvinas: Advancing a new therapeutic modality to patients

PROTEIN DEGRADATION

- PROTAC® (proteolysis-targeting chimeras) protein degraders **eliminate** vs. inhibit disease-causing proteins
- Combines the power of genetic knockdown technology with the benefits of small-molecule therapeutics

PIPELINE

2 Programs in Phase 2
Clear efficacy signals in patients with difficult-to-treat breast and prostate cancers

1 Program in Phase 1

20+ Pipeline Programs
in oncology, I-O, and neuroscience

ARVINAS

350+ team members

- Founded in 2013 by the original PROTAC pioneer
- Protein degradation platform with clinical proof of concept

PARTNERED FOR SUCCESS

ARVINAS
Pfizer

Global collaboration with Pfizer to co-develop and co-commercialize ARV-471 in ER+ breast cancer announced in July 2021

A history of pioneering since our founding

JULY 2001
Arvinas' founder Craig Crews publishes first paper describing PROTAC® degraders

JULY 2013
Arvinas founded to turn protein degraders into patient therapies

LATE 2016
Arvinas creates first oral PROTAC

EARLY 2018
Arvinas creates first BBB-crossing PROTAC degraders

2019
PROTAC clinical studies: Bavdegalutamide and ARV-471

2020
Efficacy proof of concept for bavdegalutamide and ARV-471

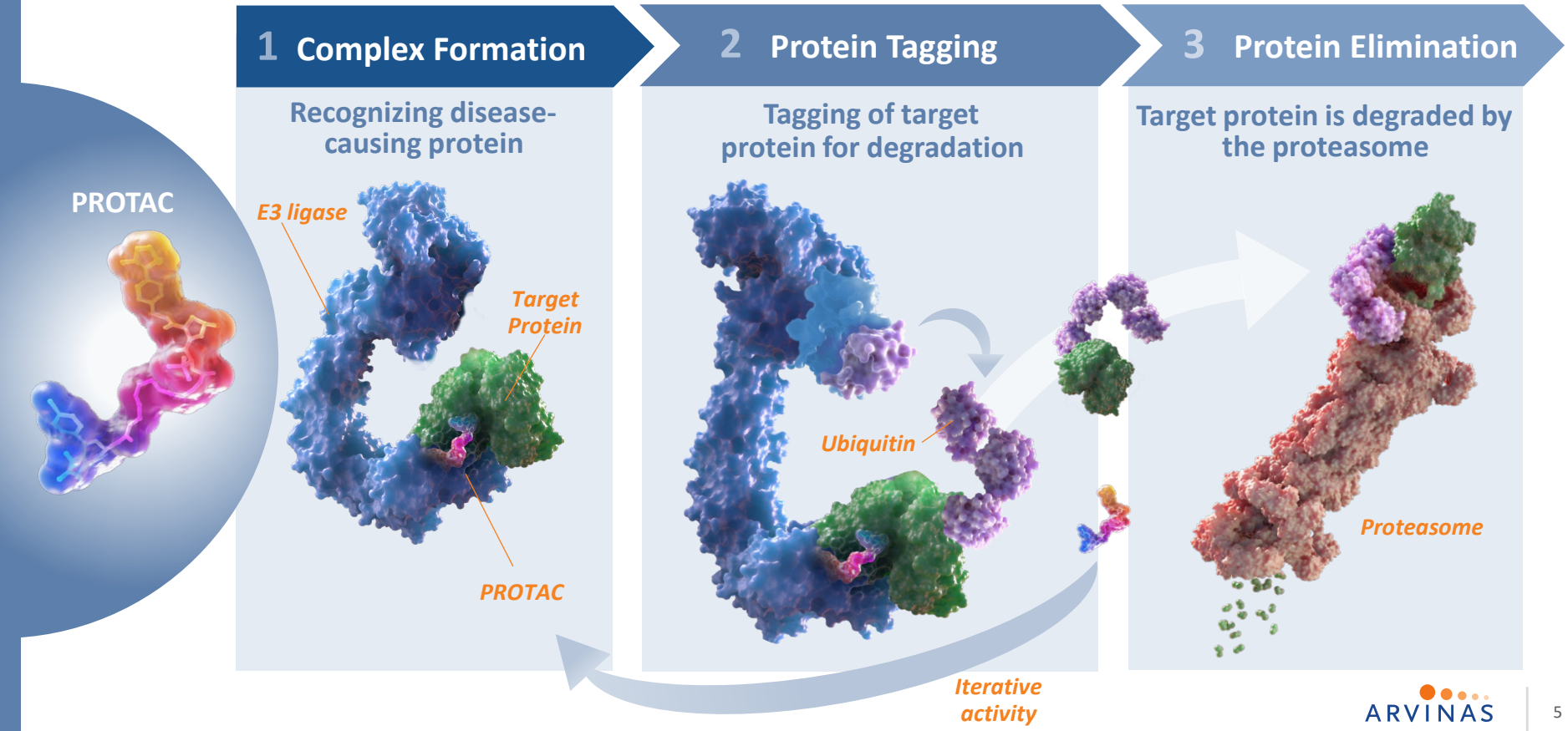
2020
Bavdegalutamide Phase 2 initiated

2021
ARV-471 Phase 2 initiated

JULY 2021
Partnered with Pfizer to co-develop and co-commercialize ARV-471

BBB, blood-brain barrier

PROTAC® protein degraders harness the ubiquitin-proteasome system to induce the degradation of disease-causing proteins

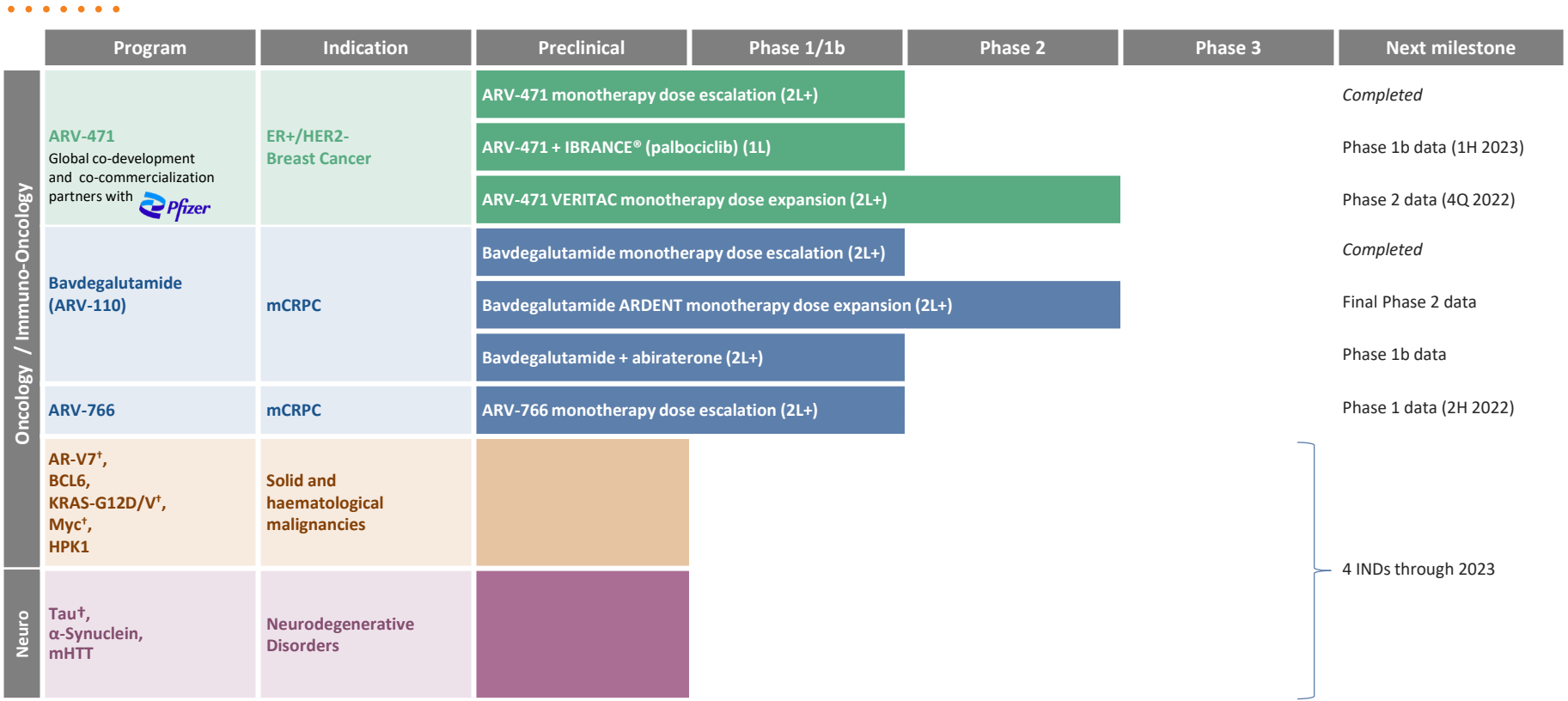


ARVINAS

PROTAC® protein degraders combine the benefits of small molecules and gene-based knockdown technologies

