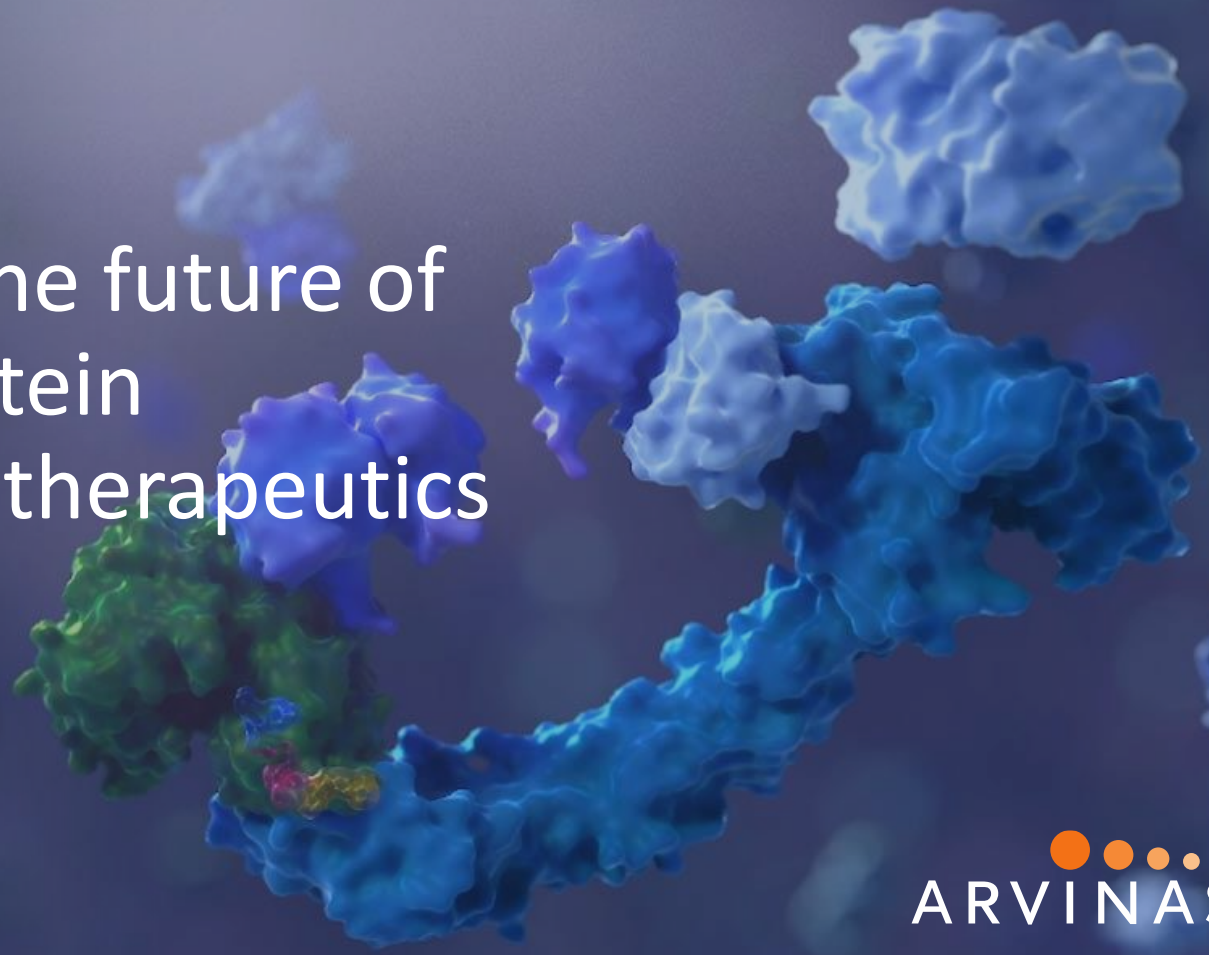
Pioneering the future of targeted protein degradation therapeutics



ARVINAS

June 2021

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 development and regulatory status of our product candidates, such as statements with respect to our lead product candidates, 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 and therapeutic potential of our product candidates, the potential commercialization of any of our product candidates, 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: whether we will be able to successfully conduct Phase 1/2 clinical trials for ARV-110 and ARV-471, initiate and complete other 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.

The Arvinas name and logo are our trademarks. We also own the service mark and the registered U.S. trademark for PROTAC®. 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.

