ARVINAS

The PROTAC® Company

PROTAC® Targeted Protein Degraders

A New Therapeutic Modality

Wedbush Conference

August 2019

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 and our collaborative partnerships, the potential benefits of the Bayer joint venture in the agricultural field, 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 a Phase 1 clinical trial for ARV-110, successfully initiate and conduct a Phase 1 clinical trial for 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.

ARVINAS

Mission statement

To *create a new class of medicines* that

degrade pathogenic proteins

to *treat diseases with serious unmet medical need*

and *improve human health*

Oncology; Neurology

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

Novel PROTAC® (proteolysis-targeting chimera) degrader platform

- Built with foundational technology and foremost experts from Yale University
- Combines the strengths of small molecule inhibitors and gene-based medicines

Full worldwide development and commercialization rights for lead programs

- ARV-110 - Metastatic castration-resistant prostate cancer; Phase 1 initiated 1Q19, and received "Fast Track" designation from FDA in May 2019
- ARV-471 - Estrogen receptor-positive / HER2-negative locally advanced or metastatic breast cancer; FDA "Safe to Proceed" received 2Q19, and Phase 1 initiation expected 3Q19
- Brain-penetrant PROTAC programs targeting tauopathies and α-synucleinopathies

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

- Up to $2.1B in potential milestones plus tiered royalties
- Partnerships across broad set of therapeutic areas and a JV for agricultural applications

Strong cash and IP positions

- First targeted protein degradation company to IPO (NASDAQ: ARVN; September 2018)
- ~$211M in proforma cash, cash equivalents, and marketable securities as of 6/30/19[1]
- Broad platform IP, complemented by specific product IP

Team built for success

- Strong leadership team with unparalleled protein degrader development experience
- World-class Board and scientific advisors, including Craig Crews (PROTAC inventor)

1 Proforma for the Bayer license and collaboration agreement and private placement which closed on July 16, 2019 and provides $51.5 M in cash to Arvinas

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High potential PROTAC® pipeline, focused on cancer and neurology[1]

1 Pipeline as of August 4, 2019
2SP, progressive supranuclear palsy
3 MSA, multiple systems atrophy

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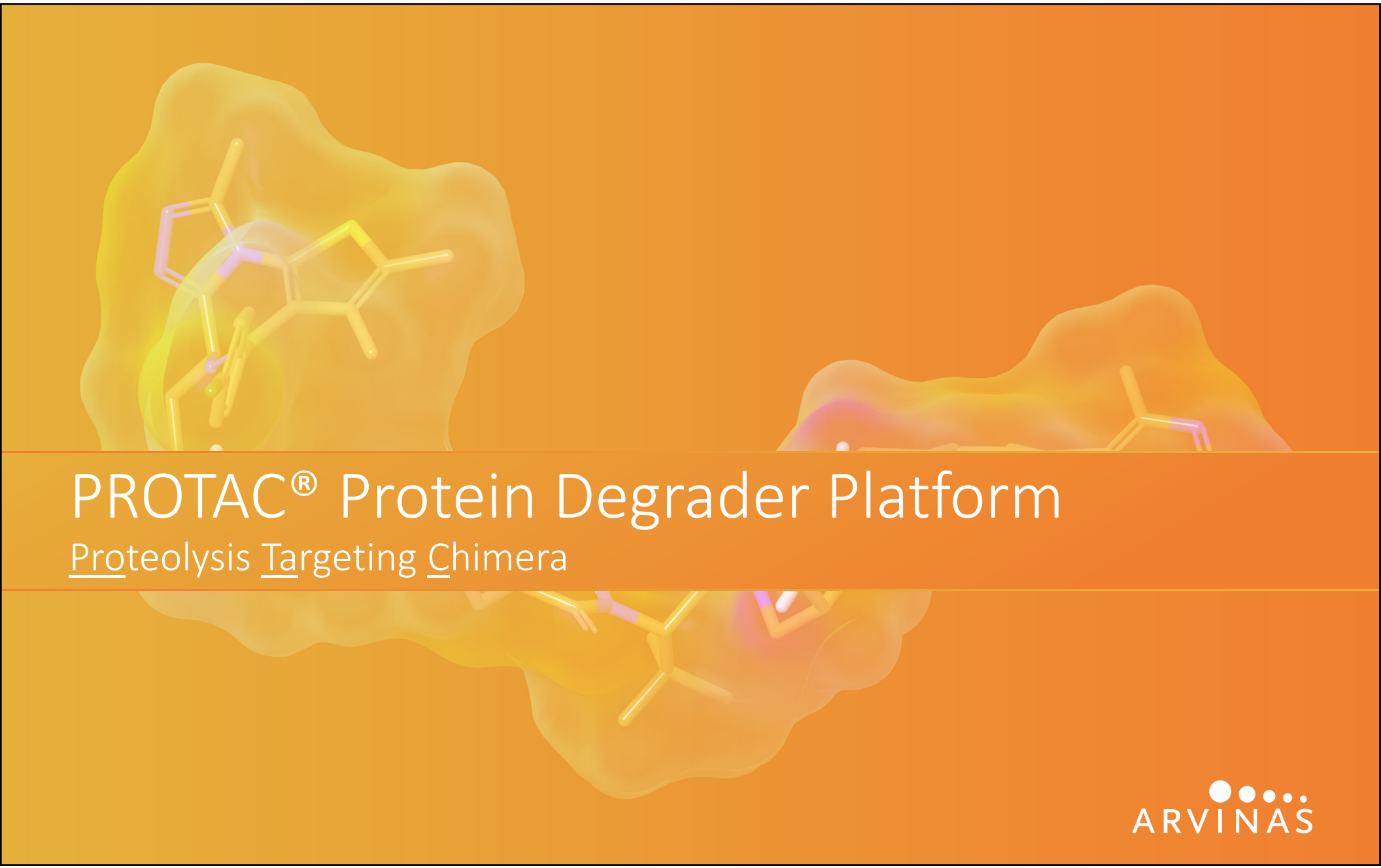

PROTAC® Protein Degrader Platform

Proteolysis Targeting Chimera

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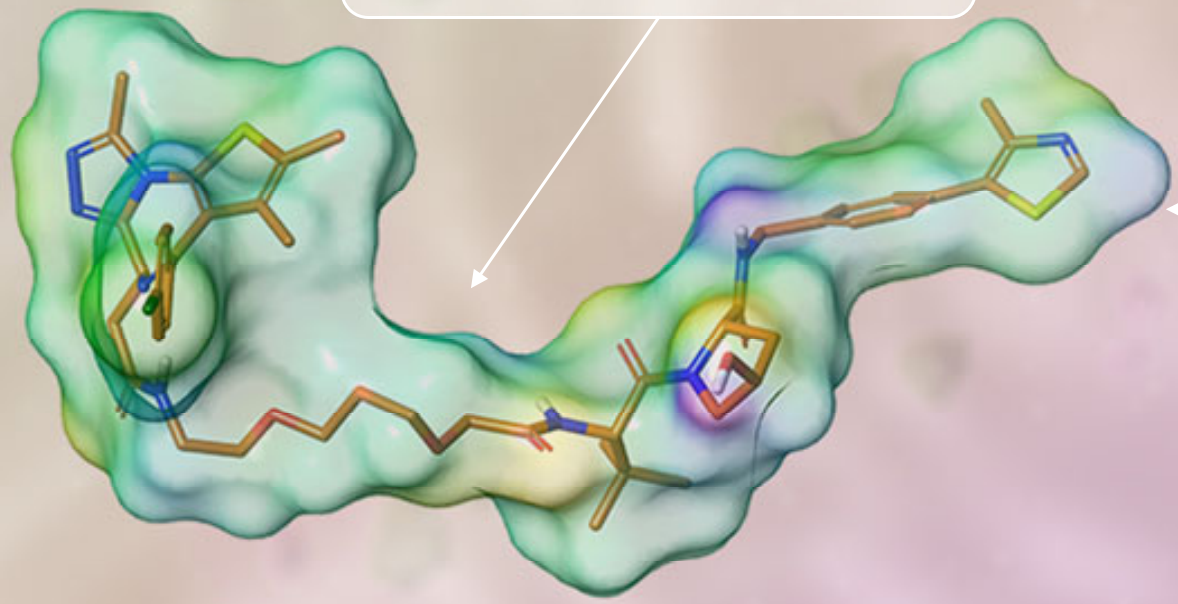
What is a PROTAC® protein degrader?