PROTAC protein degraders have distinct advantages over both small molecule inhibitors and gene-based medicines	PROTAC Protein Degraders	Small Molecule Inhibitors	Gene-Based Medicines
Eliminate disease-causing proteins	✓		✓
Disrupt scaffolding function	✓		✓
Potential to treat “undruggable” proteins	✓		✓
Iterative mechanism of action	✓		
Broad tissue penetration	✓	✓	
Oral dosing	✓	✓	
Ease of manufacturing	✓	✓	

Strong pipeline with multiple compounds nearing pivotal trials



Note: Pipeline is non-exhaustive

† Denotes historically undruggable proteins; †† Trial may potentially be part of a planned umbrella study with Pfizer to explore multiple combination agents

mCRPC, metastatic castration-resistant prostate cancer; ER+/HER2-, estrogen receptor+/human epidermal growth factor receptor 2-; IND, investigational new drug

ARVINAS

Our strategic collaboration with Pfizer accelerates global development and commercialization of ARV-471

Collaboration Summary

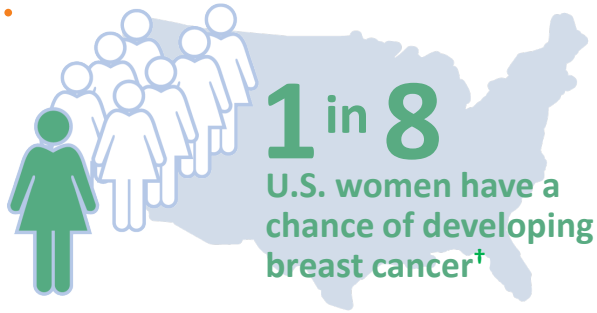
Upfront Payment & Equity Investment	$1B†
Development Expenses & Commercial Costs	50% Arvinas / 50% Pfizer
Approval & Commercial Milestones	Up to $1.4B
Profit Share	50% Arvinas / 50% Pfizer Worldwide

Broad Impact to Arvinas

- Accelerates and broadens global development and commercialization of ARV-471
- Leveraging of Pfizer's breadth of expertise and experience successfully driving trials to approval
- Provides access to Pfizer's global clinical, regulatory, medical, patient advocacy, and commercial footprint
- Accelerates Arvinas' strategy to build a fully integrated biotech
- Shares development costs and risks while progressing ARV-471 as part of Arvinas' pipeline
- Further enables the advancement of our deep pipeline in oncology, I-O, and neuroscience

† $650 million in cash and $350 million equity investment at $101.22 per share

ARV-471: First-in-class ER-degrading PROTAC in advanced breast cancer



Resistance is the greatest challenge to current therapies

In 2021, there will be an estimated

192,134 new cases

of ER+/HER2- breast cancer in the U.S.††

The unmet need in ER+/HER2- breast cancer represents a

>$15b market opportunity†††

ARV-471

An investigational oral PROTAC® protein degrader for the treatment of ER+ metastatic breast cancer

- **The injectable SERD fulvestrant established the importance of ER degradation for delivering benefit to patients with advanced breast cancer**
- **ARV-471 has the potential to degrade ER better than fulvestrant and become an oral, best-in-class ER-directed therapy**

† ACS: https://www.cancer.org/cancer/breast-cancer/about/how-common-is-breast-cancer.html; accessed 2/22/22; †† US SEER database; †††Arvinas estimate. SERD, selective estrogen receptor degrader.

Pfizer ARVINAS

ARV-471 shows signals of efficacy in a heavily pretreated patient population

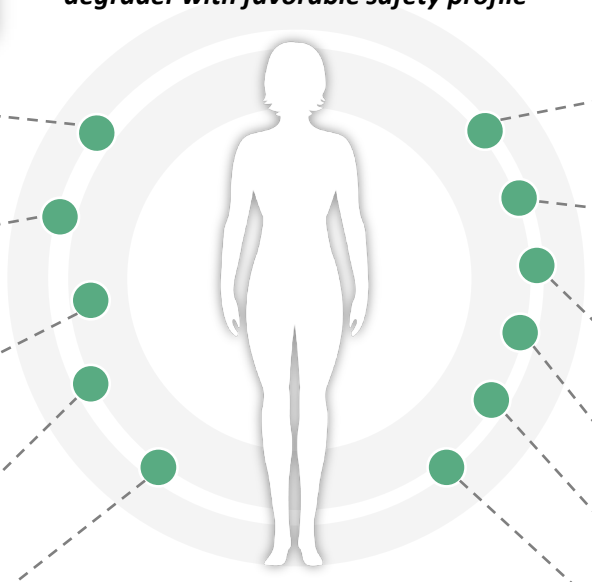
Data as presented at SABCS 2021

Heavily Pretreated Patient Population

4	Median number of previous therapies
100%	Patients were treated with prior CDK4/6 inhibitors
78%	Patients treated with prior chemo (55% in the metastatic setting)
80%	Patients were previously treated with fulvestrant
66%	Patients **expected** to have ER-independent disease[1]

ARV-471

Designed to be an oral, high-potency ER degrader with favorable safety profile



…with potential to become an endocrine backbone of choice for ER+/HER2- breast cancer treatment

Clinical Benefit and Safety Profile in Phase 1

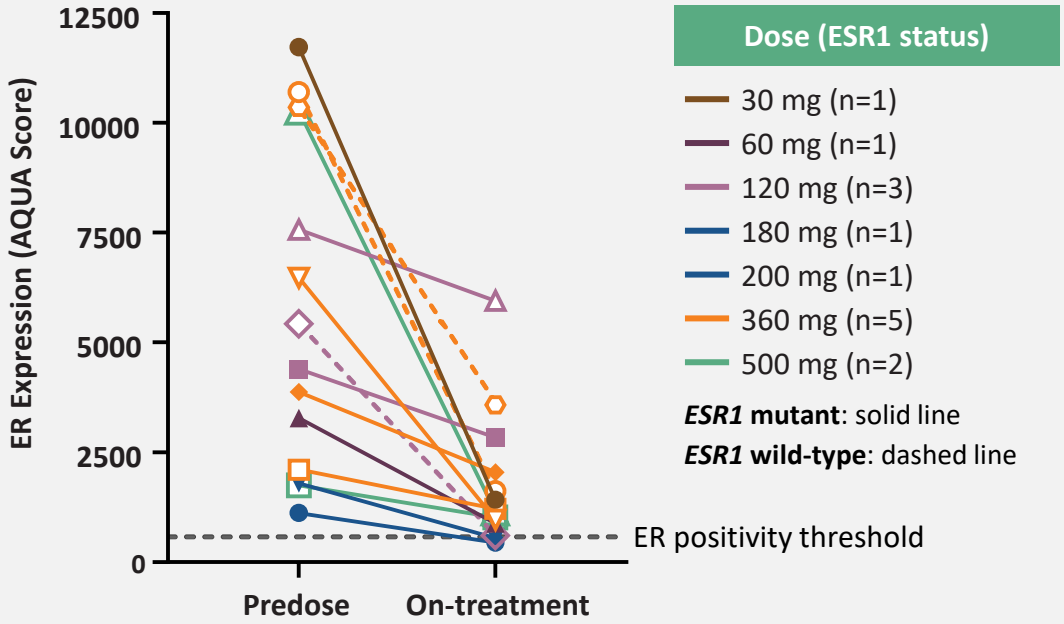
40%	Clinical benefit rate (CBR)[2] in 47 evaluable patients[3]
3	Confirmed partial responses (PR); tumor shrinkage in other patients
89%	Maximum ER degradation (mean 64%, median 67%)
0	DLTs in 60 patients
1 of 60	Patients required dose reduction
1 of 60	Patients discontinued due to AE

[1] Patient population expected to have highly ER-independent disease due to pretreatment with CDK4/6 inhibitors (Wander 2020).
[2] CBR is defined as the percentage of patients achieving a confirmed CR, PR, or SD ≥ 24 weeks.
[3] Excludes patients unable to complete cycle 1 due to reasons other than PD, toxicity, or death.
AE, adverse event; CBR, clinical benefit rate; DLT, dose limiting toxicity; TRAE, treatment related adverse event

