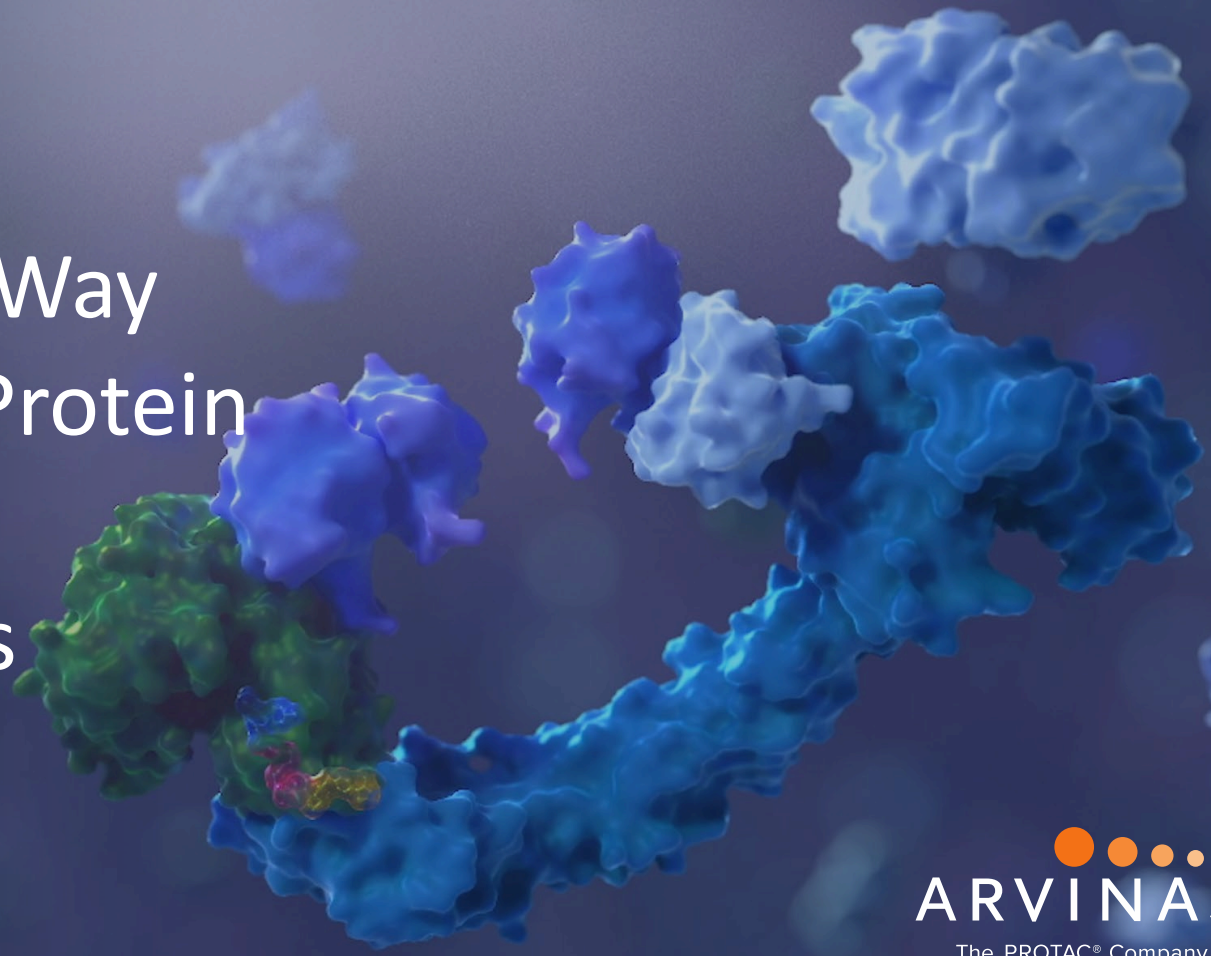
Leading the Way in Targeted Protein Degradation Therapeutics



ARVINAS

The PROTAC® Company

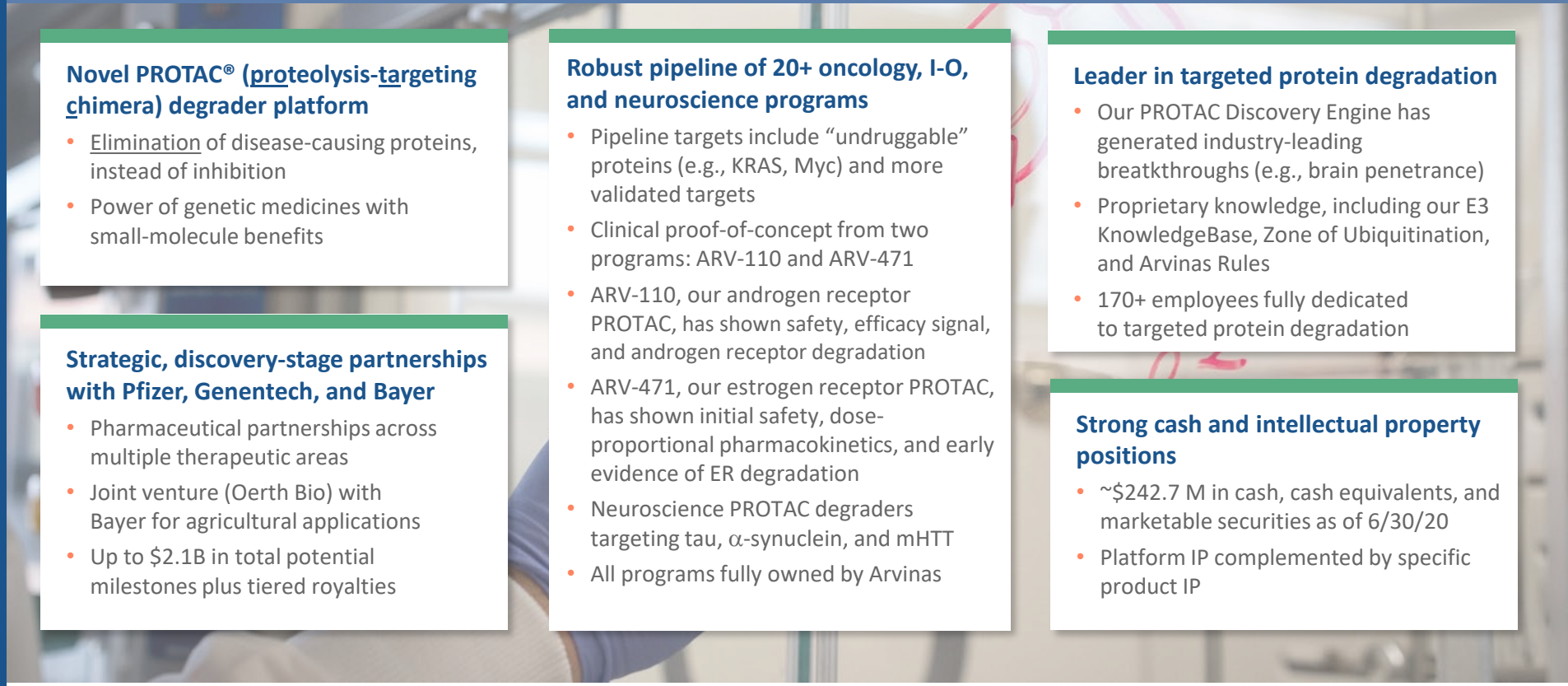
OCTOBER 2020

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 and ARV-471, and the timing of clinical trials and data from those trials 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, the potential benefits of our arrangements with Yale University, our collaborative partnerships, and the Bayer joint venture, 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, 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, each party’s ability to perform its obligations under our collaborations and/or the Bayer joint venture, our expected timeline 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.

Clinical-stage leader in protein degradation, a powerful new modality



Novel PROTAC® (proteolysis-targeting chimera) degrader platform

- Elimination of disease-causing proteins, instead of inhibition
- Power of genetic medicines with small-molecule benefits

Strategic, discovery-stage partnerships with Pfizer, Genentech, and Bayer

- Pharmaceutical partnerships across multiple therapeutic areas
- Joint venture (Oerth Bio) with Bayer for agricultural applications
- Up to $2.1B in total potential milestones plus tiered royalties

Robust pipeline of 20+ oncology, I-O, and neuroscience programs

- Pipeline targets include “undruggable” proteins (e.g., KRAS, Myc) and more validated targets
- Clinical proof-of-concept from two programs: ARV-110 and ARV-471
- ARV-110, our androgen receptor PROTAC, has shown safety, efficacy signal, and androgen receptor degradation
- ARV-471, our estrogen receptor PROTAC, has shown initial safety, dose-proportional pharmacokinetics, and early evidence of ER degradation
- Neuroscience PROTAC degraders targeting tau, α-synuclein, and mHTT
- All programs fully owned by Arvinas

Leader in targeted protein degradation

- Our PROTAC Discovery Engine has generated industry-leading breatkthroughs (e.g., brain penetrance)
- Proprietary knowledge, including our E3 KnowledgeBase, Zone of Ubiquitination, and Arvinas Rules
- 170+ employees fully dedicated to targeted protein degradation

Strong cash and intellectual property positions

- ~$242.7 M in cash, cash equivalents, and marketable securities as of 6/30/20
- Platform IP complemented by specific product IP