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

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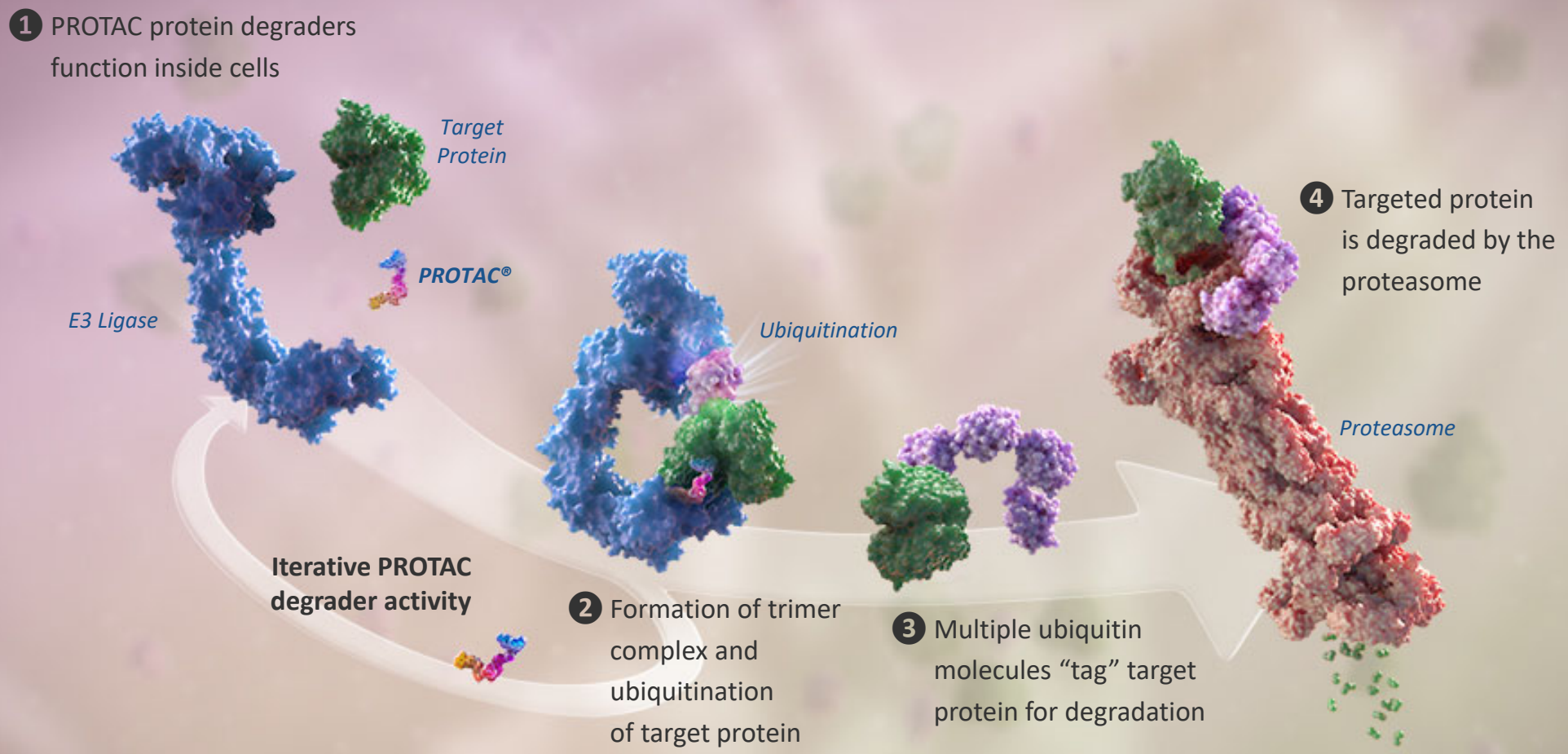
PROTAC® protein degraders harness the ubiquitin-proteasome system to induce the degradation of disease-causing proteins



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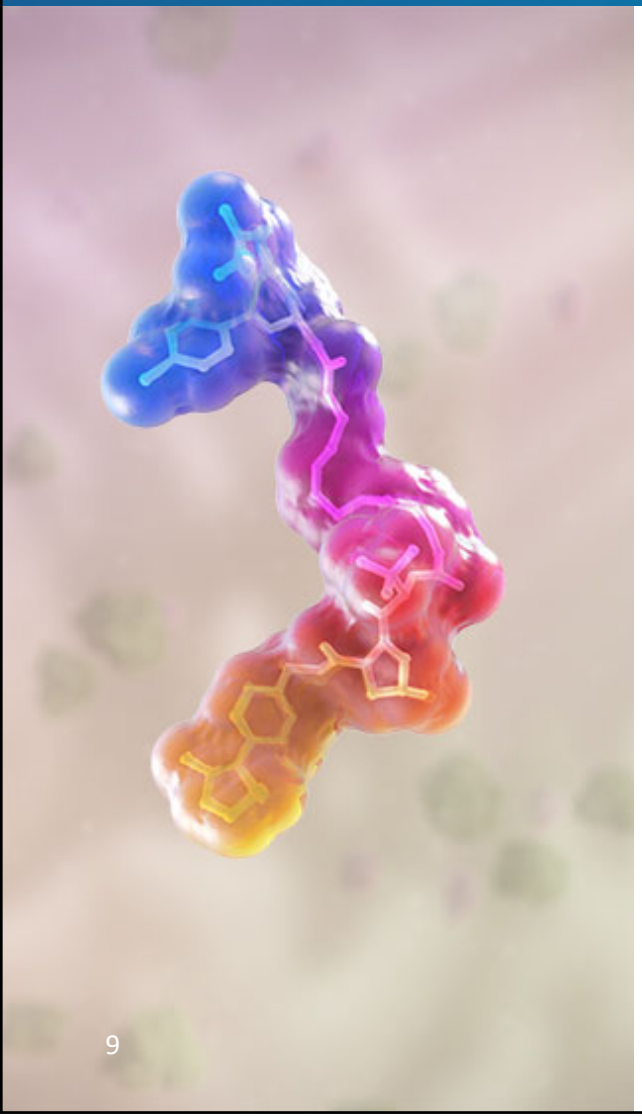
PROTAC® protein degraders combine the advantages of gene-based medicines with the benefits of small molecule therapies



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	✓	✓	✗

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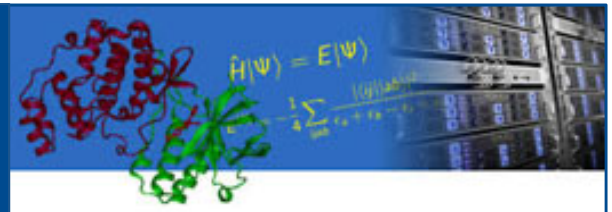
Arvinas' technology and expertise enable effective hit ID and optimized development candidates

Capabilities



Computational Chemistry
- Molecular Dynamics Simulations
- Site Directed Mutagenesis
- GPU-enabled

Structural Information
- X-Ray, SAR
- Ligand Optimization

Medicinal Chemistry
- Fit-for-Purpose PROTAC™ Matrix
- Rapid Synthesis
- Diversity

Discovery Process

Target Selection
- Differential Biology/Profile
- Chemical Equity
- Biophysics

Predictive Hit Identification
- >90% Success Rate
- Efficient, Rapid Process
- Linker SAR Generated

Clinical Candidate Optimization
- PROTAC-Specific Design Metrics
- Holistic Optimization Strategy
- Advanced bRo5 Profiles (oral)

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Research and Development Programs

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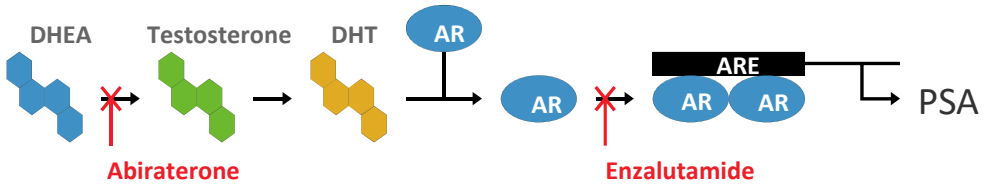
ARV-110 is Arvinas' AR degrader for men with metastatic castration-resistant prostate cancer (mCRPC)[1]

Androgen Receptor (AR) Activity Drives Prostate Cancer

- 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**)
- **Resistance mechanisms** to abiraterone and enzalutamide include:
  - **AR gene amplification** (40-60% of patients)
  - **AR gene enhancer amplification** (>70% of patients)
  - **AR point mutations** (~15% of patients)
  - **Intra-tumoral androgen production**

PROTAC® Degrader ARV-110

- First-in-class AR degrader being tested in men with metastatic castration-resistant prostate cancer who have progressed on standards of care (enzalutamide, abiraterone)
- In preclinical models, overcomes known resistance mechanisms to enzalutamide and abiraterone
- Highly selective degradation of AR
- **Phase 1 clinical trial initiated 1Q19; preliminary data expected 4Q19**
- **Received FDA "Fast Track" designation in May 2019**



1. According to the American Cancer Society, prostate cancer is the second leading cause of cancer death in men in the U.S. (~174k diagnosed/yr1); 35-45k new incidences of mCRPC in the U.S. each year