ARV-471 degraded ER up to 89% through the 500 mg dose level

Data as presented at SABCS 2021

ER Expression in Paired Tumor Biopsies†

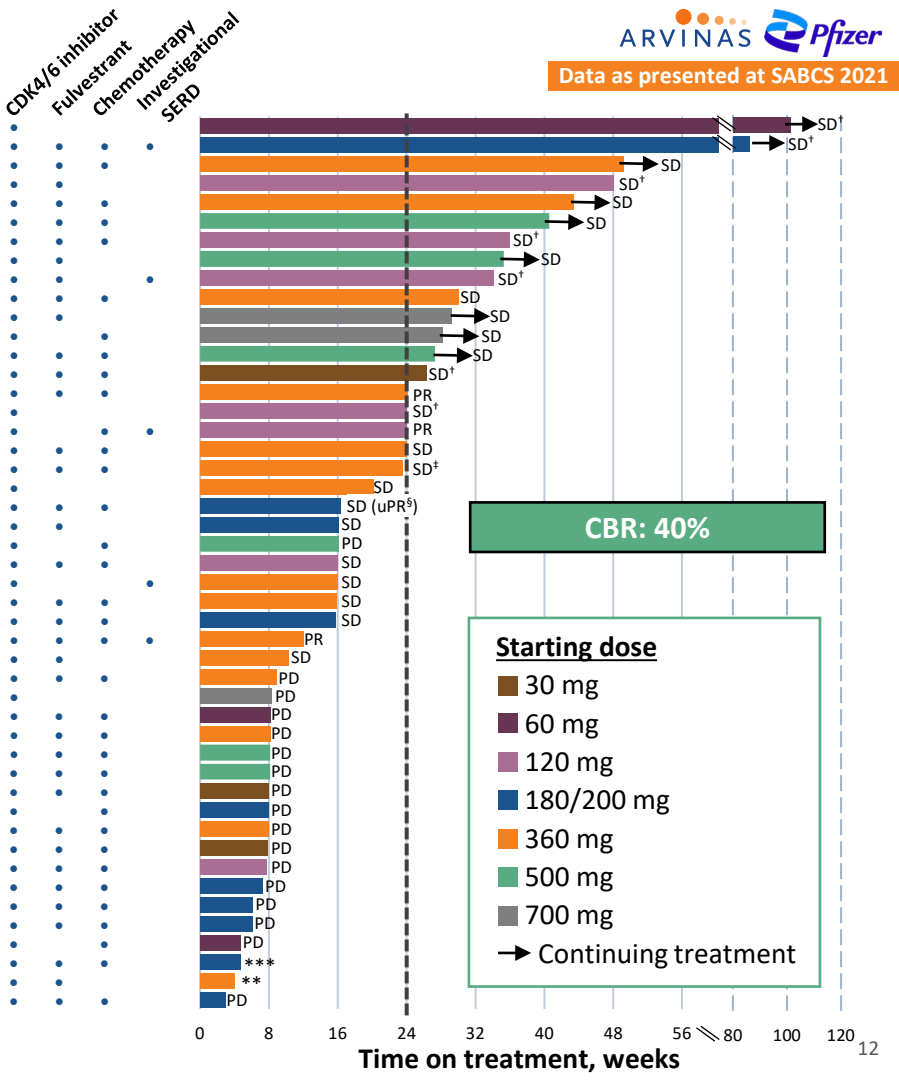


- Degradation up to **89%**; median **67%**; mean of **64%**
- Degradation **exceeds** reported data for **fulvestrant** (previously reported: 40-50%)††
- Degradation of **wild type ER and ESR1** mutant proteins

† Data available as of September 3, 2021; median time on treatment at biopsy: 31 days (range: 16–77). ER immunoreactivity analyzed by QIF using the AQUA method, and ER positivity threshold derived by examining AQUA scores and visually inspecting all samples in the dataset to determine a cut-point for ER positivity
†† Fulvestrant degradation reported as 40-50% in Robertson et al., Breast Cancer Research (2013) and Kuter et al., Breast Cancer Res Treat (2012).
AQUA, automated quantitative analysis; ER, estrogen receptor; QIF, quantitative immunofluorescence

ARV-471 achieved a high CBR (40%) in a heavily pretreated population

- **40% clinical benefit rate** (CBR) in 47 evaluable patients*
  - CBR = rate of confirmed CR or PR or SD ≥24 weeks
- **3** patients had **confirmed PRs**
- 14 out of 60 patients were ongoing at the time of data cutoff, including **2 who have been on treatment for >18 months**

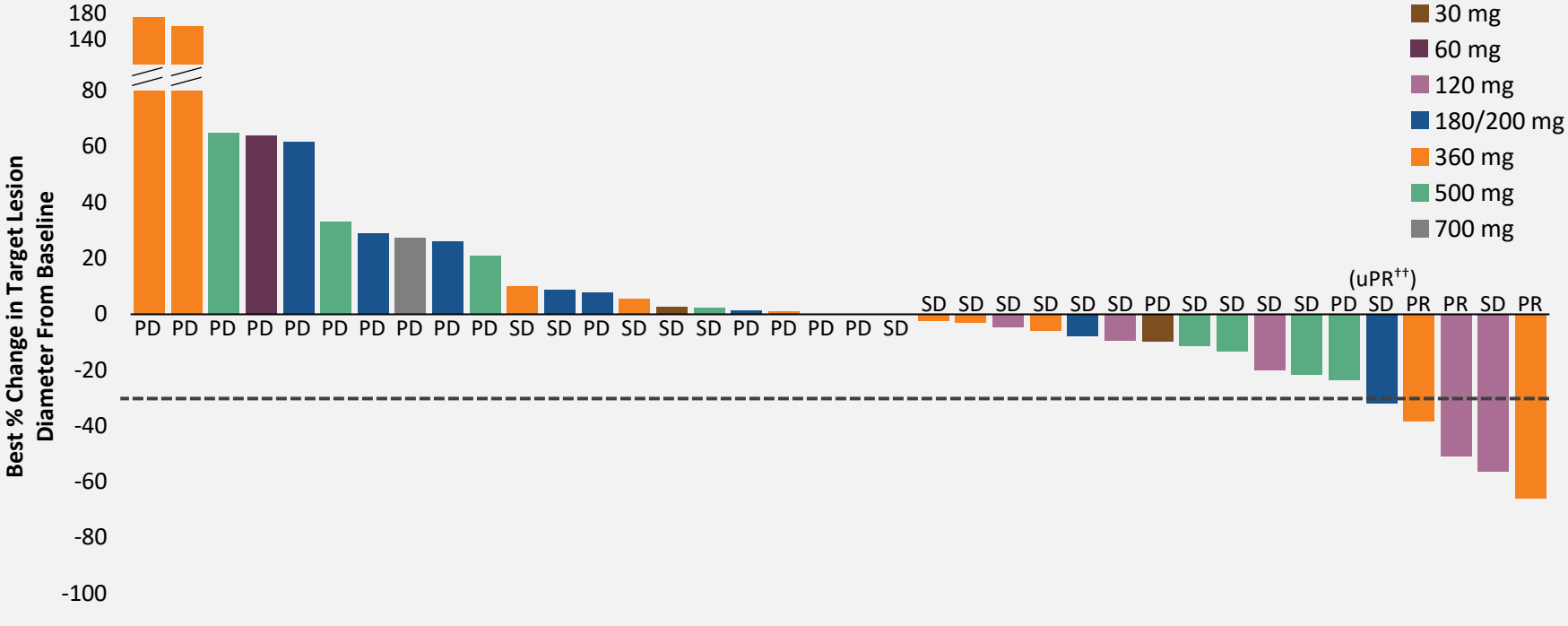


*Excludes patients unable to complete cycle 1 due to reasons other than PD, toxicity, or death
**Patient discontinued treatment due to venous embolism before first on-study scan
***Patient discontinued treatment due to clinical progression before first on-study scan
†Patient had dose escalation from starting dose
‡Week 24 imaging assessment performed at 23.4 weeks (within the window allowed per protocol)
§Patient had disease progression on subsequent scan and discontinued treatment
CBR=clinical benefit rate; CDK=cyclin-dependent kinase; PD=progressive disease; PR=confirmed partial response; SD=stable disease; SERD=selective estrogen receptor degrader; uPR=unconfirmed partial response

ARV-471 demonstrates promising anti-tumor activity in late-line patients

Data as presented at SABCS 2021

Antitumor activity in response-evaluable patients (n=38)†



† Patients with measurable disease at baseline who had a baseline and ≥1 on-treatment scan
†† Patient had disease progression on subsequent scan and discontinued treatment
PD, progressive disease; PR, confirmed partial response; SD, stable disease; uPR, unconfirmed partial response