Note: mHTT, mutant huntingtin

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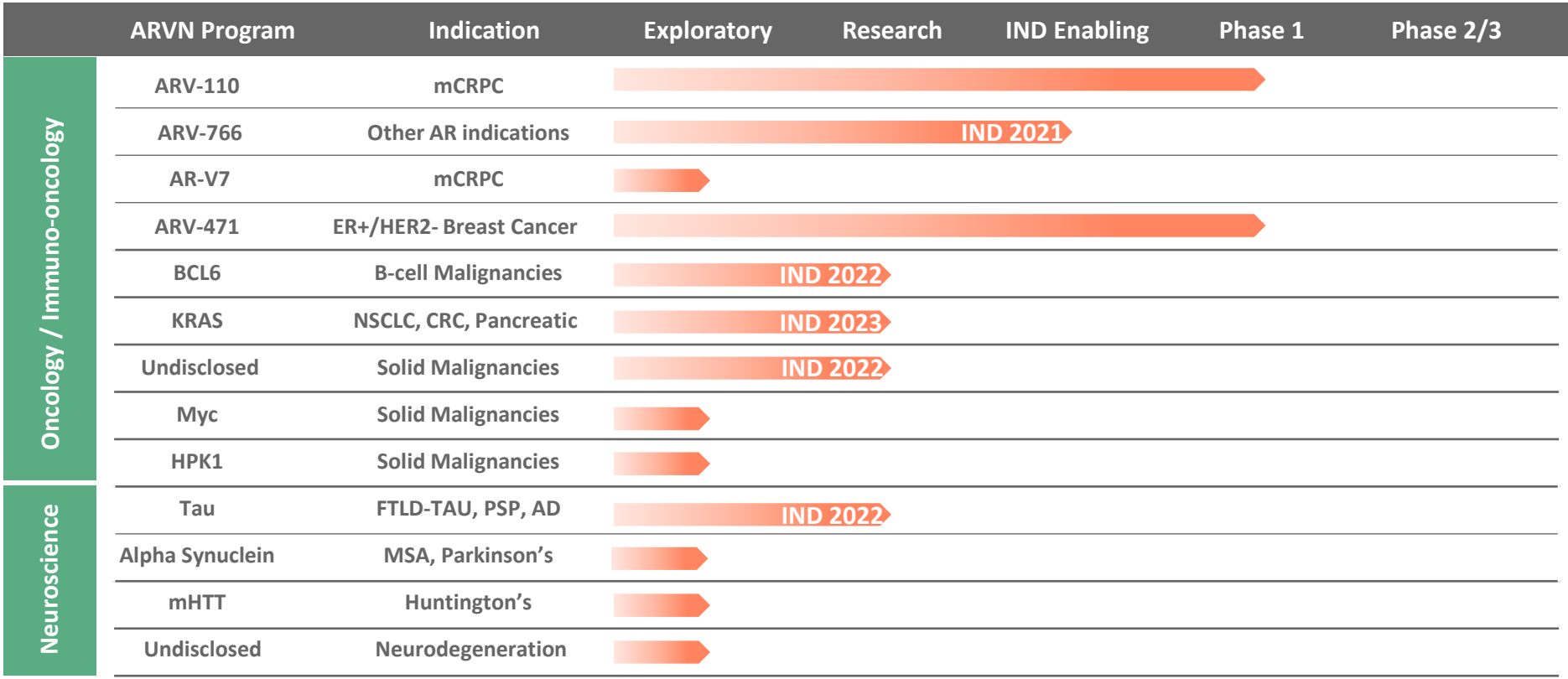
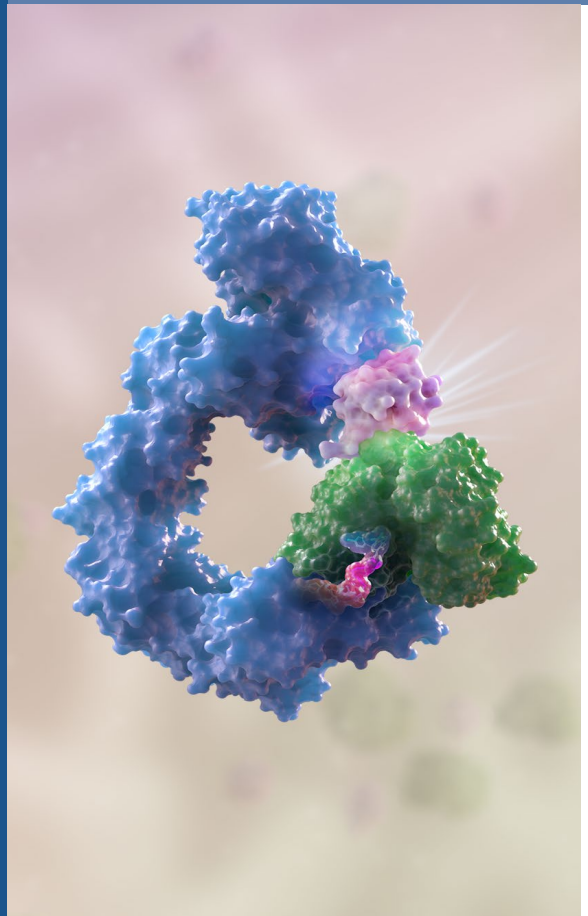
Arvinas' pipeline encompasses a range of validated and undruggable targets in oncology, I-O, and neuroscience



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

ARVINAS

ARV-110 and ARV-471 have provided clinical proof-of-concept for PROTAC® degraders



- ✓ Degradation of AR and ER demonstrates proof-of-mechanism in human patients
- ✓ Safety initially observed in two different programs in two different patient populations
- ✓ ARV-110 overcame prior resistance to AR therapy, showing the translation of ARV-110's preclinical profile into patient benefit
- ✓ Reinforces our confidence in Arvinas' extensive and promising preclinical pipeline

We anticipate a rapid pace of milestones in 2021-22

	2020 Q4	2021	2022
ARV-110 (mCRPC)	• Program update • Initiation of Phase 2	• Completed Phase 1 data • Phase 2 interim data • Initiation of combination study	• Full Phase 2 data • Combination study data
ARV-471 (ER+/HER2- breast cancer)	• Interim Phase 1 data • Initiation of combination study with CDK4/6i	• Completed Phase 1 data • Initiation of Phase 2 • CDK4/6i combination study data	• Interim Phase 2 data
ARV-766 (AR PROTAC®)		• Initiate Phase 1	• Phase 1 data • Initiate Phase 2
INDs		• ARV-766	• BCL6 • Undisclosed (oncology) • Tau

Note: mCRPC, metastatic castration-resistant prostate cancer; ER+/HER2-, estrogen receptor+/human epidermal growth factor receptor 2-

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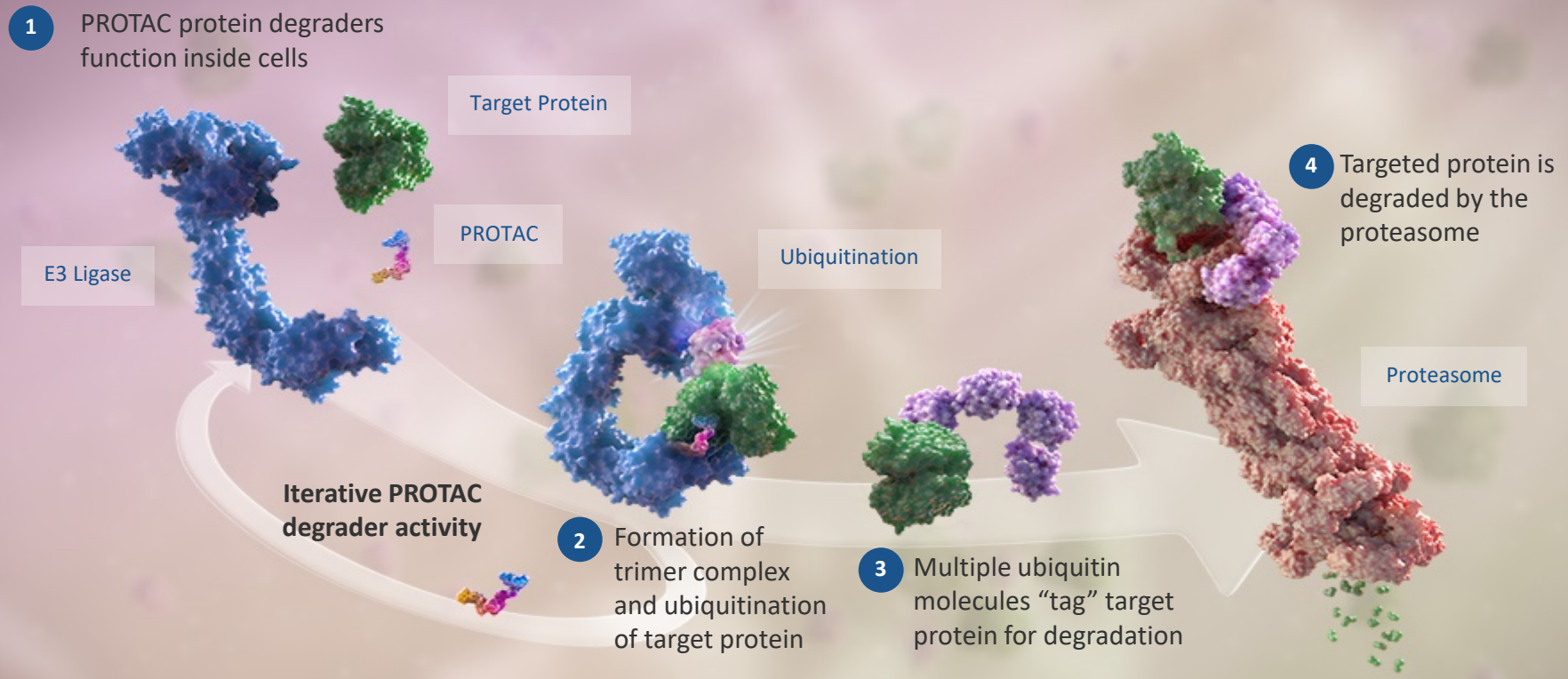
Arvinas’ PROTAC® Discovery Engine

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



PROTAC® protein degraders combine the advantages of gene-based medicines with the benefits of small molecule therapies

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 pathogenic proteins	✓	✗	
Target scaffolding function	✓	✗	
Potential to treat “undruggable” proteins	✓	✗	
Iterative mechanism of action	✓	✗	✗
Broad tissue penetration	✓		✗
Orally bioavailable	✓		✗
Ease of manufacturing	✓		✗

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

PROTAC Discovery Engine

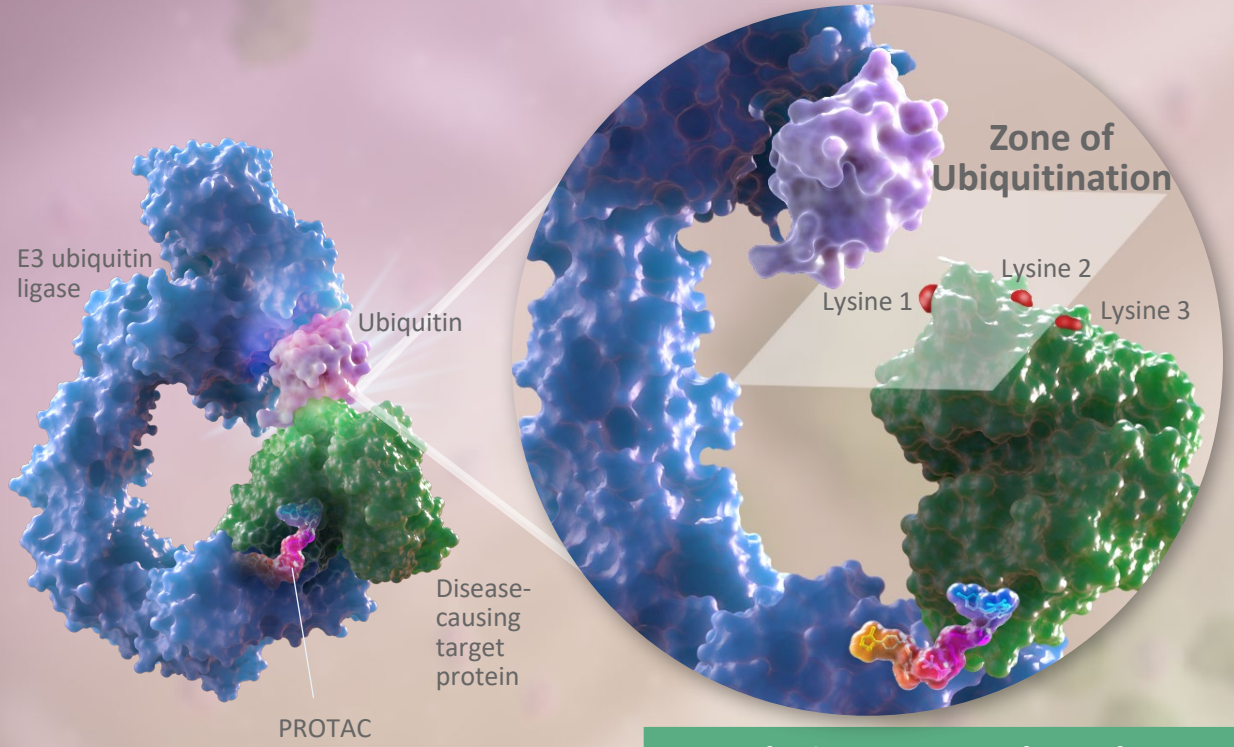
- E3 KnowledgeBASE of novel E3 ligases
- Novel warheads for undruggable targets and new ligands for E3 ligases
- Advanced screening capabilities, including proprietary DNA-encoded libraries tailored for PROTAC development