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

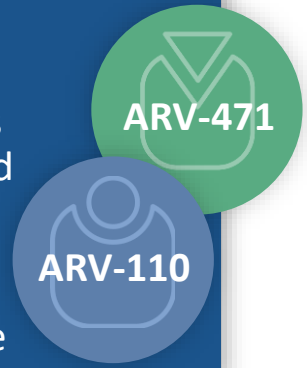
PROTEIN DEGRADATION

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

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



ARVINAS

200+ team members

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

FINANCIAL SNAPSHOT†

~$651 Million

Funded into 2024

† As of 3/31/21

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: ARV-110 and ARV-471

2020
Efficacy proof of concept for ARV-110 and ARV-471

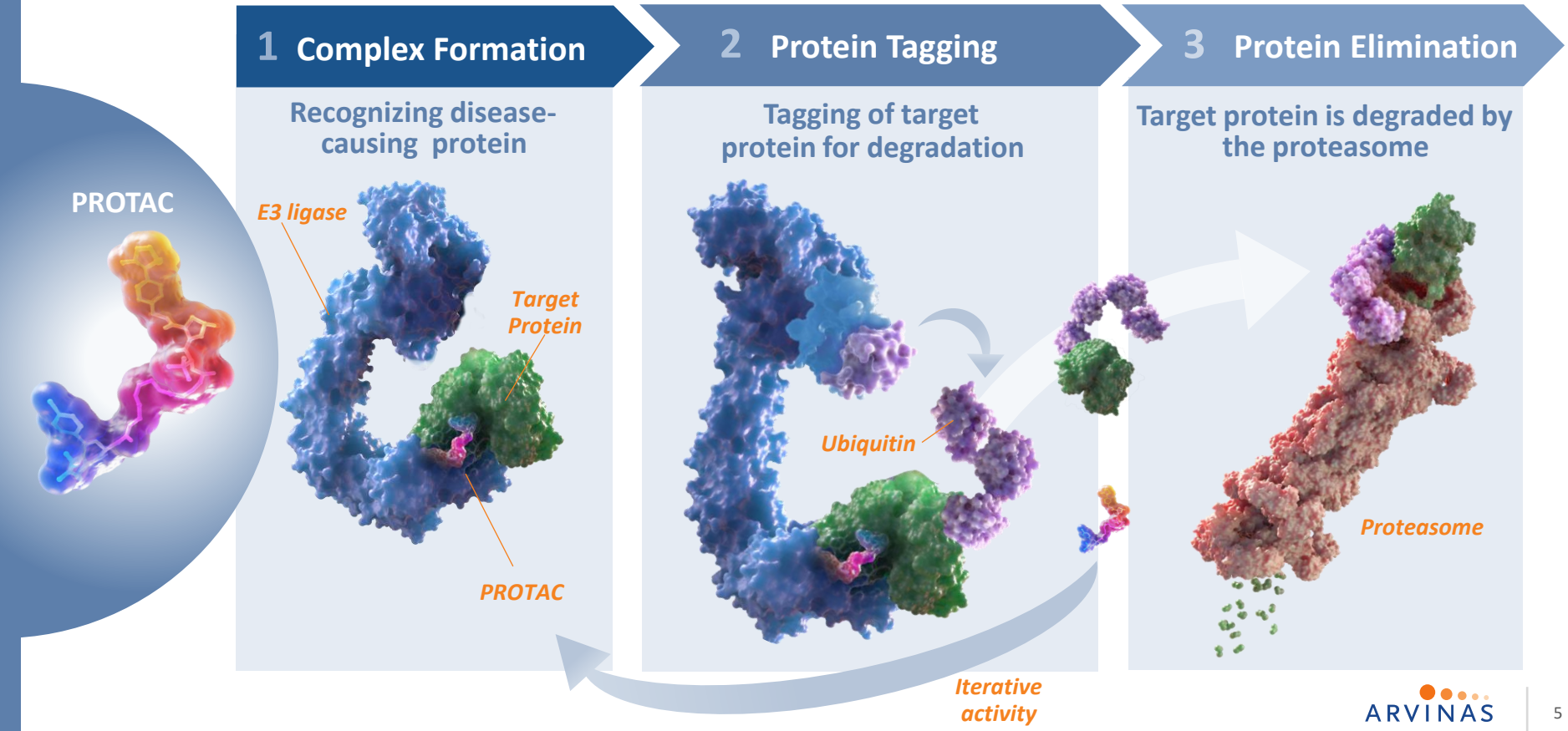
2020
ARV-110 Phase 2 initiated

2021
ARV-471 Phase 2 initiated

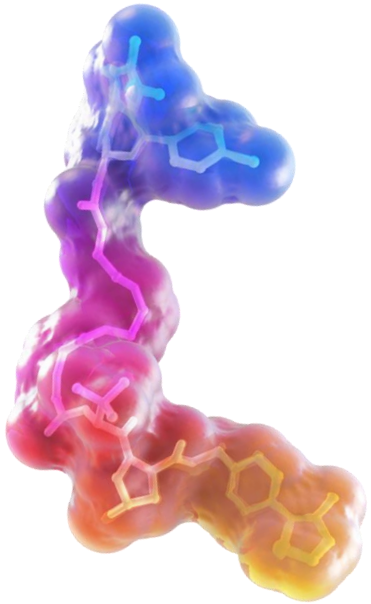
2021
Expected: Phase 2 data for ARV-110

BBB, blood-brain barrier

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



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	✓	✓	

Developing therapeutics for validated and “undruggable” targets

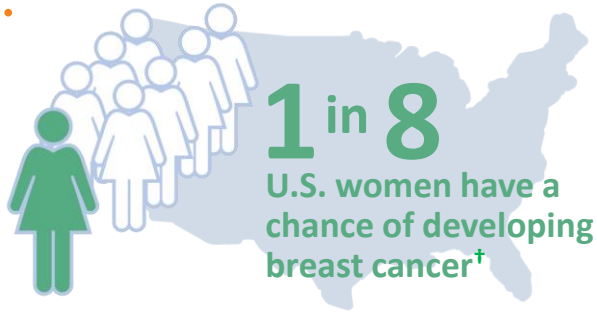
	ARVN Program	Indication	Exploratory	Research	IND Enabling	Phase 1	Phase 2	Phase 3
Oncology / Immuno-oncology	ARV-110	mCRPC						
	ARV-766	mCRPC		IND 2021				
	AR-V7†	mCRPC						
	ARV-471	ER+/HER2- Breast Cancer						
	BCL6	B-cell Malignancies		IND 2022				
	KRAS G12D/V†	NSCLC, CRC, Pancreatic		IND 2023				
	Undisclosed	Solid Malignancies		IND 2022				
	Myc†	Solid Malignancies						
	HPK1	Solid Malignancies						
Neuroscience	Tau†	FTLD-TAU, PSP, AD		IND 2022				
	Alpha Synuclein	MSA, Parkinson’s						
	mHTT	Huntington’s						
	Undisclosed	Neurodegeneration						

†Denotes historically undruggable proteins

Note: Pipeline is non-exhaustive and IND dates are anticipated.

mCRPC, metastatic castration-resistant prostate cancer; ER+/HER2-, estrogen receptor+/human epidermal growth factor receptor 2-; NSCLC, non-small-cell lung carcinoma; CRC, colorectal cancer; FTLD-tau, frontotemporal lobar degeneration-tau; PSP, progressive supranuclear palsy; MSA, multiple systems atrophy

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**

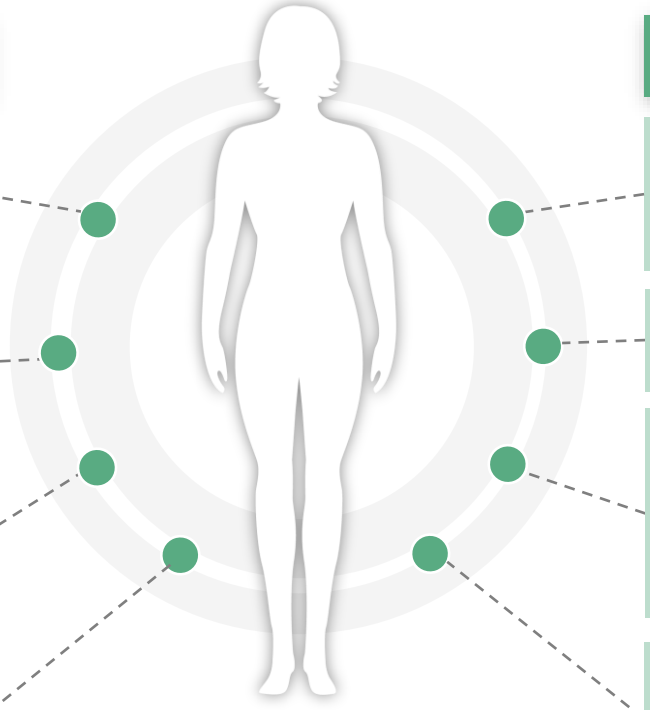
† US CDC: https://www.cdc.gov/cancer/breast/young_women/bringyourbrave/breast_cancer_young_women/index.htm; accessed 5/25/21; †† US SEER database; †††Arvinas estimate. SERD, selective estrogen receptor degrader.