ARV-471 was well tolerated at all dose levels; MTD not reached

Data as presented at SABCS 2021

TRAE in ≥ 10% of patients	30 mg (n=3)		60 mg (n=3)		120 mg (n=7)		180/200 mg (n=11)		360 mg (n=15)		500 mg (n=17)		700 mg (n=4)		Total (N=60)	
	Gr 1/2	Gr 3	Gr 1/2	Gr 3	Gr 1/2	Gr 3	Gr 1/2	Gr 3	Gr 1/2	Gr 3	Gr 1/2	Gr 3	Gr 1/2	Gr 3	Gr 1/2	Gr 3
Any TRAE	0	0	3 (50%)	0	6 (86%)	0	6 (55%)	1 (9%)	10 (67%)	1 (7%)	7 (41%)	2 (12%)	2 (50%)	0	34 (57%)	4 (7%)
Nausea	0	0	2 (33%)	0	2 (29%)	0	4 (36%)	0	3 (20%)	0	4 (24%)	1 (6%)	1 (25%)	0	16 (27%)	1 (2%)
Fatigue	0	0	1 (17%)	0	0	0	1 (9%)	0	3 (20%)	0	5 (29%)	0	2 (50%)	0	12 (20%)	0
Vomiting	0	0	0	0	2 (29%)	0	1 (9%)	0	2 (13%)	0	1 (6%)	0	0	0	6 (10%)	0
AST increased	0	0	0	0	1 (14%)	0	2 (18%)	0	0	0	1 (6%)	0	2 (50%)	0	6 (10%)	0

Discontinuation rate <2% (1 out of 60)
Dose reductions <2% (1 out of 60)

Four patients experienced Gr 3 events potentially related to ARV-471 (headache lasting 1-day, single occurrence of asymptomatic increased amylase and lipase, nausea and asymptomatic QTc prolongation, and venous embolism after a minor procedure†)

Data cut-off: 09/30/21

† Advanced breast cancer is highly associated with venous embolisms. Event was included as potentially treatment related, so treatment with ARV-471 was stopped.

MTD, maximum tolerated dose; TRAE, Treatment related adverse event

ARV-471: Moving forward rapidly across the continuum of disease

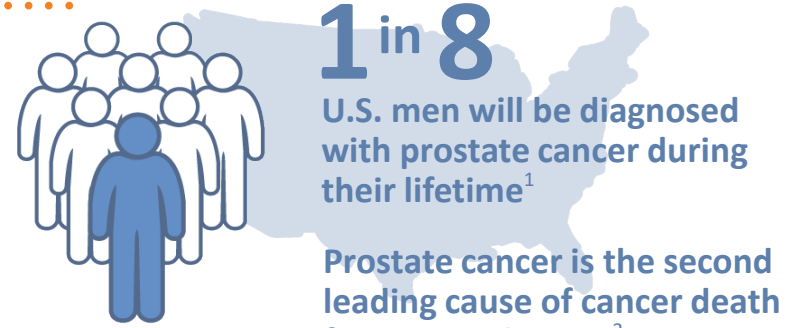
US ER+/HER2- Breast Cancer Treatment Paradigm (~200,000 US patients†)

	Adjuvant (Post-Surgical) Breast Cancer (~160K)	Metastatic Breast Cancer (~50K)		
		First Line	First Line	Second/Third Line
Supportive Trials to Define Registration Paths	Initiate in 2H 2022 **Neoadjuvant** *(Randomized vs Control)* ARV-471, or ARV-471 + CDK4/6i	Initiated Dec 2020 **Phase 1b (enabling trial):** Combo: ARV-471 + IBRANCE® (palbociclib)	Initiated 1Q21 **Phase 2:** VERITAC Expansion: ARV-471	Initiate in 2H 2022 **Phase 1b** Combo: ARV-471 + CDKi or other Targeted Therapies††
Pivotal Trials		Initiate in 2H 2022 **Phase 3 registrational:** Two Phase 3 trials: ARV-471 as monotherapy and combo		

† SEER database; includes US patient population only, †† E.g., everolimus or as part of umbrella study with multiple combination agents
CDK, cyclin-dependent kinases Pi3Ki; phosphoinositide 3-kinase inhibitor; mTORi: mammalian target of rapamycin inhibitors

Pfizer ARVINAS

Bavdegalutamide: AR-degrading PROTAC in metastatic prostate cancer



1 in 8

U.S. men will be diagnosed with prostate cancer during their lifetime[1]

Prostate cancer is the second leading cause of cancer death for men in the U.S.[2]

In 2021 alone, there were an estimated

248,530 new cases

of prostate cancer[3]

34,130 deaths are attributed to the disease[3]

High unmet need in prostate cancer treatment represents

$8b market in the US alone[4]

Bavdegalutamide (ARV-110)

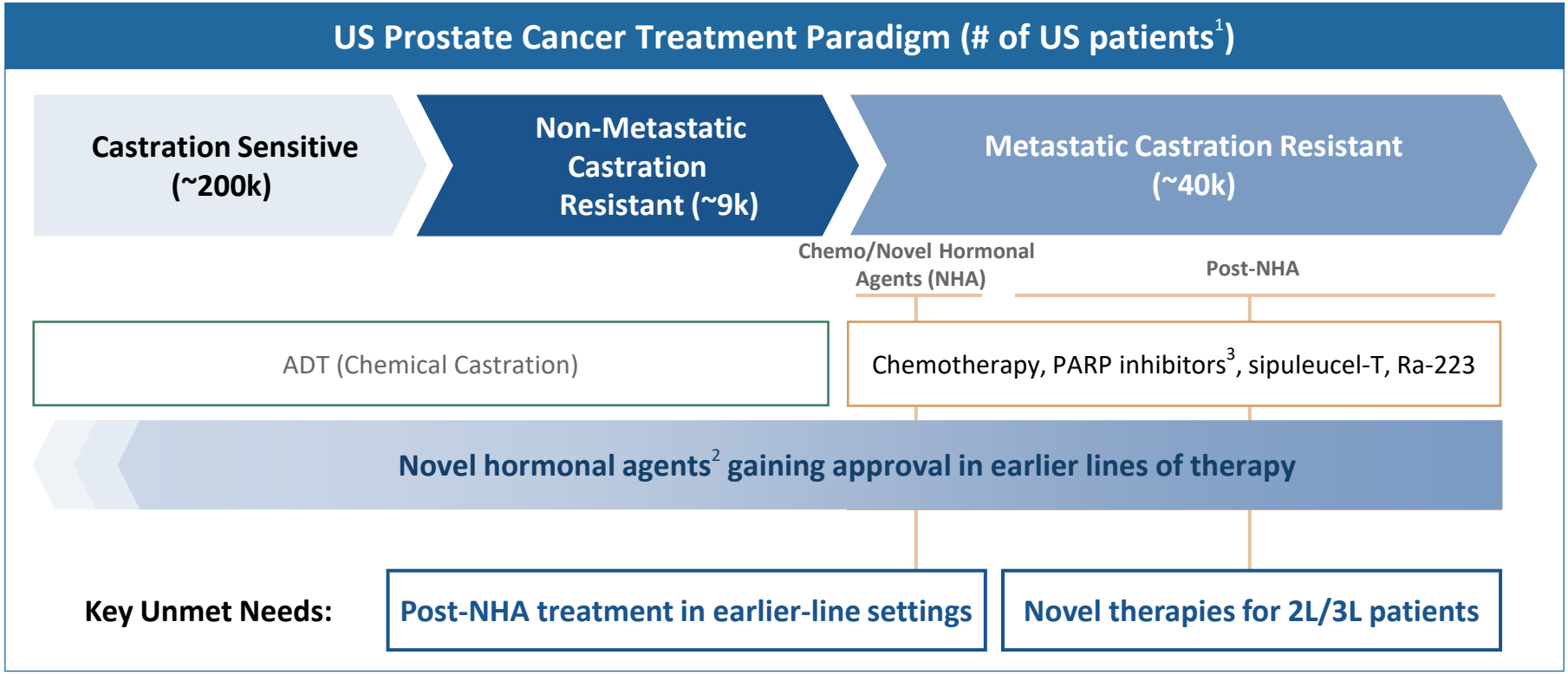
An investigational oral PROTAC® protein degrader that targets the androgen receptor (AR) for the treatment of prostate cancer

- **AR is a critical target in prostate cancer therapy**
- **Tumors develop resistance to standard-of-care AR inhibitors**
- **Bavdegalutamide may overcome point mutations and other drivers of resistance**
- **Activity in late-line settings suggests potential for even stronger benefit in earlier-line, less-pretreated patients**

[1] ACS: https://www.cancer.org/cancer/prostate-cancer/about/key-statistics.html, accessed 2/22/22; [2] American Cancer Society; [3] US SEER database; [4] Arvinas estimate.