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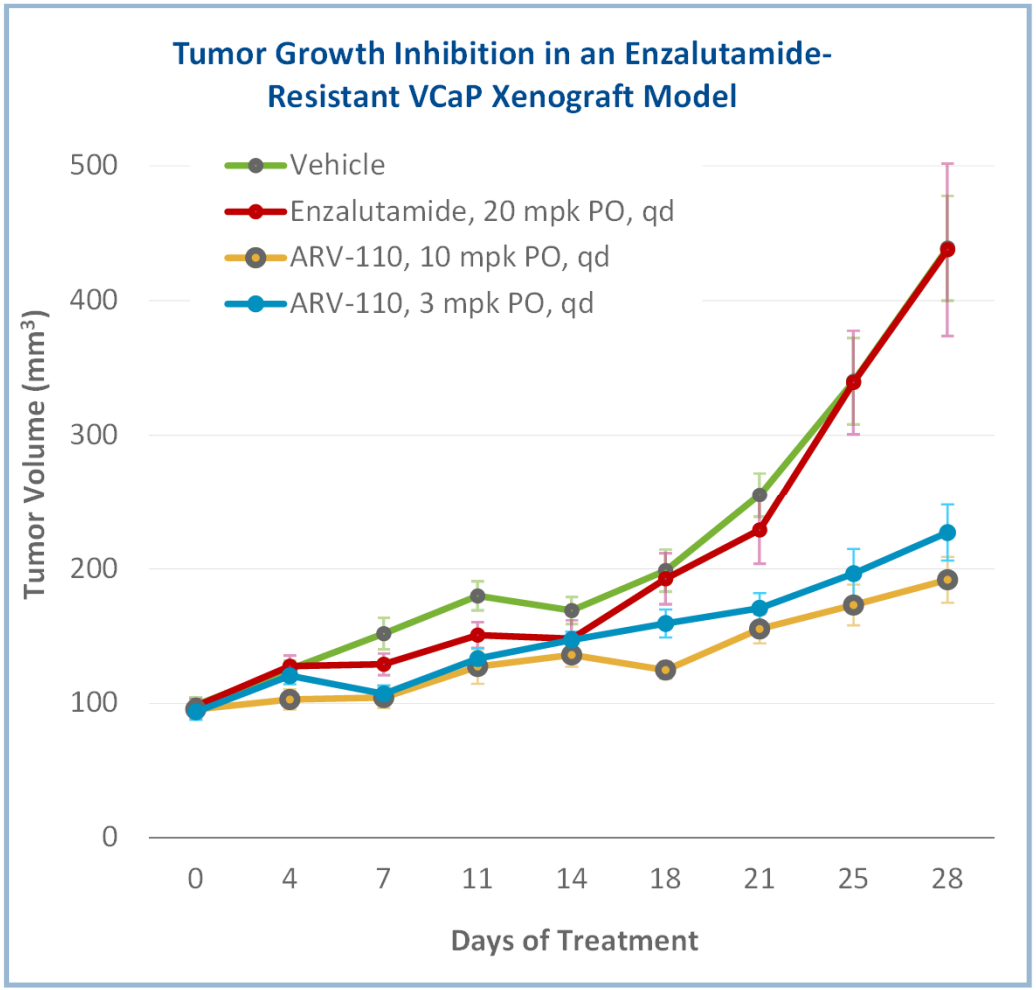
ARV-110 inhibits tumor growth in an *in vivo* model of acquired enzalutamide resistance

- *In vivo* mouse xenograft model of **acquired enzalutamide resistance** developed at Arvinas
- In this model, VCaP tumors acquired resistance to enzalutamide after being continuously propagated in castrated, enzalutamide treated mice for ~3 years
- Daily and orally delivered ARV-110 significantly inhibited tumor growth (*at right*)
  - 10 mpk ARV-110: 70% tumor growth inhibition



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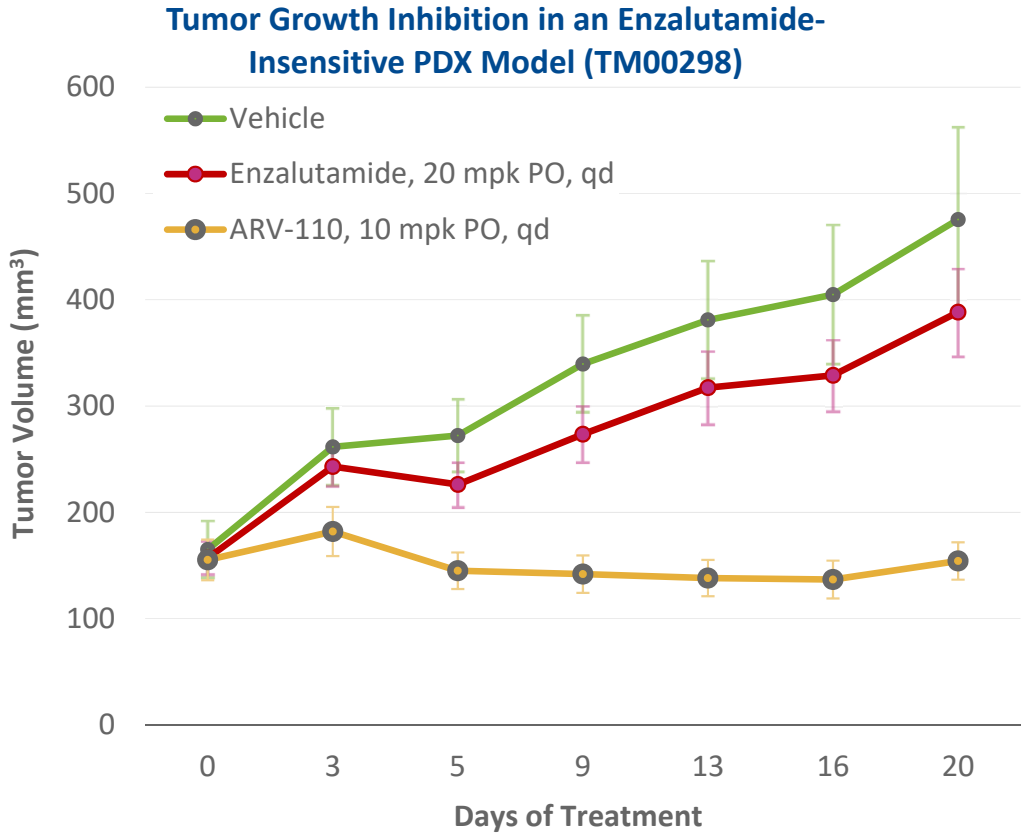
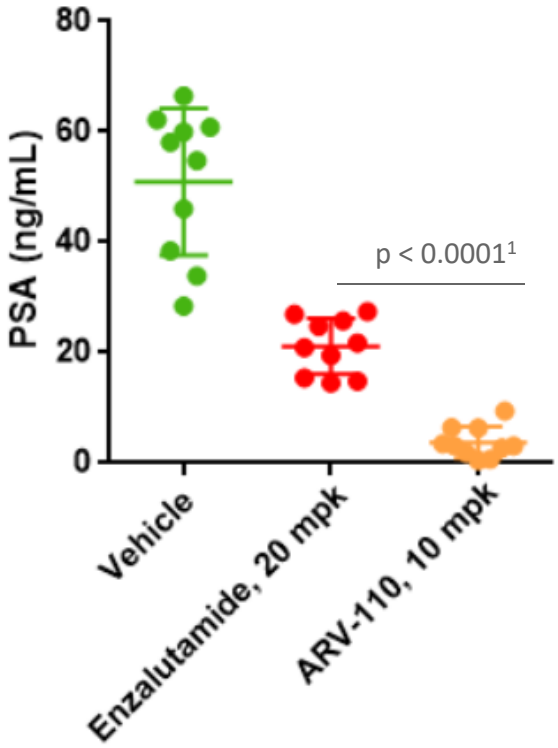
ARV-110 demonstrates efficacy and plasma PSA reduction in an enzalutamide-insensitive patient derived xenograft model

- Orally delivered ARV-110 significantly inhibited tumor growth in these **intrinsically enza-insensitive** tumors (TGI: 100%)



- **Plasma PSA levels** following ARV-110 treatment **significantly decreased** vs. mice treated with vehicle or enzalutamide



1 p value refers to ARV-110 vs. enzalutamide

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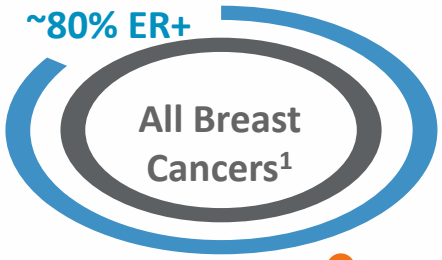
ARV-471 is Arvinas' ER degrader for patients with locally advanced or metastatic breast cancer

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

- ~268,000 women are expected to be diagnosed with invasive breast cancer in the US in 2019[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
- 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+ locally advanced or metastatic breast cancer
- Investigational New Drug (IND) clearance from FDA in 2Q19
- **Ph 1 trial expected to begin 3Q2019**
- After Phase 1 dose escalation, a Phase 1b trial in combination with CDK4/6 inhibitor is planned