ARV-471 has shown robust signals of efficacy in a challenging patient population

Data as presented 12/14/2020

A challenging patient population

5	Median of prior therapies
100%	Patients treated with CDK4/6 inhibitors
71%	Patients with prior fulvestrant treatment
66%	**Patients expected to have ER-independent disease**†



Early clinical benefit: Interim Phase 1

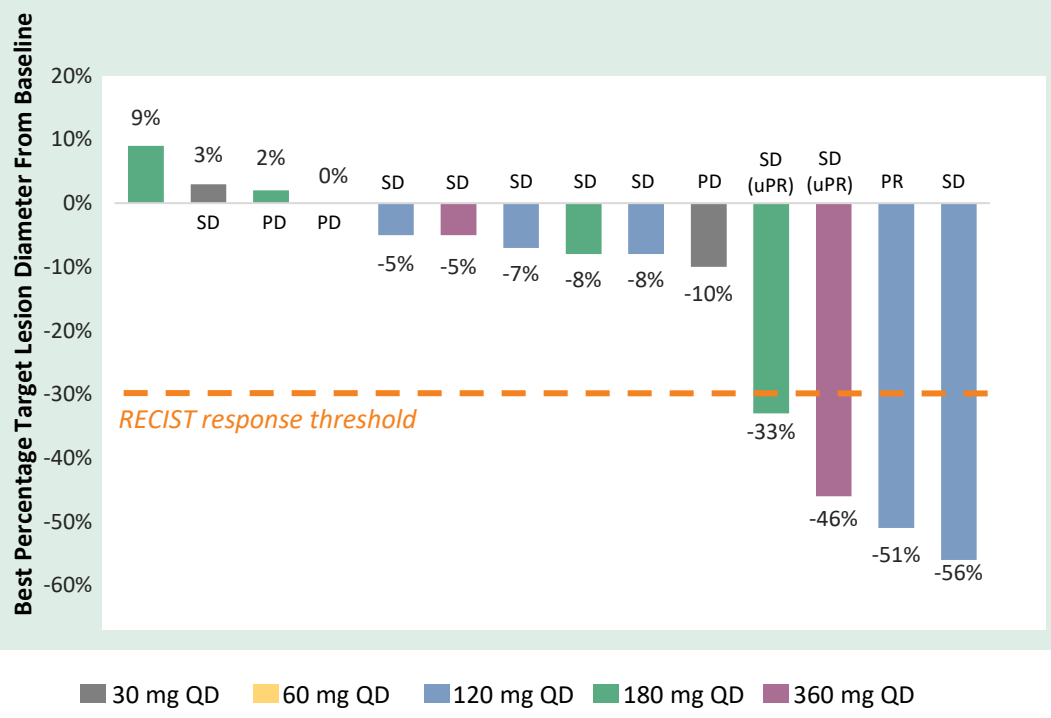
- Maximum ER degradation of **90%** (mean, **62%**) in first 3 dose levels (*fulvestrant reported at 40-50%)*††
- **Outstanding tolerability** profile
- **Confirmed partial response** (PR) in patient who progressed on multiple treatments; **tumor shrinkage** in other patients
- **Clinical benefit rate** (CBR) of **42%** (5/12)†††

† Wander 2020; †† Fulvestrant degradation reported as 40-50% in Robertson et al., Breast Cancer Research (2013) and Kuter et al., Breast Cancer Res Treat (2012);
††† CBR defined as SD persisting ≥ 24 weeks, or a best response of confirmed CR or PR. ESR1, Estrogen Receptor 1.

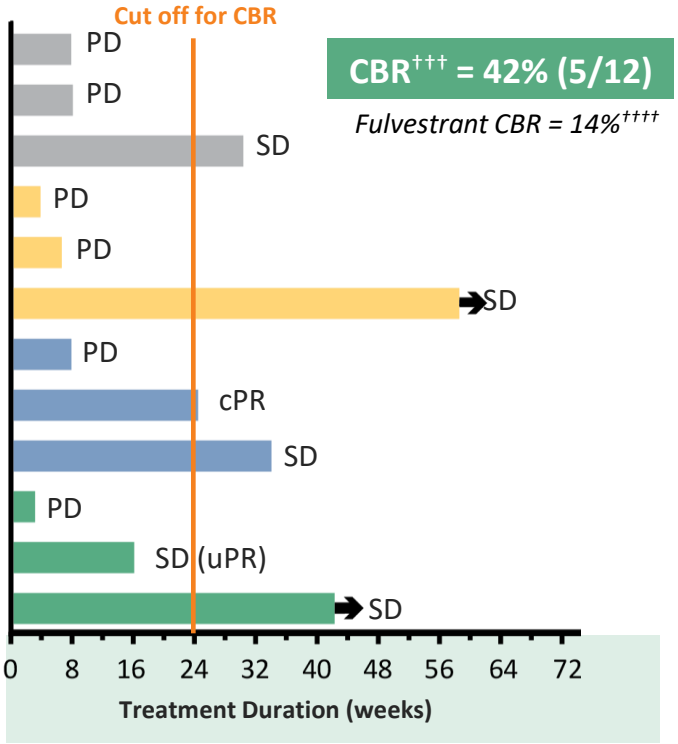
Interim Phase 1 data for ARV-471 demonstrate anti-tumor activity and a high CBR in heavily pretreated patients

Data as presented 12/14/2020

Antitumor Activity in Eligible Patients (N=14)†



Treatment Duration (weeks) and Response in Eligible Patients (N=12)††



† 7 patients out of 21 are excluded from graph due to no measurable disease at baseline (n=4), discontinuation of treatment without post-treatment target lesion measurements (n=2), and discontinuation after 2 doses due to non-compliance (n=1); †† Excludes 8 patients enrolled < 24 weeks prior to the data cut-off of November 28, 2020 and 1 patient who received -471 and discontinued due to non-compliance, ††† CBR defined as SD persisting ≥ 24 weeks, or a best response of confirmed CR or PR. †††† Based on VERONICA trial of fulvestrant post-CDK4/6i, ASCO 2021. QD, once daily; CBR, clinical benefit rate; RECIST, response evaluation criteria in solid tumors; SD stable disease; PD, progressive disease; cPR, confirmed partial response; uPR, unconfirmed partial response.