ARVINAS

Migration of novel hormonal agents to earlier settings has created substantial unmet need for new treatments in mCRPC



[1] SEER database, [2] Includes enzalutamide, abiraterone, darolutamide, apalutamide, [3] Approved for patients with BRCA mutation or homologous recombination repair (HRR) gene-mutated mCRPC that has progressed after AR-directed therapies. ADT=androgen deprivation therapy; mCRPC=metastatic castration resistant prostate cancer; NHA=novel hormonal agent; PARP=poly (ADP-ribose) polymerase; 2L=second-line; 3L=third-line

ARVINAS

Bavdegalutamide has shown robust signals of clinical activity in heavily pretreated patients with mCRPC who received 1–2 prior novel hormonal agents

Data as presented at ASCO GU 2022

Heavily pretreated patient population in Ph 1 and Ph 2†

Ph 1: 5 Ph 2: 4	Median number of previous therapies
Ph 1: 82% Ph 2: 39%	Patients were treated with both abiraterone and enzalutamide
Ph 1: 76% Ph 2: 31%	Patients were treated with prior chemotherapy

Evidence of clinical benefit and manageable tolerability profile in Ph 1 and Ph 2 patients with AR T878X/H875Y-mutant tumors‡

46%	PSA_{50} response rate in patients with AR T878X/H875Y-positive tumors
2 of 7	RECIST-evaluable patients had durable confirmed partial responses
43%	Patients remained on treatment for 24 weeks or more
0%	Gr ≥4 TRAEs; low rates of discontinuation and dose reduction

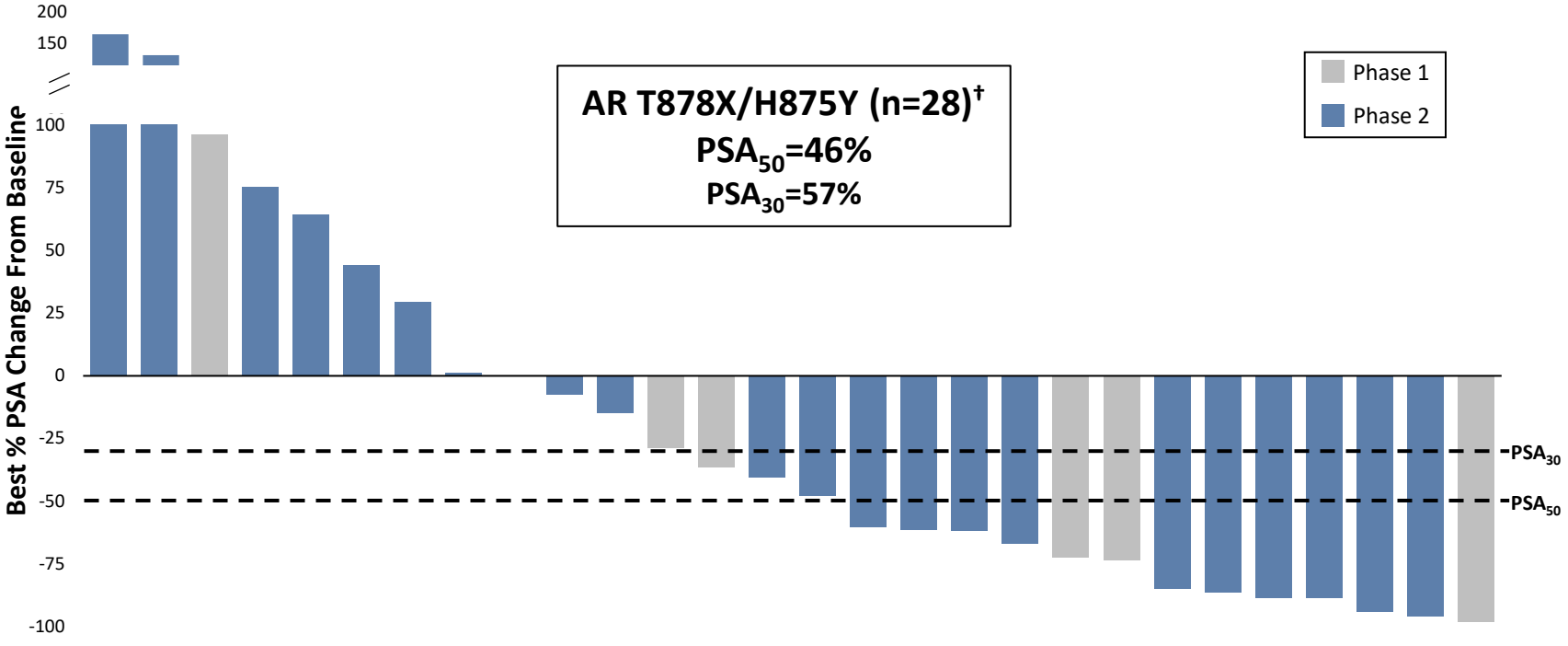
† Phase 2 study enrollment ongoing (December 20,2021 data cutoff date); ‡ All patients with AR T878X/H875Y tumor mutations in Phase 1 and 2 studies; T878X = T878A or T878S; AR, androgen receptor; mCRPC, metastatic castration resistant prostate cancer; TRAE, treatment-related adverse event

ARVINAS

46% PSA_{50} in all patients with AR T878X/H875Y tumor mutations in Ph1 and Ph2 expansion (ARDENT) subgroups supports advancing to pivotal trials in molecularly defined tumors

Data as presented at ASCO GU 2022

Best PSA Change in all patients with AR T878X/H875Y tumor mutations in Phase 1 and Phase 2



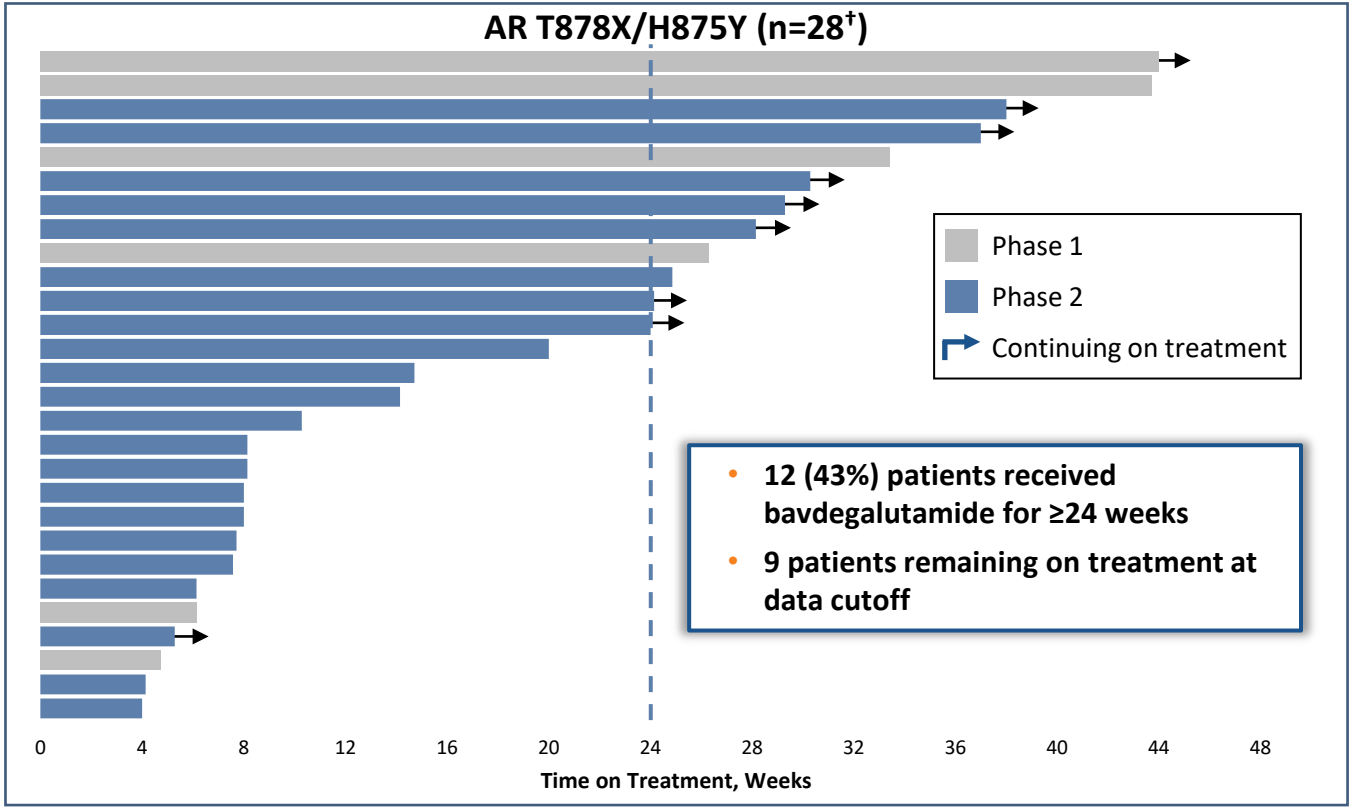
† Includes biomarker-evaluable patients treated at or above the RP2D (phase 1 and 2) or with exposure above the minimum efficacious threshold in nonclinical xenograft tumor models (phase 1) and with ≥4 weeks of PSA follow-up