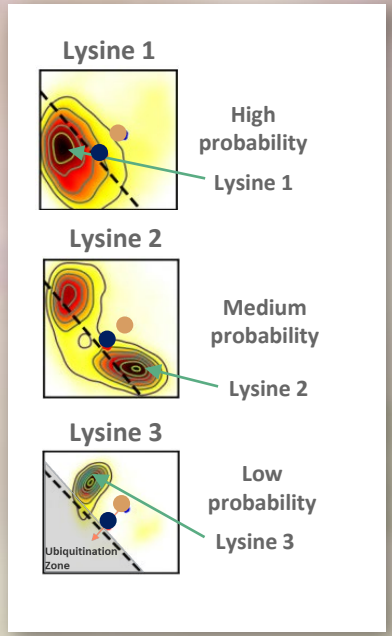
- Optimizing the Zone of Ubiquitination
- Arvinas Next Generation Linker Evolution (ANGLE)
- Predictive computational modeling
- State-of-the-art proteomics capabilities

- "Arvinas Rules" for drug-like properties, including blood-brain barrier penetration and oral bioavailability in humans
- Deep knowledge of *in vivo* PK/PD and efficacy relationships

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

Our deep understanding of the Zone of Ubiquitination informs the structure-based design of PROTAC® degraders





We design PROTAC degraders to optimize the position of lysine residues within the Zone of Ubiquitination

Strategic partnerships expand the impact of our PROTAC® Discovery Engine

These partnerships expand the impact of PROTAC degraders beyond oncology and beyond human therapeutics, while maintaining full ownership of our pipeline

Clinical-stage Oncology Programs: ARV-110

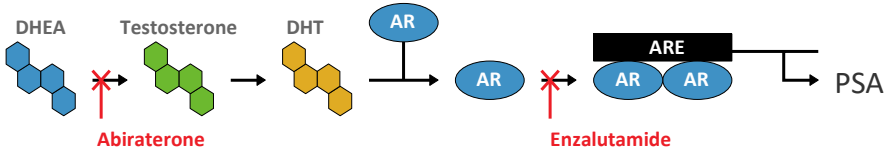
ARV-110 is Arvinas' AR degrader for men with metastatic castration-resistant prostate cancer (mCRPC)

Androgen Receptor (AR) Activity Drives Prostate Cancer

- Prostate cancer is the second leading cause of cancer death in men in the US[1]
- Current agents work by decreasing androgen levels (abiraterone) or blocking androgen binding to AR (enzalutamide)
- **15-25%** of patients never respond to abiraterone or enzalutamide (**intrinsic resistance**)
- **Acquired resistance mechanisms** to abiraterone and enzalutamide include:
  - **AR gene amplification** (40-60% of patients)
  - **AR gene enhancer amplification** (>70% of patients)
  - **AR point mutations** (up to 25% of patients)
  - **Intra-tumoral androgen production**
- Despite rapid and dramatic responses to standards of care, all patients with metastatic disease progress to the castration resistant state and their tumors continue to be dependent on the AR signaling axis[2]

PROTAC® Degrader ARV-110

- First-in-class AR degrader
- In preclinical models, overcomes known resistance mechanisms to enzalutamide and abiraterone
- Highly selective degradation of AR; not brain penetrant
- Received FDA "Fast Track" designation in May 2019
- **Interim phase 1 data disclosed in 2Q20 showed AR degradation and efficacy signal, validating the PROTAC® mechanism and platform**



[1]American Cancer Society; [2]Cancers 2017, 9, 67; doi:10.3390/cancers9060067

Phase 1 study of ARV-110 is a traditional "3+3" dose escalation study in patients that have received ≥2 prior systemic therapies for mCRPC

Design

- "3 + 3" dose escalation; starting dose = 35 mg, orally, once daily with food
- Dose increases dependent on toxicities
  - Range 25% to 100% based on severity of AEs

Inclusion criteria

- Men with mCRPC, regardless of AR status
- At least two prior systemic therapies, at least one of which was abiraterone or enzalutamide
- Disease progression on most recent therapy
  - Rising PSA or 2+ new lesions upon bone scan

Endpoints

Primary:

- Define the maximum tolerated dose and recommended phase 2 dose

Secondary:

- Pharmacokinetics
- Anti-tumor activity (PSA50, RECIST criteria)

Exploratory:

- Biomarkers
  - ctDNA mutational profiling
  - AR levels in optional paired biopsies
  - AR and AR-V7 levels in circulating tumor cells (CTCs)

mCRPC= metastatic castration-resistant prostate cancer. RECIST= Response Evaluation Criteria in Solid Tumors. ctDNA, circulating tumor DNA. PSA, prostate-specific antigen

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Enrolled patients in the ARV-110 clinical trial have been highly pretreated at baseline

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

Patient characteristics	Parameter	N (%)	
Median age (years)		67.5	
ECOG Performance Status	0	15	(68)
	1	7	(32)
Number of prior regimens in mCRPC	≥2	22	(100)
	Mean	5	(NA)
	Median (range, 2-9)	6	(NA)
Prior 2nd generation AR treatment	**Abiraterone acetate (ABI)**	**22**	**(100)**
	Enzalutamide (ENZA)	**17**	**(77)**
	BOTH	**17**	**(77)**
Prior chemotherapy	**Any Chemotherapy**	**17**	**(77)**
	Docetaxel	**13**	**(59)**
	Cabazitaxel	**9**	**(41)**
	Docetaxel and Cabazitaxel	**5**	**(23)**
Other agents	Lutetium	2	(9)
	Radium RA 223	5	(23)
	Sipuleucel-T	5	(23)
	PARP inhibitor	5	(23)

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ARV-110 has been generally well tolerated; potential drug-drug interaction in the two patients taking concomitant rosuvastatin

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

Related TEAE	35 mg (N=3)		70 mg (N=4)		140 mg (N=8)		280 mg (N=7)		Total (N=22)	
	Gr ≤2	Gr ≥3	Gr ≤2	Gr ≥3	Gr ≤2	Gr ≥3	Gr ≤2	Gr ≥3	N (%)	
Any	-	-	1	1	4	1	5	1	13	(59)
Nausea	-	-	-	-	2	-	4	-	6	(27)
Diarrhea	-	-	1	-	3	-	2	-	6	(27)
Fatigue	-	-	1	-	2	-	2	-	5	(23)
ALT increased	-	-	-	1†	1	-	1	1†	4	(18)
AST increased	-	-	-	1†	2	-	-	1†	4	(18)
Lymphocyte count decreased	-	-	-	-	-	1	3	-	4	(18)
Vomiting	-	-	1	-	1	-	2	-	4	(18)

- Related TEAE in ≥ 10% of patients (N=22)
- 1 of 22 patients had a DLT with ALT/AST Grade 3/4 and renal failure (280 mg)

†Patients on rosuvastatin (N=2)

ARVINAS

Preliminary evidence supports a potential drug-drug interaction with rosuvastatin (Crestor®)

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

Clinical observations

- 2 of 22 patients received concomitant rosuvastatin
  - First patient with DLT: Grade 3/4 ALT/AST and renal failure
  - Second patient with Grade 3 ALT/AST; re-challenge off rosuvastatin supported contribution of rosuvastatin. Patient was restarted on ARV-110 with no further toxicity

Pharmacologic data supporting rosuvastatin interaction[1]

- Rosuvastatin concentrations increased in both patients with LFT rise compared to baseline
- Subsequent *in vitro* transport pump studies indicated BCRP transporter inhibition by ARV-110[2]

Following introduction of rosuvastatin restriction, no further elevation in LFTs observed

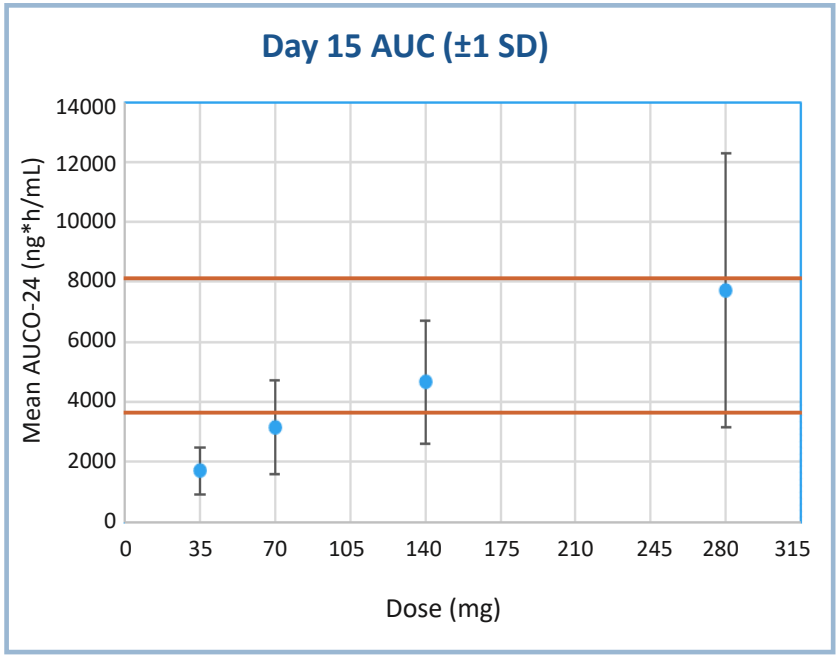
- 6 patients were on other statins, including 3 on atorvastatin (Lipitor®) and no ALT/AST adverse events

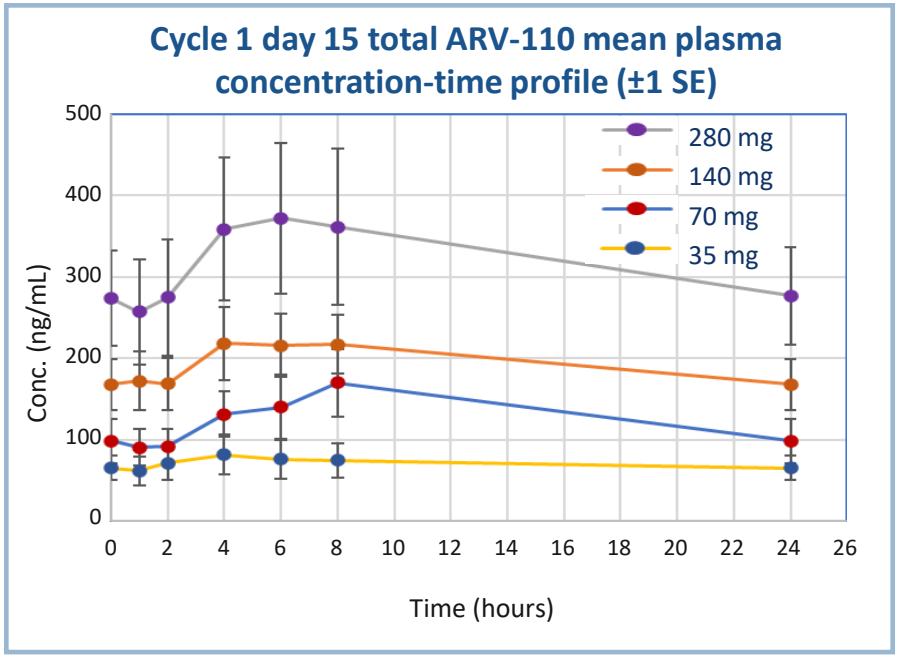
FT= liver function tests; DLT= dose-limiting toxicity; BCRP= breast cancer resistance protein; [1]Analyses are exploratory (validated but not GLP compliant)
[2]Following new in vitro BCRP data, restriction has been broadened to include substrates with high risk of clinically significant interactions

ARVINAS

ARV-110 exposures are dose-proportional and demonstrate drug-like pharmacokinetics, with a half life that supports daily dosing

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





The orange lines represent the minimum efficacious exposures for tumor growth inhibition in various preclinical models[1]

$T_{1/2} \approx$ 110 hours

[1]Upper line based on enzalutamide-resistant vertebral cancer of the prostate (VCaP) models. Lower line based on castrated and non-castrated VCaP model
QD, once per day. AUC, area under the curve. Cmax, maximum serum concentration. SD, standard deviation. SE, standard error.