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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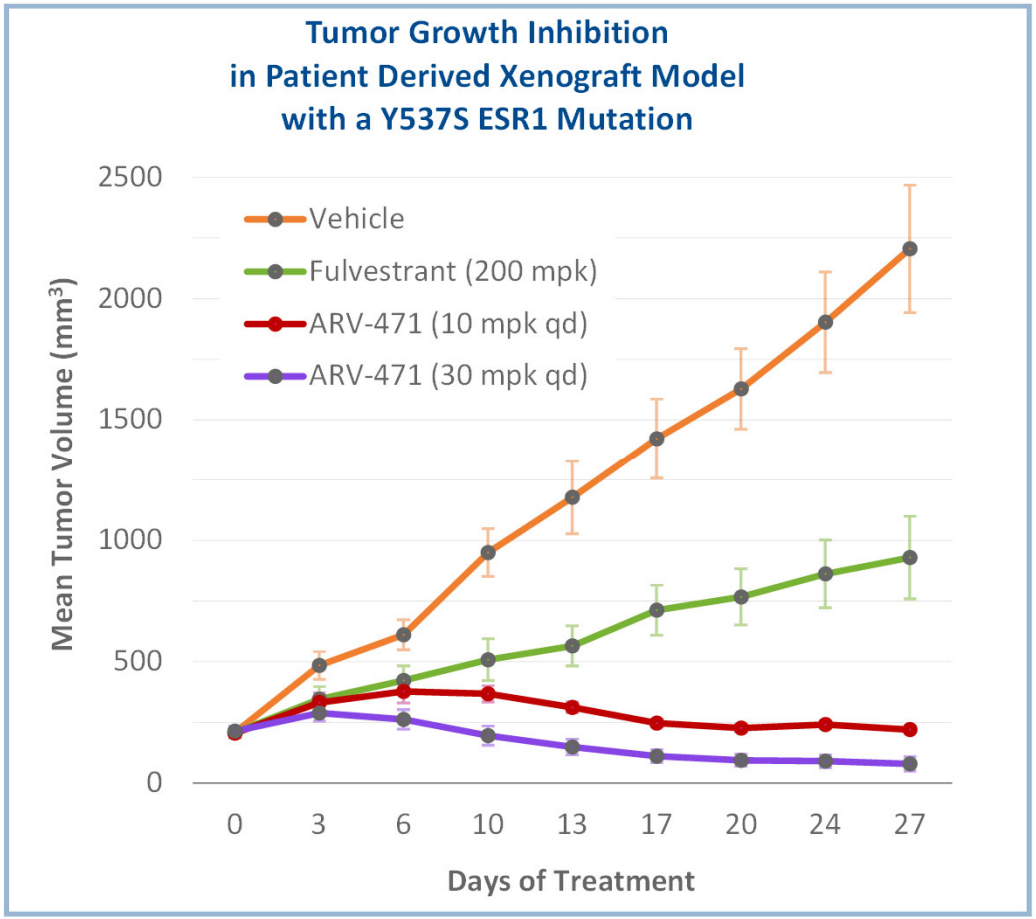
ARV-471: superior tumor growth inhibition versus fulvestrant in a Y537S (ER gene mutation) PDX model

ARV-471 *In Vivo* Preclinical Development

- Oral, daily dose of ARV-471 inhibited tumor growth by 99% at 10 mpk and 106% at 30 mpk in an **ESR1 mutant PDX model** (*at right*)
- Superior inhibitor of tumor growth compared to fulvestrant[1]
- In corresponding quantitative western blots, ER is reduced by 79% and 88% in the 10 mpk and 30 mpk arms, respectively, vs. 63% for fulvestrant



1 Fulvestrant schedule: 2x weekly x2 / q7dx2

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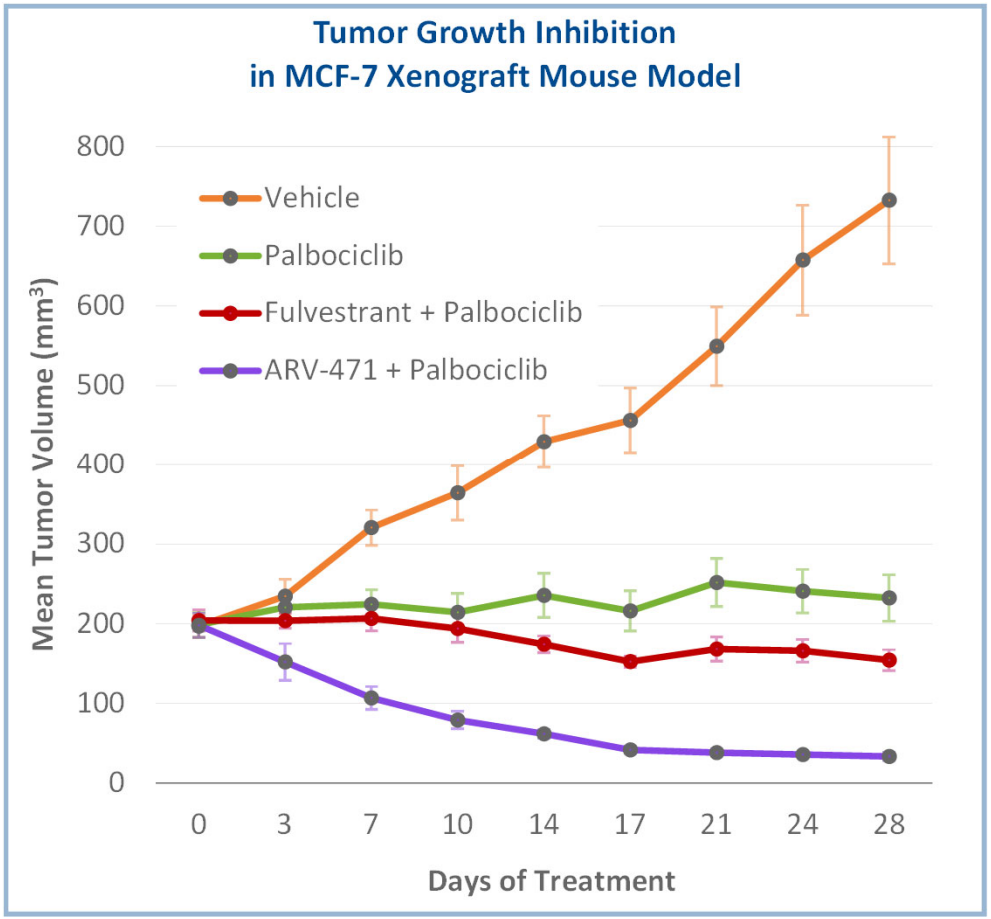
In combination with palbociclib, ARV-471 exhibits superior tumor shrinkage versus fulvestrant

ARV-471 *In Vivo* Preclinical Development

- Achieved significant tumor shrinkage in combination with palbociclib (131% TGI) in an MCF-7 xenograft mouse model
  - In all 10 mice in experiment, tumors reduced by >80%
- Superior tumor shrinkage (in combination with palbociclib) compared to fulvestrant (108% TGI)



Palbociclib arm: 60 mpk po qd; 94% TGI.
Fulvestrant + Palbociclib arm: Fulvestrant 200 mpk sc biwx 2, qwx 3 + palbociclib 60 mpk po qd; 108% TGI
ARV-471 + Palbociclib arm: ARV-471 30 mpk po qd + palbociclib 60 mpk po qd; 131% TGI

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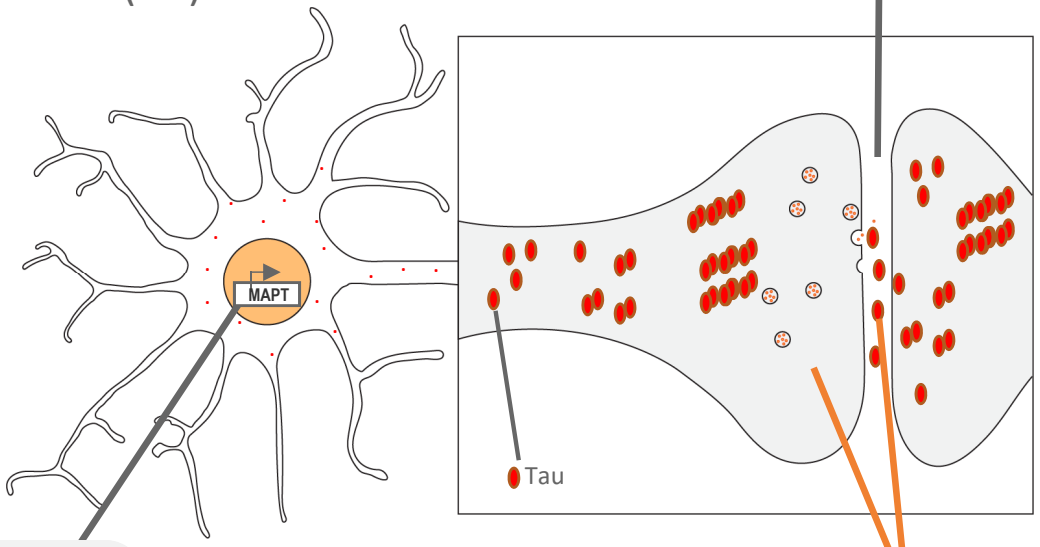
Mutant-specific PROTAC® degraders may reduce intra- and extracellular tau, creating a strong opportunity in neuroscience