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ARV-471 has the potential to be a best-in-class ER-directed therapy

Comparison of Phase 1 data with clinical-stage SERDs

Data as presented 12/14/2020

Phase 1 Data Comparison										
	Drug Candidate	CDK4/6i Pretreated Patients *(0 – 100%)*	Clinical Benefit Rate	Mean ER Degradation in Patient Tumors	Select TRAEs (> 5% of Patients)					
					Gastrointestinal AEs			Other AEs		
					Diarrhea	Nausea	Vomiting	Bradycardia	Visual disturbance	
	ARV-471	**100%**	**42%**	**62%** *Interim*		●	●			
SERDs	H3B-6545	87%	34%	*Not reported*	●	●	●	●		
SERDs	ZN-C5	87%	40%	*Not reported*	●	●	●			
SERDs	Rintodestrant	70%	30%	28%	●	●	●			
SERDs	SAR439859	63%	34%	*Not reported*	●	●	●			
SERDs	AZD9833†	62%	35%	<50%††		●	●	●	●	
SERDs	GDC9545	59%	41%	<50%††	●	●		●		

Source: H3B-6545 SABCS 2020 Poster, ZN-C5 SABCS 2020 Poster, Rintodestrant SABCS 2020, SAR439859 SABCS 2020 Poster, AZD9833 SABCS 2020 and ASCO 2020 Posters, GDC-9545 SABCS 2019 Poster. This comparison utilizes data from different Phase 1 trials and presents a non-head-to-head summary comparison.

† Reported AEs are from ASCO 2020 Poster; ††Visual estimation based on ER degradation data provided by each company.

AE, adverse event; ER, estrogen receptor; SERD, selective estrogen receptor degrader; TRAE, treatment-related adverse event.

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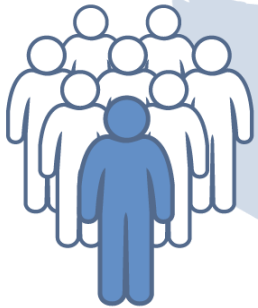
ARV-471: Moving forward rapidly across the continuum of disease

Four clinical trials ongoing by the end of 2021

FUTURE STUDY SETTING		ENABLING TRIAL	REGIMEN	INITIATION
ARV-471 ACROSS THE TREATMENT PARADIGM	3L Metastatic Patients	**Phase 1b**	ARV-471 + Targeted Therapy†	**2H 2021**
	2L Metastatic Patients	**VERITAC Phase 2** *Expansion trial*	ARV-471	**1Q 2021**
	1L Metastatic Patients	**Phase 1b** *Supportive study in 2/3L patients to enable 1L study*	ARV-471 + CDK4/6i (palbociclib)	**Dec 2020**
	Adjuvant	**Window of Opportunity** *Enabling trial in neoadjuvant setting*	ARV-471 **or** ARV-471 + CDK4/6i (palbociclib)	**2H 2021**

† E.g., everolimus or alpelisib. CDK, cyclin-dependent kinase

ARV-110: AR-degrading PROTAC in metastatic prostate cancer



1 in 8

U.S. men will be diagnosed with prostate cancer during their lifetime†

Prostate cancer is the second leading cause of cancer death for men in the U.S.††

In 2021 alone, there will be an estimated

248,530 new cases

of prostate cancer†††

34,130 deaths are attributed to the disease†††

High unmet need in prostate cancer treatment represents

$8b market in the US alone††††

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**
- **ARV-110 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**

† CDC: https://www.cdc.gov/cancer/prostate/basic_info/risk_factors.htm; accessed 5/25/21; †† American Cancer Society; ††† US SEER database; †††† Arvinas estimate.

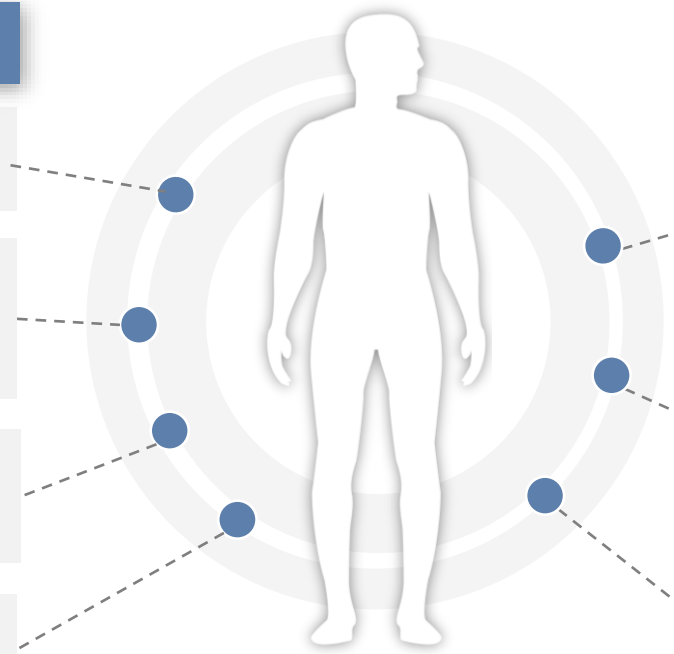
ARVINAS

ARV-110 has shown clinical benefit in Phase 1 in a highly refractory patient population

Data as presented 12/14/2020

A challenging patient population

5	Median number of previous therapies
82%	Patients were treated with both abiraterone and enzalutamide
76%	Patients were treated with prior chemotherapy
84%	Patients have non-AR mutations



Clinical benefit in Phase 1

ARV-110 is well tolerated, allowing continued dose escalation up to 700 mg daily†, and potentially **supporting use in earlier lines of therapy**

AR degradation and late-line activity suggest **strong potential across multiple disease states**

AR molecular profiling identifies **a molecularly defined**, late line population that may **offer a possible path to accelerated approval**

† As of 12/14/2020

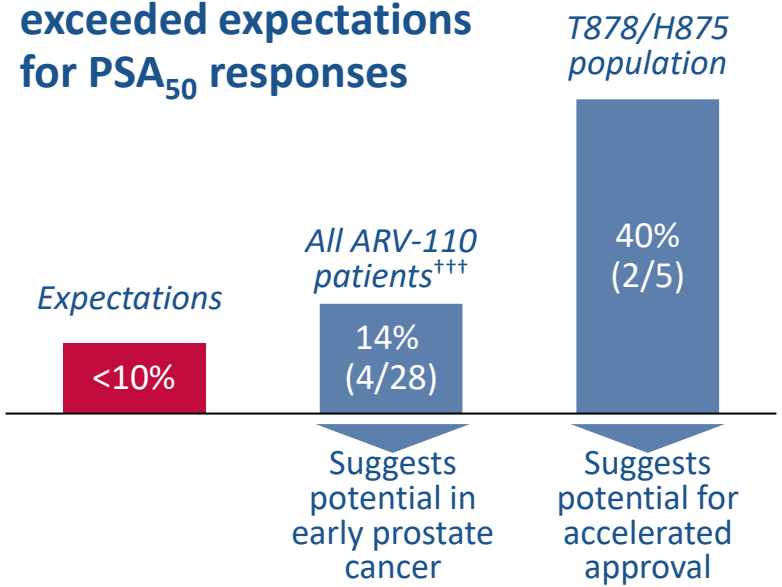
ARV-110 has shown encouraging efficacy signals in patients with extensive prior therapy and few to no treatment options