AR, androgen receptor; PSA, prostate-specific antigen; PSA30, best PSA declines ≥30%; PSA50, best PSA declines ≥50%; T878X, T878A or T878S

ARVINAS

Bavdegalutamide showed robust duration of treatment in Ph 1 and Ph 2 expansion (ARDENT) trial patients with AR T878X/H875Y mutant tumors

Data as presented at ASCO GU 2022



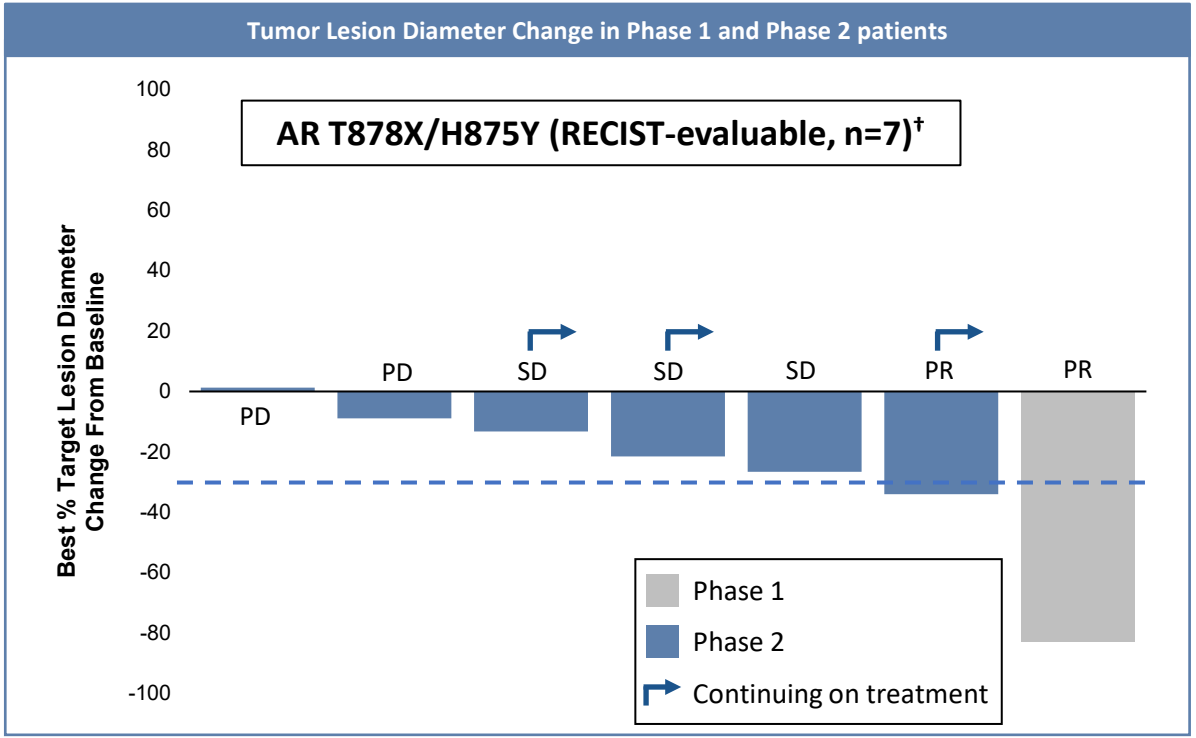
† Includes biomarker-evaluable patients treated at or above the recommended phase 2 dose (phase 1 and 2) or with exposure above the minimum efficacious threshold in nonclinical xenograft tumor models (phase 1); Phase 2 enrollment ongoing (December 20, 2021 data cutoff date)

AR=androgen receptor; T878X=T878A or T878S

ARVINAS

Durable partial responses in 2 of 7 RECIST-evaluable patients with AR T878X/H875Y mutant tumors

Data as presented at ASCO GU 2022



- Activity was durable; patients with confirmed partial responses (PR) remained on treatment for approximately 9 (ongoing) and 10 months
- 6 of 7 patients had tumor reductions

† Includes biomarker-evaluable patients treated at or above the recommended phase 2 dose (phase 1 and 2) or with exposure above the minimum efficacious threshold in nonclinical xenograft tumor models (phase 1); Includes patients with measurable disease at baseline and ≥1 on-treatment scan; patients with SD as best response and <12 weeks follow-up were excluded

PD, progressive disease; PR, confirmed partial response; RECIST, Response Evaluation Criteria in Solid Tumors; SD, stable disease; T878X=T878A or T878S

Profile of bavdegalutamide potentially supports a clear precision medicine opportunity in mCRPC

Near-term, precision opportunity in T878/H875-positive mCRPC

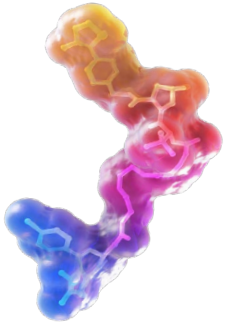
- Initiate global Phase 3 trial in 2H 2023, with opportunity for simultaneous global regulatory submissions
- Unmet need and potential opportunity expected to increase as NHAs move earlier

Additional opportunities to explore in a broader patient population

- Monotherapy or in combination (e.g., abiraterone)
- Pre-NHA (e.g., HSPC)

AR, androgen receptor; FDA, Food and Drug Administration; CSPC, castration-sensitive prostate cancer; mCRPC. metastatic castration resistant prostate cancer; NHA, novel hormonal agents

Drugging an undruggable: KRAS

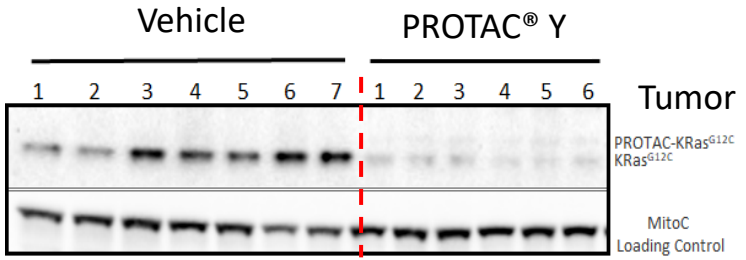


KRAS is the most frequently mutated gene in human cancer and is a classic "undruggable" target due to its lack of deep "pockets"

KRAS is associated with poor prognosis and resistance to standards of care in several tumor types

As a proof of concept, we have successfully developed in vivo active KRAS G12C-specific PROTAC® degraders

Six hours after a single dose, PROTAC Y degraded >80% of KRAS G12C in vivo†

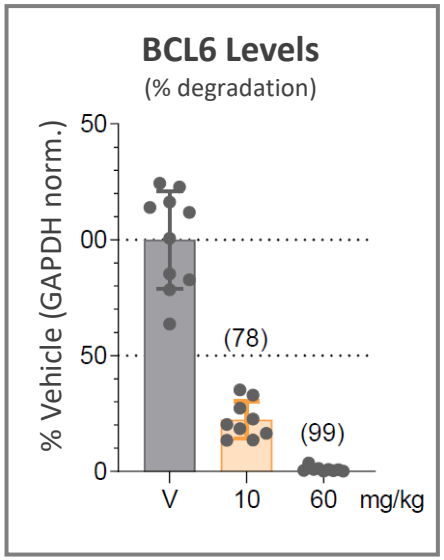


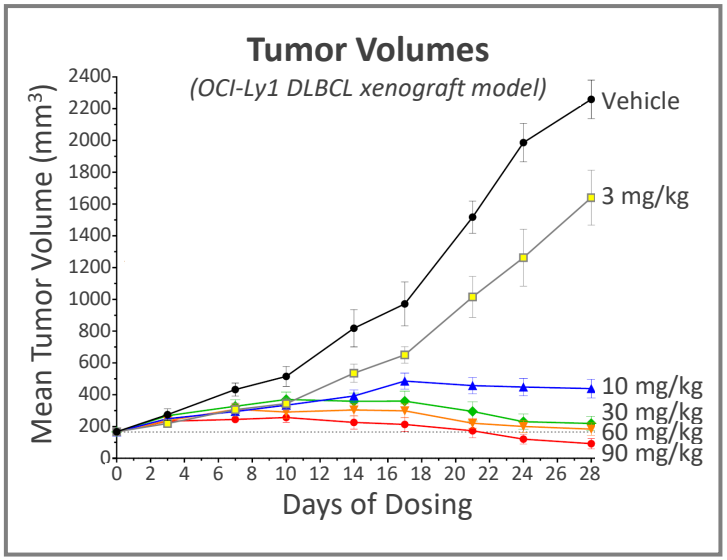
Leveraging learnings from KRAS G12C development to accelerate other KRAS degraders' development