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

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



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

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

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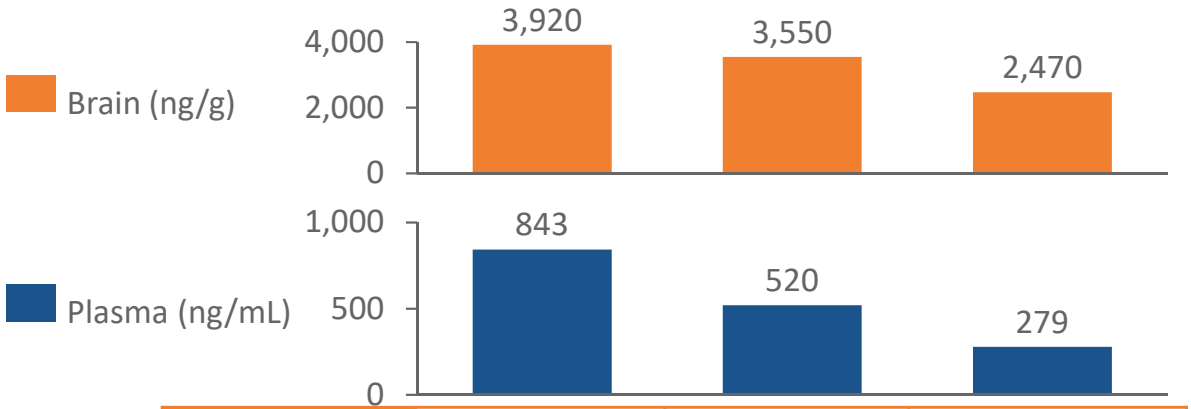
Our PROTAC® degraders can be engineered to cross the blood-brain barrier (BBB)

- 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

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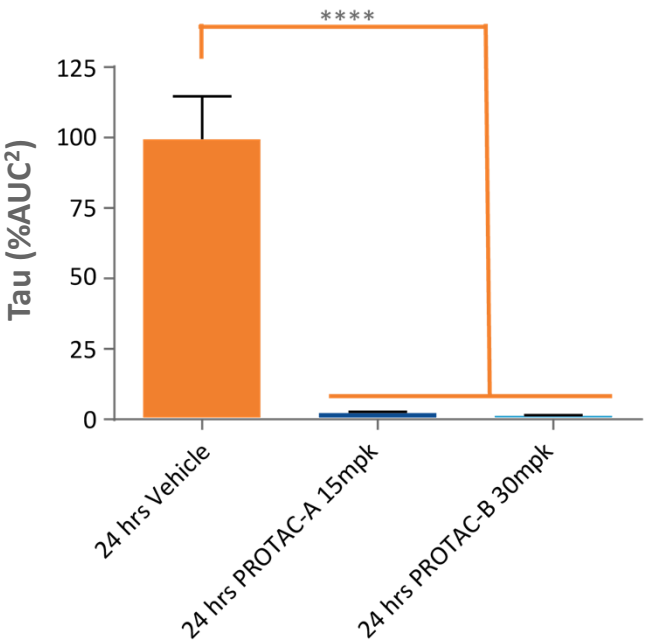
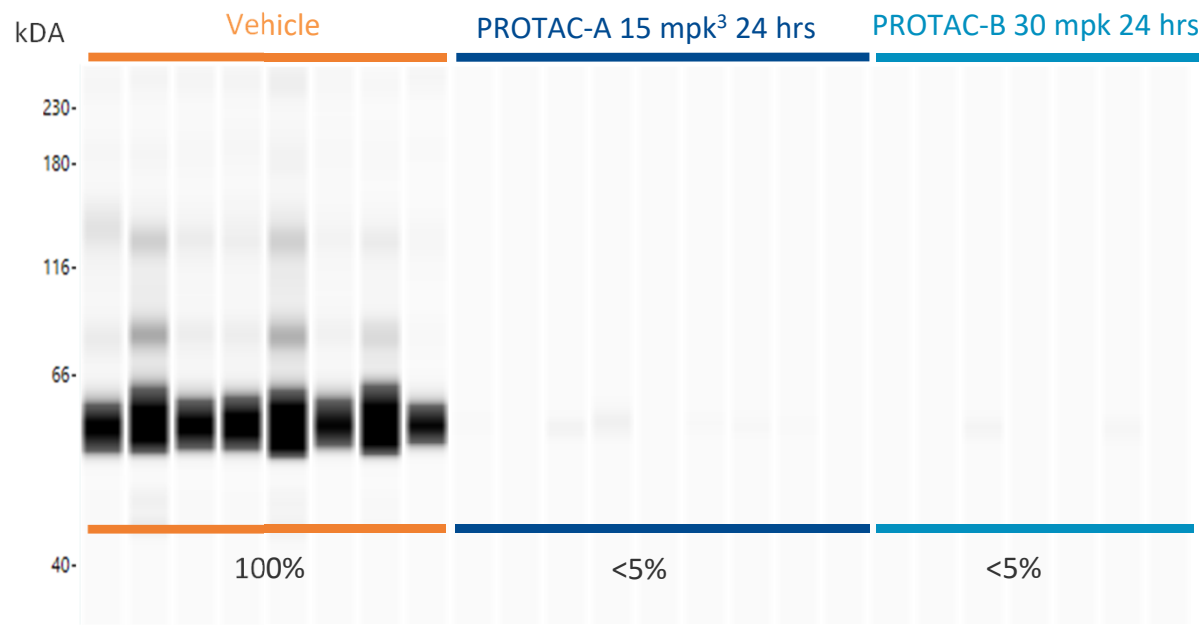
In vivo, tau-directed PROTAC® degraders eliminate >95% of pathologic tau following parenteral administration

Pathologic tau in Tg2508[1] mouse cortex



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)

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

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Arvinas' approach in neuroscience

Approach: 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; "2018 Alzheimer's Disease Facts and Figures." Alzheimer's and Dementia; V.14; No.3; 2018; p36

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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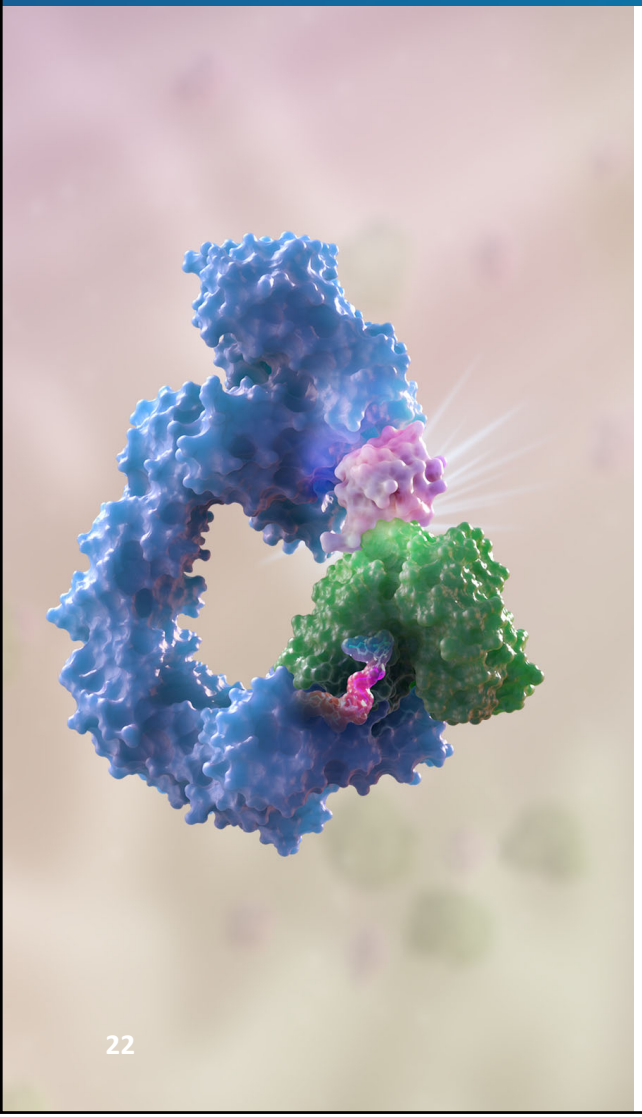
Future targets and platform expansion



"Undruggable" Targets

- The ~80% of proteins not addressable by small-molecule inhibition may be degradable by PROTAC® protein degraders
- These targets are difficult to drug because they lack active sites or accessible binding pockets
- PROTAC degraders do not require tight target binding in order to be effective; the MOA is event-driven rather than occupancy-driven
- Arvinas has multiple, classically undruggable targets in the pipeline; expect to share further data in 2020 and beyond

Platform Expansion

- Identifying and leveraging tissue and disease-specific E3 ligases
- Enhanced prediction of degradation selectivity

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Corporate Overview

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Financial snapshot

$211 Million[1]

Proforma cash, cash equivalents, and marketable securities

as of 6/30/19

Guidance

Expect cash, cash equivalents, marketable securities, and Bayer proceeds to fund planned operations into 2H21

33.5 Million[2]

Proforma common shares outstanding

as of 6/30/19

Analyst Coverage

Cantor Fitzgerald, Citibank, Evercore ISI, Goldman Sachs, Piper Jaffray[3]

1 Proforma for the Bayer license and collaboration agreement and private placement proceeds of $51.5M, which closed on July 16, 2019

2 Proforma for the Bayer private placement of 1.3 M shares of common stock, which closed on July 16, 2019

3 The foregoing list includes the names of all brokerage firms known by the company as of 8/12/19 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.