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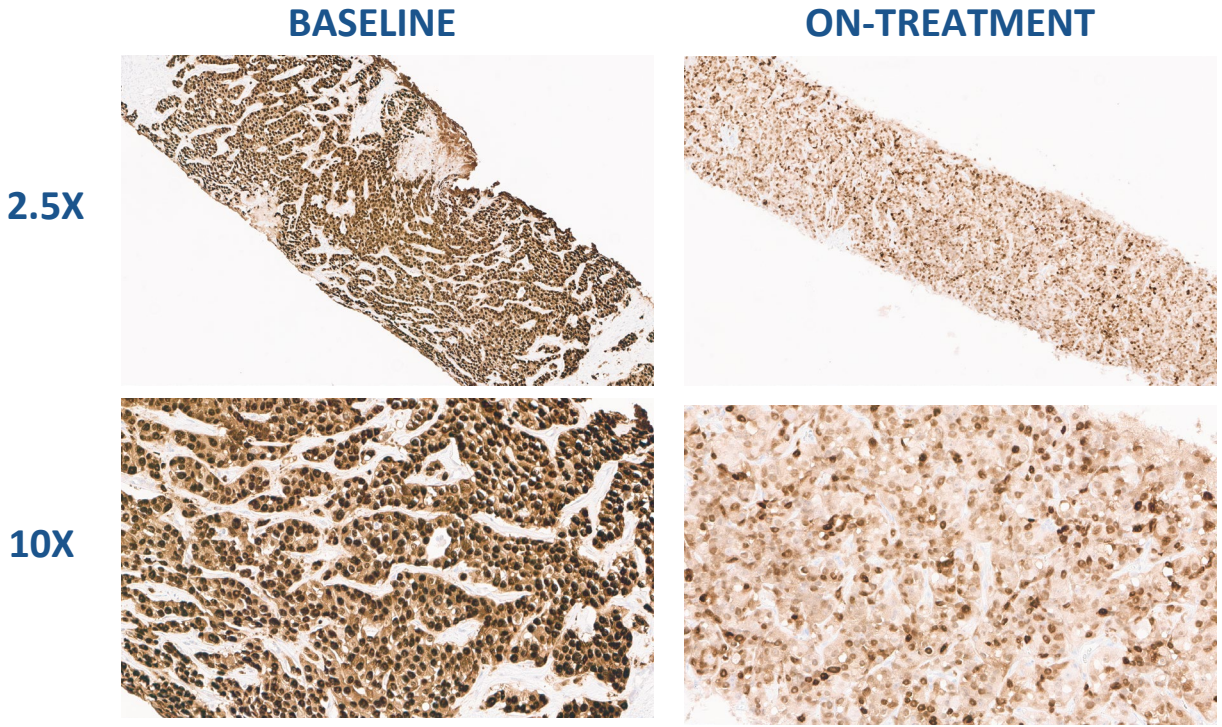
ARV-110 degrades AR in tumor tissue, demonstrating the first proof of mechanism for PROTAC® protein degraders

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



Decreased AR protein levels in an AR wildtype/amplified tumor from a patient following 6 weeks of ARV-110 dosing (280 mg)

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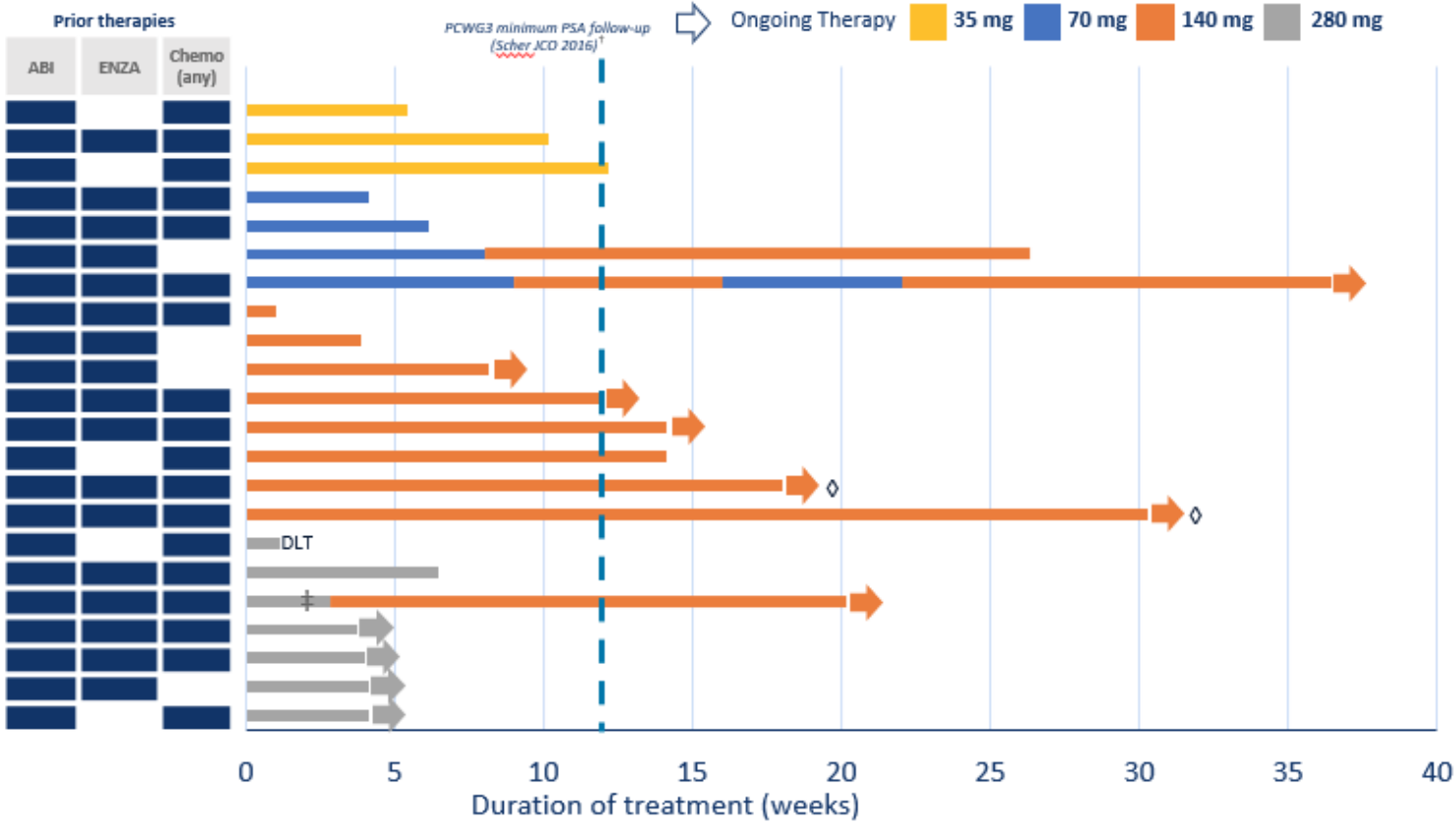
Duration of patient therapy in the ARV-110 dose escalation trial

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



Duration of therapy increases in patients at higher doses of ARV-110

>1/3 of patients across all dose levels remained on therapy longer than 3 months

Limited follow-up on most recent cohort at 280 mg

Data as of April 20, 2020

†PCWG3, Prostate Cancer Working Group 3; ‡Dose reduced for non-safety reasons; ◊PSA50 responder

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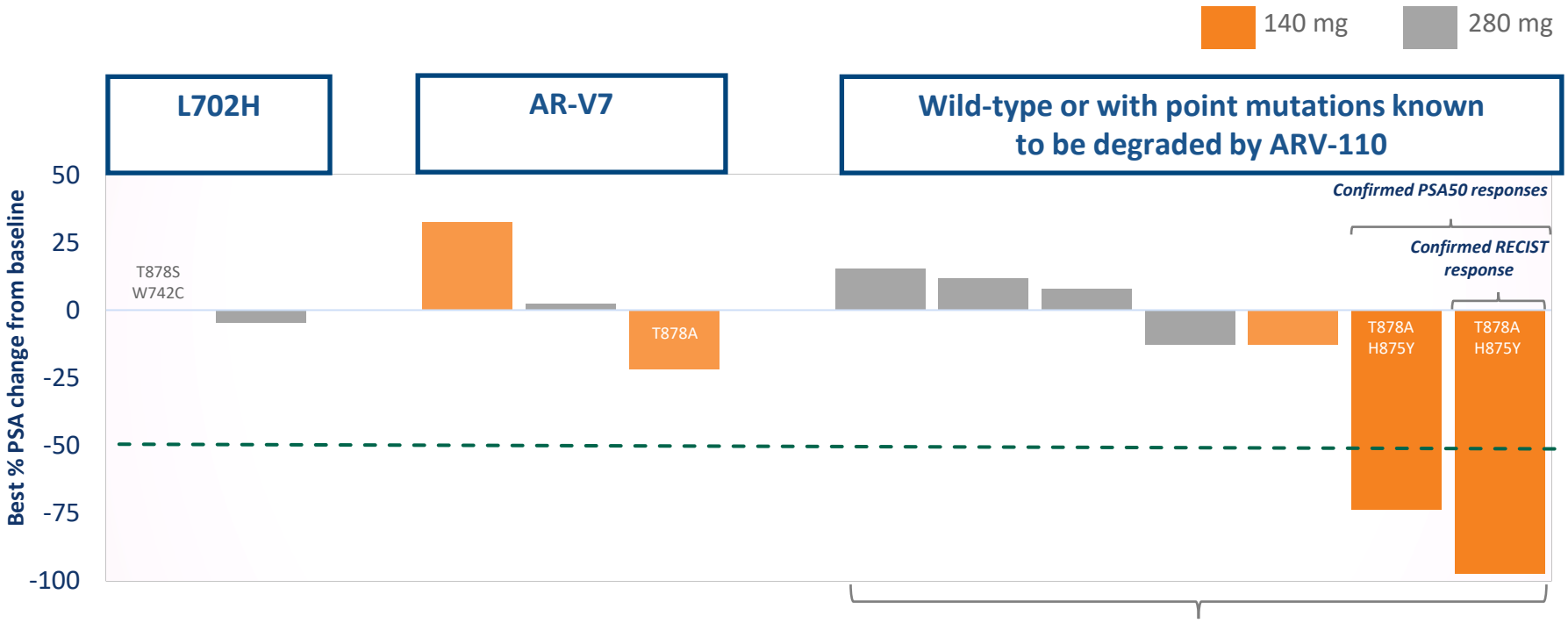
In patients with only ARV-110-degradable forms of AR, 2 of 7 had PSA decreases >50%[1]