† MiaPaCa-2 xenograft mode

Targeting scaffolding proteins: Arvinas' BCL6 PROTAC degrader demonstrates robust efficacy in pre-clinical models of DLBCL

Orally administered BCL6 PROTAC degrader shows dose-responsive degradation and tumor regressions in vivo





- BCL6 is a transcriptional repressor that is a driver of B-cell malignancies and a therapeutic target in DLBCL
- We have developed specific, potent, and orally bioavailable BCL6 PROTAC degraders that are efficacious in multiple pre-clinical DLBCL models

DLBCL, diffuse large B-cell lymphoma

Neuroscience: High potential in an area of tremendous unmet need

Each year, >6 million patients in the U.S. are diagnosed with Alzheimer's, Parkinson's, and Huntington's diseases alone†

Opportunity for PROTAC® Degraders:

- **Very few disease-modifying therapies exist**
- **Blood-brain barrier penetration is a challenge for other modalities**
- **Traditional therapies have difficult routes of administration, e.g., intra-thecal**

Arvinas Neuroscience Pipeline

PROTAC degraders could revolutionize the treatment of neuroscience diseases

- Cross the blood brain barrier and degrade disease-causing proteins inside cells
- Target pathogenic proteins in the brain without impacting healthy proteins
- Potential for oral therapies

† Globaldata, DecisionResources.

mHTT, mutant Huntingtin protein; MSA, multiple systems atrophy; PSP, progressive supranuclear palsy

Strategic, target-based partnerships expand the impact of our PROTAC® Discovery Engine

ARVINAS

Genentech
A Member of the Roche Group

September 2015
(expanded in November 2017)
Target discovery deal

Pfizer

December 2017
Target discovery deal

Orthbio™ BAYER

June 2019
Target discovery deal and agriculture-focused joint-venture to fight crop disease and other challenges facing the global food supply

Partnerships expand PROTAC® degraders beyond oncology and beyond human therapeutics

Rapid pace of upcoming milestones

Program	Anticipated Milestones in 2022/2023
ARV-471 (ER PROTAC®)	• Present data from the VERITAC Phase 2 expansion trial (200 mg and 500 mg) (4Q 2022) • Initiate two Phase 3 trials in patients with metastatic breast cancer (as monotherapy and in combination) (2H 2022) • Initiate a Phase 1b combination trial with CDK inhibitors or other targeted therapies (2H 2022) • Initiate a Phase 1b combination trial with everolimus (2H 2022) • Initiate a Phase 2 neoadjuvant trial in patients with early breast cancer (2H 2022) • Present safety data from the Phase 1b combination trial with palbociclib (1H 2023)
Bavdegalutamide (AR PROTAC®)	• Initiate randomized Phase 3 trial in mCRPC for patients with AR T878/H875 tumor mutations (2H 2023)
ARV-766 (AR PROTAC®)	• Share Phase 1 dose escalation data in mCRPC (2H 2022) • Initiate Phase 2 expansion trial in mCRPC (2H 2022)
INDs	• Four INDs through the end of 2023