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Strategic partnerships are validating our PROTAC® protein degrader technology

Genentech
A Member of the Roche Group

September 2015

(expanded in November 2017)

- Target discovery deal
- Upfront, development, and commercial milestone aggregate payments in excess of $650M
- Tiered royalties

Pfizer

December 2017

- Target discovery deal
- Upfront, development, and commercial milestone aggregate payments up to $830M
- Tiered royalties

BAYER

June 2019

- Pharma target discovery deal, including cardiovascular, gynecologic, and oncologic disease
- Agricultural JV (50:50 share)
- Private equity placement
- ~$115M in total upfront and committed funds

Potential for nearly $2.1 billion in milestones

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The PROTAC® Company:
Leading in protein degradation therapeutics

- ARV-110: Believed to be the first PROTAC degrader in the clinic
- Leading platform and product IP, driven by nearly two decades of PROTAC protein degradation research
- First to publish data on orally available PROTAC protein degraders
- Leadership team with experience getting drugs to market
- Strong financial position to advance the platform and product candidates

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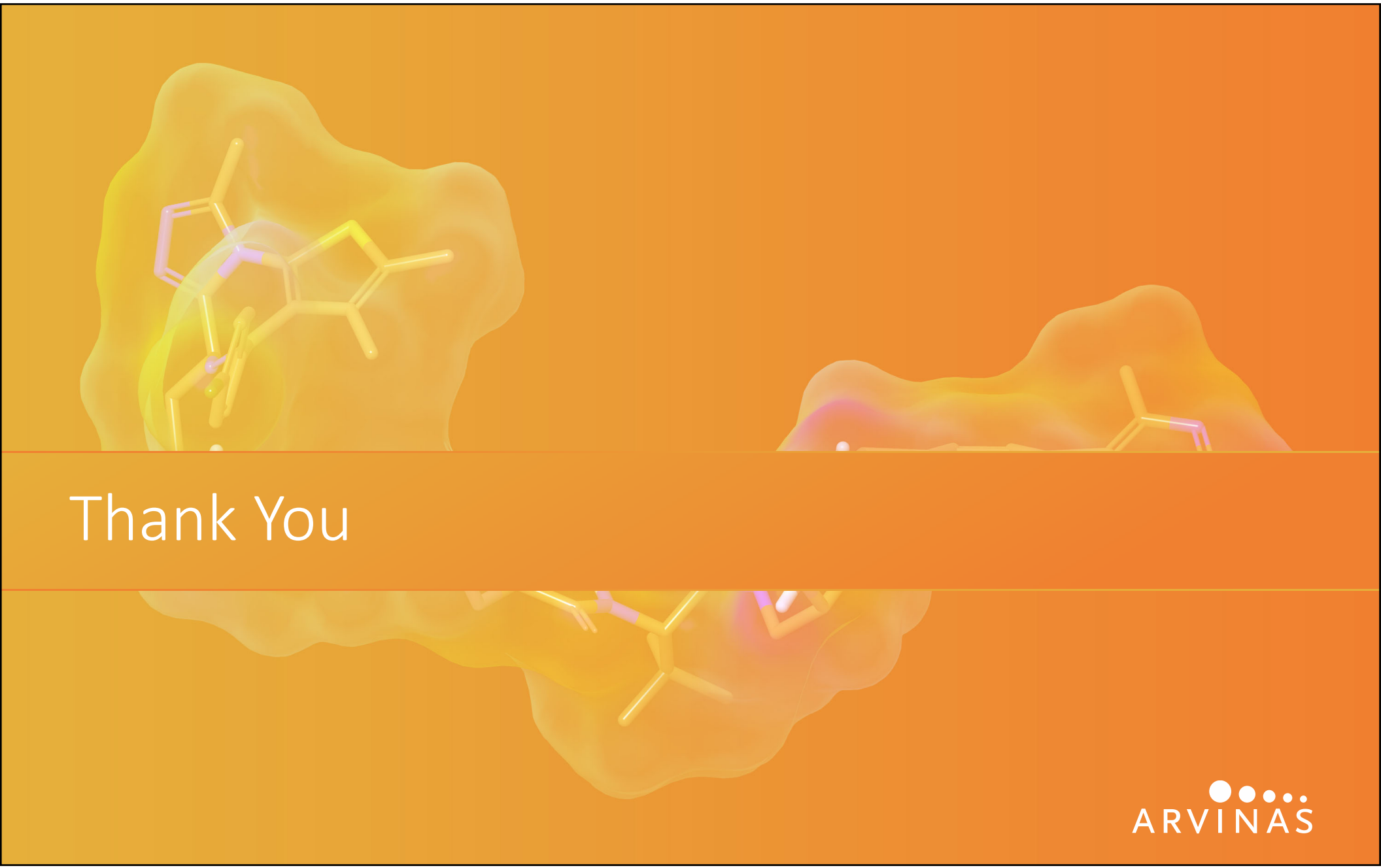
Thank You
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Appendix

The need for a new approach

Our understanding of the proteins responsible for causing certain diseases has greatly outpaced innovation

Up to 80% of the human proteome is still considered “undruggable” and not addressable via small molecule inhibitors

Current treatment options for many diseases are suboptimal and/or suffer from rapid onset of resistance

Nucleic acid-based approaches (siRNA, gene therapy) lack many of the drug-like properties of traditional small molecules

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Our strategic approach to proving and delivering a novel technology platform

Clinically validate the PROTAC® protein degrader concept with well-defined targets

Prioritize additional targets where degradation has the potential to be superior to existing modalities

Treat patients with diseases inaccessible to current therapies by degrading "undruggable" targets

- Invest in our pipeline and our platform and grow our IP to expand our leadership in protein degradation
- Selectively collaborate with strong partners to expand the impact of PROTAC protein degraders into new areas

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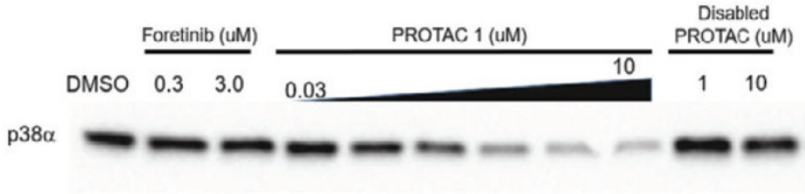
Weak or promiscuous ligands can be converted into potent and selective PROTAC® degraders

When developed into PROTAC degraders, weak binders can become potent degraders

- Foretinib is a relatively weak binder to p38α
- PROTAC 1 is a foretinib-based PROTAC degrader with a p38α binding affinity of 11 μM
- Despite its 11 μM binding affinity, PROTAC 1 has a DC_{50} of 210 nM[1]
  - Based on experience, optimization of potency better than 210 nM is likely

A PROTAC degrader based on foretinib has a nanomolar DC_{50} despite a 11 μM binding affinity

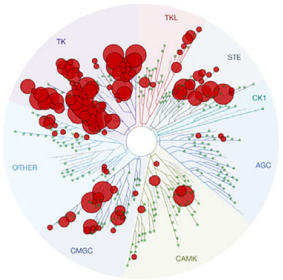


DC_{50} = 210 nM[1]

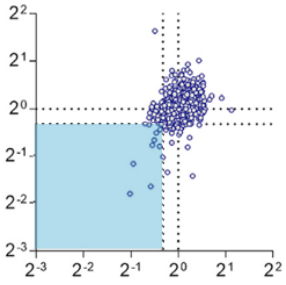
When developed into PROTAC degraders, promiscuous ligands can become selective degraders

- Foretinib binds to 133 protein kinases (*left panel*)
- In cells treated with a foretinib-based PROTAC degrader, only a small subset of cellular proteins are degraded (*blue-shaded quadrant of the right panel*)

Binds 133 Kinases



Degrades <10 Proteins



Source: Bondeson et al., 2018, Cell Chemical Biology
1 DC_{50} = Half-maximal degradation concentration