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

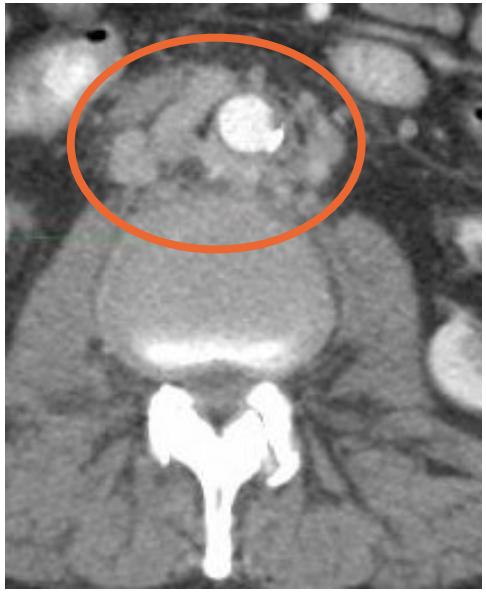


[1]Twelve patients shown exclude one patient with DLT associated with rosuvastatin

Two confirmed PSA50 responders, including one confirmed RECIST partial response

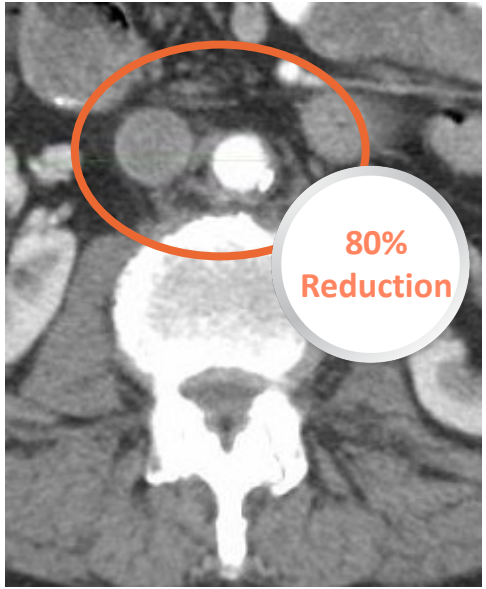
Data as presented at ASCO 2020 and as of 4/20/20

Responding Patient	1	2
PSA response	**74%** decline	**97%** decline
RECIST response	Not measurable	**80%** reduction
Duration of ARV-110	30+ weeks ongoing	18+ weeks ongoing
Biomarker status	AR H875Y and T878A mutations (associated with resistance to abiraterone or enzalutamide)[1]	
Common prior therapies	Enzalutamide, Abiraterone, Bicalutamide	
Other prior therapies	Docetaxel Radium	Provenge Cabazitaxel
History	Extensive bone metastases (sternum, left first rib, T3, T10 vertebral bodies)	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

[1]Jernberg E, Endocrine Connections, 2017

We are developing ARV-110 to be a potentially first- and best-in-class AR-targeted therapy for prostate cancer

Clear initial efficacy signal in dose escalation, in the most advanced patient population tested with an AR-directed therapy

Safety profile acceptable for potential use in frontline settings

Exploring a fast-to-market, biomarker-driven strategy for accelerated approval in 2L+ mCRPC

Potential to address unmet patient need in 1L mCRPC and mCSPC (~45k patients)

Next ARV-110 update anticipated Q4 2020

Note: Data presented at ASCO 2020 and as of 4/20/20. mCRPC, metastatic castration-resistant prostate cancer; mCSPC, metastatic castration-sensitive prostate cancer

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Clinical-stage Oncology Programs: ARV-471

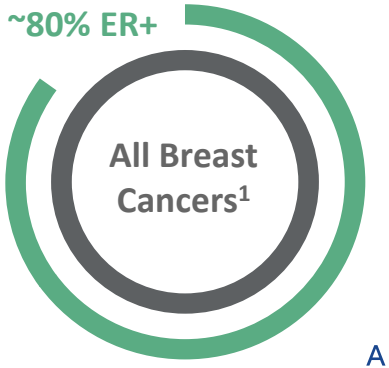
ARV-471 is Arvinas' ER degrader for patients with locally advanced or metastatic breast cancer

Breast cancer is the second most common cancer in women[1]

- **~276,000 women** are expected to be diagnosed with invasive breast cancer in the US in 2020[1]
- Metastatic breast cancer accounts for **~6% of newly diagnosed cases**[2]
- **80% of breast cancers** are estrogen receptor (ER) positive[3]
- **Fulvestrant has demonstrated the value of ER degradation** in breast cancer. However, after 6 months of fulvestrant treatment, **up to 50% of ER baseline levels remain**[4]

PROTAC® Degrader ARV-471

- ARV-471 is in development for the treatment of patients with ER+/HER2- locally advanced or metastatic breast cancer
- **Superior ER degradation and tumor inhibition** in preclinical studies
- Clinical strategy is to ultimately **develop ARV-471 for 1L settings**



[1]American Cancer Society; [2]Malmgren, J.A., Breast Cancer Res Treat (2018) 167:579–590; [3]National Cancer Institute, Hormone Therapy for Breast Cancer; [4]Gutteridge et. Al., Breast Cancer Res Treat 2004;**88** suppl 1:S177

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Phase 1 study of ARV-471 is a traditional “3+3” dose escalation study

Design

- “3 + 3” dose escalation; starting dose at 30 mg orally, once daily (po, qd) with food
- Dose increases dependent on toxicities: range 25% (if 1 DLT in 6 pts) to 100% (≤Grade 1 Adverse Events)

Inclusion criteria

- ER+/HER2- advanced breast cancer
- At least two prior endocrine therapies in any setting, and a CDK4/6 inhibitor
- Up to three prior cytotoxic chemotherapy regimens

Endpoints

Primary:

- Maximum tolerated dose and recommended phase 2 dose

Secondary:

- Pharmacokinetics
- Anti-tumor activity (RECIST, CBR)

Exploratory:

- Biomarkers
  - ER gene (ESR1) mutational status in ctDNA and/or tumor tissue
  - ER, Progesterone Receptor and Ki-67 levels in pre- and post-treatment tumor biopsies in patients with accessible tumor tissue

RECIST, response evaluation criteria in solid tumors; CBR, clinical benefit rate

In the first dose cohort of the 3+3 escalation study of ARV-471, exposure reached the predicted efficacious range

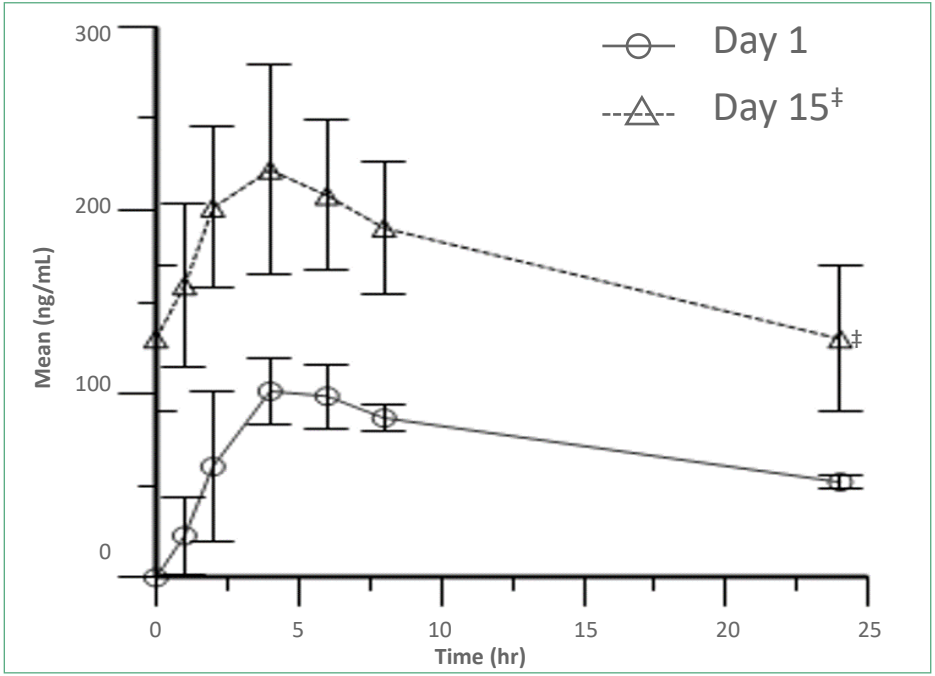
Data as of October 2019

Preclinical Efficacious Exposure Range

Dose (po, qd)	Mean AUC_{0-24} (ng*hr/ml)	Mean C_{max} (ng/ml)
3 mpk	658	84
10 mpk	2538	312
30 mpk	5717	962

30 mg Cohort Phase 1 Data

Day	AUC_{0-24} (ng*h/mL) Mean	C_{max} (ng/ml) Mean
Day 1	1690	109
Day 15	4100†	224



- Exposure at 30 mg entered the preclinical efficacious range associated with tumor growth inhibition
- No treatment-related AEs or DLTs were observed

T_{max} = 4 hrs
$t_{1/2}$ = ~24 hrs

† Day 15 AUCs calculated using imputed 24 hour values; ‡Day 15 24 hour value is imputed from time zero

ARV-471 is a potential first- and best-in-class ER degrader for ER+ locally advanced or metastatic breast cancer

Strong clinical profile[1]:

- Early evidence of ER degradation in the Phase 1 dose escalation
- No DLTs; dose escalation continues
- Dose-proportional pharmacokinetics

Superior ER degradation and tumor inhibition in preclinical studies

Fast-to-market strategy with potential indication in 2L+ ER+/HER2- mBC

Potential expansion to 1L ER+ breast cancer (~50k patients) in combination with CDK4/6i

Next ARV-471 update anticipated Q4 2020

[1]As of 5/29/2020.
DLT, dose-limiting toxicity

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Preclinical Programs

For recently introduced targets, PROTAC® protein degraders are likely to differentiate from other drug modalities

Target	Differential Biology Based on the Tenets of PROTAC® Degraders
BCL6 *Transcription factor implicated in B cell lymphomas*	Target scaffolding function of BCL6
KRAS *Oncogenic cell growth regulator*	Target "undruggable" KRAS mutants (e.g., G12V, G12D)
Myc *Oncogenic transcription factor driving tumor cell proliferation*	Directly degrade "undruggable" Myc vs. other indirect approaches
HPK1 *Suppressor of T cell activation; immuno-oncology target*	Address potential scaffolding function
mHTT *Key target for Huntington's disease*	Selectively degrade mutant huntingtin (mHTT) protein

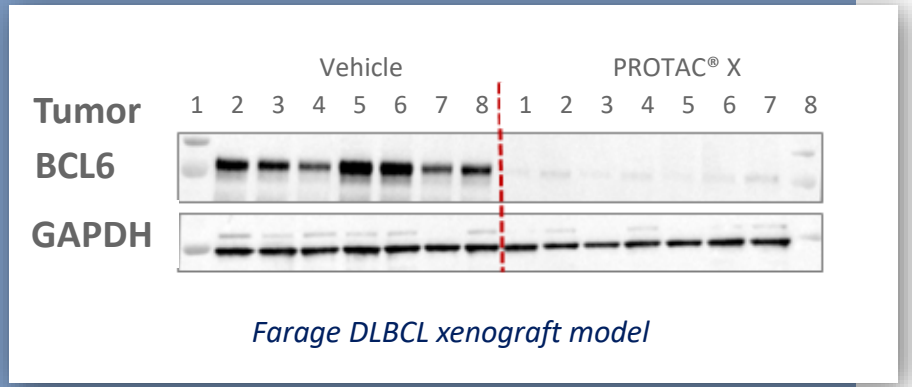
Detail follows

Arvinas' BCL6 program is aiming for an oral, best-in-class targeted therapy for B-cell malignancies