Data as presented 12/14/2020

- Up to 90% of early-stage prostate cancer patients treated with enzalutamide experience PSA reductions†
- PSA_{50} responses drop to 8-15% in patients with **3L** mCRPC††

In the ARV-110 Phase 1 population, we expected <10% PSA_{50} response rate

ARV-110 substantially exceeded expectations for PSA_{50} responses



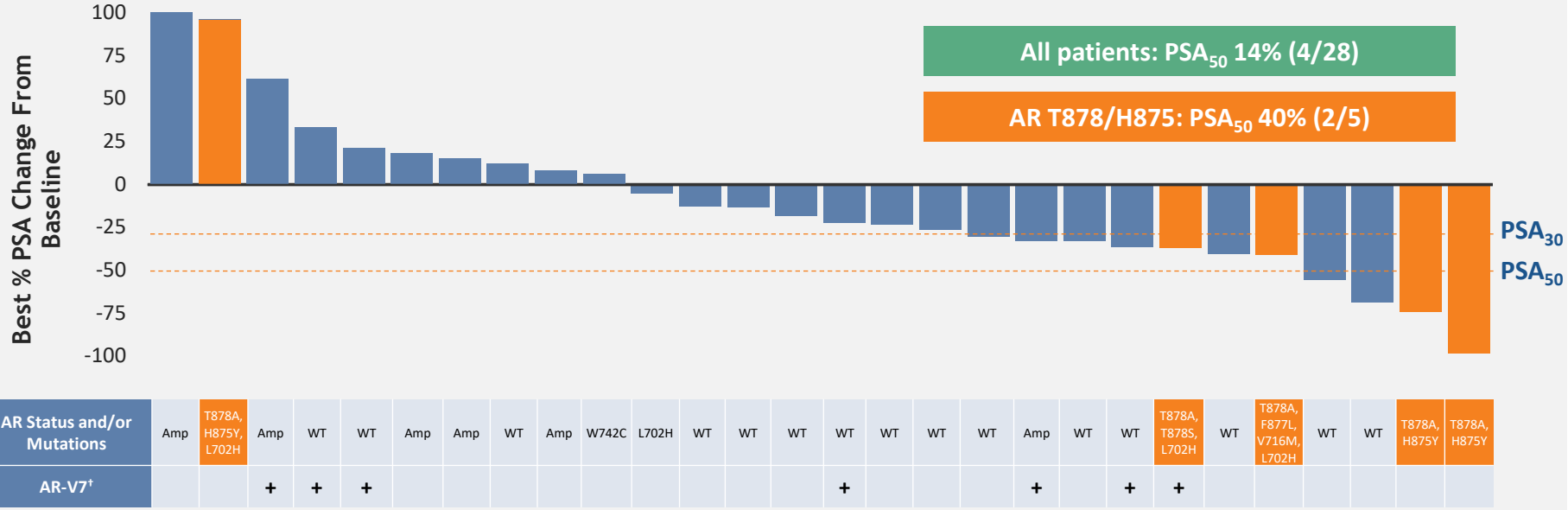
† Tombal, Lancet Oncology 2014; †† de Wit R, N Engl J Med. 2019; Hussain, ESMO 2019; ††† Includes all patients with exposures above a threshold that predicted efficacy in preclinical models. mCRPC, metastatic castrate resistant prostate cancer; PSA50, prostate-specific antigen reduction >50%

ARVINAS

ARV-110 has an efficacy signal in a highly resistant population and a 40% PSA_{50} in a molecularly defined subgroup

Data as presented 12/14/2020

Best PSA Change In All Patients Above Minimum Exposure Threshold (N= 28)††



AR Status and/or Mutations	Amp	T878A, H875Y, L702H	Amp	WT	WT	Amp	Amp	WT	Amp	W742C	L702H	WT	WT	WT	WT	WT	WT	WT	Amp	WT	WT	T878A, T878S, L702H	WT	T878A, F877L, V716M, L702H	WT	WT	T878A, H875Y	T878A, H875Y
AR-V7†			+	+	+										+				+		+	+						

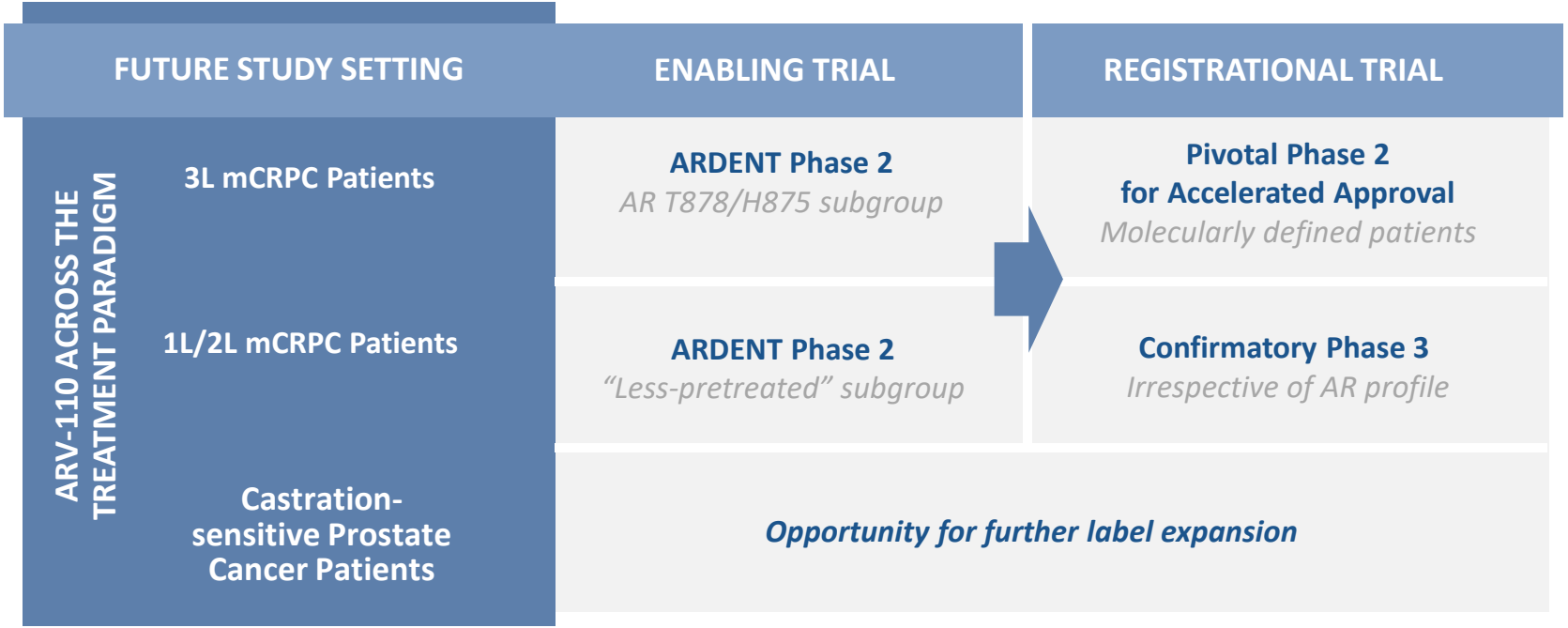
20/28 (71%) of patients have either T878/H875 or wild-type AR