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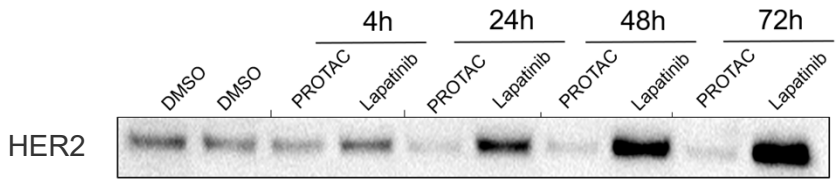
Potential advantages of PROTAC® protein degraders over inhibitors

Overcome Target Protein Overexpression

PROTAC degraders can disable this common tumor resistance mechanism

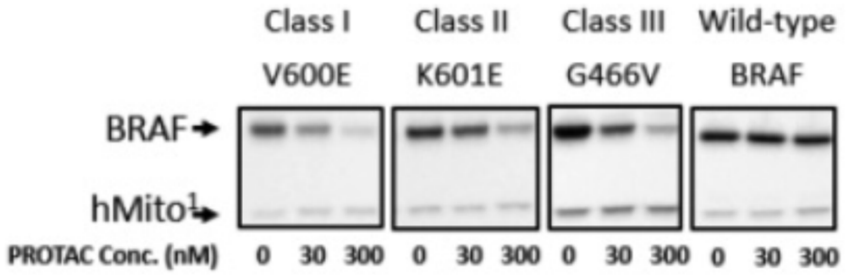
- Lapatinib alone results in HER2-overexpression, but a PROTAC created with lapatinib as the "warhead" degrades natural and overexpressed HER2
- HER2 degraded despite increased RNA levels



Selectively Eliminate Mutated Proteins

PROTAC degraders can differentiate between mutant and wild type proteins

- The three mutants of BRAF shown (V600E, K601E, G466V) differ from the wild type by a single point mutation, but are degraded by a BRAF-targeted PROTAC that spares the wild type

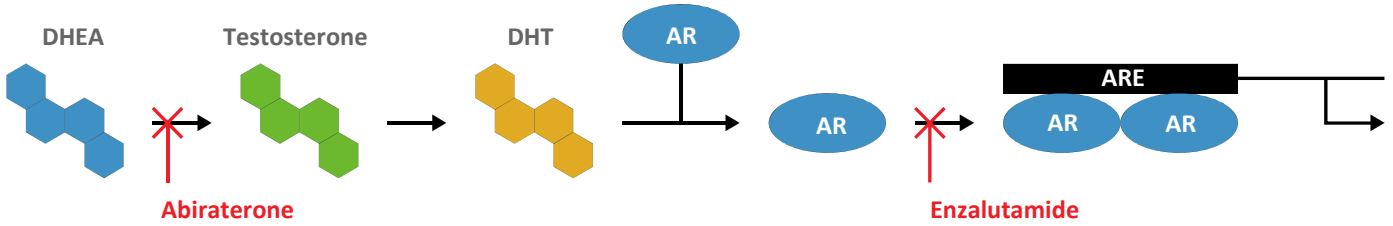


1 hMito is a protein this particular PROTAC degrader is not targeted to degrade, and is included as a loading control

ARVINAS

Androgen receptor and metastatic castration resistant prostate cancer (mCRPC)

Prostate cancer is the second leading cause of cancer death in men in the U.S. (~174k diagnosed/yr[1]); 35-45k new incidences of mCRPC in the U.S. each year



- Downstream PSA production
- Disease progression

Androgen Receptor (AR) Activity Drives Prostate Cancer[2]

- Current agents work by decreasing androgen levels (abiraterone) or blocking androgen binding to AR (enzalutamide)
- 15-25% of patients do not respond to abiraterone or enzalutamide (intrinsic resistance)

Acquired Resistance Mechanisms to Abiraterone and Enzalutamide

- AR gene amplification (40-60% of patients)
- AR gene enhancer amplification (>70% of patients)
- AR point mutations (~15% of patients)
- Intra-tumoral androgen production

In resistant patients, PSA levels rise, suggesting that AR remains the principal driver of disease

1. American Cancer Society
2. DHEA, dehydroepiandrosterone; DHT, dihydrotestosterone; AR, androgen receptor; ARE, androgen response element

ARVINAS

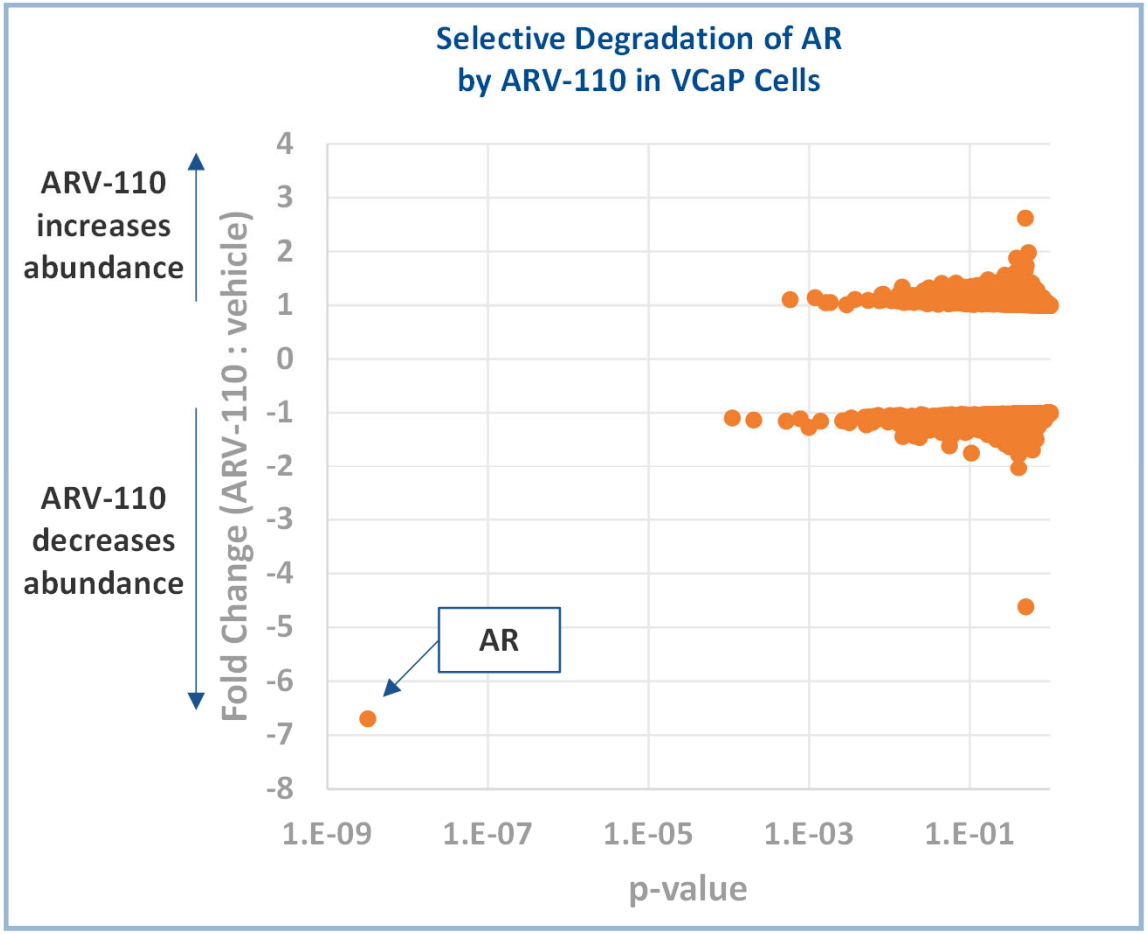
ARV-110 selectively degrades AR

Orally bioavailable androgen receptor-targeted PROTAC protein degrader

- ARV-110 is in development for the treatment of men with mCRPC who have progressed on abiraterone and/or enzalutamide
- Appears to overcome mechanisms of resistance to current standards of care
- **DC_{50} = 1 nM** in VCaP cells[1]

ARV-110 Selectively Degrades AR

- After 8 hours of treatment of VCaP cells with 10 nM ARV-110 *in vitro*, AR was the only degraded protein among the nearly 4,000 proteins measured
  - 85% D_{max}[2]
  - p-value: $3x10^{-9}$