BCL6

- Most B cell lymphomas are dependent on constitutive or deregulated expression of BCL6, a transcriptional repressor of:
  - Cell cycle checkpoints
  - Terminal differentiation
  - Apoptosis
  - DNA damage response
- PROTAC® degradation would address the scaffolding function of BCL6

After oral dosing, PROTAC® X achieved ≥95% degradation of BCL6 *in vivo*



Farage DLBCL xenograft model

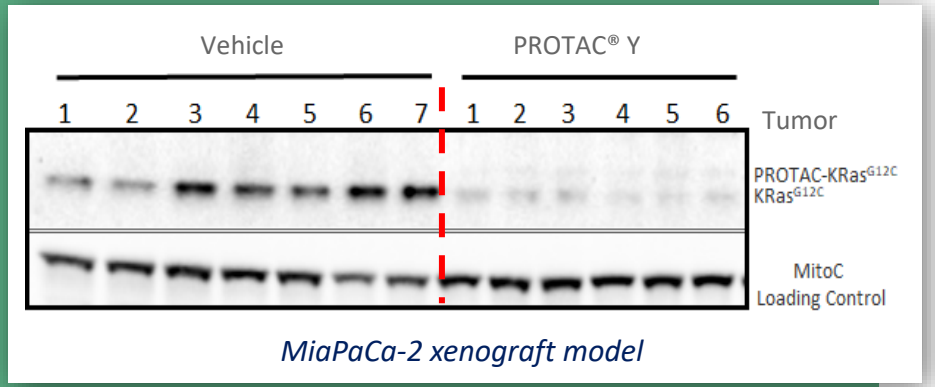
Optimizing* in vivo *tumor growth inhibition activity and selecting a candidate to take forward with anticipated IND in 2022

We are taking a comprehensive approach to degrading KRAS

KRAS

- KRAS is the most frequently mutated gene in human cancer and is a classic "undruggable" target due to its lack of deep "pockets"
- We are creating pan-KRAS mutant, in addition to mutant-specific (e.g., G12D and G12V), degraders
- 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*

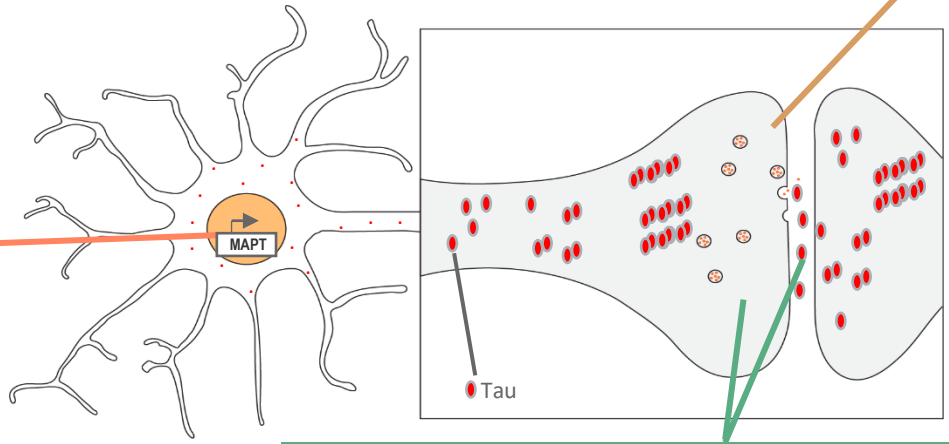


MiaPaCa-2 xenograft model

Leveraging learnings from KRAS G12C development to accelerate other KRAS degraders' development with anticipated IND in 2023

Mutant-specific PROTAC® degraders may reduce intra- and extracellular tau, creating a strong opportunity in neuroscience

PROTAC degraders may overcome the limitations of other platforms, including antisense oligonucleotides (ASO) and monoclonal antibodies (Ab)



ASO

- Degrades mRNA, impacting intra- and extracellular tau
- Does not discriminate between wild type and pathologic tau
- Requires intrathecal dosing

Ab

- Blocks only extracellular pathologic tau
- IV dosing results in only 0.5% in CSF

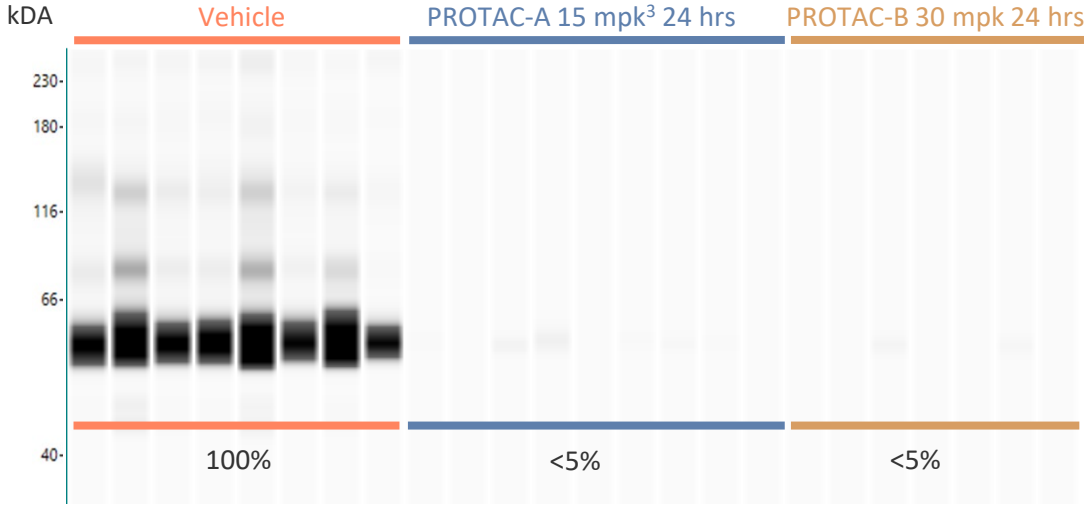
PROTAC *Potential*

- Reduce intra- and extracellular pathologic tau
- Discriminate between wild type and pathologic tau
- Oral administration with BBB biodistribution

ASO, antisense oligonucleotide; Ab, antibody; CSF, cerebrospinal fluid; BBB, blood-brain barrier

In vivo, tau-directed PROTAC® degraders eliminate >95% of pathologic tau in the brain following parenteral administration

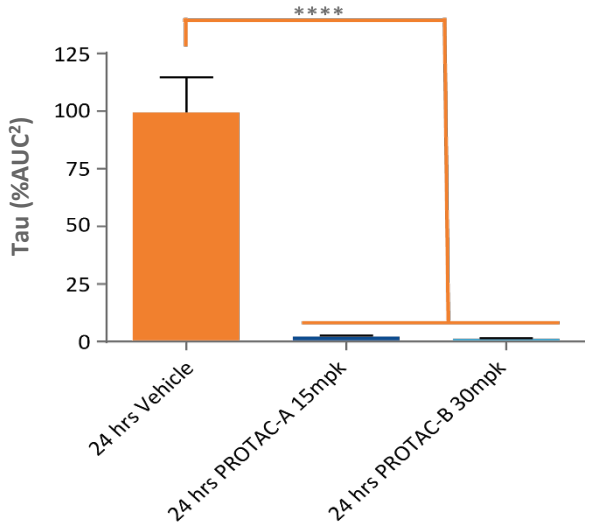
Tau Detection (protein capillary electrophoresis)



24 hours post dose:

- >95% of pathologic tau is degraded
- No significant change in total soluble tau 24 h post dose (data not shown)

Pathologic tau in Tg2508[1] mouse cortex

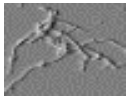


1 Tg2508 is a murine pathologic tau model (P301L). 2 AUC, area under the curve; 3 mpk, milligrams per kilogram
**** Tukey's multiple comparisons test P < 0.0001

ARVINAS

Tau-directed PROTAC® protein degraders inhibit *ex-vivo* tau seeding

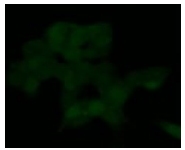
Tau Seeding Reporter Assay



Tau Seed
(Pre-formed fibrils[2] or Cortex Lysates[3])

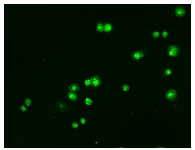
Modified from Holmes et al., 2014

24h



- Tau /- PFF Seeding

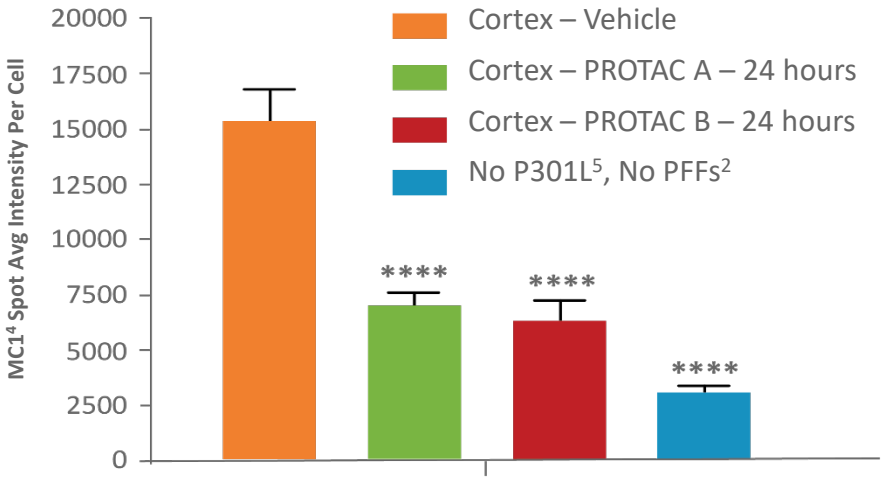
OR



+Tau /+ PFF Seeding

Dox-inducible Tau P301L CHO-K1[1]

PROTAC Treatment Inhibits Tau Seeding *ex-vivo*[4]



1 Tau P301L CHO-K1 is a cell line expressing a doxycycline-inducible tau mutation linked to FTDP-17 (frontotemporal dementia and parkinsonism linked to chromosome 17). 2 Pre-formed fibrils (PFFs) are used to "seed" tau aggregation. 3 Cortex lysates are from Tg2508 mice. 4 MC1 is an antibody that detects a pathologic conformation of tau. 5 "No P301L," no doxycycline induction.