Each column represents one patient. † Epic Sciences, Genetic profiling: FoundationOne®Liquid (70-gene panel), †† Data as of 30-Nov-2020. The "minimum exposure threshold" refers to drug exposure corresponding to tumor inhibition in preclinical models; see Arvinas' 12/14/2020 data release for more information.

ARVINAS

ARV-110: Potential path forward in both molecularly defined and earlier-line patients with prostate cancer

Expecting interim ARDENT Phase 2 data in the second half of 2021



mCRPC, metastatic castration-resistant prostate cancer

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

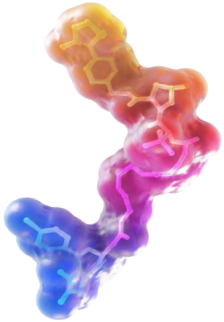
PROTAC degraders could revolutionize the treatment of neuroscience diseases

- 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

Drugging an undruggable: KRAS

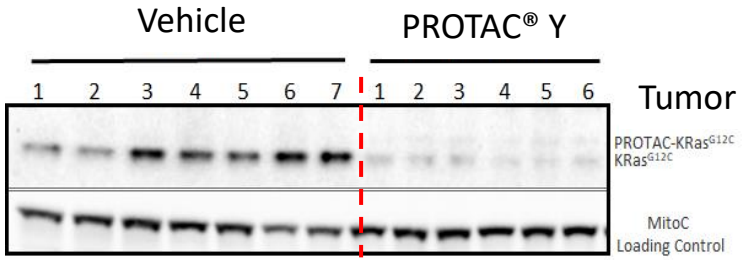


KRAS is the most frequently mutated gene in human cancer and is a classic “undruggable” target due to its lack of deep “pockets”

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 with anticipated IND in 2023

† MiaPaCa-2 xenograft mode

Rapid pace of anticipated milestones in 2021-2022

	2021	2022
ARV-471 (ER PROTAC®)	• **Share completed Phase 1 data** • **Share interim CDK4/6i combination study data** • Initiate Window of Opportunity study • Initiate non-CDK4/6i combination study(s)	• **Share interim VERITAC Phase 2 data** • **Share completed CDK4/6i combination data** • Share interim data from non-CDK4/6i combination(s)
ARV-110 (AR PROTAC®)	• **Share completed Phase 1 data** • **Share ARDENT Phase 2 interim data** • Initiate combination study(s)	• **Share full ARDENT Phase 2 data** • **Share interim combination data**
ARV-766 (AR PROTAC®)	• Initiate Phase 1	• Share Phase 1 data • Initiate Phase 2
INDs	• ARV-766	• BCL6 • Tau • Undisclosed (oncology)

With proof-of-concept established, we are building toward an integrated Arvinas

Relentlessly driving toward patient impact

- Delivering on the promise of turning degraders into therapeutics for patients
- Pipeline built to nominate ≥1 clinical candidate per year
- Anticipating our first Phase 2 data in 2H2021
- Continuing to build new components into our PROTAC Discovery Engine
- Planning a robust build-out of the company, resources, and capabilities needed to bring the first PROTAC® therapeutics to patients

For more information

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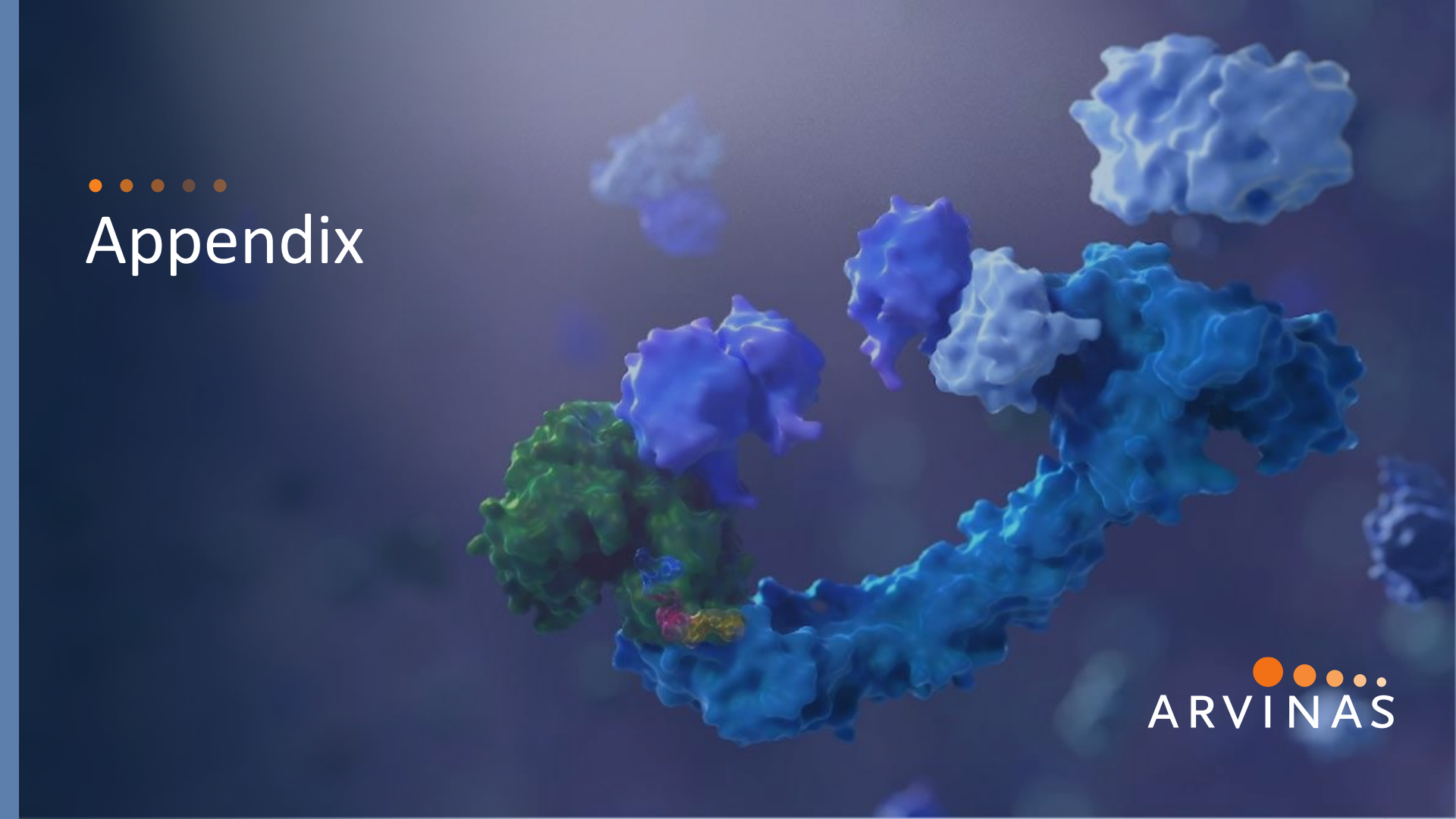
Appendix