1 VCaP, Vertebral Cancer of the Prostate
2 D_{max}, maximal degradation

ARVINAS

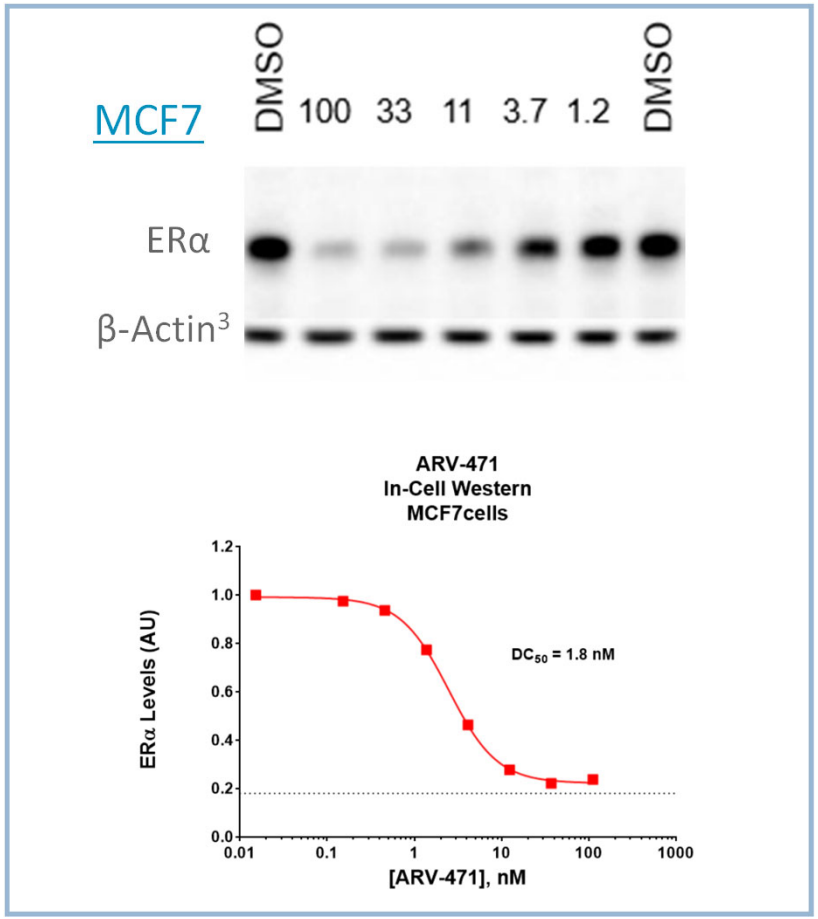
Our estrogen receptor-targeting PROTAC® degrader: ARV-471

Orally bioavailable estrogen receptor-targeted PROTAC protein degrader

- ARV-471 is in development for the treatment of patients with ER+ locally advanced or metastatic breast cancer
- Potential as both a single agent and in combination with CDK4/6 inhibitors

ARV-471 Degrades ER in ER+ Breast Cancer Cell Lines

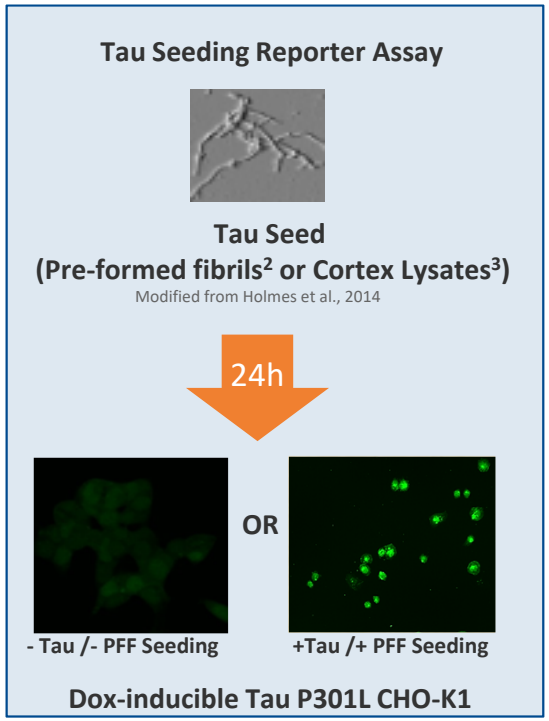
- ARV-471 induces ER degradation in multiple ER+ breast cancer cell lines, including MCF-7 cells and ESR1-mutant lines[1]
- **DC_{50} = 1.8 nM** in MCF7 cells[2]

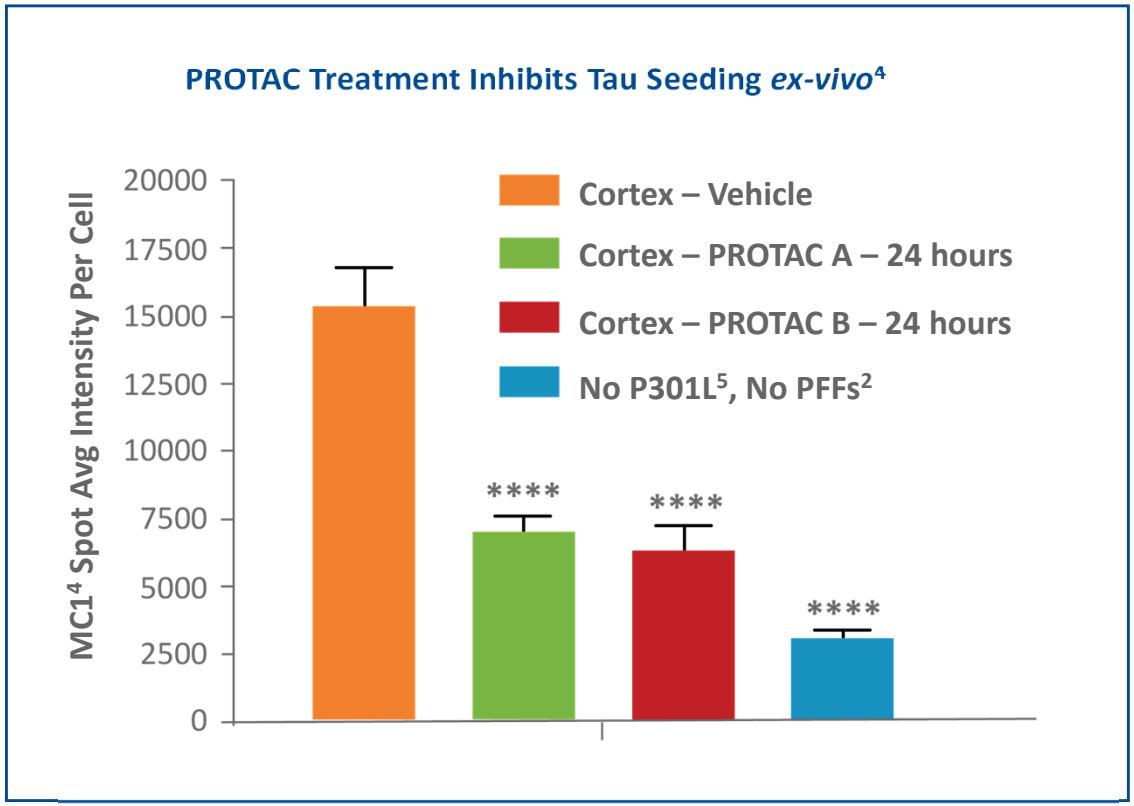


1 Also tested: MB-134-VI, T47D, D538G, Y537S, ZR-75-1, BT474, CAMA-1
2 DC_{50} = Half-maximal degradation concentration
3 Beta-actin is a protein ARV-471 and fulvestrant are not targeted to degrade, and is included as a loading control

ARVINAS

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





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

Arvinas / Bayer collaboration

In June 2019, Bayer and Arvinas announced a $110+ million partnership to develop human PROTAC® therapies and launch a separate joint venture (JV) to develop PROTAC® degraders for agricultural applications

Pharmaceutical collaboration and direct equity investment

- Focus on gynecology, oncology, and cardiovascular disease targets
- Upfront and committed funding exceeds $60 million (including equity investment)
- Over $685 million in potential milestone payments, plus commercial royalties

Agriculture-focused joint venture

- JV to develop agricultural products using PROTAC® degrader technology
- Potential for weed, pest, and disease control applications
- Over $55 million in committed funding by Bayer to JV
- Bayer and Arvinas share ownership and governance of the JV equally

Combined with Genentech and Pfizer, potential for nearly $2.1 billion in milestones

Seasoned leadership with expertise in advancing novel technologies

Leadership Team

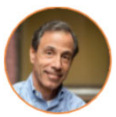
John G. Houston, PhD
President & CEO
Bristol-Myers Squibb · gsk

Sean Cassidy, CPA, MBA
Chief Financial Officer
CuraGen Corporation · Deloitte.

Ronald Peck, MD
Chief Medical Officer
gsk · TESARO (A GSK company) · Bristol-Myers Squibb

Ian Taylor, PhD
Chief Scientific Officer
Pfizer · Bayer

Angela Cacace, PhD
VP Neuro and Platform Biology
Fulcrum Therapeutics · Bristol-Myers Squibb



John A. Grosso, PhD
VP Chemistry, Mfg. & Controls
Bristol-Myers Squibb · vitae Pharmaceuticals

Matthew Batters, JD
VP Bus. Development & Counsel
ALEXION · Skadden

Marcia Dougan Moore, MPH
VP Development Operations
ALEXION · Bristol-Myers Squibb

Randy Teel, PhD
VP Corporate Development
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Liam Ratcliffe, M.D., Ph.D.

ARVINAS

For more information

www.arvinas.com

Press/Media

pr@arvinas.com

Business Development

bd@arvinas.com

Investors

ir@arvinas.com

Careers

careers@arvinas.com

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