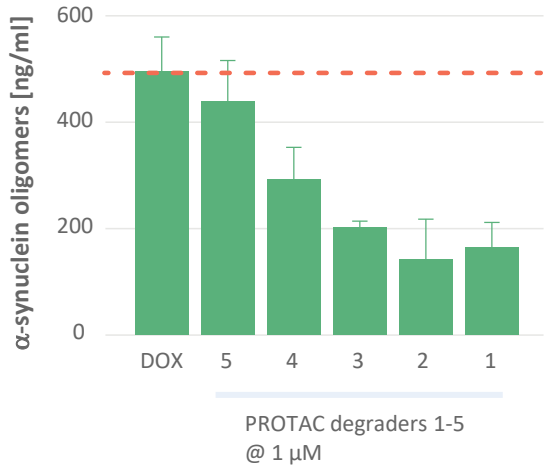
**** Tukey's multiple comparisons test $P < 0.0001$. Comparisons are between the Cortex-Vehicle value and all other values (individually)

ARVINAS

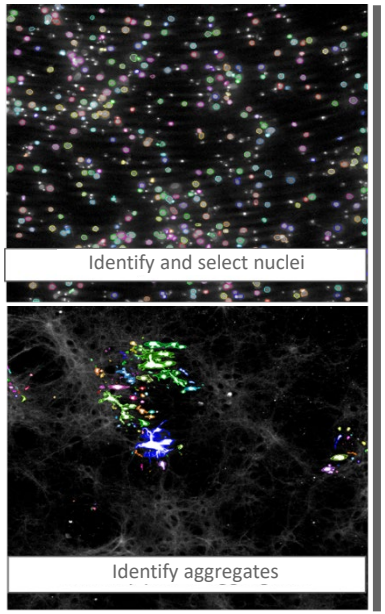
Oligomer-specific PROTAC® molecules degrade human α-synuclein aggregates in primary rat neurons

PROTAC molecules degrade oligomeric α-synuclein species

PROTAC degraders were identified that specifically remove oligomeric α-synuclein

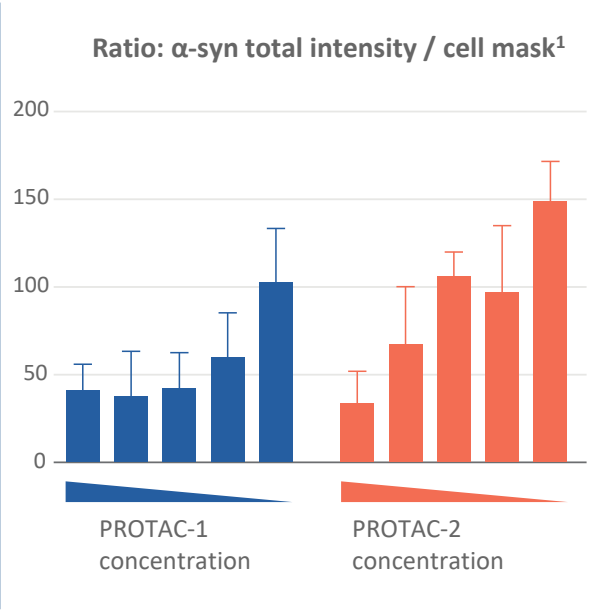


PROTAC-1 and PROTAC-2 degrade α-synuclein aggregates in primary rat neurons expressing human α-synuclein



Neuronal α-synuclein +PFF induction assays[1]

Intensity and area features of α-synuclein aggregates calculated



1 Assay is of primary rat neurons expressing A53T human α-synuclein, with pre-formed fibrils (PFF) added or not. In the absence of α-synuclein-specific PROTAC degraders, α-synuclein forms aggregates induced by PFFs (green fluorescence in cellular images). When PROTAC degraders specific for oligomeric α-synuclein are added, the ratio of oligomeric α-synuclein:cell mask (background fluorescence) is decreased (right panel).

ARVINAS

Corporate Overview

Arvinas is 170+ colleagues strong and growing, benefitting from the experience and resources of the Connecticut biotech sector

Mission

We invent PROTAC® protein degraders designed to destroy disease-causing proteins and improve the lives of patients suffering from cancer, neurological disorders, and other serious diseases

Core Values

Pioneering, Excellence, Community, & Commitment

People

- 170+ highly experienced drug development professionals in New Haven, Connecticut
- 200+ FTEs at contract research organizations

Bioscience in Connecticut

- 39,000 employees across 2,500 companies[1]
- Strong academic base for R&D partnerships

[1]BioCT 2019 Report (link)

Financial snapshot

$243 Million

Cash, cash equivalents, and marketable securities (*as of 6/30/20)*

Guidance

Expect cash, cash equivalents, and marketable securities to fund planned operations into 2022

39.2 Million

Common shares outstanding (*as of 7/24/20)*

Analyst Coverage[1]

BMO, Cantor, Citibank, Evercore, Goldman Sachs, Guggenheim, HC Wainwright, Oppenheimer, Piper Sandler, Roth, Wedbush

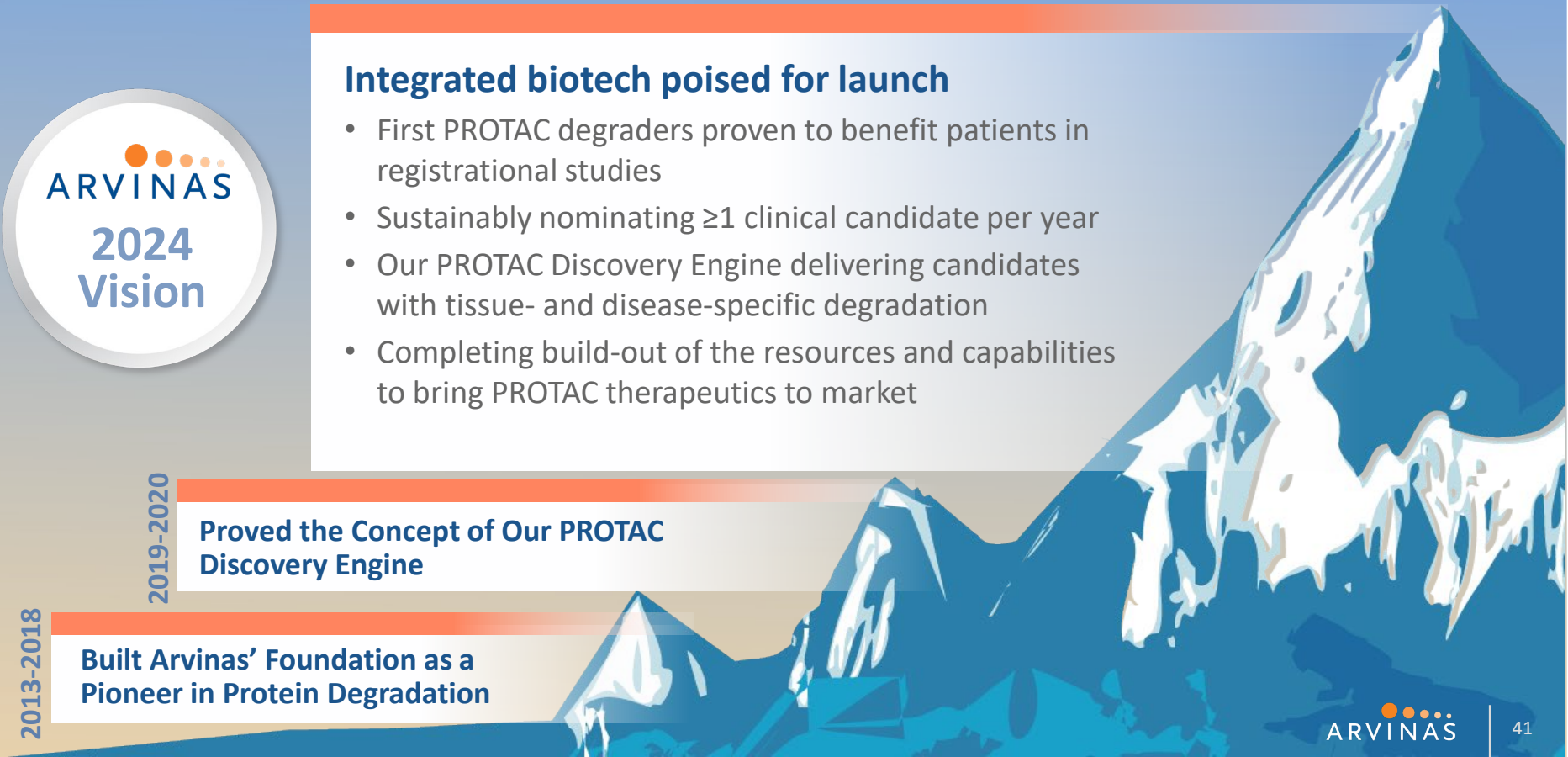
1 The foregoing list includes the names of all brokerage firms known by the company as of 10/12/20 to have analysts covering the company. This list may not be complete and is subject to change as firms add or delete coverage. Please note that any opinions, estimates or forecasts regarding the company made by these analysts are theirs alone and may not represent the opinions, estimates or forecasts of the company.

Arvinas 2024 Vision: Ascending to new heights in bringing the benefits of PROTAC® degraders to patients

ARVINAS
2024 Vision

Integrated biotech poised for launch

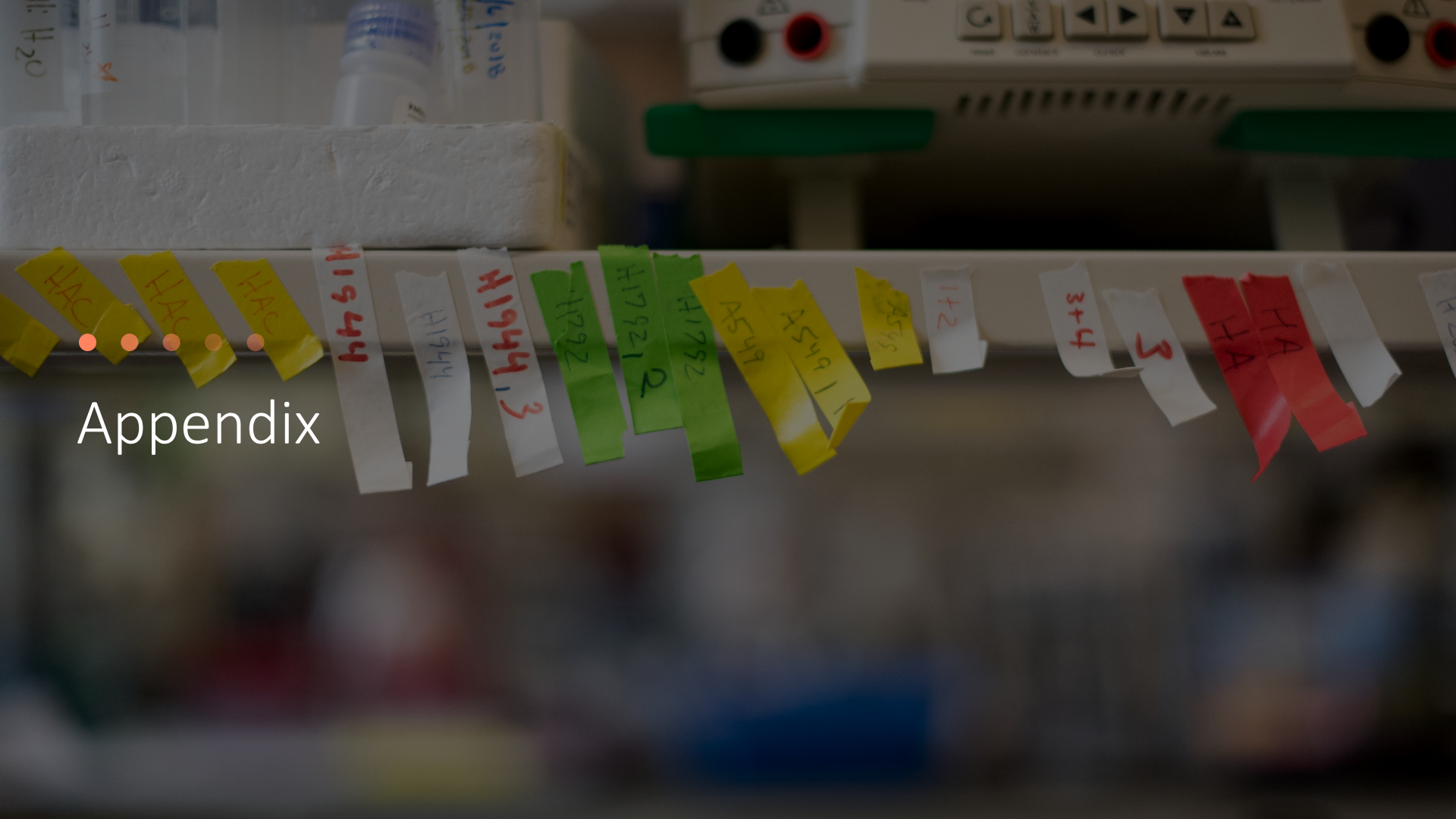
- First PROTAC degraders proven to benefit patients in registrational studies
- Sustainably nominating ≥1 clinical candidate per year
- Our PROTAC Discovery Engine delivering candidates with tissue- and disease-specific degradation
- Completing build-out of the resources and capabilities to bring PROTAC therapeutics to market



2019-2020 Proved the Concept of Our PROTAC Discovery Engine

2013-2018 Built Arvinas' Foundation as a Pioneer in Protein Degradation

Thank You!