High-performing leadership team

Proven track record of driving success with 20+ novel drugs

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

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

John A. Grosso, Ph.D.
VP Chemistry, Mfg. & Controls

Ron Peck, M.D.
Chief Medical Officer

Ian Taylor, Ph.D.
Chief Scientific Officer

Kimberly Wehger
VP Information Technology

Matthew Batters, J.D.
General Counsel

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

Marcia Dougan Moore, M.P.H.
SVP Strategic Operations

Larry Snyder, Ph.D.
ED Medicinal Chemistry

Randy Teel, Ph.D.
VP Corporate Development

Steve Weiss
VP Human Resources

ULTOMIRIS (ravulizumab-cwvz) injection for intravenous use 300 mg/3 mL vial

ABILIFY (aripiprazole) 2, 5, 10, 15, 20, 30 mg Tablets

IBRANCE palbociclib 125 mg tablets

Nexavar (sorafenib) tablets

Nurtec ODT (rimegepant) orally disintegrating tablets 75 mg

OPDIVO (nivolumab)

VIZIMPRO DACOMITINIB

SOLIRIS (eculizumab) Injection for Intravenous Use

Once-daily oral Zejula niraparib capsules 100 mg

YERVOY (ipilimumab)

farxiga (dapagliflozin) 5mg & 10mg tablets

SPRYCEL dasatinib 100 mg tablets

Stivarga (regorafenib) tablets

Strensiq (asfotase alfa) for injection 40 mg/mL

Daklinza (daclatasvir) 60 mg tablets

Aliqopa (copanlisib) 60 mg vial for injection

XALKORI CRIZOTINIB

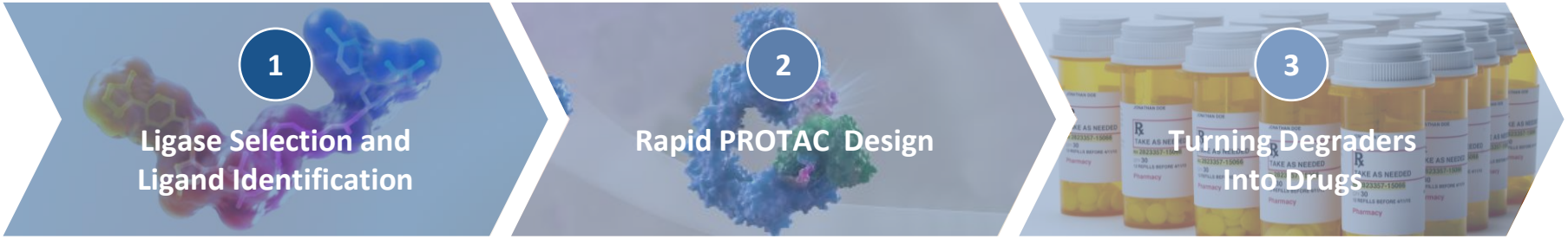
IXEMPRA (ixabepilone) for injection, for intravenous infusion

Sunvepra Asunaprevir

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
- Arvinas' DNA-encoded libraries for advanced screening
- Identification of new "warheads" for previously undruggable targets

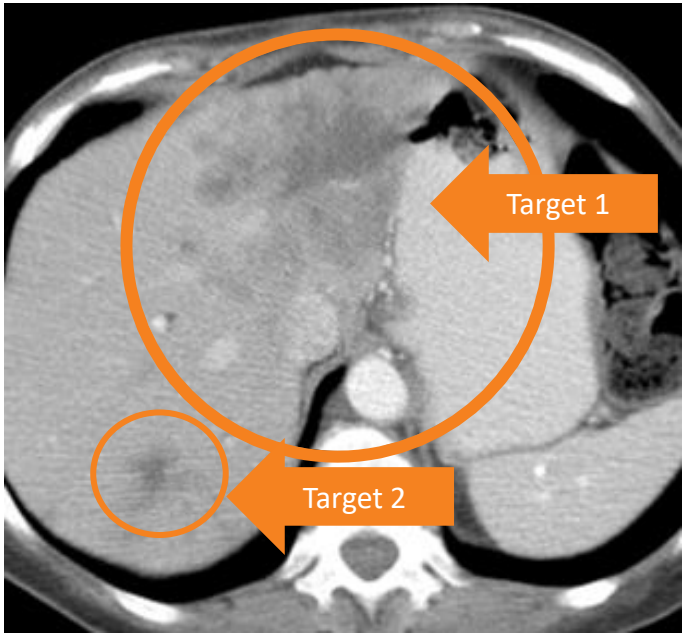
- Zone of Ubiquitination – we design PROTAC degraders to predict the precise location where a protein can be tagged
- Predictive computational modeling
- State-of-the-art proteomics capabilities