For more information

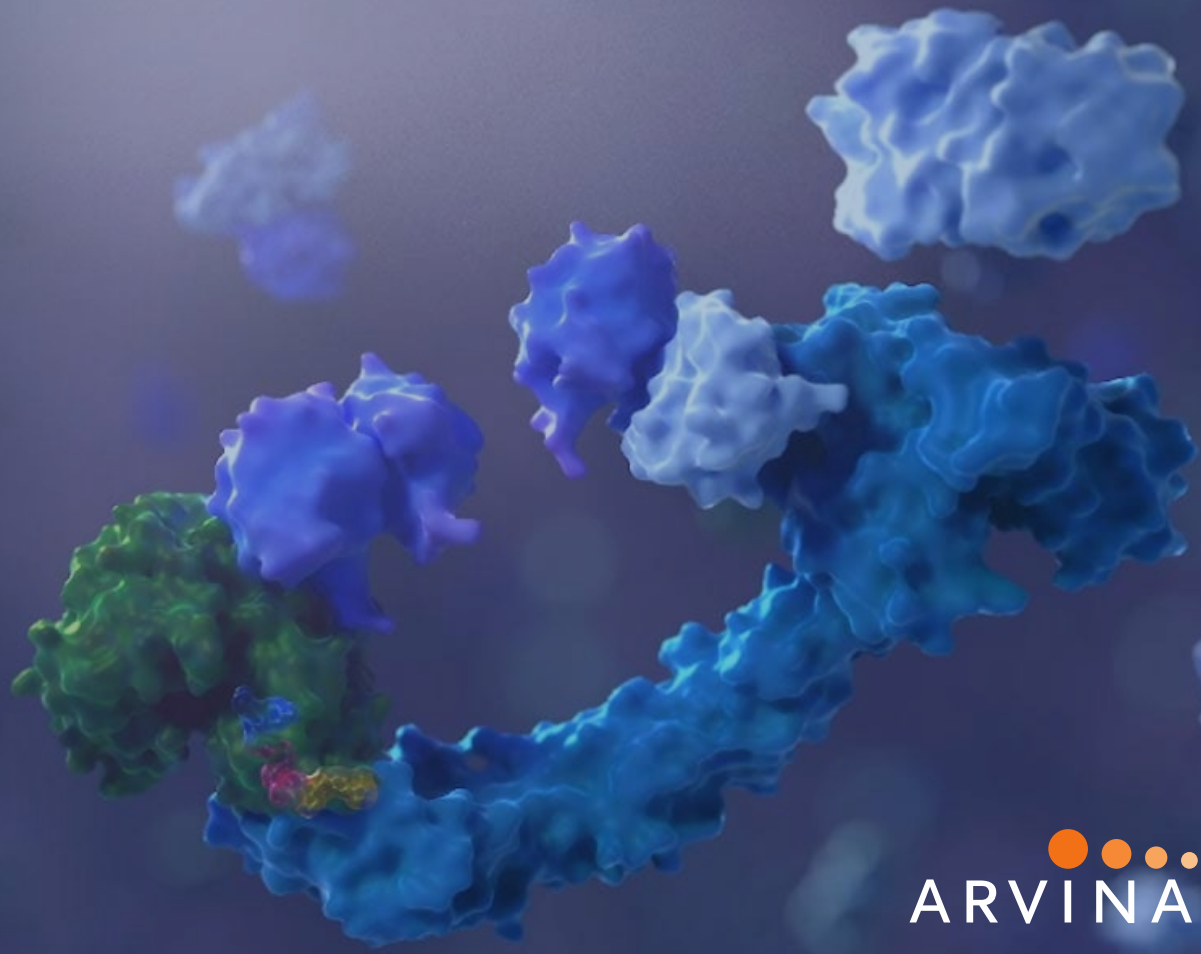
PRESS/MEDIA
pr@arvinas.com

INVESTORS
ir@arvinas.com

BUSINESS DEVELOPMENT
bd@arvinas.com

CAREERS
careers@arvinas.com

Appendix



ARVINAS

High-performing leadership team

Proven track record of driving success with 20+ novel drugs

John G. Houston, Ph.D.
President & CEO

Matthew Batters, J.D.
General Counsel

Jeff Boyle
VP Investor Relations

Angela Cacace, Ph.D.
SVP Neuro & Platform Biology

Sean Cassidy, M.B.A., C.P.A.
Chief Financial Officer

Debbie Chirnomas, M.D., M.P.H.
VP Oncology Clinical Research

John A. Grosso, Ph.D.
SVP R&D Technical Operations

Eric Mason, PharmD
VP, Early Clinical Development

John Northcott
Chief Commercial Officer

Achal Pashine, Ph.D.
VP, Oncology & Immuno-Oncology Research

Ron Peck, M.D.
Chief Medical Officer

Christina Sheil, M.D.
VP Pharmacovigilance

Lisa Sinclair
SVP Corporate Operations

Larry Snyder, Ph.D.
VP Medicinal Chemistry

Ian Taylor, Ph.D.
Chief Scientific Officer

Randy Teel, Ph.D.
SVP Corporate & Business Development

Kimberly Wehger
VP Information Technology

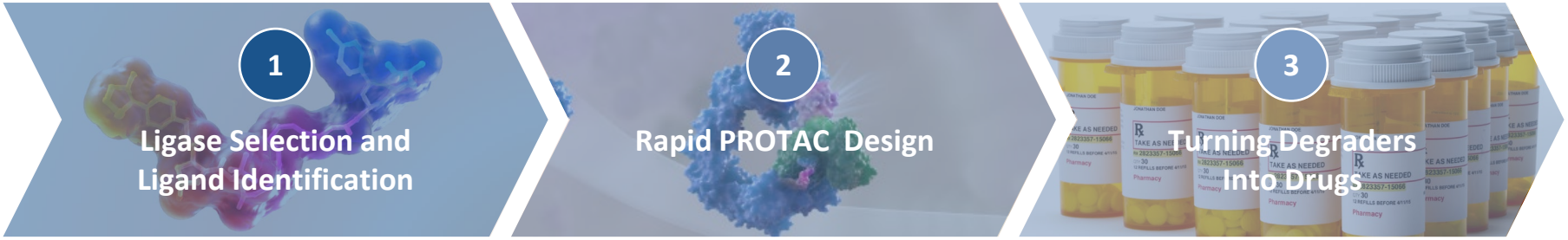
Steve Weiss
SVP Human Resources

OPDIVO (nivolumab)
Aliqopa (copanlisib) 60 mg vial for injection
XALKORI CRIZOTINIB
Stivarga (regorafenib) tablets
ULTOMIRIS (ravulizumab-cwvz) injection for intravenous use 300 mg/3 mL vial
Nexavar (sorafenib) tablets
ABILIFY (aripiprazole) 2, 5, 10, 15, 20, 30 mg Tablets
VIZIMPRO DACOMITINIB
Nurtec ODT (rimegepant) orally disintegrating tablets 75 mg
SPRYCEL dasatinib 100 mg tablets
IBRANCE palbociclib 125 mg tablets
YERVOY (ipilimumab)
farxiga (dapagliflozin) 5mg & 10mg tablets
IXEMPRA (ixabepilone) for injection, for intravenous infusion
SOLIRIS (eculizumab) Injection for Intravenous Use
Strensiq (asfotase alfa) for injection 40 mg/mL
Once-daily oral Zejula niraparib capsules 100 mg
Daklinza (daclatasvir) 60 mg tablets

Arvinas' breakthroughs are driven by our integrated PROTAC® Discovery Engine

Arvinas' platform is built from nearly 20 years of experience, know-how, and IP

PROTAC Discovery Engine



- E3 KnowledgeBase – matching the correct E3 ligase to correct target
- Leveraging AI and structural understanding of ligases to identify and design ligands
- Arvinas' DNA-encoded libraries for advanced screening
- Identification of new "warheads" for previously undruggable targets

- Zone of Ubiquitination – we design PROTAC degraders to predict the precise location where a protein can be tagged
- Predictive computational modeling
- State-of-the-art proteomics capabilities

- "Arvinas Rules" for drug-like properties, including blood-brain barrier penetration and oral bioavailability in humans
- Deep knowledge of molecular features allow us to create PROTAC degraders with drug-like properties and activities

AI, artificial intelligence

ARV-471 achieved confirmed RECIST Partial Response (cPR) in a patient with extensive prior therapy and an ESR1 mutation at 120 mg

Data as presented 12/14/2020

Extensive prior therapy

CDK4/6 inhibitor: Palbociclib

Endocrine therapies: 6 Agents
- Aromatase inhibitors x 3
- Tamoxifen
- Investigational SERDs X 2†

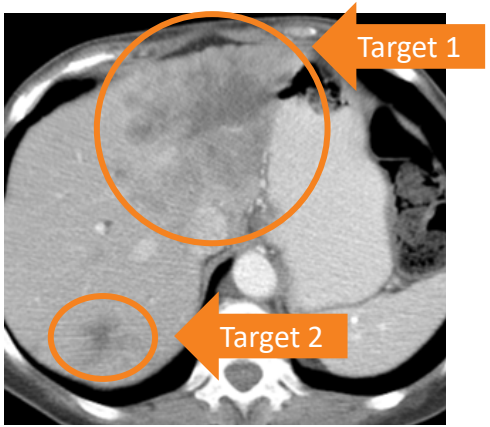
Other targeted agents: Everolimus

Chemotherapy: 2 Regimens
- 1 neoadjuvant + 1 metastatic

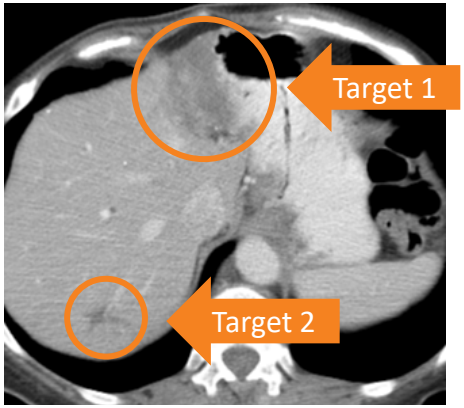
ESR1 mutations

D538G

Baseline CT Scan



After 4 Cycles



51% reduction in target lesions (RECIST partial response)

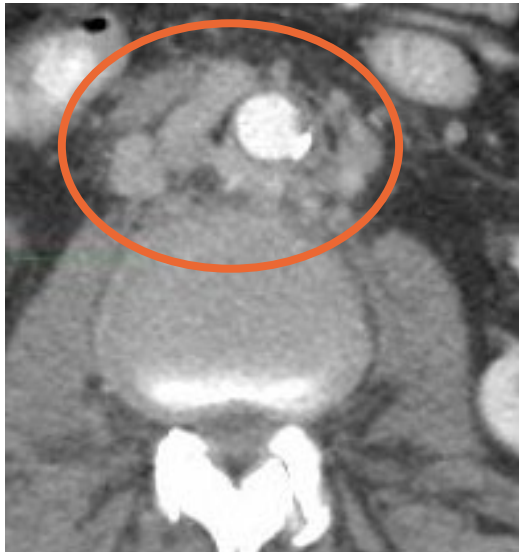
† Includes one selective ERα covalent antagonist.
CDK: cyclin-dependent kinases

Bavdegalutamide achieved RECIST confirmed response in a patient with extensive prior treatment

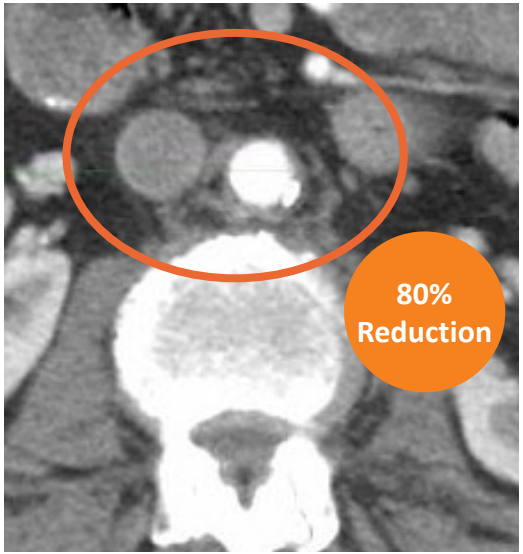
Data as presented at ASCO 2020 and as of 4/20/20

Patient Characteristics	
PSA response	**97%** decline
RECIST response	**80%** reduction
Duration of bavdegalutamide	18+ weeks ongoing
Biomarker status	AR H875Y and T878A mutations (associated with resistance to abiraterone or enzalutamide)†
Common prior therapies	Enzalutamide, Abiraterone, Bicalutamide
Other prior therapies	Provenge Cabazitaxel
History	Extensive disease involving adrenal gland, aortocaval nodes, multiple cone metastases

Baseline CT Scan
Extensive retroperitoneal adenopathy compressing the inferior vena cava



After 4 Cycles
Near complete regression of adenopathy



RECIST: Response evaluation criteria in solid tumors
† Jernberg E, Endocrine Connections, 2017