HAC
HAC
HAC
H1N1
H1N1
H1N1
H1N1
H1N1
H1N1
A549
A549
1+2
3+4
3
HA
HA

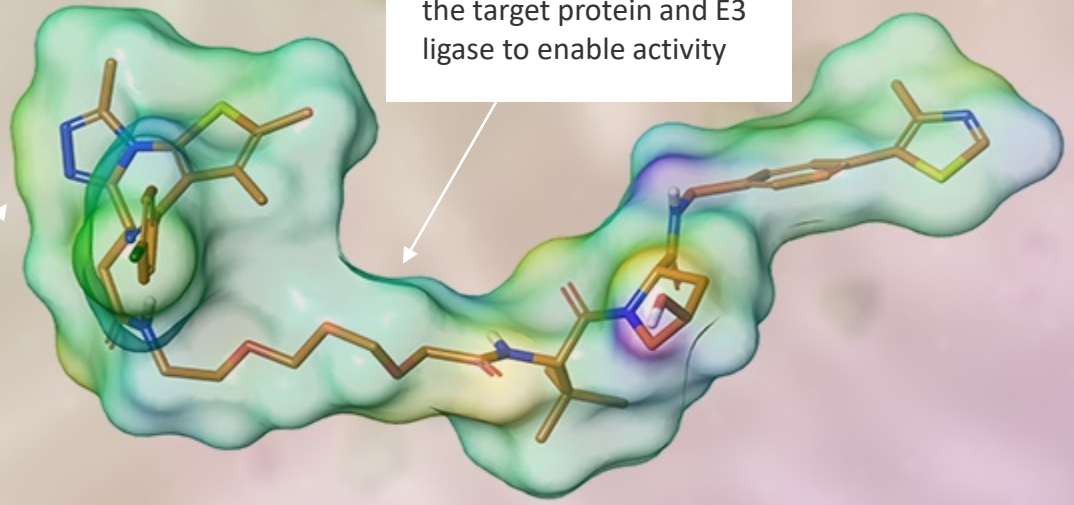
Appendix

What is a PROTAC® protein degrader?

A proteolysis-targeting chimera (PROTAC) degrader is a chimeric, modular small molecule engineered to induce the degradation of disease-causing proteins by the ubiquitin-proteasome system

A linker region orients the target protein and E3 ligase to enable activity

Protein ligand domain (“warhead”) targets a specific protein



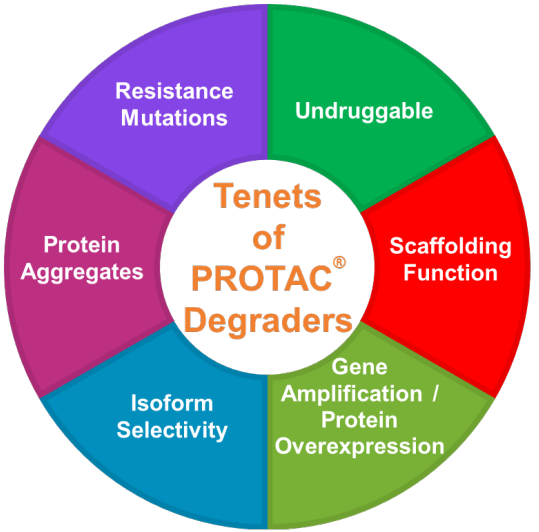
Ligase ligand recruits a specific E3 ubiquitin ligase

All three regions of the PROTAC degrader play a role in the specificity and potency of target degradation

Our target selection strategy is designed to build the optimal portfolio of PROTAC® protein degraders

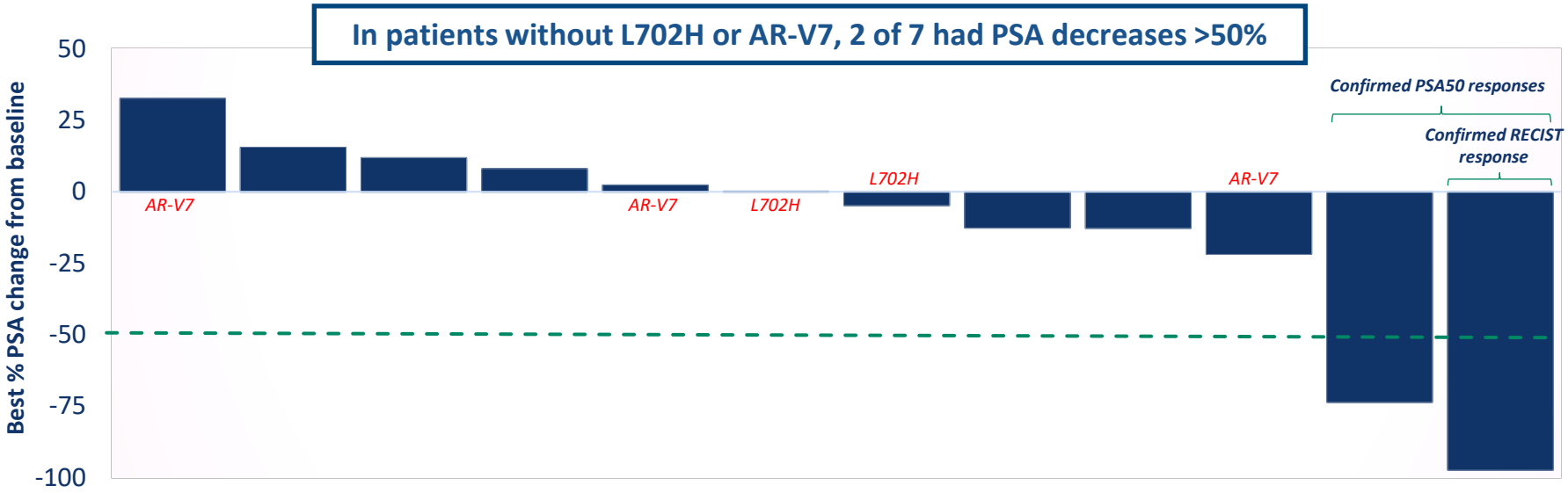
Guiding principles for our portfolio strategy

- **Focus on targets where degradation of the disease-causing protein will result in differential biology and patient outcomes versus other modalities**
- **Build on our established expertise and capabilities in oncology, immuno-oncology, and neuroscience**
- **Create a diversified, risk-balanced portfolio of validated and undruggable targets**



AR biomarker status and best % PSA change in patients at ≥140 mg (excludes DLT patient; N=12)[1]

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



AR Status:	AR-V7	Wildtype Amplif.	Wildtype	Wildtype Amplif.	AR-V7	L702H T878S W742C	L702H	Wildtype	Wildtype	T878A Amplif. AR-V7	T878A H875Y	T878A H875Y

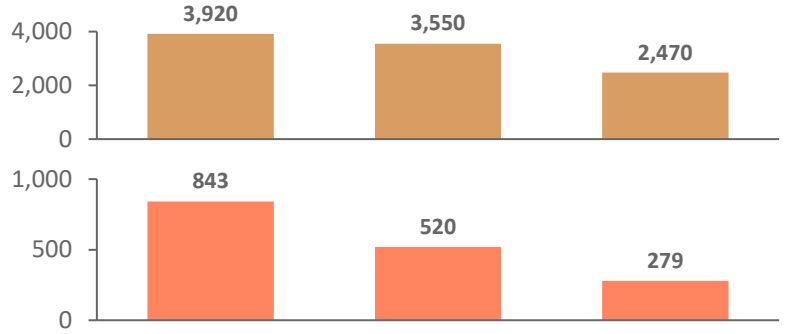
[1]One patient discontinued after 2 weeks due to DLT associated with rosuvastatin; AR status based on assays from Epic Sciences, Foundation Medicine (RUO), and OHSU/KDL

PROTAC® degraders can be engineered to cross the blood-brain barrier (BBB)

- Micromolar rodent brain exposure achieved after peripheral (IV) administration
- Brain-to-plasma ratio >0.5 achievable with PROTAC degraders

PROTAC	Species	Dose (mg/kg)	[Plasma 1h] (ng/ml)	[Brain 1h] (ng/g)	B/P ratio
1	mouse	10	309	227	0.8
2	mouse	10	843	3920	4.7
3	mouse	10	285	1425	5.0

Over a 4-hour time course, PROTAC degraders are more durable in the brain than in plasma

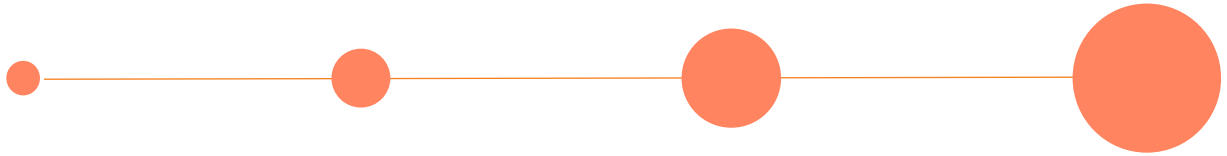


Time (hours)	1	2	4
B/P Ratio	4.7	6.8	8.9

Arvinas' approach in neuroscience reduces risk while proving the concept of protein degradation

Prove the concept with PROTAC® degraders in defined populations while pursuing larger, multifactorial indications

Conceptual



Tau	FTDP (~3K)	Progressive supranuclear palsy (~20K)	ApoE4 AD risk allele carriers (600-900K)	Alzheimer's (~6M)[1]
α-synuclein	Synuclein mutations, e.g., duplication/ triplication (~4K)[2]	Multiple systems atrophy (~50K)[3]	GBA PD risk allele carriers (~500K)	Parkinson's (~1M)[4]

FTDP, frontotemporal dementia and parkinsonism; GBA, glucocerebrosidase gene; AD, Alzheimer's disease; PD, Parkinson's disease

1 Alzheimer's Association: "2019 Alzheimer's Disease Facts and Figures" video; https://www.alz.org/alzheimers-dementia/facts-figures

2 Kowal. Movement Disorders 2013, 28: 311-319; Nishioka. Intechopen 2011

3 NINDS; https://www.ninds.nih.gov/Disorders/Patient-Caregiver-Education/Fact-Sheets/Multiple-System-Atrophy

4 Parkinson's Foundation: http://parkinson.org/Understanding-Parkinsons/Causes-and-Statistics/Statistics

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