- "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

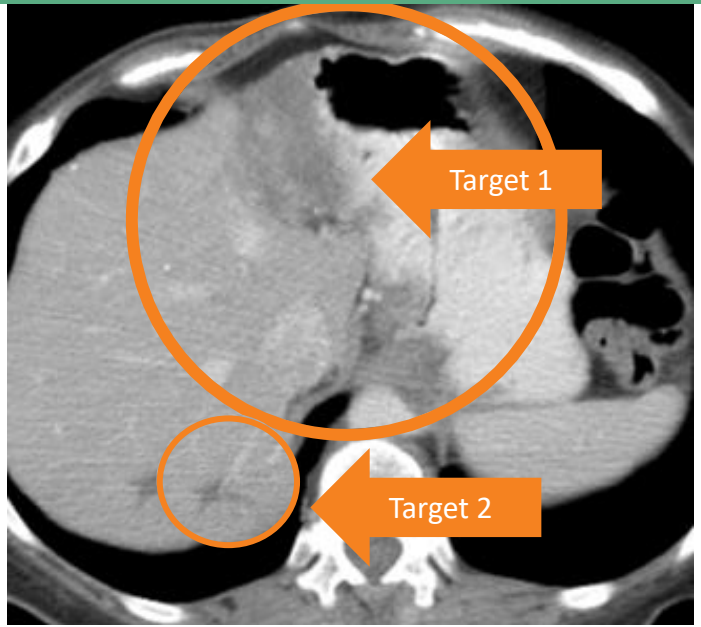
Breast cancer: confirmed 51% tumor reduction in clinical trial

Data as presented 12/14/2020

Baseline CT Scan



After 4 cycles

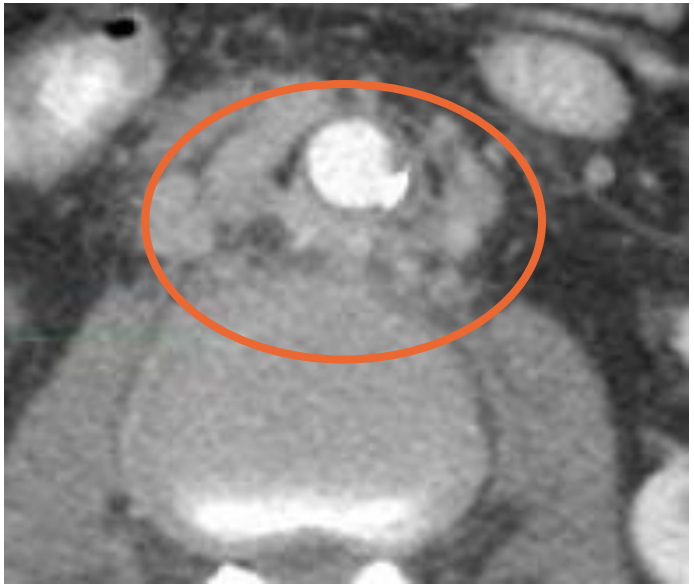


51% reduction in target lesions

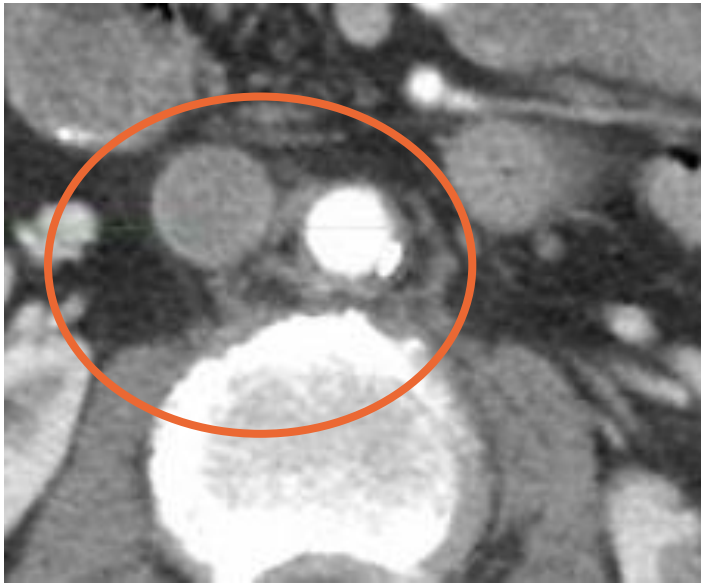
Prostate cancer: confirmed 80% tumor reduction in clinical trial

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

BASELINE CT SCAN
Extensive retroperitoneal adenopathy compressing the inferior vena cava



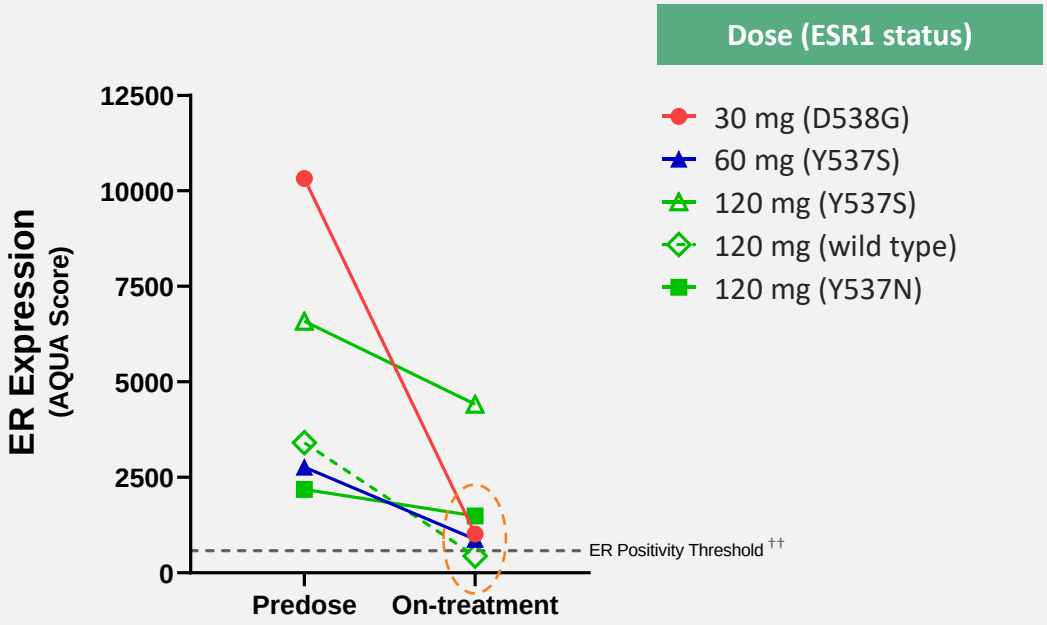
AFTER 4 CYCLES
Near complete regression of adenopathy



80% reduction in target lesions

ARV-471 degraded ER up to 90% through the 120 mg dose level

ER Expression in Paired Tumor Biopsies†



Degradation up to 90%; average of 62%

Degradation **superior to fulvestrant** *(previously reported: 40-50%)†††*

Degradation of **wild type ER and ESR1** mutant proteins

† ER immunoreactivity analyzed by quantitative immunofluorescence (QIF) using the automated quantitative analysis (AQUA) method. †† 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). ESR1, Estrogen Receptor 1